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Wintering of the biotrophic fungus *Puccinia lagenophorae* within the annual plant *Senecio vulgaris*: implications for biological weed control

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Epidemics of the obligate biotrophic fungus *Puccinia lagenophorae* might be used to control populations of the annual plant, groundsel, *Senecio vulgaris*. Insight into the mechanisms of survival of *P. lagenophorae* over winter may help to explain the number of inoculum sources, and their strength (assessed by number and size of pustules), present in an *S. vulgaris* population in spring, indicating the probability and rate of progress of a subsequent epidemic. Results of the study indicated survival of the rust as mycelium within the host over winter. Survival outside the host is unlikely, because aecidiospores lost their capacity to germinate over winter and teliospores have not been reported to be infectious. Survival of *S. vulgaris* plants over winter was reduced by rust infection in autumn. The mortality of *S. vulgaris* was 30–100% depending on the date of infection. All plants infected early in autumn died but those infected late in autumn were more likely to survive. In turn, poor survival of the host impacted on the survival of *P. lagenophorae* over winter. Consequently, the results of the study suggest that no inoculum sources, or only a few weak ones, are present in *vulgaris* populations in spring. This suggestion was supported by observations of an S. *vulgaris* population at a ruderal site. Therefore, research on biological weed control should focus on increasing the negative impact of *P. lagenophorae* on *S. vulgaris* populations while augmenting the probability of survival of the rust over winter to start new epidemics in spring.

Keywords: aecidiospores, biological weed control, groundsel, *Puccinia lagenophorae*, rust, *Senecio vulgaris*.

Introduction

The annual plant *Senecio vulgaris* is a troublesome weed, if the level of herbicide resistance is high in a population and mechanical control is not possible (Frantzen & Hatcher, 1997). Under such conditions, biological control might be an alternative to traditional methods of weed control.

Biological weed control using pathogens is a relatively new field of research and is based on both weed science and plant pathology. Weed science provides the knowledge about crop losses caused by weeds (either reducing crop quality by contamination, or competing for resources and reducing crop quantity). Plant pathology provides knowledge about mechanisms underlying disease epidemics that may increase the mortality of weeds and reduce their competitiveness.

Various approaches may be used to control weeds using pathogens. The classical, or inoculative, approach involves the introduction of natural enemies from the native area of a weed into areas that are colonized by the weed, but where the natural enemies are absent (Watson, 1991). An example is the introduction of *Puccinia chondrillina* into Australia to control *Chondrilla juncea* (Cullen *et al*., 1973). The inundative approach has been developed for areas where weeds and pathogens live in close association, but where populations of pathogens cannot build up sufficiently to have deleterious effects on weeds (Templeton *et al*., 1979; Hasan & Ayres, 1990). Fungi are cultured and periodically applied, like herbicides, to control populations of a target weed. An example is the control of *Aeschynomene virginica* by *Colletotrichum gloeosporioides* (Templeton *et al*., 1984). The system management approach aims at controlling weeds where no immediate and complete control is required, the introduction of an exotic natural enemy is not possible, and the natural enemy cannot be produced in large quantities (Müller-Schärer & Frantzen, 1996). This approach takes into account the fresh view in agro-ecology that eradication of a weed is not always necessary. The term 'system management approach' replaces the older terms 'augmentative approach' and 'conservation approach',

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which were not clearly defined. The system management approach is still a concept without application in weed control.

Reducing the competitiveness of a weed by stimulating a disease epidemic is the aim of the system management approach (Frantzen & Müller-Schärer, 1998). Disease epidemics on the target weed population have to progress sufficiently rapidly to reduce the competitiveness of the weed before the crop enters the critical period when it is rather sensitive to competition.

The start of an epidemic depends on the presence of inoculum sources within a host population or the arrival of inoculum from sources outside the population. Epidemics can be classified according to the number and strength (number and size of pustules in a source) of the inoculum sources within and around a host population. Focal epidemics start at relatively low levels of inoculum and general epidemics at high levels, when inoculum is well-dispersed (Zadoks & Schein, 1979).

Incidence of the rust fungus *Puccinia lagenophorae* on *Senecio vulgaris* is higher in autumn than in spring (Paul & Ayres, 1986a). This seasonal effect suggests that survival of *P. lagenophorae* in winter is critical to the start of an epidemic in an S. *vulgaris* population.

Epidemics of *P. lagenophorae* might control S. *vulgaris* if they can be managed effectively (Frantzen & Hatcher, 1997). Insight into the mechanisms of survival of *P. lagenophorae* over winter may help to explain the number of inoculum sources, and their strength, in a population of S. *vulgaris* in spring, indicating the probability and rate of progress of a subsequent epidemic. If the number, or strength, of inoculum sources is low, management of them may be required for an epidemic to progress rapidly enough to control the weed population.

P. lagenophorae probably originated from Australia and was first reported in Europe in 1961 (Viennot-Bourgin, 1964), where it is now common, and infects S. *squalidus,* S. *vulgaris,* S. *viscosus, Pericallis hybrida* (= S. *cruentus*), *Bellis perennis, and Calendula officinalis* (Wilson *et al*., 1965). The life cycle consists of an asexual cycle, the aecidial infection, and a sexual cycle, the telial infection (Fig. 1). However, the sexual process seems to be incomplete because pycnidia and pycnidiospores have not yet been detected either in the field or in experiments under controlled conditions (Wilson & Henderson, 1966; and the authors' personal observations). This suggests that only the asexual cycle is completed and survival of the fungus during winter by way of teliospores, which are the common survival units of rust fungi, does not result in new infections in spring. Overwintering may therefore depend on survival of aecidiospores, or survival of mycelium inside the host plant. These two options are further explored in the present study by:

1 following the fate of *P. lagenophorae* on *vulgaris* plants over winter;

2 assessing the survival of aecidiospores under various conditions of storage;

3 quantifying the impact of *P. lagenophorae* on plant fitness parameters over winter.

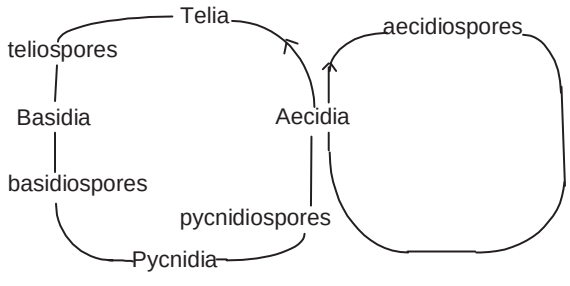

Figure 1 Hypothetical life cycle of *P. lagenophorae*, adapted from Wilson & Henderson (1966). Pycnidia and pycnidiospores of this fungus have not been detected.

Materials and methods

Plant and fungus

A maternal half-sibling *S. vulgaris* ssp. *vulgaris* line was used (G.S. Wyss, Federal Research Station Wädenswil, personal communication), named after its origin in Unterehrendingen (Switzerland). Aecidiospores of *P. lagenophorae* were collected at the same place and date and plants of the line Unterehrendingen were inoculated with the isolate (Wyss, 1997). Subsequently, aecidiospores of one aecium were used to produce the pathogen line Unterehrendingen. The lines of both the host and the pathogen were maintained in a climate room with a day/night cycle of 23°C and 60% r.h. during the 16 h day period (light intensity about 150 $\mu mol\,m^{-2}\,s^{-1}$) and 15°C and 80% r.h. during the dark period. Plant and rust fungus were cultured in the same climate room, but not at the same time.

Plants were inoculated by spraying a suspension of 0·5 mg aecidiospores per ml distilled water. Microscope slides covered with water agar were placed between the plants to determine the density of aecidiospores (*c.* 1200 per cm^2 agar). After inoculation, plants were enclosed in plastic bags for about 16 h.

Sporulation experiment 1995–96

Two cohorts of *S. vulgaris* plants were used. Plants of cohort 1 were grown from seed in pots containing NPK-fertilized compost or nutrient amended peat. Plants of cohort 2 were grown in a mixture of NPK-fertilized compost and peat. The diameter of all pots was 9 cm and no additional fertilizer was provided during the experiment. All plants were kept in the greenhouse until the date of inoculation.

Plants of cohort 1 were inoculated on 16 October 1995 and the pots were transferred to the botanical garden. This date is further referred to as time zero of this experiment and subsequent times are indicated as weeks ahead. Plants were about 4 weeks old and

still in a vegetative stage, or flowering, at inoculation. From the 16th of October onwards, plants were removed from the garden at intervals of 2–3 weeks. On each occasion, 25 randomly selected plants were placed in a heated greenhouse with a minimum temperature of 15°C for two to three weeks, then were classified as alive or dead (without any green tissue), and bearing aecidia or not. Further infection by *P. lagenphorae* in the greenhouse was prevented effectively by watering pots from below to keep the surface of the plants dry.

Plants of cohort 2 (still in a vegetative state) were inoculated on 15 November 1995 and the pots were transferred to the botanical garden. Thereafter, the same procedure was followed as for cohort 1. The last 27 plants of cohort 2 were not transferred to the greenhouse and remained in the garden until 9 April 1996. At this date, the percentage of plants surviving the winter and the percentage of plants with aecidia was determined.

Temperature and relative humidity were measured (Grant 1200 Squirrel, Grant Instruments, Cambridge, United Kingdom) in the garden four times a day, at 1:40, 7:40, 13:40, and 19:40, and at four different points at plant level. The minimum and maximum temperatures per week were used in analysis of the data.

Survival of aecidiospores

Aecidiospores were collected from plants about 6 weeks old in a climate room with a cyclone spore collector. The temperature inside the room was 18–20°C and the relative humidity was 60–70%. A day/night cycle of 12 h light and 12 h dark was maintained. Aecidiospores were collected about 4 weeks after inoculation. Subsamples were placed in four glass containers. The relative humidity was adjusted to 75% in two containers and to 34% in the other two, using salt solutions (Winston & Bates, 1960). One container at each humidity was placed in an incubator at 5°C and the other at 18°C, both in the dark. The capacity of spores to germinate was tested after 0, 1, 7, 14, 28, 42 days and (at 5°C only) 65 days of storage. Germination was tested by settling aecidiospores onto slides covered with 1% water agar in a settling tower. Each slide was placed in a separate Petri dish lined with wet blotting paper and the dishes were placed in an incubator at 18°C in the dark. Germination was determined after 24 h using four slides for each condition and period of storage. Aecidiospores were considered to be germinated if the germ tube was longer than half the diameter of the spore. Significant differences in germination between storage conditions were tested for using Friedman's Test for Randomized Blocks (Sokal & Rohlf, 1981).

In another trial, aecidiospores were collected from plants belonging to cohort 1 of the sporulation experiment. These plants had been returned to the heated greenhouse on 13 November 1995 to stimulate sporulation and aecidiospores were collected on 4 December (day zero in this experiment). The spores were stored in an Eppendorf tube placed between the plants of cohort 1 in the botanical garden. On 4 January (day 31), 7 February (day 65), 29 February (day 87) and 1 April 1996 (day 119), aecidiospores were removed for a germination test similar to the one described above, except that spores were settled onto slides by suspending them in distilled water and spraying the suspension with a de Vilbiss sprayer.

Host fitness experiment 1996–97

Two cohorts of S. *vulgaris* were used. Seeds were sown in small peat pots ($5 \times 5 \times 5$ cm), filled with nutrient-amended peat (TKS2, Floragard). Plants were grown in incubators until they had on average four true leaves, under a day/night cycle of 16 h ($150\ \mu mol\ m^{-2}\ s^{-1}$) light at 23°C and 8 h dark at 15°C and r.h. about 70%. Half the plants were inoculated and half served as control. One day later they were transplanted to experimental plots in the garden, plants of cohort 1 on the 1 October (time zero) and plants of cohort 2 on 29 October.

Twelve plots, each of 1·1 m by 1·1 m, and about 2 m apart, were prepared in the botanical garden. Six plots were randomly assigned to cohort 1 and six to cohort 2. Three of the six plots were randomly assigned to the inoculation treatment and three to the control. Planting density was 50 plants per plot. Each plot was divided into 11 cells each of 10 cm^2. One plant was placed at the centre of a cell and the 50 plants per plot were randomly assigned to the cells, resulting in a random pattern of plants within each plot.

After transplanting, each plant was checked every fortnight for presence of flowers, seed heads and aecidia of *P. lagenophorae*, except between the start of January to the end of February when they were covered by snow. The percentages of plants alive, flowering, with seed heads, and with aecidia, were calculated per plot, i.e. per 50 plants planted in autumn, and means were calculated per cohort and treatment for three replicated plots. Data were analysed after checking that they conformed to the assumptions underlying an ANOVA (Sokal & Rohlf, 1981).

Temperature and relative humidity were measured as for the sporulation experiment and for the same set of treatments.

Field observations 1996–97

Plants of S. *vulgaris* were marked on a ruderal site (a gravel pit) in the vicinity of Fribourg (Switzerland) in November 1996. The area was about 10 m by 8 m with 150 marked plants. Plants were classified as bearing aecidia or not and also into the following categories: (1) seedling (only cotyledons present); (2) vegetative (one or more true leaves present but no reproductive organs); (3) bolting (flower buds but no flowers visible); (4) flowering (flowers but no seeds); (5) seed

setting (at least one capitulum containing seeds). The marked plants were examined again in March 1997 for aecidia. Data were analysed using the chi-squared test (Sokal & Rohlf, 1981).

Results

Sporulation experiment 1995–96

The mortality of cohort 1 increased to 100%, with all plants dead by the end of February (Fig. 2a). Mortality of plants of cohort 2 was lower and living plants were still present in the spring (week 25).

Disease symptoms were visible on plants of cohort 1 at 16 days after inoculation, i.e. 16 days after transfer of the plants from the greenhouse to the botanical garden. Aecidia were seen 12 days later. Sporulation on plants of cohort 1 out of doors steadily decreased during autumn and early winter and no more aecidia appeared later on in the winter. The plants of cohort 2 did not show disease symptoms out of doors until spring.

Inoculated plants that did not have aecidia at the time of transfer from the garden to the greenhouse developed them about two weeks later. The percentage of plants bearing aecidia decreased with time for both cohorts (Fig. 2b) and was lower for cohort 1 than for cohort 2. No cohort 1 plants with aecidia were present by the end of February, when all plants of this cohort had died.

The last 27 plants of cohort 2 were left out of doors and 21 plants were still alive on 4 April 1996 (data not shown). Three plants of the cohort had aecidia on that date. On 10 April, 20 plants were alive and six had a few small aecidia.

Measurement of the temperature started at week 8 of the experiment (Fig. 2c). The minimum temperature was near, or below, 0°C during the course of the experiment. The maximum temperature increased to above 30°C in spring. Plants were frequently covered by snow from mid-December (week 8) to the end of February (week 19).

Survival of aecidiospores

Aecidiospores stored at 18°C lost their capacity to germinate sooner than did those stored at 5°C, and the percentage of spores germinated approached zero after 14 days of storage irrespective of the relative humidity (Fig. 3). The germination curves between days 1 and 42 differed significantly between the four types of incubation conditions (Friedman test, $P < 0{\cdot}01$).

Aecidiospores stored in the garden lost their capacity to germinate with time (Fig. 3). At day 87, the percentage

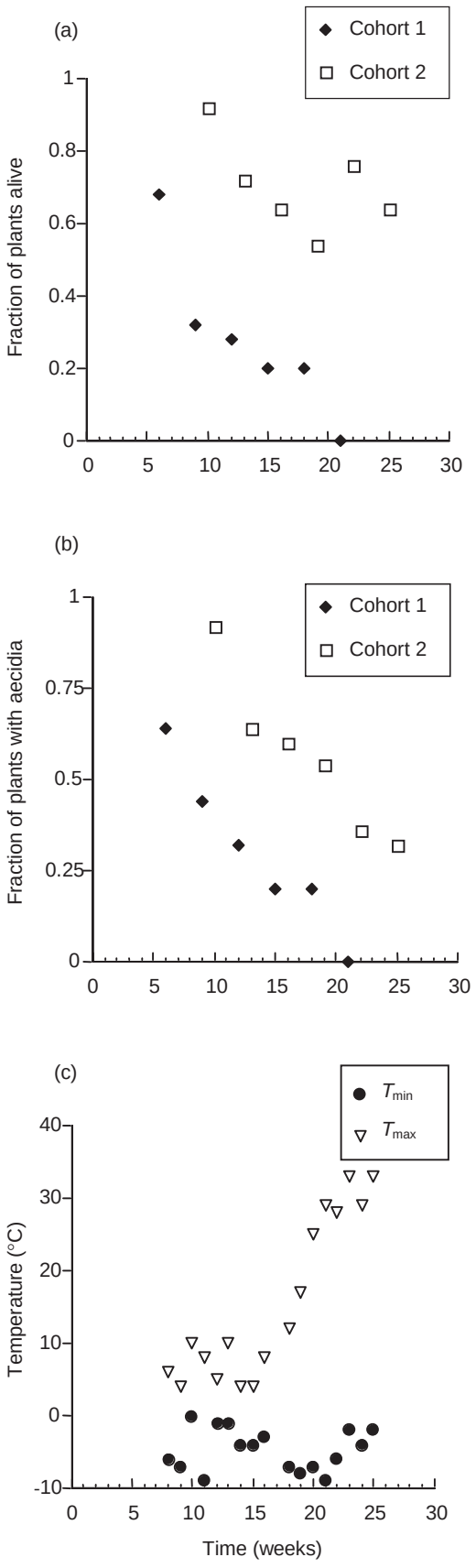

Figure 2 Sporulation experiment. (a) Fraction of plants surviving (b) fraction with aecidia of two cohorts and (c) the minimum and maximum temperature from autumn 1995 to spring 1996. Time 0 is 16 October 1995.

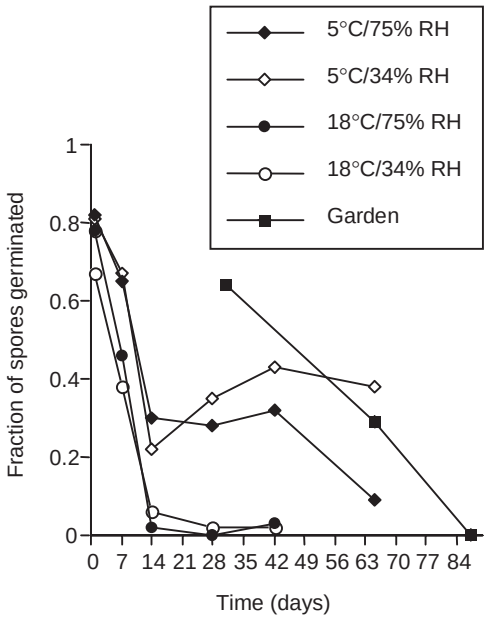

Figure 3 Fraction of aecidiospores germinated after various periods of storage under various conditions. Entries are means of counts on four replicated microscope slides. A minimum of 200 spores was observed on each slide. The temperature in the garden is presented in Fig. 2 (see weeks 8–20 for t = 0 to t = 87).

germinated approached zero and most of the spores had lost their orange colour, suggesting they were no longer alive.

Host fitness experiment 1996–97

Inoculation with the rust reduced survival of plants of cohort 1 significantly (ANOVA, $P < 0{\cdot}05$) before winter (Table 1, time = 12 weeks) and none of these inoculated plants survived the winter. Inoculated plants of cohort 2 tended to have a lower survival rate than the control plants, but the effect of inoculation on survival was not significant (ANOVA, $P > 0{\cdot}05$) at any occasion.

Most plants of cohort 1 flowered before winter, compared with only a small percentage of plants of cohort 2 (Table 1). The percentage of plants flowering did not increase after winter for the inoculated plants of cohort 1, because all these plants had died. Inoculated plants of cohort 2 flowered significantly (ANOVA, $P < 0{\cdot}05$) less than control plants at week 25.

Inoculated plants of cohort 1 did not produce seeds, whereas nearly 80% of the control plants had produced seeds at week 25 (Table 1). The percentage of plants producing seeds was relatively low for cohort 2, compared with the control plants of cohort 1, and no significant differences could be detected between control and inoculated plants.

No plants of cohort 1 had aecidia at two weeks after inoculation. At four weeks, nearly all plants inoculated had abundant aecidia (Fig. 4a). The percentage of control plants with aecidia increased during autumn but only a few aecidia were present per plant. After winter, one or two small aecidia appeared again on a small percentage of the control plants. Plants of cohort 2 had no aecidia before winter and the mean percentage of plants with aecidia in spring was 2% for the control plants and 3% for the plants inoculated (data not presented). Only one or two small aecidia were present on a plant.

The minimum temperature was near, or below, 0°C during the course of the experiment (Fig. 4b). The maximum temperature was about 30°C in autumn and approached this value again in spring. Plants were briefly covered by snow at the end of November and permanently covered during the period January–February.

Table 1 Host fitness experiment. Cumulative percentage of plants surviving, flowering, and with seed heads, of two cohorts planted on 1 and 29 October. Plants were inoculated, or not, with *P. lagenophorae*, the day before transplanting. Assessments were made from 4 to 25 weeks after 1 October 1996 (25 weeks was 25th March 1997). Each entry represents a mean percentage (standard error of mean in parentheses) of three replicated plots

	Cohort 1		Cohort 2	
Time (weeks)	control	inoculated	control	inoculated
Alive				
4	1·00 (0·00)	0·99 (0·01)	1·00 (0·00)	1·00 (0·00)
12	1·00 (0·00)	0·67 (0·09)	0·99 (0·01)	0·99 (0·01)
23	0·99 (0·01)	0·00 (0·00)	0·91 (0·04)	0·61 (0·13)
25	0·96 (0·01)	0·00 (0·00)	0·86 (0·08)	0·53 (0·14)
Flowering				
4	0·36 (0·09)	0·35 (0·04)	0·00 (0·00)	0·00 (0·00)
12	0·87 (0·01)	0·82 (0·01)	0·13 (0·02)	0·27 (0·02)
23	0·89 (0·02)	0·82 (0·01)	0·27 (0·01)	0·31 (0·04)
25	0·96 (0·02)	0·82 (0·01)	0·66 (0·07)	0·42 (0·02)
Seed set				
4	0·00 (0·00)	0·00 (0·00)	0·00 (0·00)	0·00 (0·00)
12	0·01 (0·01)	0·00 (0·00)	0·00 (0·00)	0·00 (0·00)
23	0·69 (0·06)	0·00 (0·00)	0·03 (0·01)	0·03 (0·02)
25	0·79 (0·04)	0·00 (0·00)	0·07 (0·01)	0·06 (0·02)

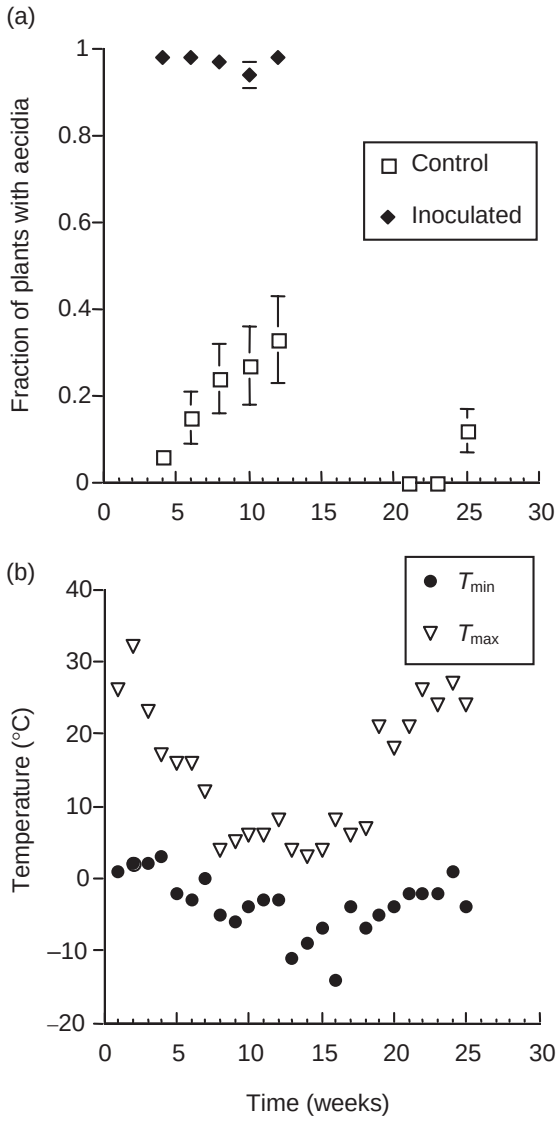

Figure 4 Host fitness experiment. (a) percentage of plants of cohort 1 with aecidia (b) the minimum and maximum temperature from autumn 1996 to spring 1997. Entries are the means and standard errors of mean of three replicated plots. Plants were covered by snow from week 14 to week 21 and no assessments were made. Values for the inoculated plants are not shown for week 21 and subsequent weeks because all plants had died during the winter. Temperatures were measured during the period 1 October 1996 (week 1) to 24 March 1997 (week 25). Each entry is the minimum, or maximum, of measurements by four sensors.

Field observations 1996–97

Most plants marked in autumn 1996 were in the flowering stage and only a few seedlings were present (Table 2). The developmental stage had a significant effect on the percentage of plants with aecidia (chi-squared test, $P<0{\cdot}01$) and plants in a more advanced developmental stage were more likely to bear aecidia.

Plants in a vegetative stage, or at bolting, appeared to survive the winter better than plants in other stages (Table 2), but this effect was not significant (chi-square test, $P>0{\cdot}05$). The total number of plants surviving was rather low. A fraction, 0·07, of the plants with aecidia in autumn, and 0·08 of those without, survived the winter (data not presented). No marked plants had aecidia in spring.

Table 2 Field observations at a ruderal site. Plants of *S. vulgaris* classified according to developmental stage, where entries represent the total numbers of plants per stage, the numbers and percentages of these plants with *P. lagenophorae* aecidia in November 1996, and numbers and percentages of the same plants alive in March 1997

		With aecidia		Alive	
Stage	No. of plants	*n*	%	*n*	%
Seeding	4	2	50	0	0
Vegetative	50	30	60	6	12
Bolting	16	13	81	2	13
Flowering	72	62	86	3	04
Seedsetting	8	7	88	0	00
Total	150	114	76	11	07

The number of plants with aecidia remained low during the summer compared with the total number of plants present at the ruderal site. One plant among the 2395 counted in June, one of the 2823 in July, and three of the 1651 plants in August, had aecidia.

Discussion

Three different life strategies of pathogens may be distinguished (Zadoks & Schein, 1979). One, the pathogen has a long infectious period, which means that dispersal units such as spores are produced over a long time, maximizing the probability of infecting a new host. In the case of biotrophic pathogens, this strategy is possible only when the host is present throughout the year. Two, the pathogen has a long latent period, during which it lives inside the host and produces dispersal units at a time when the probability to infect a new host is high. Again, for a biotroph, this strategy requires the continuous presence of the host. Three, the pathogen survives outside the host during a part of the year by producing specific survival units such as teliospores, or as mycelium utilizing food sources other than the host. This strategy may be optimum if the host is absent during some period of the year, or if conditions are not suitable for infection.

The borders between the three strategies are not always clearly defined and the strategies are not mutually exclusive. Pathogens may have a combination of strategies with more or less emphasis on one of them. *P. lagenophorae* fits best into the second strategy, according to the results presented.

Transfer of plants without aecidia from the garden to the greenhouse in winter resulted in production of aecidia by *P. lagenophorae*, which could only be

explained by latent infection. Sori of the rust fungus *Puccinia chondrillina* were also observed by Adams & Line (1984) after transfer of *Chondrilla juncea* plants from the field into the greenhouse during winter. The development of sori from latent infections may be governed by temperature. A study of Shearer & Zadoks (1972) demonstrated that the latent period of *Septoria nodorum* on wheat had a minimum of 6 days at 22°C and a maximum of 49 days at 4°C. A similar mechanism is suggested for *P. lagenophorae*. If the temperature is about 20°C the latent period is about 10 days (Paul & Ayres, 1984; Wyss, 1997) whereas, at the lower winter temperature in the present study, the latent period was about 5 months. Thus, aecidia were produced because of the change from the low temperature in the garden to the relatively high temperature in the greenhouse. A similar change could also explain the appearance of aecidia on plants in the garden in spring when the day temperature rose to 30°C. Although temperature may be a major factor governing the latent period, other factors such as humidity and density of sori may also be involved (Shearer & Zadoks, 1972). Genotype of the host and of the pathogen are other factors that may affect the latent period (e.g. Parlevliet, 1989) and temperature may interact with these factors. However, such interactions were not investigated in the present study.

Survival of *P. lagenophorae* outside the host during winter seems unlikely. As stated in the introduction, infection of plants by teliospores has not been observed or demonstrated. Survival of aecidiospores out of doors during winter could not be demonstrated in the present study. The laboratory experiment demonstrated a reduction of aecidiospore survival with prolonged storage and, comparing and extrapolating these results with those of the germination tests of spores stored in the garden, survival of aecidiospores in the field for the whole winter cannot be expected.

The strategy of *P. lagenophorae* to overwinter inside its host, *S. vulgaris*, by having a prolonged latent period, requires survival of the host over winter. Survival of *S. vulgaris* plants is reduced by *P. lagenophorae* itself, as demonstrated by Paul & Ayres (1986b). These authors, however, did not consider date of inoculation as a factor. The results presented here indicate that plants inoculated early in autumn, and already bearing aecidia before winter, have a higher mortality rate than plants inoculated later in autumn and bearing no aecidia before winter. One explanation for this difference could be that aecidia are used by secondary pathogens as entries to the plant and these increase the mortality rate (Hallett & Ayres, 1992). Increasing this rate to 100%, as determined in the present study for cohort 1 in the two experiments, means that neither host nor pathogen survives the winter. The effects of *P. lagenophorae* may also be less severe, as for cohort 2. A fraction of the plants infected in autumn survived and the pathogen also survived in limited amounts.

Field observations did not support the findings of the experiments in the garden. In the field, the mortality over winter did not differ between plants bearing aecidia in autumn and those without. An explanation for this divergence might be that the number of surviving plants in the field over winter was too low, i.e. 11, to demonstrate a significant difference between plants with and without aecidia in autumn. The results of field observations indicated, however, that a high mortality of the host over winter, i.e. 93% of the plants marked in autumn, causes an extinction of the pathogen, and its re-establishment in the population may take a long time, so that no epidemic could be observed in the following spring and summer.

Epidemics will have to be started and stimulated to control *S. vulgaris* with *P. lagenophorae*. A theory has been proposed to calculate the number and spatial distribution of inoculum sources of a pathogen within a weed population needed to control the weed (Frantzen & Müller-Schärer, 1998), based on an assumption of a focal epidemic. The data presented here suggest that focal epidemics are more likely than general epidemics in spring because of the poor survival of *P. lagenophorae* over winter and the theory of Frantzen & Müller-Schärer (1998) may apply to the control of *S. vulgaris*. The data suggest that few and weak (less than 10 small pustules) inoculum sources are present to start an epidemic in spring and may have to be increased artificially, as already suggested by Paul & Ayres (1986a).

The rust *P. lagenophorae* is balancing between extinction and survival within an *S. vulgaris* population. Infection may result in death of the host and subsequent extinction of the rust. Research on biological weed control should focus on increasing the negative impact of *P. lagenophorae* on *S. vulgaris* populations while augmenting the probability of survival of the rust over winter to start new epidemics in spring.

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References

Adams EB, Line RF, 1984. Biology of *Puccinia chondrillina* in Washington. *Phytopathology* **74**, 742–5.

Cullen JM, Kable PF, Catt M, 1973. Epidemic spread of a rust imported for biological control. *Nature* **244**, 462–4.

Frantzen J, Hatcher PE, 1997. A fresh view on the control of the annual plant *Senecio vulgaris*. *Integrated Pest Management Reviews* **2**, 77–85.

Frantzen J, Müller-Schärer H, 1998. A theory relating focal epidemics to crop–weed interactions. *Phytopathology* **88**, 180–4.

Hallett SG, Ayres PG, 1992. Invasion of rust *(Puccinia lagenophorae)* aecia on groundsel *(Senecio vulgaris)* by secondary pathogens: death of the host. *Mycological Research* **96,** 142–4.

Hasan S, Ayres PG, 1990. The control of weeds through fungi: principles and prospects. *New Phytologist* **115,** 201–22.

Müller-Schärer H, Frantzen J, 1996. An emerging system management approach for biological weed control in crops: *Senecio vulgaris* as a research model. *Weed Research* **36,** 483–91.

Parlevliet JE, 1989. Identification and evaluation of quantitative resistance. In: Leonard KJ, Fry WE, eds. *Plant Disease Epidemiology. 2. Genetics, Resistance, and Management*. New York, USA: McGraw-Hill, 215–48.

Paul ND, Ayres PG, 1984. Effects of rust and post-infection drought on photosynthesis, growth and water relations in groundsel. *Plant Pathology* **33,** 561–9.

Paul ND, Ayres PG, 1986a. Seasonal effects on rust disease (*Puccinia lagenophorae*) of *Senecio vulgaris*. *Symbiosis* **2,** 165–74.

Paul ND, Ayres PG, 1986b. The impact of a pathogen *(Puccinia lagenophorae)* on populations of groundsel *(Senecio vulgaris)* overwintering in the field I: mortality, vegetative growth and the development of size hierarchies. *Journal of Ecology* **74,** 1069–84.

Shearer BL, Zadoks JC, 1972. The latent period of *Septoria nodorum* in wheat 1: the effect of temperature and moisture treatments under controlled conditions. *Netherlands Journal of Plant Pathology* **78,** 231–41.

Sokal RR, Rohlf FJ, 1981. *Biometry*. New York, USA: Freeman.

Templeton GE, TeBeest DO, Smith RJ Jr, 1979. Biological weed control with mycoherbicides. *Annual Review of Phytopathology* **17,** 301–10.

Templeton GE, TeBeest DO, Smith RJ Jr, 1984. Biological weed control in rice with a strain of *Colletotrichum gloeosporioides* (Penz.) Sacc. used as a mycoherbicide. *Crop Protection* **3,** 409–22.

Viennot-Bourgin G, 1964. La rouille australienne du Séneçon. *Revue de Mycologie* **29,** 241–58.

Watson AK, 1991. The classical approach with plant pathogens. In: TeBeest DO, ed. *Microbial Control of Weeds*. New York, USA: Chapman & Hall, 3–23.

Wilson M, Henderson HM, 1966. *British Rust Fungi*. Cambridge, UK: Cambridge University Press.

Wilson IM, Walshaw DF, Walker J, 1965. The new groundsel rust in Britain and its relationship to certain Australasian rusts. *Transactions of the British Mycological Society* **48,** 501–11.

Winston PW, Bates DH, 1960. Saturated solutions for the control of humidity in biological research. *Ecology* **41,** 232–7.

Wyss GS, 1997. *Quantitative Resistance in the Weed-pathosystem* Senecio vulgaris *L.* Puccinia Lagenophorae *Cooke*. PhD Thesis, Zurich, Switzerland: Swiss Federal Institute of Technology.

Zadoks JC, Schein RD, 1979. *Epidemiology and Plant Disease Management*. New York, USA: Oxford University Press.