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High disease incidence and apparent disease tolerance in a North American Great Basin plant community

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Abstract. Patterns and consequences of plant disease at the community level have rarely been studied. We surveyed fungal infection in a Great Basin community of perennial shrubs over 4 years. Repeat surveys in fixed plots and along transects showed that disease incidence in the dominant perennial species was often very high, with up to 100% of all individuals infected. Despite the widespread prevalence of infection, and its severity on individual plants (which sometimes had over 1/3 of their leaves covered in pustules), its effects on survival and flowering were undetectably small. Thus, this perennial community appears to be stable, despite widespread disease. There are two potential explanations for this pattern; either the pathogens have evolved to be avirulent, or the hosts have become tolerant to being infected. Avirulence is not likely, because multiple infections are common in this system, and multiple infections have been shown in other species to favor strains that are faster reproducing and thus more virulent. Instead, it is more likely that tolerance has evolved in these host species, because infection in each year is practically inevitable and because the host plants are long-lived, giving little opportunity for new resistance genotypes to evolve.

Key words: avirulence, community ecology, resistance, rust fungi, tolerance, virulence

Introduction

Plant ecologists have only recently turned their attention to the consequences of disease in natural populations. Prior to about 1987, few studies had quantified the incidence and impact of disease in natural plant communities, and it was generally assumed that disease was rare and inconsequential (Browning, 1974; Harlan, 1976; Dinoor and Eshed, 1984). More recent studies of individual species have revealed that disease is common in natural populations, and that it usually reduces plant fitness (Alexander and Burdon, 1984; Augspurger and Kelley, 1984; Parker, 1986; Paul and Ayres, 1986a, b; Parker, 1987; Clay, 1991; Wennström and Ericson, 1991; Jarosz and Burdon, 1992; Roy, 1993a; Roy and Bierzychudek, 1993; Lively *et al.*, 1995). Because

pathogens can affect host fitness, they can influence biodiversity in several important ways: by altering the abundance of particular genotypes in a population, by altering the abundance of species in a community, and by altering the competitive dynamics among species in a community.

Although plant pathogens can influence biodiversity, few studies have quantified their effects at the community level (reviewed by Kranz, 1990; Burdon, 1993; Dobson and Crawley, 1994). Most community-level studies of plant disease have documented the impact of pathogens that were accidentally introduced into populations where they had not occurred before. Diseases such as Dutch elm disease and chestnut blight, when introduced into a non-resistant host population, can completely change forest community structure (Weste, 1981, 1986; Hiers and Evans, 1997). For example, Day and Monk (1974) showed that over the 30 years following the introduction of chestnut blight from Asia, the American chestnut decreased from 31% to 0.1% of total basal area in the Appalachian Mountains. As the chestnuts died, they were replaced by oak, maple, hickory, and tulip trees. Cases such as this demonstrate that when pathogens are introduced into defenseless host populations, they can drastically restructure whole plant communities. By contrast, endemic (native) pathogens may have less drastic consequences than introduced pathogens, because the hosts have had an opportunity to evolve with them (Browning, 1974).

We surveyed the incidence and fitness consequences of disease in a relatively simple Great Basin plant community at Turtle Mountain, in the Fort Sage Mountains of Northeastern California. We chose this locality because two of us had noticed high disease incidence in the perennial shrubs at this site. We originally suspected that we were witnessing a short-term epidemic due to unusually wet conditions, and expected that the shrub community would undergo a massive die-off as a result of being severely infected. To determine the influence of disease in the community, we assessed disease incidence, survival, and flowering in the four dominant shrub species over a 4-year period. We expected to see reductions in the survival and reproduction of diseased plants, and expected that this would lead to rapid changes in community composition. These expectations were not borne out because pathogen infection had little effect on host survival or flowering.

Materials and methods

Turtle Mountain is the northernmost arm of the Fort Sage Mountains, which lie at the eastern boundary of the basin and range province of Western North America. The Fort Sage plant community belongs to the Great Basin floristic province (Hickman, 1993). This region is defined by having entirely internal drainage and it is characterized geomorphologically by fault-block mountain

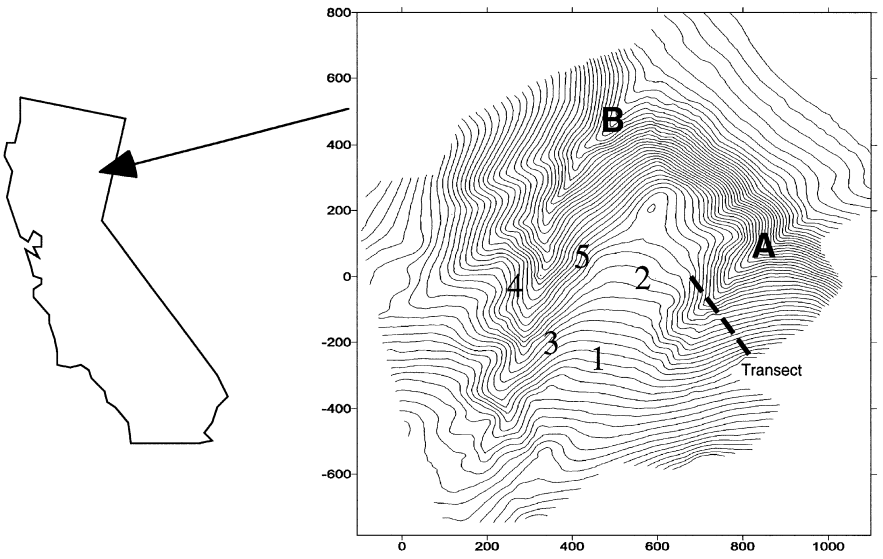

Figure 1. Map of Fort Sage study area in California showing Catchments A and B, the position of permanent plots 1–5 in Catchment B, and the transect across Catchment A. Scale is in meters.

ranges separated by basins (Bloom, 1978). The basin and range region is essentially a high-elevation desert with hot summers, cold winters, and low precipitation.

We used a combination of linear transects and five 30-by-30 m plots to characterize the perennial community and to assess disease incidence. The plots were chosen without a priori knowledge of the plants in each location. To choose the plots, we used a contour map prepared by Granger *et al.* (1996) to identify areas with different slopes and aspects within the watershed of Catchment B (Fig. 1). Within each plot, we established three equally spaced, parallel, 30 m permanent transects. The locations and physical characteristics of the plots are given in Table 1.

Table 1. Location and physical characteristics of the five permanent plots[a]

Plot	N	W	Slope (%)	Aspect (°)
1	40°05′21″	120°03′51″	16	347
2	40°05′31″	120°03′52″	14	349
3	40°05′22″	120°03′56″	35	274
4	40°05′29″	120°04′02″	48	88
5	40°05′31″	120°03′57″	42	275

[a]Locations were determined by averaging two GPS readings. Aspect is in degrees magnetic.

We estimated the frequency of perennial plant species in the permanent plots by counting each individual touching a measuring tape strung along each of the three transects in each plot, and dividing the count for each species by the tally of all species. Cover was estimated by measuring the distance along the tape that each species occupied, then dividing that number by the total distance measured. In total there were three 30 m transects for each of five plots, totaling 450 linear meters surveyed across all plots. We censused all plants along the transects in 1995 and 1998; in 1996 a subset (one transect per plot) was censused. Because the transects were in the same place each year, we could estimate the survival of each individual between years.

In addition to the permanent plots in Catchment B, in 1995 we ran a 300 m transect across Catchment A (shown in Fig. 1). Cover, frequency, and disease incidence were calculated for this transect in the same way as for the plot transects. This transect was not permanent and was only censused once, on 20 October 1995.

Infection status was determined for every individual of all perennial shrub and sub-shrub species encountered on the transects and in the plots. We also estimated the fitness consequences of infection by measuring survivorship and flowering over a 4-year period. Our estimate of disease incidence (percent of plants infected) will thus be conservative, because it does not account for possible latent (symptomless) infections. In other words, infection does not necessarily lead to disease. However, since the majority of damage that pathogens cause to their hosts occurs when the pathogens reproduce, and thus express symptoms (Power, 1992; Bull, 1994; Ebert, 1998), we are not concerned that an underestimate of infection will badly bias the results. We thus use the words infected and diseased interchangeably. Of our two estimates of host fitness, the ability to flower, however, is clearly an incomplete measure of fitness. Given that the hosts were long-lived and often large individuals, the presence or absence of flowers was the most practical measure we could make in this field study.

Because *Eriogonum microthecum* Nutt. (Polygonaceae) was the most common woody perennial encountered in the permanent plots, we increased our sample size of this species within the permanent plots. We permanently marked all individuals of *E. microthecum* in the three plots where there were fewer than 100 individuals (plots 1, 2, 4) and marked a sub-sample of *E. microthecum* in two plots where there were more than 100 individuals (plots 3 and 5). In plots 3 and 5 we marked all of the plants within 129 (182 individuals) and 417.6 m^2 (126 individuals) respectively, by working inwards systematically from one edge of the plot.

Eriogonum microthecum is a sub-shrub or shrub up to 150 cm tall and up to 160 cm in diameter (Hickman, 1993). Once the shrubs reach about 5 cm in height, they typically flower, and continue doing so each year thereafter

(personal observations). The flowers are tiny, numerous, and are borne in cyme-like, more or less flat-topped inflorescences. On each marked individual, we measured height, diameter, presence or absence of flowers, and the presence or absence of its common foliar disease *Uromyces intricatus* Cooke. We censused *E. microthecum* in all plots five times: 20 October 1995, 11 May 1996 (for systemic infections), 26 October 1996, 7 June 1997 (for systemic infections), 19 July 1998 (sub-sample), and 3 September 1998. To estimate the degree to which *E. microthecum* plants were infected, we calculated the percent of infected leaves by randomly collecting 10 leaves from each plant, each of which was then examined for spores. We performed this measure in October 1995 and 1996.

To determine whether infection had a significant effect on flowering (yes or no) and survival (yes or no), we used logistic regression with plant size as a covariate. Due to the small number of symptomless plants, these analyses were possible for only two of the dominant species, *E. microthecum* and the *Chrysothamnus*. For analysis, we assumed that each year was independent because (1) different individuals were diseased in different years, (2) with the exception of the systemically infected plants (a small percentage), each year the plants had to become newly infected, and (3) it is well known that environmental conditions, which can vary sharply from year to year (cf. Table 6), are important for plant infection (Jarosz and Davelos, 1995). All statistical analyses were performed with the program JMP (SAS, 1994).

Results

Hosts, pathogens, and disease incidence

The woody perennial community was composed of nine species (Table 2). The four dominants (frequency greater than 10%) were *Balsamorhiza sagittata* (Pursh) Nutt., *E. microthecum* Nutt., *Stephanomeria spinosa* (Nutt.) Tomb., and *Chrysothamnus* sp. (in the *nauseosus* species complex). In Catchment A (Table 2) the most common species was *B. sagittata* and it also had the greatest cover. In Catchment B (Table 2) the most common plants were *Chrysothamnus* sp. with a frequency of 39.4% and cover of 41.7%.

Each of the dominant plant species was infected by different fungal species. *Balsamorhiza sagittata* was infected by *Puccinia balsamorhizae* Peck, *E. microthecum* was infected by *U. intricatus* Cooke, and *S. spinosa* was infected by *Puccinia hieracii* var. *stephanomeriae* (P. Syd. & Syd.) Cummins. All of these rust fungi have similar lifecycles: they are host genus specific, autoecious (do not alternate to a different host), have five different spore stages, and can become systemic, over-wintering in roots and stems (personal observation and

Table 2. Frequency and cover for each woody perennial species encountered on October 1995 along the three 30 m permanent transects in each of the 5 plots in Catchment A, and the 280 m long transect across Catchment B

Plant species		Catchment A			Catchment B		
	Overall frequency	*N*	Frequency	Cover	*N*	Frequency	Cover
Balsamorhiza sagittata	28.9	65	33.9	11.1	41	23.4	14.5
Chrysothamnus sp. (*nauseosus* group)	25.1	23	12.0	2.0	69	39.4	41.7
Eriogonum microthecum	19.1	41	21.4	2.5	29	16.6	10.1
Stephanomeria spinosa	11.4	35	18.2	9.2	7	4.0	3.1
Ephedra viridis	6.8	14	7.3	2.3	11	6.3	8.6
Prunus andersoni	5.2	11	5.7	0.7	8	4.6	14.1
Purshia tridentata	1.9	0	0	0	7	4	3.1
Artemisia tridentata	1.4	2	1.0	0.3	3	1.7	3.1
Ribes velutinum	0.3	1	0.5	0.1	0	0	0
Annuals/grasses/dirt		Remainder			Remainder		

Frequency: # of individuals of a species divided by the sum of all species. Cover: area per species divided by the total for all species. Sorted by overall frequency of occurrence (i.e., frequency summed over both catchments).

Arthur, 1934). New infections in the spring can take place from two different spore sources: from infections on old leaves, and from fresh aeciospores produced on systemically infected plants. Over the summer, new infections can also arise from asexual urediniospores. One major difference between the *Chrysothamnus* species, which had the least infection, and the other dominant host species is that it was attacked by an unidentified ascomycete, whereas the others were all attacked by rust fungi.

Despite very high disease incidence in some of the species, rates of survival and flowering were high for all the species (Tables 3 and 5). Disease incidence was variable within and among years and species, but was often higher than 60% (Tables 3 and 4). Three of the four dominant species were attacked by rust fungi, and exhibited high disease incidence. For example, in 1995 and 1996, more than 89% of all individuals of *B. sagittata*, *E. microthecum*, and *S. spinosa* were infected with rust fungi by late summer (Table 3). In comparison with 1995 and 1996, disease incidence was lower in 1998 for *B. sagittata* and *S. spinosa*, but remained at nearly 100% for *E. microthecum* over the entire 4-year period. By contrast with the other dominants, the *Chrysothamnus* species had little or no infection in 1995 and 1998, though it too had considerable infection in 1996 (60%, Table 3).

The lower incidence of infection recorded for *B. sagittata* and *S. spinosa* in 1998 are not the result of a slightly earlier census date (September vs. October). In this high desert environment, heat and lack of water limit leaf production to the early summer. By mid-July, *B. sagittata* is completely dried up and no new

Table 3. Summary statistics for infection, flowering, and survival for the four dominant perennial species found on the transects

Censused factor (%)	Catchment	Date	*Balsamorhiza*	*Eriogonum*	*Stephanomeria*	*Chrysothamnus*
Infected						
	A	20 Oct. 95	96.9	95.1	100.0	8.7
	B	20 Oct. 95	100.0	93.1	100.0	13.0
	A	26 Oct. 96	89.3	93.1	90.3	60.0
	A	3 Sept. 98	11.3	100.0	8.3	0
Flowered						
	A	20 Oct. 95	87.7	90.0	74.3	85.7
	B	20 Oct. 95	95.1	93.1	100.0	91.3
	A	3 Sept. 98	94.3	97.1	72.0	63.6
Survival						
	A	1995–1998	81.5	85.0	71.4	100.0

The data shown for Catchment A are only from the transects within the five permanent plots, the data shown for Catchment B are from the temporary transect that was censused only once. A more complete census of *Eriogonum* was also made within the plots (i.e., was not limited to the transects; the results from the full data set are shown in Table 5).

Table 4. Infection, flowering rate, and cumulative survival for *E. microthecum* in the permanent plots over a 4-year period. Systemic infection was only recorded in 1996 and 1997

	Infected (%) (% of total systemically infected)	Flowered (%)	Survival (%)
October 1995	97.38	89.80	100.00
October 1996	85.49 (29.41)[a]	89.97	99.13
June 1997	– (27.17)[b]	86.07	94.77
September 1998	90.99	90.73	90.99

[a]Systemic infections censused 11 May 1996.
[b]Systemic infections censused 7 June 1997.

infections can occur (personal observations). The other species remain photosynthetic, but new infections are likely to be rare due to low humidity and a lack of new leaves. We established that infections are already high by July by doing a partial census of the two densest plots of *E. microthecum* (Tables 3 and 5). All 168 individuals censused were infected. The advantage to doing late summer censuses (September, October) was that we could tally both the total number of infected plants over the season, and whether or not they flowered. Three of the species flower only in the late summer (*E. microthecum*, *Chrysothamnus* sp., *S. spinosa*). *Balsamorhiza sagittata* flowers in May and June, but the flower heads remain on the plants, and thus can be observed later in the year.

Table 5. Logistic regressions testing the influence of infection (yes or no) and plant size (in 1995) on flowering (yes or no in the year of infection and in the year for survival (yes or no in the year following infection) for *E. microthecum*. Based on census data from all of the marked plants in the plots (i.e., including, b plants on the transects)

Parameters	Reproduction year 1		Reproduction year 2		Survival to next year	
	Wald χ^2	p	Wald χ^2	p	Wald χ^2	p
	Flowered in 1995		Flowered in 1996		Survival 1995–1996	
Infected or not by October 1995	0.10	0.7502	0.31	0.5758	3.40	0.0665
Plant diameter	32.69	<0.00001	30.89	<0.0001	1.70	0.1991
	Flowered in 1996		Flowered in 1997		Survival 1996–1997	
Systemically infected or not May 1996	0.30	0.5848	4.20	0.0404	0.77	0.3801
Plant diameter	29.76	<0.0001	29.09	<0.0000	0.76	0.3840
	Flowered in 1996		Flowered in 1997		Survival 1996–1997	
Infected or not by October 1996	0.62	0.4306	0.55	0.4593	0.39	0.5322
Plant diameter	29.08	<0.0001	20.17	<0.0001	0.21	0.6468
	Flowered in 1997		Flowered in 1998		Survival 1997–1998	
Systemically infected or not June 1997	0.03	0.8672	5.77	0.0163	1.25	0.5342
Plant diamter	21.15	<0.0001	10.00	0.0016	0.04	0.8491
	Flowered in 1998					
Infected or not by September 1998	0.02	0.8957				
Plant diameter	8.27	0.0040				

There was no effect of infection on *Chrysothamnus* survival; all plants survived from 1995 to 1998. We were unable to calculate whether infection in 1995 influenced flowering in 1995 or survival to 1998 of either *B. sagittata* or *S. spinosa* because too few plants were uninfected in 1995 (2/65 and 0/35, respectively). For the *Chrysothamnus* species, infection in 1995 had no influence on flowering in 1995 (Wald $\chi^2 = 0.0017$, $p = 0.9671$) but as was also true for *E. microthecum*, there was a tendency for small plants to flower less often (Wald $\chi^2 = 2.78$, $p = 0.0956$).

Consequences of infection for E. microthecum

Most rust-infected *E. microthecum* individuals were extensively infected; rust pustules were present on every leaf of some individuals, and up to 50% of each leaf could be covered by lesions. We estimated the percentage of leaves infected on each plant by closing our eyes and sampling 10 leaves from different parts of the bush. The average percentage of infected leaves per infected plant was $44 \pm 1.6\%$ ($N = 344$) in 1995 and $31.5 \pm 3.6\%$ ($N = 95$) in 1996. Although we did not quantify the percentage of leaves infected in the other years of the study, in every census similar levels of infection were observed. Indeed, there was little evidence of resistance in this system; of 344 marked individuals, only four escaped infection in all 4 years.

We measured density of *E. microthecum* in each plot because infection is usually positively density dependent (Burdon and Chilvers, 1982). Host densities in the plots ranged between 0.002 and 1.4 plants m^{-2} with an average across all plots of about one shrub per 10 m^2 (0.11 m^{-2}). Given that most individuals became infected in all years of the study, host density was apparently high enough in all the plots that pathogen transmission was not limited by it.

We estimated the fitness of *E. microthecum* by measuring the frequency of flowering within each year and survival between years (Table 5). Although *E. microthecum* plants often had 1/3 or more of their leaves covered with rust pustules, they nonetheless had nearly 100% rates of survival and flowering (Tables 3 and 4). There were no significant differences in reproduction (as assayed by flowering rates) between uninfected and non-systemically infected plants for any of the 4 years, but there was a significant effect of plant size, with smaller plants flowering less often than larger ones (Table 5). There were no significant differences in survival between infected and uninfected plants for any year (Table 5), but for 1995–1996, uninfected plants tended to have lower survivorship than infected plants (Wald likelihood ratio $\chi^2 = 3.40$, $p = 0.0665$). The unexpected result that uninfected plants sometimes had lower survival than infected plants does not seem to be the result of differences in plant size, as

size was never significantly associated with survival in any of the years of the study (Table 5).

Uromyces intricatus sometimes causes systemic infections in *E. microthecum*. We expected that systemic infections would harm the plants more than local lesions do, because infection is more extensively spread within the plants (Jarosz and Davelos, 1995). We thus recorded systemic infections of *E. microthecum* by recording the presence of aeciospores in May 1996 and June 1997 (Table 4). Systemic infections did not decrease survival (Table 5), but they did influence flowering in *E. microthecum*, but not until 1 year later (Table 5, reproduction year 2), and not consistently so. The consequences of systemic infection for flowering differed between the 2 years in which systemic infections were measured. For plants recorded as systemically infected in 1996, the percent of plants that flowered in 1997 was slightly higher than the non-systemically infected ones (86.96 vs. 86.06, Wald $\chi^2 = 4.20$, $p = 0.0404$ with plant diameter as a covariate). For plants systemically infected in 1997, however, the percent of plants that flowered in 1998 was slightly lower than the non-systemically infected ones (87.76 vs. 92.49, Wald $\chi^2 = 5.77$, $p = 0.0163$ with plant diameter as a covariate). Systemically infected plants were, on average larger than non-systemically infected plants in both years (1996: systemic $= 34.23 \pm 2.29$ cm, non-systemic $= 23.50 \pm 1.01$ cm, $t = -4.77$, $p < 0.0001$; 1997: systemic $= 31.86 \pm 1.76$ cm, non-systemic $= 22.67 \pm 1.10$ cm, $t = -5.33$, $p < 0.0001$). Non-systemic infection had no discernible effect on the presence or absence of flowering during a given year or in the following year (Tables 3 and 5).

Discussion

The perennial plant community in the Fort Sage Mountains often had high levels of rust fungus infection. For example, in 1995 and 1996, most individuals of three dominant species were infected by rust fungi, with up to 100% of all leaves of *E. microthecum* covered in lesions. We expected that, if the high incidence of rust disease impaired host reproduction and survival, then there would be massive die-offs of *E. microthecum*, *B. sagittata*, and *S. spinosa*. However, after four seasons of observation there was little evidence that infection decreased host fitness. For example, survival and flowering rates for *E. microthecum* were nearly 100% (Table 5), despite nearly 100% infection in each of the 4 years (Tables 3 and 4). There was also no significant effect of infection on survival and flowering of *B. sagittata* and *S. spinosa*, despite high levels of infection in 1995 and 1996 (Table 3).

Of our two estimates of fitness, survival and flowering, survivorship is undoubtedly the most demographically critical; the longer a perennial survives,

the more likely it will ultimately reproduce (Silvertown, 1987). We detected no significant decreases in survival over a 4-year period for four species. Annual reproduction is a more problematic measure because it is usually linked to plant size in perennials, and size is often determined by the growth environment (Chiariello and Gulmon, 1991). In our study, whether a plant flowered or not depended strongly on plant size, and was not influenced by non-systemic infections. A similar result was found for rust-infected perennial *Linum* over a 4-year period in Australia (Jarosz and Burdon, 1992). However, for the 1/3 of *E. microthecum* plants that were systemically infected, flowering significantly increased once and decreased once in the year following the recorded infection. These inconsistent effects of systemic infections on flowering suggest that other factors besides disease were influencing flowering. Plant size does not appear to be the explanation as systemically infected plants were, on average, larger than non-systemically infected plants and these relationships did not change between years. Nor does rainfall appear to be a good explanation. The period in which more flowering occurred in the systemically infected plants was the very dry 1996–1997 period, whereas the period of less flowering was the relatively wetter 1997–1998 period (Table 6). Although more complete measures of reproduction than our plus or minus measure might have allowed more resolution of these short-term fitness consequences, the fact that most infected plants flowered was interesting in itself, because many systemic rust infections eliminate flowering (Parker, 1987; Wennström and Ericson, 1991; Roy, 1993b; Roy and Bierzychudek, 1993).

The traditional view of natural populations is that they do not show high levels of disease unless they are disrupted by some factor, such as unusual weather conditions, disturbance, or human introductions of new pathogens

Table 6. Precipitation data from the National Weather Service gauge that is closest to the Fort Sage Mountains (at Doyle, about 8 km from the field site). The months April–October were considered to be the frost-free growing period. Averages were calculated from the complete data set (1931–1998, but with no measurements for water years 1957–1959)

Year	Rainfall for the month (cm)							Growing season
	April	May	June	July	August	September	October	Total[a]
1995	6.8	6.8	3.9	4.4	0	0	0	21.9
1996	2.7	2.6	1.3	1.0	0.5	0.8	2.6	11.5
1997	2.3	0.8	3.7	1.0	0	0.6	1.6	10.0
1998	1.6	4.3	7.3	0.9	1.4	6.6	1.7	23.8
64 year average	1.5	1.8	1.4	0.7	0.5	1.0	1.8	8.7

[a]Precipitation totals for the 'frost-free' growing season of April–October.

(Harlan, 1976). At our site, the pathogens are native and the high disease levels were sustained over a 4-year period with a twofold difference in rainfall during the growing season (Table 6), suggesting that we were not seeing a one-time epidemic caused by a single wet year. Nonetheless, because all of these years were wetter-than-normal years, disease incidence might have been higher or the plants might have been better able to withstand infection than normal. In this regard, it is noteworthy that there were not large year-to-year differences in tolerance or disease incidence, despite a factor of two difference in precipitation (in this desert environment).

Our finding of high disease incidence with low fitness consequences in natural populations is not unique. Roy and Kirchner (2000) recently summarized the studies that quantified both rust fungus disease incidence and its fitness consequences in natural plant populations. In roughly half the populations studied (for example, Alexander, 1991; Wennström *et al*., 1995; Davelos *et al*., 1996), the fitness consequences of infection were very low, despite levels of disease incidence ranging from 50% to 100%, suggesting low resistance and either high host tolerance or low pathogen virulence. In the other half of the studies (for example, Parker, 1987; Wennström and Ericson, 1991; Roy, 1993b; Roy and Bierzychudek, 1993), disease incidences were low, but when plants were infected, the fitness consequences of infection were high, suggesting that resistance was common but that once a plant became infected it had little or no tolerance to infection.

Our observations suggest that disease is endemic at Fort Sage, and that infection is high either because the hosts are highly tolerant to it, or because the pathogens have somehow become avirulent while at the same time preserving a high degree of infectiousness. Can we distinguish between the two alternatives, tolerant hosts or avirulent parasites, that are consistent with high disease levels and low fitness consequences? Clearly the best way to differentiate these mechanisms is by experimentally infecting a series of different host genotypes with different pathogen isolates. Evidence for host tolerance would be indicated if host genotypes that were exposed to the same pathogen isolate, and that showed identical amounts of infection (and thus exhibiting similar resistances), had different fitness consequences of infection (Fig. 2a). On the other hand, if the fitness consequences of infection varied among pathogen strains, but not among host strains, then one could reasonably attribute low fitness consequences to pathogen avirulence (Fig. 2b). The experiment we have just outlined is necessary to disentangle the effects of pathogen virulence from host tolerance, a problem which all future studies with pathogens (and potentially, herbivores) should consider. Unfortunately, it is difficult to perform such experimental work with species such as the woody perennials discussed here, because they take years to grow to reproductive maturity, making it difficult to measure their lifetime fitness in experimental settings. Although we cannot

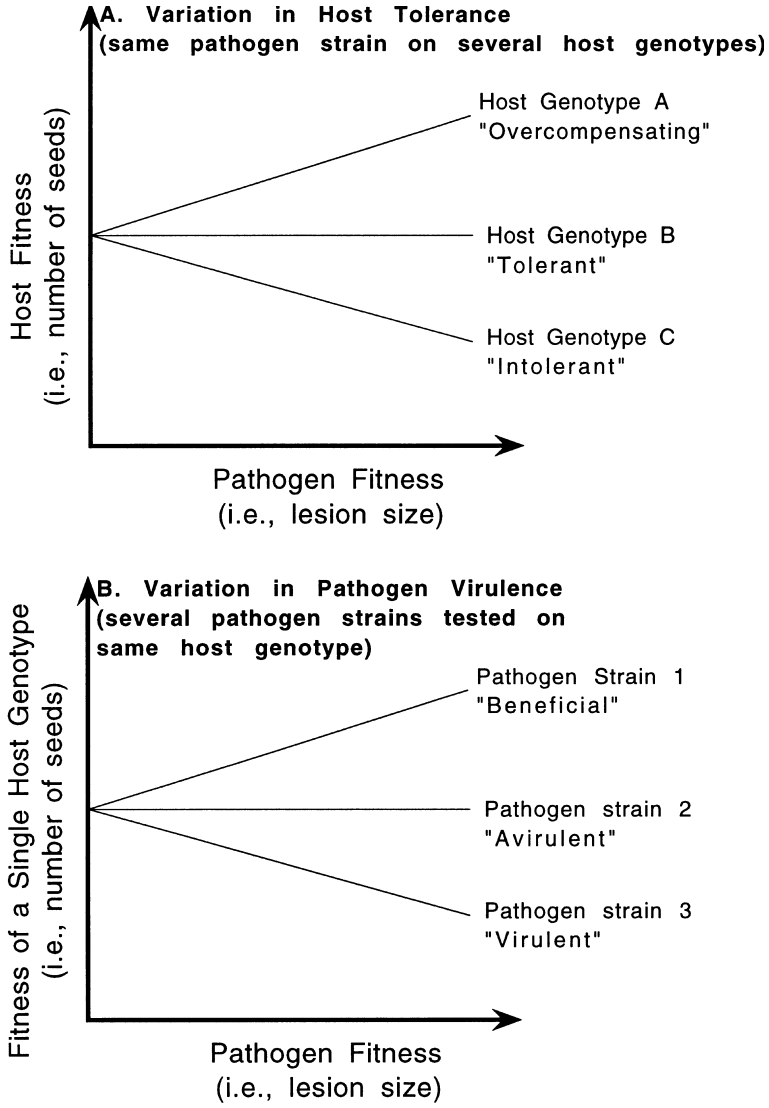

Figure 2. Norms of reaction diagrams differentiating host tolerance from pathogen virulence. To differentiate tolerance of the hosts to disease from pathogen avirulence it is necessary to do experiments with multiple strains of both the host and the pathogen to determine whether the host or the pathogen strain has more influence over host fitness. At the same time, these experiments can also determine whether there is genetic variation in pathogen virulence or host tolerance. (A) Differences among three host genotypes in their ability to tolerate one particular pathogen strain. Each line represents an idealized regression line, the slope of which measures tolerance. Host genotype A can compensate for infection, host genotype B is tolerating infection, host genotype C has no tolerance to infection. (B) Differences among three pathogen strains in virulence on one particular host genotype. Pathogen strain 1 is beneficial, pathogen strain 2 is avirulent, whereas pathogen strain 2 is virulent. Note that in the 'real world' the regression lines would not necessarily be linear.

easily experimentally test for tolerance vs. avirulence in this system, theory and empirical findings from more easily studied systems suggest that we are observing host tolerance, rather than pathogen avirulence in the Fort Sage perennial community.

The traditional view of host–parasite relationships would suggest that the rust fungi attacking three of the dominant shrubs at Fort Sage have evolved to become avirulent. The reasoning behind this idea is that because pathogens typically evolve more rapidly than their hosts, they can more readily evolve to become avirulent than their hosts can evolve to become tolerant. However, conditions at the Fort Sage locality should favor pathogen virulence rather than avirulence. First, the ubiquity of lesions and spores indicates that the pathogens are reproducing often and in large quantities. High rates of pathogen reproduction are typically strongly correlated with virulence (Power, 1992; Bull, 1994; Ebert, 1998), because they entail significant physiological burdens and damage to the host. Second, rust fungi are only transmitted horizontally (no direct transmission to host offspring). Greater reliance on vertical transmission tends to lead to lower virulence because pathogens will have higher fitness if their hosts can produce more offspring (Ewald, 1987; Ewald and Schubert, 1989; Lipsitch *et al.*, 1996). It is less clear that horizontal transmission necessarily leads to higher virulence (Lipsitch *et al.*, 1996), but most of the available evidence suggests that it usually does. Third, multiple infections are common, and individual plants can be simultaneously infected by multiple pathogen strains (B.A. Roy and M. Pfunder, unpublished data). Fourth, host density and frequency are high, leading to high pathogen transmission rates. These conditions – almost unlimited opportunity for transmission leading to frequent colonization and recolonization of hosts – are exactly the conditions that ought to lead to increased pathogen virulence due to competition among pathogen strains. Fast growing parasites, or those with the most rapid transmission rates, ought to have an advantage over slower growing ones, and fast growing parasites are usually more virulent because their rapid growth and reproduction are a drain on the host's resources (Power, 1992; Bull, 1994; Ewald, 1994; Lipsitch *et al.*, 1996; Ebert, 1998). However, the pathogens at Fort Sage cause little apparent damage to host fitness.

An alternative to the evolution of reduced virulence in the pathogens is the evolution of tolerance in the hosts. Although tolerance is likely to have physiological costs due to shifting allocation away from growth to regrowth (Van der Meijden *et al.*, 1988; Simms and Triplett, 1994), tolerance is still likely to be evolutionarily favored when disease is present (Strauss and Agrawal, 1999; Tiffin, 1999; Roy and Kirchner, 2000). Given that so many factors would appear not to favor the evolution of pathogen avirulence in this system, we suggest that host tolerance is more likely.

There are two factors that may be critically important in the evolution of the plants and their pathogens in the Fort Sage Mountains: perennial, long-lived hosts and virtually unlimited pathogen dispersal. The perennials at the Fort Sage site are all relatively long-lived (at least 25 years, except for *S. spinosa* which our survivorship data suggest may have about a 10-year lifecycle), and there are few establishment events because seedlings can recruit only during rare wet seasons. This demographic pattern means that the same host genotypes are available for colonization for long periods of time. The rusts have almost unlimited opportunities to infect their hosts and sexual reproduction in every year gives them the opportunity to rapidly evolve to evade host resistance defenses. The climate is ideal for rust fungi: relative humidity is low between thunderstorms so spores disperse well, and summer thunderstorms are frequent enough that infection can occur. Wind is a constant presence in this habitat, and because the canopy is not closed in this shrub-dominated community, wind borne spores can readily disperse to new hosts (as is suggested by the high rates of infection in our data). In addition, all the host-specific pathogens produce infective spores from early spring through late fall, and infections can also become systemic. Thus the pathogen has almost unlimited opportunities for transmission to host genotypes that stay the same year after year due to low rates of population turnover. Under these circumstances, where the host has little opportunity to evolve new resistance, and pathogens have widespread opportunities to cause infection and to evolve tactics to overcome host resistance, hosts' survival may depend on their capacity to tolerate, rather than resist, infection.

In conclusion, contrary to our expectations that widespread disease would cause extensive mortality and thus would drastically alter this plant community, we found no evidence that plant pathogens were currently adversely affecting the ability of hosts to survive and flower. This pattern can be accounted for either by low virulence in the pathogens, or by high tolerance to infection in the hosts. However, several factors – including multiple infections and high transmission rates – should favor pathogen virulence rather than avirulence. Thus, host tolerance to infection is the more likely explanation. Experimentation is necessary to determine whether host tolerance or avirulence is present, but the critical experiments are not possible with these long-lived perennial plants. Our results are nonetheless interesting because they document high disease incidence in a natural plant community with negligible current consequences for host survival and flowering. We report these results with the hope that they will stimulate research on the evolution of pathogen avirulence and host tolerance in long-lived, perennial plant–pathogen systems that are more experimentally tractable than the desert shrubs described here.

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