Effects of Habitat and Population Structure on Powdery Mildew Epidemics in Experimental *Phlox* Populations

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Based on a portion of a Ph.D. dissertation by the first author.
Research supported in part by NSF grant DEB 7800522 (ML) and a David Ross Fellowship from Purdue Research Foundation.
We thank Warren Müller for assistance with the statistical analyses and an anonymous reviewer for constructive suggestions on the text.
Accepted for publication 15 September 1987.

ABSTRACT

Jarosz, A. M., and Levy, M. 1988. Effects of habitat and population structure on powdery mildew epidemics in experimental *Phlox* populations. Phytopathology 78:358-362.

The development of powdery mildew epidemics caused by *Erysiphe cichoracearum* on its natural *Phlox* hosts was monitored from 1979 to 1982 in a series of experimental populations provided with a central inoculum source. Populations were established in both shaded woodland and adjacent, exposed old-field habitats and contained 33, 67, or 100% of individuals susceptible to the local pathogen isolate used. Generally, shaded populations accrued 3–5 times more infected plants and 5–40 times higher disease severity (i.e., area under the disease progress curve) than comparably structured, exposed populations. The persistence of infections was also greater on shaded plants. Between-year variation in disease severity was largely explained by temperature and rainfall effects on pathogen growth. Population structure did not appear to affect disease severity. Expected reductions in epidemic severity with increasing proportions of resistant plants may have been negated by the low host density (typical of woodland *Phlox* taxa) and large inoculum sources used. A previous survey established that resistance levels among wild *Phlox* populations were significantly correlated with the degree of habitat shading; in fully shaded sites, populations also tended to be homogeneous for high levels of resistance. Consequently, the experimental epidemiology supported the hypothesis that natural *Phlox* populations have evolved and/or maintained resistance levels primarily in response to habitat-mediated levels of *E. cichoracearum* pathogen pressure.

Additional key words: environmental regulation of epidemics, host mixtures.

Three primary determinants of plant disease are the genetic structure of the host, genetic structure of the pathogen population, and the favorability of the environment for the growth of both (2). In agriculture, human control of the composition of host populations predisposes such systems to sporadic, often devastating epidemics (10,31). The artificially selected, high-density monocultures of genetically uniform crops provide an ideal resource for pathogens which, commonly, are highly host-specific and capable of rapid epidemic increase. The environment, by influencing the infection potential and growth rate of the pathogen, regulates the severity of disease and the extent of economic loss (25,27).

In natural systems, severe epidemics are rarely reported except where alien pathogens have been introduced (12,13). Selection is thought to foster coevolved polymorphisms in host resistance and pathogen virulence. This heterogeneity per se may buffer the host against epidemics because of the reduced density of susceptible tissue per compatible interaction, physical interference with effective pathogen transmission, and general decreases in effective inoculum volume (3). Environmental regulation of disease epidemiology would further influence the strength of reciprocal selection pressures governing the host-pathogen interaction.

This view of the dynamics of plant disease outside of agriculture is inferred from a patchwork of evidence. First, surveys of wild relatives of crops demonstrate that natural populations may harbor substantial resistance polymorphism to pathogens (6,8,16,17,24,28,32). Second, in some cases, the distribution of resistance appears to be concentrated in geographic or altitudinal zones in which climates are more favorable for pathogen transmission and growth (8,11,19,22,28). For example, the distribution of resistance to *Erysiphe graminis* among natural populations of *Hordeum spontaneum* and *Triticum dicoccoides* is correlated with gross temperature and moisture availability factors that promote powdery mildew infections (20,21). Third, several studies of epidemiology in crop multilines and varietal/species mixtures have shown that disease severity is lower than in comparable homogeneous stands (7,9,15,29).

In this report, we consider the epidemiology of *Erysiphe cichoracearum* D.C. ex Merat in experimental populations composed of wild species of *Phlox* L. Polemoniaceae. A previous survey of responses to pathogen challenge among 112 populations representing 10 host taxa indicated that resistance was more prevalent in populations from shaded habitats (15). Shading has been reported to increase the severity of *Erysiphe* infections on other host genera (12,23,30). Accordingly, we hypothesized that the observed patterns of powdery mildew resistance in *Phlox* largely reflected the history of pathogen pressure as mediated by habitat regulation of disease epidemiology. To test the habitat aspect of this hypothesis, we determined powdery mildew epidemiology for *Phlox* populations of varied resistance composition in both shaded and exposed habitats typically occupied by the host taxa.

MATERIALS AND METHODS

Populations containing 84 or 90 uniformly spaced plants were established at the Ross Biological Reserve Station of Purdue University, Tippecanoe Co., IN, each year from 1979 to 1982. Populations were located in either shaded woodland or in neighboring, exposed old-field habitats with 25-m separations between treatments. Populations contained either a low (33%; L), medium (67%; M), or high (100%; H) proportion of susceptible plants. Because of the difficulty of continuously maintaining stocks of the genetically heterogeneous host materials, only a limited number of unreplicated experimental treatments was possible each year (Table 1). Within a habitat, populations were located randomly. Within populations, plants were grown in 15-cm ceramic pots sunk to ground level, and the intervening vegetation was periodically mowed. The plants occupied randomly assigned positions on a hexagonal grid with pots centered at 61-cm

intervals and with plants separated edge-to-edge by about 48 cm. The total area of the grid was about 28 m^2. The test plant density approximated that of natural *Phlox* populations in woodland habitats.

From 1979 to 1981, perennial *Phlox* (*P. aspera*, *P. carolina*, *P. glaberrima*, *P. pilosa*, *P. pulcherrima*, and *P. villosissima*) were used as test plants. These plants were prescreened for their susceptibility to a local isolate of *E. cichoracearum*. Plants used as susceptible hosts had a minimum of 25% hyphal cover at 14 days postinoculation under growth chamber environments favorable to pathogen growth. Resistant hosts had 0% cover under similar screening conditions. After screening, all aerial tissue was removed and the plants were allowed to regenerate from rhizome stock. All test plants were mildew-free and adult-sized (about 20 cm tall with 3–5 stems) at the beginning of the experiment. In 1982, plants from the annual *P. drummondii* were used. Test plants were propagated from seed of uniformly susceptible parents.

At the center of each population, a core of 1, 6, or 7 laboratory-infected plants (Table 1) served as a primary inoculum source of *E. cichoracearum*. The core was designed to imitate a persistent infection focus; it was occasionally reprovisioned with fresh, conidia-producing plants and maintained throughout each experiment.

Disease spread and severity were monitored by periodically scoring the percent mildew cover on all living aerial tissues. The season-long severity of infection per susceptible plant was estimated by calculating the area under the disease progress curve (AUDPC) using the formula of Shaner and Finney (26). In the shaded habitat, a few plants categorized as resistant became lightly infected during some experiments. These plants were considered to be susceptible and their responses were included in the AUDPC calculations for their respective populations.

Because of the absence of replicate treatments within years and the nonorthogonal design overall, the statistical analysis was performed in two stages. Initially, we tested for gross differences among the 17 total populations by analyzing variance of individual plant AUDPC values. The data were log transformed, i.e., log 10 (1 + AUDPC), to make the variances homogeneous. This was a conservative test since between-plant variation, which includes both random differences and systematic position effects, was used to test for significance. Subsequently, we tested for treatment effects with a nonorthogonal analysis of variance of the mean-transformed AUDPC values of populations. Various linear regression models were calculated using a range of combinations and permutations of main effects, i.e., time of experiment (year/season), population structure, habitat, and population location within a habitat, and two-way interactions. The significance of each main effect was evaluated after being adjusted for other main effects. Each two-way interaction was also evaluated separately, after being adjusted for all main effects.

TABLE 1. Experimental conditions and resultant disease levels

Start date	No. plants		Treatment[a]	AUDPC[b]	Percent of susceptibles infected
	Test	Core			
8/28/79	84	7	L-shaded	553.1	94
			M-shaded	397.3	84
			H-shaded	640.3	89
6/11/80	84	6	L-shaded	73.9	74
			L-exposed	51.9	21
			M-shaded	47.7	62
			M-exposed	7.3	13
			H-shaded	34.9	40
6/21/81	84	6	L-shaded	116.4	55
			L-exposed	99.5	50
			M-shaded	207.5	67
			M-exposed	38.7	21
			H-shaded	525.7	86
5/25/82	90	1	H-shaded	46.1	73
			H-exposed	1.1	9
8/31/82	90	1	H-shaded	76.8	77
			H-exposed	3.8	28

[a] Proportion of susceptible plants per population: L = 33%; M = 67%; H = 100%.

[b] Area under the disease progress curve. Mean of untransformed data for susceptible plants only.

RESULTS

Mean AUDPC values for the 17 experimental populations are presented in Table 1. The analysis of variance of individual log-transformed AUDPC values indicated significant differences among populations (F = 47.88, with 16, 1,055 df, $P < 0.001$). Subsequent analyses of the mean-transformed AUDPC values of the populations indicated that disease severity was significantly influenced by habitat ($F = 18.54$, with 1, 5 df, $P < 0.01$) and by time ($F = 7.44$, with 4, 9 df, $P < 0.01$) but not by population structure ($F = 0.67$, with 2, 6 df, $P > 0.50$), population location within a habitat (F = 0.23, with 3, 6 df, $P > 0.75$), or any two-way interactions between main effects.

AUDPC values, adjusted for time and population structure, were significantly higher for shaded populations (Table 2). Generally, the greater disease level reflected that shaded populations had 3–5 times more infected plants than comparably structured, exposed populations as well as higher AUDPC values per infected plant (Table 1). The only exceptions to this trend were the L populations of 1980 and 1981. The 1980 L-exposed population had a relatively high AUDPC value considering that its percentage of susceptibles infected was less than a third of that of the 1980 L-shaded population. However, more than 85% of the AUDPC value for the 1980 L-exposed population was accounted for by one heavily infected plant. If this unusual plant were excluded, the AUDPC value would decrease to 7.2, or about a tenth that of the shaded comparison. The 1981 L-exposed population was unusual because, for reasons unknown, its AUDPC and proportion of susceptibles infected were almost twice those of any other exposed population in the study.

Habitat effects also were evident in the differential persistence of infections. Once infected, plants from shaded populations tended to remain infected throughout the experiment, whereas disease was generally transient on plants in exposed populations. Discounting plants infected only at a final census and including only contemporaneous habitat comparisons, 62% (121 of 192) of infected plants were persistently diseased in the shaded habitat versus 42% (22 of 52) in the exposed habitat. This difference was most extreme in the spring of 1982, when disease was persistent on 37 of 49 infected shaded plants and on 0 of 8 infected exposed plants.

AUDPC values, adjusted for habitat and population structure, showed significant between-year variation in disease severity (Table 2). Although there were differences in experimental design from year to year, the annual variation in AUDPC appeared to be influenced primarily by gross climatic factors. The growth of *E. cichoracearum* on a variety of crop hosts has been shown to be strongly regulated by temperature (with minima, optima, and maxima being about 9, 22, and 34 C, respectively [30]) and to be promoted by high relative humidity but inhibited by direct rainfall and free moisture on leaf surfaces (23). Here, disease severity was

TABLE 2. Habitat and time effects on disease epidemics

Effect	Treatment	AUDPC (S.E.)[a]
Habitat	Shade	1.348 (± 0.098)
	Exposed	0.598 (± 0.135)
Time	1979	1.934 (± 0.193)
	1980	0.693 (± 0.142)
	1981	1.216 (± 0.142)
	1982 spring	0.688 (± 0.225)
	1982 fall	0.844 (± 0.225)

[a] All values are means of log 10 (AUDPC + 1) values calculated for individual susceptible plants within populations and adjusted for other main effects.

high in the fall of 1979, low in the summer of 1980, and intermediate in the summer of 1981 when the respective average maximum daily temperatures at the local weather station were 22.3, 29.2, and 27.6 C, respectively (18). Maximum temperatures during the 1979 experiments never exceeded 30 C, while those during the 1980 experiments were often at or above the 34 C limit for pathogen growth. Low temperatures, including three frosts, curtailed disease development during the last third of the fall 1979 experiment (Fig. 1). Heavy rains (>11 cm) inhibited epidemic development during the first 10 days of the spring 1982 experiments (Fig. 1). Hot and very dry conditions during the first 20 days of the fall 1982 experiments had a similar effect.

In contrast to the environmental influences on epidemic severity, no significant effect was observed for population structure. While the absence of replicated experiments prevented a conclusive statement about such an effect, there was no consistent trend either within or between years (Table 1; Fig. 1). The expected hierarchy of disease levels was commensurate with the proportion of susceptible plants only in the shaded population series of 1981. The exposed series of the same year, as well as both series in 1980, displayed hierarchies the reverse of that expected. In 1979, the L population had a higher AUDPC than its M comparison and the highest percentage of susceptibles infected in the series.

One factor that may have diminished any consistent influence of population structure on disease severity was the use of 6 or 7 plants as the core inoculum source in the experiments from 1979 to 1981. Patterns of disease spread within H-shaded populations (Table 3) suggested that primary inoculum volumes were sometimes large enough to supercede potential interference with disease transmission by resistant plants in mixed arrays.

In 1979, when there was no obvious relationship between AUDPC and population structure, the inoculum core contained 7 plants and disease spread in the H-shaded population was fairly rapid. The mean interval to first infection for all plants ultimately infected was 22.0 days. In fact, 50% of the plants in the outer rank and 69% of all plants ultimately infected were infected at day 17,

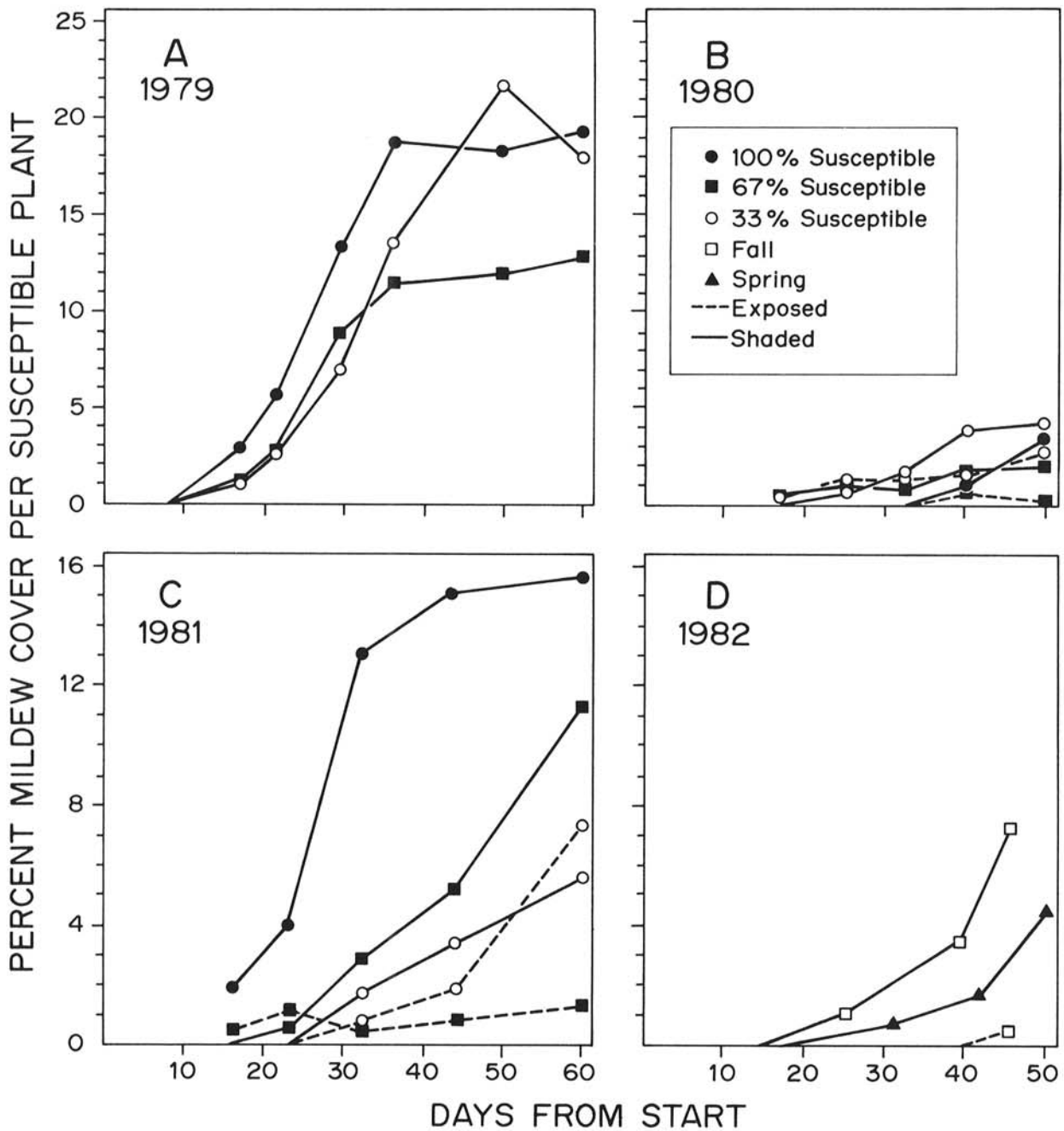

Fig. 1. Disease progress curves for individual populations. Values for the 1982 spring-exposed population were too small to graph.

the first census when macrosymptoms were observed. Further, 79% of all plants ultimately infected were infected by the next census on day 22. At both censuses, disease severity was very low (Fig. 1). These features suggested that the core inoculum volumes in 1979 were sufficient to infect most susceptible plants early in epidemic development and that ultimate disease severity was due largely to continued pathogen growth on these plants by autoinfection.

In 1981, when the shaded series displayed AUDPC levels commensurate with population structure, the inoculum core contained 6 plants, and disease spread in the H-shaded population was slower than in 1979. The mean interval to first infection for all plants ultimately infected was 29.0 days (Table 3). When macrosymptoms were first observed on day 16, they were apparent on only 20% of the plants in the outer rank and 44% of those ultimately infected. The latter value increased to only 58% by the next census on day 23. Disease severity was very low at both censuses (Fig. 1). These features suggested that effective inoculum volumes in 1981 were smaller than in 1979 and, consequently, that there was more opportunity for resistant plants to interfere with disease transmission and influence epidemic severity.

Further evidence of the importance of core inoculum volume for epidemic development was apparent in the H-shaded population in fall 1982 when a single inoculum source plant was used. The mean interval to first infection was 33.4 days, and only 10% of the plants were infected at day 15. Although 77% of the population was ultimately infected, the proportion of plants infected decreased significantly with distance from the inoculum core ($r = -0.97$; $P < 0.01$).

DISCUSSION

Environmental regulation of epidemics. This study demonstrated that the field epidemiology of *E. cichoracearum* parasitism on wild *Phlox* hosts was strongly environmentally regulated. Habitat-specific factors were the predominant influence on epidemic development. Proportions of infected plants, AUDPC indices of disease severity, and the persistence of infection were significantly greater in shaded than exposed habitats with otherwise common macroclimates. Gross weather factors, especially temperature, also appeared to explain significant annual variation in disease levels.

Habitat differentials for powdery mildew severity in natural *Phlox* populations may be greater than was observed in this study, in which a large and persistent primary infection focus was artificially imposed. The two-to-one average difference in the persistence of infections in shaded versus exposed experiments almost certainly implies that in nature relatively few epidemics would be initiated, and even fewer well developed, in exposed habitats.

In the extreme, susceptible *Phlox* populations that occupy exposed field and prairie habitats may escape all but sporadic infections until, perhaps, mid-fall when climates are usually conducive to pathogen establishment. However, such late and low-severity infections should have little or negligible impact on host fitness. Most *Phlox* species have completed sexual reproduction and exhibit little aerial vegetative growth by this time.

Conversely, susceptible *Phlox* populations that occupy woodland or otherwise shaded habitats may be subject to primary infection throughout the growing season, perhaps even chronically for multiple seasons when the pathogen successfully overwinters. Once established, epidemic development should be rapid; under the variety of local weather conditions from 1979 to 1982, all shaded populations had 50% incidence of infection within 2–8 wk. The resultant high severity of disease may be expected to significantly decrease both survivorship and fecundity components of host fitness (Levy et al, *unpublished*).

The pattern of environmental regulation of epidemic development indicated in this study is consistent with the ecogeographic distribution of resistance to powdery mildew among natural *Phlox* populations. A survey of 112 population samples indicated that shaded populations had significantly higher levels of resistance than exposed populations and that among exposed populations there was a weak negative correlation between resistance level and mean annual temperature (14). Thus, both studies supported the hypothesis that host populations have evolved and/or maintained resistance in response to pathogen pressure primarily mediated by site-specific microenvironments.

The importance of the environment for disease dynamics in natural plant-pathogen systems has been generally inferred but rarely tested (12,13). Several studies have noted patterns of resistance in wild plants that are best explained by large-scale climate differences regulating pathogen pressure (8,11,19–22, 28). This study demonstrated that environmental regulation of pathogen pressure can operate on a much finer ecogeographic scale and, by extension, that resultant patterns of host resistance can be correspondingly complex.

Influences of population structure. The proportion of resistant plants in our experimental populations did not consistently or significantly affect epidemic development. This contrasted with the general observation in crop host mixtures that infection rates and disease severity decrease as the proportion of resistant plants increases (7,9,15,29). Because it was not possible to replicate our experiments within years, the influence of population structure could not be resolved conclusively. However, patterns of disease spread within H-shaded populations suggested that potential reductions in AUDPC within mixed populations may have been diminished by the large inoculum sources and the low plant density used.

Barrett (1) has noted that in host mixtures the proportion of susceptible plants infected initially by a primary inoculum source depends largely on inoculum volume and spore dispersal gradients and not on the proportion of resistant plants. When alloinfections have little influence on subsequent disease spread, ultimate disease severity will correspondingly depend on the rate of primary infection from the source and the rates of autoinfection, which likewise are independent of the resistant component of the mixture. Furthermore, controlled epidemics of powdery mildew on barley have indicated that at very low plant densities (<31 plants per m^2) alloinfections have little influence on epidemic development (4,5). In our experiments, the test plant density was about 3.0 plants per m^2 with nearest-neighbor separations of about 48 cm edge-to-edge. Under these conditions, by comparison, resistant plants should have had little opportunity to influence AUDPC values.

The absence of consistent epidemic buffering by the resistant component of our experimental mixtures may not be unusual in

TABLE 3. Spread of disease within the 100%-shaded populations of 1979, 1981, and fall 1982

Start date	Rank[a]	Percent initially infected[b]	Percent ultimately infected	Mean no. days to first infection
8/28/79	1	67	92	18.3 ± 4.9[c]
	2	83	94	18.3 ± 8.7
	3	58	92	24.0 ± 14.1
	4	50	83	24.4 ± 12.5
6/21/81	1	50	83	23.6 ± 10.4
	2	50	89	24.9 ± 15.1
	3	46	92	30.1 ± 17.9
	4	20	80	33.0 ± 16.0
8/31/82	1	0	100	27.3 ± 5.7
	2	17	92	27.0 ± 8.6
	3	11	83	36.3 ± 11.9
	4	8	79	35.4 ± 11.1
	5	3	60	34.9 ± 10.0

[a] Rank 1 is closest to inoculum source. In 1979 and 1981, ranks 1 to 4 contained 12, 18, 24, and 30 plants, respectively. In 1982, ranks 1 to 5 contained 6, 12, 18, 24, and 30 plants, respectively.

[b] Infections were initially detected at day 17 in 1979, day 16 in 1981, and day 15 in fall 1982.

[c] Standard deviation.

certain natural situations. The plant density employed here typifies that of wild *Phlox* populations occupying shaded woodland habitats, an environment that, according to this study, will promote chronically high pathogen pressure. Generally, such populations have been shown to be uniform or highly skewed for high resistance levels (14). This implies that, in these natural populations, highly susceptible plants are always selectively disadvantaged irrespective of the resistance heterogeneity present.

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