DISEASE SPREAD AND POPULATION DYNAMICS OF ANther-Smut Infection Of Silene alba CAUSED BY THE FUNgus Ustilago Violacea

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SUMMARY

(1) The anther-smut fungus, Ustilago violacea, sterilizes its dioecious host plant Silene alba by transforming plant reproductive parts into stamen-like structures that produce and disperse spores. To examine the dynamics of the plant–fungus interaction, a population of S. alba in Virginia, U.S.A. was mapped, and followed over two years, to study the demography of healthy and diseased individuals and spread of the disease. Additional field experiments explored the processes of spore dispersal and floral infection.

(2) The number of spores deposited on male, but not female, flowers was related to the number and proportion of diseased flowers close-by.

(3) Within a flowering season, both natural levels of floral infection and infection levels resulting from any inoculation experiment were about 20%. Observation of marked plants revealed, however, that only 4% of plants healthy in one year were diseased the following year, indicating that floral infection does not always lead to successful systemic infection.

(4) A deterministic model was used to explore the effects of plant recruitment and disease-spread on the fate of infected populations. Either elimination of the fungus, coexistence of plant and fungus, or local extinction of both organisms could occur depending on circumstances.

INTRODUCTION

The detrimental effect that pathogens often have on their hosts has led to the suggestion that disease-causing organisms can regulate host population size, affect genetic variability in host populations, and influence species coexistence in communities (e.g. Burdon & Shattock 1980; Burdon 1982; May & Anderson 1983a,b; Holt & Pickering 1985). Evaluation of these suggestions requires an understanding of the transmission of the pathogen and the effect of the pathogen on the survival and reproduction of host individuals. A fungal pathogen with a very large effect on the fitness of the plants it infects is Ustilago violacea (Pers.) Fuckel, the anther-smut fungus, which infects members of the plant family Caryophyllaceae. Anther-smut infection of the dioecious Silene alba (Miller) Krause causes a striking change in floral morphology: stamens with anther sacs filled with fungal spores develop in both male and female flowers (Baker 1947b). The infected flowers are sterile; males do not produce pollen and in females, the ovary aborts early in development. Naturally diseased plants are usually systemically infected, with all flowers infected, and thus completely sterile.

Anther-smut infections have long been of interest to biologists because of the effect of U. violacea on floral structure and its implications for plant/insect interactions and plant sex determination (e.g. Baker 1947b; Fischer & Holton 1957). The sterilizing effect of the fungus and the fact that plant populations can have a substantial percentage of diseased individuals (up to 67%: Baker 1947b) suggests that U. violacea is likely to be ecologically

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important to S. alba and raises the question of how host and pathogen coexist. Past ecological studies of anther-smut diseases (e.g. Baker 1947b; Lee 1981) have been largely descriptive and have not focused on quantitative studies of spore or disease spread nor on the population dynamics of this interaction. This study examines infection of S. alba by U. violacea using several approaches. A population containing both healthy and diseased plants was mapped for a two-year period; this provided a description of the population and allowed estimation of rates of recruitment, mortality, and disease spread through observation of known individuals over time. Within a flowering season, detailed observation of flowers and individual plants, and experimental inoculation studies were used to document the processes of spore dispersal and floral infection. The long-term potential fates of infected populations of S. alba were explored by integrating demographic data in a population model.

METHODS

Study organisms and sites

Silene alba, a dioecious biennial or short-lived perennial native to Europe, was introduced to North America in the early nineteenth century and has become naturalized in open, weedy habitats (McNeill 1977). Flowers open in the evening and remain open the following morning; numerous types of insects visit the flowers (Diptera, Hymenoptera, Lepidoptera, Thysanoptera) although butterflies and moths are probably the primary pollinators (H. M. Alexander, unpublished data; Baker 1947a). The obligate plant pathogen, U. violacea, is commonly found in European populations of the plant, but is rarer in North America (Baker 1947b).

Floral infection can occur when insects that have visited diseased flowers deposit spores on flowers of healthy plants (Baker 1947b). Following spore germination, meiosis and conjugation, a dikaryotic mycelium can invade the plant tissue. The original flower that is the site of infection is not affected, but diseased flowers will be subsequently produced as the fungus spreads within the plant. If the fungus invades the root system, diseased flowers will be produced the following year. Liro (in Baker 1947b) has reported smutted plants of S. dioica to produce diseased flowers for up to four years. Without infection of the root system, the death of the above-ground parts in the autumn can eliminate the fungal infection within the plant. Baker (1947b) also found that seedlings from clean seed had a high percentage of infection if they grew up beneath diseased plants, thus suggesting a prefloral mode of infection. Nearly 100% infection can be achieved artificially by soaking seedlings in a spore suspension (H. M. Alexander, unpublished data). Seed transmission of the fungus does not occur because sporulating flowers are sterile (Baker 1947b).

Our field studies were performed in Giles Co., Virginia, U.S.A. in 1984 and 1985. Silene alba is found along roadsides, railroad tracks, and in abandoned fields to altitudes of up to 1000 m. Many, but not all, sites contain plants infected by U. violacea with the percentage of diseased individuals usually ranging from around 1 to 30% (Fig. 1). The demographic study was conducted in a roadside site (37°3’N, 80°6’W) at 954 m with approximately 25% diseased plants (site 1). Observations of spore deposition and floral infection were made in this field and in a similar field 40 m away (site 2). Artificial inoculation of healthy plants was carried out in twelve roadside sites ranging in altitude from 615 to 769 m; eight sites contained naturally diseased plants (Fig. 1).
Fig. 1. Map of field sites along roads in Giles Co., Virginia, U.S.A. used in a study of Silene alba and Ustilago violacea. Size of circle reflects population size of Silene alba (○ < 50 flowering plants, ○ 100–300 flowering plants). The proportion shaded refers to proportion of flowering plants infected by Ustilago violacea. Sites 1 and 2 refer to the locations of a demographic study and a study of natural floral infection; other sites were used in an artificial inoculation experiment.

Demography

In late May 1984, all flowering plants in a 225-m² area at site 1 were mapped and labelled with metal tags anchored to the ground next to each plant. The sex and the disease status of the plants (healthy, completely diseased, or partly diseased) were recorded. In mid-July 1984, new flowering plants were mapped and labelled, and the status of all previously labelled individuals was noted. In late May and June 1985, we recorded the position and status of additional newly-appearing flowering plants and the survivorship and, if living, the disease status, of individuals recorded in 1984.

Types of partly-diseased plants

Partly diseased plants were of two forms: type I (diseased and healthy flowers on the same stems) or type II (separate diseased and healthy stems). Plants of both types were separately marked in sites 1 and 2 in 1984 and examined in 1985 to determine the outcome of infection, i.e. whether the plant was completely diseased, partly diseased or disease-free.

Spore dispersal

Levels of spore dispersal to healthy flowers were determined for male and female flowers of similar age in site 2 in 1984. Pairs of large male and female buds that were of equal distance to diseased flowers were marked. These buds flowered synchronously and
flowers were collected individually after being open for two days. The number and percentage of diseased flowers in a 1-m radius around each collected flower were also recorded. This procedure was followed for nine pairs of flowers marked on 24 June 1984 and for ten pairs of flowers marked on 25 June 1984. To estimate spore deposition per flower, single flowers were agitated for 1 min in 1 ml of water. Spores in four independent samples of the spore suspension were counted using a haemacytometer and results averaged to estimate spores ml\(^{-1}\) and therefore spores per flower.

*Natural floral infection*

In 1985, the incidence of natural floral infection was studied by observing twenty-four healthy male and twenty-three healthy female plants at two-day intervals from late May until late July in site 2. Total floral production of these plants was determined by counting and marking all flowers present on a plant every other day, and at each new visit counting and marking any newly produced flowers. Plants were observed for signs of floral infection (type I partial disease).

*Artificial inoculation*

To determine the effect of artificial inoculation in the field, forty-five healthy males and forty-five healthy females were labelled and inoculated with the fungus in early June, 1985. Inoculations were performed by gently inserting a toothpick dusted with fungal spores into the floral tube of healthy flowers. Spores used to inoculate a plant came from flowers from between five and ten diseased plants. All of the flowers present on each plant were inoculated on one day, up to a maximum of ten flowers. Plants were observed at weekly intervals until early August 1985 for the presence of diseased flowers.

**RESULTS**

*Demography*

In 1984, there were 255 flowering plants in the census area. The sex ratio of flowering plants was 1:1, males flowered before females, and 21\% of the males were diseased compared with 33\% of the females \((\chi^2 = 4.63, P < 0.05)\) (Table 1a). In 1985, 241 of the 255 1984 plant locations were unambiguously relocated (tags for the remaining plants were not found). Thirty-six per cent of the 1984 flowering plants were dead in 1985, with the proportions not significantly different among sex and disease classes (Table 1b). Over 70\% of the surviving plants flowered in 1985 (Table 1c). Of the 115 healthy plants in 1984 that flowered in 1985, three were diseased in 1985 (one completely diseased, two type II partly diseased). Three plants recorded as diseased in 1984 were listed as healthy in 1985. Thirty-nine plants flowered in 1985 that were not labelled in 1984; of these twelve were diseased.

Fewer plants flowered in 1985 than 1984 (Table 1d), but the general trends of males flowering before females and a somewhat greater disease level in females were found (both non-significant). Although the flowering plant sex ratio was not significantly different from 1:1, more females flowered than males. This result may be partly due to some males (either previously labelled or unlabelled) that flowered very early in the season being recorded as non-flowering since reproductive phenology of the population was approximately two weeks earlier in 1985 than in 1984.
Table 1. Results of a demographic study of an infected population of Silene alba in Virginia.

(a) Percentage of 1984 flowering plants of each sex that were healthy, completely diseased, or partly diseased

<table>
<thead>
<tr>
<th></th>
<th>May 1984</th>
<th>July 1984</th>
<th>Total 1984</th>
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<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
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<tr>
<td>n=95</td>
<td>n=61</td>
<td>n=80</td>
<td>n=110</td>
</tr>
<tr>
<td>Healthy (%)</td>
<td>77.9</td>
<td>52.4</td>
<td>80.0</td>
</tr>
<tr>
<td>Completely diseased (%)</td>
<td>16.8</td>
<td>44.3</td>
<td>18.8</td>
</tr>
<tr>
<td>Partly diseased (%)</td>
<td>5.3</td>
<td>3.3</td>
<td>1.2</td>
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(b) Percentage of flowering plants alive in 1985 based on sex and disease status in 1984

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<tbody>
<tr>
<td>Healthy in 1984</td>
<td>59.1</td>
<td>73.2</td>
</tr>
<tr>
<td>(n=93)</td>
<td>(n=82)</td>
<td></td>
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<tr>
<td>Diseased in 1984</td>
<td>52.0</td>
<td>65.9</td>
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<tr>
<td>(includes partly diseased)</td>
<td>(n=25)</td>
<td>(n=41)</td>
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(c) Percentage of flowering in 1985 for plants that were alive in 1985, based on sex and disease status in 1984

<table>
<thead>
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<th></th>
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<th>Female</th>
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<tbody>
<tr>
<td>Healthy in 1984</td>
<td>70.9</td>
<td>70.0</td>
</tr>
<tr>
<td>(n=55)</td>
<td>(n=66)</td>
<td></td>
</tr>
<tr>
<td>Diseased in 1984</td>
<td>100.0</td>
<td>77.8</td>
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<tr>
<td>(only completely diseased)</td>
<td>(n=13)</td>
<td>(n=27)</td>
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<td>(because no 1984 partly diseased plants survived)</td>
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(d) Percentage of 1985 flowering plants of each sex that were healthy, completely diseased or partly diseased

<table>
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<tr>
<th></th>
<th>May 1985</th>
<th>June 1985</th>
<th>Total 1985</th>
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<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>n=62</td>
<td>n=72</td>
<td>n=31</td>
<td>n=58</td>
</tr>
<tr>
<td>Healthy (%)</td>
<td>64.5</td>
<td>69.4</td>
<td>80.6</td>
</tr>
<tr>
<td>Completely diseased (%)</td>
<td>32.3</td>
<td>26.4</td>
<td>19.4</td>
</tr>
<tr>
<td>Partly diseased (%)</td>
<td>3.2</td>
<td>4.2</td>
<td>0.0</td>
</tr>
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Types of partly diseased plants

Eleven plants observed to be partly diseased in 1984, flowered in 1985. Of the seven type I plants in 1984 (diseased and healthy flowers on the same stem), four produced only healthy flowers, one produced only diseased flowers, and two produced some stems with only healthy flowers and some stems with only diseased flowers (type II) in 1985. Four plants in 1984 produced healthy flowers on some stems and diseased flowers on other stems (type II); all four were completely diseased in 1985.

Spore dispersal

For flowers marked on 24 June, female flowers had more spores deposited on them than males (female $\bar{x} = 7.27 \times 10^4 \pm$ S.E. 2.71 x 10^4, males $\bar{x} = 2.02 \times 10^4 \pm$ S.E. 1.81 x 10^4, Wilcoxon Sign Rank Test, $P < 0.02$). For flowers marked on 25 June, there was no
significant difference between spore number per flower for males and females (females, $\bar{x} = 3.12 \times 10^4 \pm S.E. 1.22 \times 10^4$, males $\bar{x} = 4.39 \times 10^4 \pm S.E. 1.21 \times 10^4$). For male flowers, there were significant positive Spearman rank correlations between number of spores per flower and both the number of ($r = 0.81$, $P < 0.001$; Fig. 2) and the proportion ($r = 0.80$, $P < 0.001$) of diseased flowers found in a 1-m radius of the collected flower. For female flowers, these relationships were not significant (number of diseased flowers, $r = 0.16$, Fig. 2; proportion of diseased flowers, $r = 0.29$). For both sexes, spore number per flower was not correlated with the total density of flowers (healthy and diseased) found in the 1-m radius circle. Because these relationships did not differ significantly between the two dates for either sex, data from both dates were combined in the correlation analyses described above.

**Natural floral infection**

Thirteen per cent of the male plants (3/24) and 30% of the female plants (7/23) that were healthy at the beginning of the season in 1985 produced diseased flowers over the course of the summer; the difference between the sexes was not statistically significant. Despite a small sample-size of infected plants, for males, the plants that produced large numbers of flowers were significantly more likely to become diseased. This was true either for total flower production over the summer or for flower production until 25 June (Fig. 3) (for both tests: Mann–Whitney $U$-test, 1-sided, $P < 0.05$). The same trend was apparent in females, but was not statistically significant (total flower production, $0.05 < P < 0.1$, production until 25 June, N.S., Fig. 3). Flower production until 25 June is a relevant
measure because the first appearance of floral infection was 13 July, with the average date of floral infection 22 July. Based on the artificial inoculation experiment (described later), spore deposition had to occur a minimum of three weeks earlier than the date diseased flowers were observed. Thus, flowers produced before 25 June are probably most important to the infection observed.

**Artificial inoculation**

The percentage of artificially inoculated plants that became partly diseased (type I) was not higher in sites where diseased plants were present compared with disease-free sites (disease absent 22%, disease present 18%). Thus the appearance of diseased flowers on inoculated plants is most likely to be due to the experimental addition of inoculum, not natural infection. Overall, 20% (9/45) of the males and 18% (8/45) of the females became
partly diseased. The probability of either a male or female plant producing diseased flowers increased with the number of flowers that were inoculated (Mann Whitney U-test, 1-sided, males $P < 0.025$, females $P < 0.005$; Fig. 4). On average, the first diseased flowers appeared on inoculated plants 38 days after inoculation, with a range of 20–58 days.

**POPULATION MODEL**

**Description of the model**

A simple deterministic model was developed to simulate the dynamics of the interaction between *Silene alba* and *Ustilago violacea* and to explore the effect of plant recruitment and disease spread on the fate of infected plant populations. Data from the 1984–1985 field studies were used to estimate the model’s parameters. The equations are difference equation analogues of the differential equations reviewed by Anderson & May (1979) with the following exceptions: no immune class exists; new-borns can become diseased; diseased plants do not reproduce; a new form of the infection rate function was used; and density dependence of birth and death rates were added.

At time $t$, the plant population consists of $H_t$ healthy and $D_t$ diseased individuals, where the total number of individuals per unit area, $N_t = H_t + D_t$. The number of diseased individuals a year later (time $t + 1$) is then given by:

$$D_{t+1} = D_t(1 - d_D) + H_t (r_t + b r_p)$$

where, $d_D =$ death rate of diseased individuals, $r_t =$ rate of successful floral infection of healthy individuals between time $t$ and $t + 1$, $b =$ rate of recruitment of new-borns into the next time interval (this equals the birth rate multiplied by the probability of new-borns dying between times $t$ and $t + 1$), $r_p =$ rate of infection of new-borns that survive to time $t + 1$ (i.e. rate of 'prefloral' infection). ‘Prefloral infection’ refers to any type of infection occurring prior to flowering.

The number of healthy individuals at time $t + 1$, is then:

$$H_{t+1} = H_t (1 + b (1 - r_p) - r_t - d_H)$$

where $d_H =$ death rate of healthy individuals.

The death, birth and infection rates are themselves functions of the composition of the population. Death rates are assumed to follow the form:

$$d = 1 - \alpha e^{-\beta N_t}$$

where $\alpha =$ a constant indicating the year to year survival at zero density. The value was set at 0.8 for diseased and 0.85 for healthy individuals. The numbers used were arbitrarily chosen, but based on observations that potted plants suffer some mortality and that diseased plants in nature have slightly higher mortality rates than healthy individuals. $\beta$ is a constant indicating the impact of population density on year-to-year survival. The value was set at 0.00271 for diseased and 0.00253 for healthy individuals, giving a survival rate of 0.61 for diseased and 0.66 for healthy individuals, respectively, when population size, $N_t = 100$. These values correspond to the survival rates observed in the natural population (Table 1b).

Recruitment rates of healthy individuals were assumed to follow an asymptotic, monotonically decreasing function of population size:

$$b = b_{\text{max}} / (1 + \gamma N_t)$$
where $b_{\text{max}}$ is an input variable reflecting recruitment rate at zero population density, and $\gamma$ is a constant indicating the impact of population density. The value was set at 0·01, such that recruitment rate would be halved when $N_i = 100$ and reduced to a third when $N_i = 200$, etc.

The floral infection rate was assumed to increase linearly as the proportion of diseased individuals in the population increased. Thus,

$$r_f = r_{f_{\text{max}}} D_i / N_i$$

where $r_{f_{\text{max}}}$ is an input variable reflecting the floral infection rate when the population approaches 100% diseased individuals. The prefloral infection rate was assumed to be linearly dependent on the number of diseased individuals in the population. Thus,

$$r_p = r_i D_i$$

where $r_i$ is an input variable reflecting the probability that a healthy seedling will become diseased given that there is one diseased plant in the population, i.e. the prefloral transmission coefficient.

Simulations were carried out until populations were at equilibrium (the sum of the absolute difference of $H_{t+1} - H_t$ and the absolute difference of $D_{t+1} - D_t$ is less than 0·001).

**Assumptions of the model**

The model is age, size, genotype and sex-independent, except that prefloral infection only occurs in new recruits. Type II partly diseased individuals are considered completely diseased and diseased individuals never become disease-free.

Birth rates and infection rates were varied as input variables. The density dependence of the birth rate $[b = b_{\text{max}}/(1 + \gamma N_i)]$ was chosen to be similar to that derived by Watkinson (1980) for yield–density relationships in pine stands $[b = b_{\text{max}}(1 + \gamma N_i)^{-\beta}]$. The observed density of this population was 260 plants in a 15 m × 15 m area, which is approximately 1 plant per m². Although this suggests a fairly wide spacing, plants are clumped and probably have a density-dependent impact on each other. Actual observed recruitment rates in the study population were thirty-nine new plants in an area that had 260 plants in the previous year, i.e. the recruitment rate was 15·0% at a density of $N_i = 100$. This gives a recruitment rate at $N_i = 0$ of 30·0%.

Plant survival $[1 - d = xe^{-\beta N_i}]$ was a negative exponential function of number of individuals in the population. Such patterns have been observed by Yoda et al. (1963) and by Harper (1977). The death rates at $N_i = 100$ were chosen to reflect the actual observed values of 0·34 for healthy and 0·39 for diseased plants.

The infection rate of plants observed flowering in both 1984 and 1985 was 3/81 = 4·0%, although floral infection rates within the same season were much higher (10/47 = 21·3%). High infection rates were also achieved by artificial inoculation (17/90 = 18·9%). Only a fraction of 1984 florally-infected plants (3/7 = 42·9%) were, however, either completely or partly diseased (type II) the following year. The form of the density/frequency dependence of infection rates is not known for this system (nor for any other natural plant disease system). The classical form used by Anderson & May (1979) assumes that the encounter rate of healthy individuals with diseased individuals increases linearly with the number of diseased individuals in the population; in a wind-dispersed pathogen, this is equivalent to a linear increase in spore concentration with the density of diseased individuals. In a pollinator-transmitted disease, the density dependence is likely to be
Anther-smut infection of *Silene alba*

![Graph showing equilibrium states](image)

Fig. 5. Equilibrium states of populations of *Silene alba* depending on values of maximum recruitment rate \( (b_{\text{max}}) \), maximum rate of floral infection \( (r_{\text{max}}) \), and prefloral transmission coefficient \( (r_i) \) in a deterministic model (see text for details). Solid areas indicate values which lead only to healthy plants; stippled areas represent values for which both plant and fungus can coexist, and clear areas indicate when both plant and fungal populations become extinct.

Weakened because insects can adjust their flight distances to compensate for plant spacing (Levin & Kerster 1969). In this case, the *per capita* infection rate is likely to be a function of frequency of diseased plants which led to our choice of the linear function \( (r_{\text{max}}D/N) \) as the *per capita* rate of becoming diseased with floral infection. Given an observed 40% infection rate, where the proportion of diseased plants was 69/255 = 27.0%, the study population was estimated to have \( r_{\text{max}} \) of 14.8%. Density dependence of the form suggested by Anderson & May (1979) was used for the prefloral infection rate, because such infection occurs as a result of spores from infected plants falling on young uninfected individuals. Of the thirty-nine new flowering plants in 1985, twelve were infected. If these latter plants flowered for the first time in 1985 and there were sixty-nine diseased individuals present, this would represent a prefloral transmission coefficient \( (r_i) \) of 0.0045. This value is probably an overestimate due to the possibility of some plants missing a year in flowering.

**Results of the model**

Depending on the input values \( (r_{\text{max}}, r_i, \text{ and } b_{\text{max}}) \), three equilibrium regions were possible (Fig. 5). At low recruitment rates and high infection rates, the outcome was total...
Fig. 6. Projections of population size of *Silene alba* infected by *Ustilago violacea*, showing number of healthy (open circles) and diseased (solid circles) flowering plants with time. Initial number of healthy flowering plants is nine; initial number of diseased flowering plants is one; maximum recruitment rate is 0.3, and maximum floral infection rate is (a) 0.3, (b) 0.4, and (c) 0.5. There is no prefloral infection. Equilibrium states are (a) elimination of the fungus, (b) coexistence of plant and fungus, and (c) local extinction of plant and fungus.

infection and subsequent local extinction of both the host and the pathogen. At low infection, but high recruitment rates, populations at equilibrium were disease-free. At high infection and high recruitment rates, pathogen and host could coexist at equilibrium and regulate each other’s populations. The presence of prefloral infection at low levels broadened the region of coexistence because the population was now less likely to become disease-free. Pre-floral infection at high levels, and high birth rates, increased likelihood of population extinction (Fig. 5).

The equilibrium numbers were independent of the initial frequency or density. With initial frequencies of \( H_i = 9 \) and \( D_i = 1 \), equilibrium was reached rapidly (< 100 years) in cases where the pathogen was eliminated. As infection rates were increased, the time to equilibrium became very long (\( > 100 \) years), up to and including the stage of coexistence between plant and fungus. As infection rates were increased still further, the time to equilibrium again decreased to the point where high infection rates led to the rapid demise of both populations (Fig. 6).

Using the parameters observed in the natural population in 1984–85 (recruitment rate = 15.0%, floral infection rate = 4.0% and mortality rate = 34 and 39% for healthy and diseased plants, respectively) and if prefloral infection is zero, the model predicts that the population will purge itself of diseased individuals in 80 years and stabilize at 35 individuals (Fig. 7). If prefloral infection occurs but at rates below \( r_i = 0.015 \), the same qualitative result is obtained, while at higher rates there is coexistence of host and pathogen, but at a reduced total population size.
**DISCUSSION**

**Disease spread**

The populations of *S. alba* studied here had over 25\% of the individuals systemically infected, and therefore sterilized, by the fungal pathogen *U. violacea*. Fungal spores were deposited onto healthy flowers, presumably as a result of floral insect visitors. Floral infection was observed at relatively high rates both naturally and as a result of artificial inoculation of flowers in the field. However, the demographic studies suggested that floral infection often did not lead to systemic infection and thus the rate of disease spread among flowering plants was low.

Other studies of anther-smut on other species have noted low infection rates: Jennersten (1985) estimated a rate of 2\% by following labelled plants of *Viscaria vulgaris* in Sweden for a three-year period, and Lee’s (1981) surveys of *S. dioica* in England revealed partial disease on only eight of several thousand shoots. Our study differs in that we quantified infection rates both within and between seasons. Detection of floral infection rates of 20\% was dependent on our method of frequently examining individually labelled plants for diseased flowers (every two days in the natural infection study and every seven days in the inoculation experiment). Without frequent observation, the presence of one or two diseased flowers on an otherwise healthy plant can easily be overlooked. Furthermore, we found that diseased flowers may not be observed at every census on a partly diseased plant. Despite the 20\% within-season infection, only a small percentage (4\%) of plants that were healthy one year were diseased the following year, indicating that all floral infections do not lead to successful invasion of the whole plant.
Although other workers (e.g. Baker 1947b) have noted in anecdotal fashion that partly diseased plants exist, we have quantified two types of partly diseased plants that give insight into the transmission process. Floral infection leads to type I diseased flowers, which appear late in the summer (after mid-July) on stems which previously only produced healthy flowers. This is the result of the long period (average thirty-eight days) between spore deposition and subsequent production of diseased flowers. The type II partly diseased plants can be found any time in the summer and have separate diseased and healthy flowering shoots coming from one root system. Our observations suggest that type I can lead to healthy, type II or completely diseased plants the next year, and type II plants will probably become completely diseased in a second year. Thus, systemic infection of a plant can be a two-year process.

Model predictions

Given the low rates of successful floral infection, how can the high incidence of disease in some populations be explained? Simulation models are useful to answer this question because the perennial nature of the plant and low infection rates make it difficult to observe changes in population disease levels over time. In our model, parameter estimates were based only on a one-year transition, but the model did provide a useful framework for identifying the important factors in host/pathogen dynamics. For example, the model predicts high recruitment rates and low disease spread will lead to disease-free populations. Populations along railway tracks in the Virginia mountains are invariably disease-free. High disturbance, including periodic clearing of the vegetation by railway personnel, probably leads to low disease spread, because plants are destroyed before the fungus can reach the root system. In addition, disturbance would lead to high recruitment rates and, as the fungus is not seed-transmitted (Baker 1947b), the population could ‘cleanse’ itself of the pathogen. Baker (1947b) also found agricultural populations of S. alba, that were disturbed yearly, to be free of disease. In contrast, Baker’s undisturbed populations had disease levels up to 67%. We have observed small isolated patches of S. alba in Virginia with 80% infection and healthy plants of only one sex remaining; the fungus may eventually spread to all plants and cause local extinction. Of particular interest is the finding of possible regions of stable coexistence of host and pathogen. Many models that illustrate coexistence of host and pathogen invoke genetic variation in host and pathogen for resistance and virulence, respectively (e.g. Leonard & Czochor 1980). Although such variation may be important, the model suggests it is not a necessary condition for coexistence.

The presence or absence of prefloral infection has important effects on the model. Prefloral infection has been largely ignored in the literature on anther-smut infection; Baker (1947b) does describe its existence, but provides no quantitative information. In the sites we studied, plants are often clumped and the passive seed dispersal of S. alba increases the chance that plants will grow up underneath neighbouring diseased plants. Thus, although we have shown that floral infection is important, prefloral infection could play a crucial role in the dynamics of this interaction.

An additional question is whether or not populations of S. alba will persist for long enough to reach the equilibrium states predicted. A high probability of site destruction or community composition changes could lead to local extinctions of the plant and pathogen that are largely independent of the disease process. This is of critical importance because an equilibrium state of coexistence occurs with only a relatively narrow range of input
Anther-smut infection of Silene alba

values and requires a long period. Thus, coexistence of host and pathogen seen in the field may be a result of observing the interaction in a transient, rather than an equilibrium state.

This plant/fungus system is of particular interest in light of May & Anderson's (1983a, b) questioning of the conventional wisdom that pathogens evolve to a less virulent state to allow coexistence of host and pathogen. In the case of *U. violacea* infection of *S. alba*, the pathogen has a profound negative effect on host reproduction because fungal reproduction and transmission is dependent on sporulation in plant floral structures, where insects can act as disease vectors. This pathogen has, however, little detrimental effect on whole plant physiology because diseased plants can produce and disperse fungal spores for several years. Coexistence of this plant and fungus appears possible primarily because of the inefficient transmission of disease through floral infection and because the organisms occupy transient habitats so that equilibrium states suggested by our model may often not be reached.

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REFERENCES


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