Host Diversity Can Reduce Potato Late Blight Severity for Focal and General Patterns of Primary Inoculum

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ABSTRACT

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The use of host diversity as a tool for management of potato late blight has not been viewed as promising in the past. But the increasing importance of late blight internationally has brought new consideration to all potential management tools. We studied the effect of host diversity on epidemics of potato late blight in Oregon, where there was little outside inoculum. The experimental system consisted of susceptible potato cv. Red LaSoda and a highly resistant breeding selection, inoculated with local isolates of US-8 *Phytophthora infestans*. Potatoes were grown in single-genotype plots and also in a mixture of 10 susceptible and 26 resistant potato plants. Half of the plots received inoculation evenly throughout the plot (general inoculation) and half received an equal quantity of inoculum in only one corner of the plot (focal inoculation). The area under the disease progress curve (AUDPC) was greater in single genotype stands of susceptible cv. Red LaSoda inoculated throughout the plot than with stands inoculated in one focus. The host-diversity effect on foliar late blight was significant in both years of the investigation; the AUDPC was reduced by an average of 37% in 1997 and 36% in 1998, compared with the mean disease level for the potato genotypes grown separately. Though the evidence for influence of inoculum pattern on host-diversity effects was weak ($P = 0.15$), in both years there was a trend toward greater host-diversity effects for general inoculation. Statistical significance of host-diversity effects on tuber yield and blight were found only in one of the two years. In that year, tuber yield from both the resistant and susceptible cultivar was increased in mixtures compared with single genotype stands and tuber blight was decreased in mixtures for susceptible cv. Red LaSoda.

Additional keywords: cultivar mixtures, genetic diversity, spatial pattern of inoculum, variety mixtures.

Host diversity slows epidemics of such diseases as rusts and mildews of small grains to the degree that host diversity can be manipulated as a disease management tool (20). In contrast, the effect of host diversity on epidemics of larger plants has been predicted to be small (19). A host-diversity effect can be defined as a change in a response such as percent disease severity, when host genotypes are mixed in a population compared with when the same genotypes are grown in separate populations. We have summarized some predictors for the magnitude of host-diversity (7). In general, the greatest effect of host diversity for reduced disease might be expected for host–pathogen systems with small host plants, shallow pathogen dispersal gradients, small lesions, short pathogen generation times, and pathogen populations with strong host specialization (7). Host-diversity effects are predicted to be greater for smaller host plants such as wheat and barley, because inoculum on small plants may be more effectively mixed throughout the host population as opposed to landing mostly on the source host individual (14,15). Potatoes are relatively larger plants and so might have a greater level of autoinfection (18) than small grains for pathogens with similar dispersal gradients. Also, lesion expansion in late blight can be very important, especially for stem lesions that can expand to destroy a large portion of individuals. Overall levels of variability tend to be low in populations of the causal agent, *Phytophthora infestans*, because only one or a few clonal lineages are represented in many populations (3,9,11), so there may be little pathogen specialization to manipulate through host mixtures. The above predictors suggest that host-diversity effects would be smaller for late blight of potatoes than for rusts and mildews of small grains. On the other hand, there may be greater levels of variability in *P. infestans* populations that is relevant to host specificity than is detected in general surveys of genetic variation such as restriction fragment length polymorphism and amplified fragment length polymorphism analyses. Also, the more pathogen generations that occur during an epidemic, the greater the effect of host diversity will tend to be (10). Although late blight epidemics may be rapid on susceptible potatoes with no fungicide applications, the generation time for *P. infestans* is relatively brief (12,13). Also, though there is variability in estimates of dispersal gradients for *P. infestans*, gradients tend to be relatively shallow (16; K. A. Garrett and C. C. Mundt, *unpublished data*). In summary, these predictors give a mixed review on the likelihood that a host-diversity effect will be observed for potato late blight.

In field studies of late blight in potato genotype mixtures in France, Andrivon found evidence for a host-diversity effect (D. Andrivon, *personal communication*). But in studies of potato genotype mixtures in Ecuador, in collaboration with G. A. Forbes, R. J. Nelson, G. Chacon, and R. Jaramillo of the International Potato Center, an important host-diversity effect was found at only one of three experimental sites (6). One important difference between Ecuadorian and European epidemics is that late blight in France tended to develop from obvious foci, a typical spatial pattern for northern Europe (D. Andrivon, *personal communication*). In Ecuador, on the other hand, disease was widespread throughout experimental plots from early on in the epidemics (6). Mundt and Leonard (14) found that the effect of host diversity on rust epidemics was greater when epidemics began with a limited number of foci.

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Although potato cultivars resistant to late blight are being developed, currently management in disease-conducive environments is dependent on use of fungicides (5). Because potatoes are an important food crop for small farmers in many parts of the world, the cost of fungicides can be problematic. Better resistance to late blight is needed, as are optimal strategies for deploying resistance. If potato genotype mixtures prove to be useful for late blight management, there are several potential groups of users. Small farmers have traditionally used mixtures of crop genotypes and species (17), and it might be possible to improve strategies for combining cultivars with late blight management in mind. It might also be possible to choose true potato seed parents to optimize the variability in resistance of progeny. For large-scale commercial growers, advances in plant breeding technology may make it possible to incorporate desired variability in late blight resistance in potato mixtures while also maintaining a desired level of agronomic and culinary consistency.

Our goal in this study was to test for host-diversity effects in potato late blight under two patterns of initial inoculum, focal and general (21). For epidemics that began in foci, inoculum was applied only to one corner of experimental plots; for general epidemics, inoculum was applied evenly throughout experimental plots. We tested whether there was an effect of host diversity on foliar disease progress, tuber infection, and tuber yield, and how such effects might differ for the two spatial patterns of inoculation.

MATERIALS AND METHODS

Experimental plot design. Experimental plots were planted in a randomized complete block design on 12 and 13 June 1997 and 11 May 1998, at the Lewis-Brown Horticultural Research Farm near Corvallis, OR. Planting in 1998 was earlier to minimize the possibility of outside inoculum. Treatments were as listed in Table 1, with plants arranged as in Figure 1. Three blocks were planted in 1997 and four in 1998.

Plots were four rows wide (3.45 m) with 0.86 m between 2.06-m long rows, and with 0.23-m spacing between the nine potato plants within a row. Borders of rye (*Secale cereale*) in 1997 and oats (*Avena sativa*) in 1998 were planted around potato plots so that a plot's nearest neighboring plot was at least 4.9 m away. Overhead irrigation was used at rates comparable with commercial production, but additional brief irrigation was added in particularly dry, hot conditions to help ensure epidemics. In order to prevent infection before artificial inoculation, chlorothalonil was applied at the highest labeled rate on 14 and 21 July 1997 and 12 and 19 June 1998. In addition, cymoxanil was applied 6 June 1998. Fields were scouted regularly and no lesions were detected before plots were inoculated in either year.

Fig. 1. Planting pattern for potato genotype mixtures in experimental plots, indicating the location of the late blight-susceptible cv. Red LaSoda (Sus) and the resistant breeding selection A90586-11 (Res). Each "Sus" and "Res" indicates the position of an individual plant.

Inoculation. A local US-8 isolate collected in the previous year was used for inoculations. Cultures were grown on rye agar plates for 2 weeks prior to inoculation. Plates were washed to produce a suspension of 2.7×10^4 sporangia per ml in 1997 and 1.3×10^6 in 1998. Plants were inoculated in the evenings of 8 August 1997 and 8 July 1998, by placing 10-μl drops of the suspension on the upper surfaces of leaflets with a repeater pipette. In plots receiving general inoculation, 144 drops of the sporangial suspension was put in each plot, arranged as approximately 4 drops per plant. In plots receiving focal inoculation, 144 drops per plot was added, arranged as approximately 36 drops on each of four plants in one corner of the plot. In plots containing mixtures, genotypes were arranged so that one of the four corner plants inoculated was susceptible cv. Red LaSoda (cv. Red LaSoda made up approximately one-fourth of the plants in the plot).

Disease severity. The percentage of blighted foliage was estimated visually in each plot, for all plants in the plot regardless of genotype, and averaged for two observers. Estimates were made between 18 August and 9 September 1997 and between 23 July and 4 August 1998.

Yield measurements. Tubers were harvested 25 September 1997 and 9 September 1998 after vines had been killed with a herbicide as a standard production practice. Tubers from the outer two rows of each plot were collected and, the following day, the number of tubers with apparent bacterial soft rot and the number that appeared firm were recorded. We assumed that soft rot indicated earlier tuber infection by *P. infestans*, although firm tubers may also have been infected. Firm tubers were weighed for a measure of yield. Both counts and weights were recorded for tubers at least 2.5 cm in diameter.

Statistical analysis. For the analyses of percent severity, the area under the disease progress curve (AUDPC) was calculated according to the midpoint rule (2) to summarize the epidemic in each year. Analysis of variance (ANOVA) was performed on the AUDPCs, treating all combinations of inoculum pattern and host genotype as different treatments to make the formulation of tests clearer (Table 1). The following linear model was fit to the data

$$y_{ijk} = \mu + \tau_i + \beta_j + \gamma_k + (\tau\gamma)_{ik} + \varepsilon_{ijk}$$

where y_{ijk} is the AUDPC for the *ijkth* experimental plot; τ_i is the effect of the *ith* treatment ($i = 1,…,6$); β_j is a random effect, with variance σ_β^2, due to the *jth* block; γ_k is the effect of the *kth* year; $(\tau\gamma)_{ik}$ is the treatment by year interaction term; and ε_{ijk} ($j = 1,…,3$ in 1997 and $j = 1,…,4$ in 1998) is the residual error. Because variation in the residual error was greater for the susceptible cultivar than for the resistant cultivar, we used an analysis in which the variance of the residual error was estimated separately for the resistant cultivar, susceptible cultivar, and mixture. The SAS code for the analysis of AUDPC is as follows: Proc Mixed; class year treatment cultivar block; model AUDPC = year | treatment / ddfm = satterth; random block (year); repeated / group = cultivar. Planned linear contrasts were used to test effects as **c'**τ, where τ is the vector of treatment effects in the order of Table 1 and **c** is the

TABLE 1. Experimental treatments used to study the effects of initial inoculum pattern for *Phytophthora infestans* and host genotypic composition on epidemic progression of potato late blight [a]

Treatment	Pattern[b]	Potato genotype
1	General	36 susceptible cv. Red LaSoda
2	General	36 resistant breeding selection A90586-11
3	General	10 cv. Red LaSoda + 26 A90586-11
4	Focal	36 cv. Red LaSoda
5	Focal	36 A90586-11
6	Focal	10 cv. Red LaSoda + 26 A90586-11

[a] The spatial pattern of individual plants in mixtures is shown in Figure 1. Seed of A90586-11 was supplied courtesy of D. Corsini (University of Idaho).
[b] Pattern of inoculum within experimental plots.

vector of coefficients for a particular contrast. Most of the contrasts were straightforward and the values of c for each are given in Table 2. Because the interaction between genotype and inoculum pattern was significant in the test of linear contrasts (Table 2), we also analyzed the effect of inoculum pattern on the genotypes in separate analyses. In addition to these analyses of absolute differences in responses, we also analyzed the relative mixture response (RMR), or the ratio of the response observed in mixtures to the appropriate weighted mean of the responses in pure stands. We calculated this ratio separately in each block to maintain the independence of observations (8). The following linear model was used to fit the RMR data

$$y_{ik} = \mu + \tau_i + \gamma_k + \varepsilon_{ijk}$$

where y_{ijk} is the RMR for the $ijkth$ experimental plot; τ_i is the effect of the ith treatment (i = 3 or 6 only, corresponding to the treatment in the denominator); γ_k is the effect of the kth year; and ε_{ijk} (j = 1,…,3 in 1997 and j = 1,…,4 in 1998) is the residual error.

For the analysis of yield, tubers of the two genotypes could be separated visually by color, so we were able to analyze both total yield in mixtures and the yield of each genotype in mixtures. Contrasts were constructed similarly to those for the analysis of percent severity, but for separate analyses of yield for cv. Red LaSoda, the treatment levels listed in Table 1 collapse into only levels 1, 3, 4, and 6, and for A90586-11, into levels 2, 3, 5, and 6. The percentage of infected tubers for the two genotypes was analyzed in the same way as yield. The patterns of residuals from analyses of yield and tuber infection were acceptable for the assumptions of the ANOVA. All statistical analyses were performed using SAS software (SAS Institute Inc., Cary, NC).

RESULTS

Percent severity was higher in single-genotype plots with general inoculum. Disease levels were significantly higher under general compared with focal inoculum (Fig. 2; Table 2). The separate analyses of the two potato genotypes clarified that inoculum pattern had an important effect only for cv. Red LaSoda and not for A90586-11. Results were similar for the analysis of the RMR (Table 2).

The host-diversity effect for reduced percent severity was large and statistically significant. The reduction was statistically significant for both the absolute and the relative mixture response (Table 2). The AUDPC was reduced by an average of 37% in 1997 and 36% in 1998. The overall effect of year on disease severity was significant (P = 0.057), but there was no evidence for a

year–treatment interaction (P = 0.95). Results for the RMR were similar (Table 2).

There was a trend toward greater host-diversity effects for reduced percent severity for plots with general inoculum. The trend was consistent in both years of the study (Fig. 2), but there was only weak statistical support for the difference (P = 0.146) (Table 2).

There was a host-diversity effect on yield only in the first year. Yield was higher in the mixture plots, even for the resistant genotype (Table 3). This effect was significant for both genotypes considered individually, as well as for the total yield (Table 4). There was no evidence for an inoculum pattern effect on yield in single genotype stands, but there was some evidence for an interaction between inoculum pattern and the size of host-diversity effects, mostly from differences in A90586-11 (Table 4). There were no significant effects on yield in 1998, other than the difference between host genotypes (Table 4).

There was a host-diversity effect for decreased tuber infection in cv. Red LaSoda only in the first year. Tuber blight incidence was reduced almost by half (Table 3). In the second year, responses were more variable and there were no significant differences. There was no evidence for an effect of inoculum pattern (Table 4). No tuber blight was detected for A90586-11 in 1997. In 1998, only very low levels of infection were found for A90586-11 (Table 3), and there was no evidence for effects of host diversity or inoculum pattern.

DISCUSSION

In contrast to earlier predictions (19), but corroborating Andrivon's result (data not shown), there was a significant effect of host diversity on severity of foliar symptoms of late blight. The mixtures were composed of only a susceptible and a highly resis-
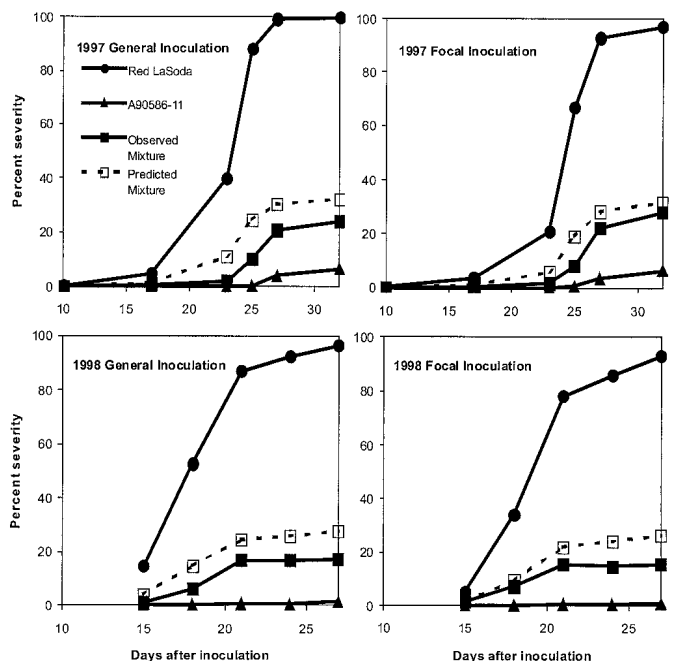

Fig. 2. Disease progress curves for potato late blight, with each solid line in 1997 representing the mean of three experimental plots and each solid line in 1998 representing the mean of four experimental plots. The upper line is the progress curve for single genotype stands of 36 plants of susceptible potato cv. Red LaSoda. The lower line is the progress curve for single genotype stands of 36 plants of the resistant potato breeding selection A90586-11. The solid middle line is the progress curve for a mixture of 10 plants of cv. Red LaSoda and 26 plants of A90586-11. The dotted middle line indicates the average of the responses in single genotype stands, weighted according to their proportion in the mixture; this is the predicted disease progress curve in the mixtures under the null hypothesis of no host-diversity effect.

TABLE 2. Analysis of variance (ANOVA) of the area under the disease progress curve (AUDPC) for late blight in single genotype and mixed genotype plots of potato

Source (single df contrast)[a]	P value (linear contrast)[b]	Form of contrast (c')	P value (RMR)[c]
IP	0.022	(1, 1, 0, –1, –1, 0)	…
Red LaSoda (susceptible)	0.023	(1, 0, 0, –1, 0, 0)	…
A90586-11 (resistant)	0.621	(0, 1, 0, 0, –1, 0)	…
IP–HG interaction	0.025	(1, –1, 0, –1, 1, 0)	…
HD effect	0.000	(10/36, 26/36, –1, 10/36, 26/36, –1)	0.000
General IP	0.000	(10/36, 26/36, –1, 0, 0, 0)	0.000
Focal IP	0.001	(0, 0, 0, 10/36, 26/36, –1)	0.000
HD–IP interaction	0.146	(10/36, 26/36, –1, –10/36, –26/36, 1)	0.217

[a] IP = inoculum pattern; HG = host genotype; and HD = host diversity. The order of treatments for contrasts is as in Table 1.

[b] P values for tests that indicate significance at the α = 0.1 level are underlined.

[c] Relative mixture response is a ratio with the AUDPC for a mixture in the numerator and the weighted mean of the AUDPC for single-genotype stands of the mixture components in the denominator.

TABLE 3. Observed mean yield and mean percentage of soft rot-infected tubers per experimental plot for a resistant and a susceptible potato genotype planted in single-genotype stands and in mixtures inoculated with *Phytophthora infestans* in two different spatial patterns [a]

| | Mean yield (kg per plant) | | | | Mean percent infected tubers | | | |
| | 1997 | | 1998 | | 1997 | | 1998 | |
Source	Focal	General	Focal	General	Focal	General	Focal	General
Red LaSoda (susceptible)								
Mixed genotype plots	0.533	0.398	0.531	0.491	20	19	0	9.4
Single genotype plots	0.338	0.310	0.695	0.791	37	36	3.0	2.3
A90586-11 (resistant)								
Mixed genotype plots	1.27	1.14	2.40	1.98	0	0	2.2	3.0
Single genotype plots	1.00	1.12	2.31	2.06	0	0	2.0	3.3

[a] Inoculum was applied in either a focal or a general pattern in the experimental plots.

tant genotype, so the mechanisms that may have contributed to the effect are limited in number (7). Host-diversity effects might have been due to the decreased proportion of susceptible tissue, physical barriers to inoculum spread, or compensation or competition between host genotypes (7). It is likely that the resistant plants produced very little inoculum while the epidemic proceeded on susceptible plants, so the inoculum level was probably greatly reduced in mixtures. The resistant plants may have acted as barriers to dispersal of sporangia. Apparently, despite the relatively large size of potato plants, inoculum was dispersed far enough from source individuals to limit the amount of autoinfection and was blocked by resistant plants so that spread to other susceptible individuals was limited. In addition, the effect of large plant size may have been mitigated by the fact that plants grew to be intertwined. Because the epidemics were rapid, growth by the resistant genotype to compensate for loss in susceptible plant matter did not seem to be a factor until disease on susceptible plants approached 100% severity (K. A. Garrett, *personal observation*). Another possible mechanism for reduction in late blight in mixtures is that competition with the resistant cultivar may have influenced the susceptibility of cv. Red LaSoda; such competitive influences have previously been suggested to operate in genotype mixtures (4).

Results of mixture studies in Ecuador were variable; some sites showed little effect of host diversity on late blight, but one site showed an important effect (6). Part of the reason for different performance at different sites in Ecuador is probably the degree of inoculum entering fields from outside sources. There may be high levels of inoculum reaching fields near other infected fields (6) and these spore showers may overwhelm effects of host diversity (20). One of our motives in performing this study was to determine how inoculum pattern influenced the effect of host diversity. The influence of inoculum pattern on host-diversity effects was not strongly statistically significant, but there was a consistent trend for a larger host-diversity effect in plots with general placement of initial inoculum. This is in contrast to the results of previous studies, in which focal inoculum resulted in greater host-diversity effects for rust (14). If the reverse effect in potato late blight is found to be real and consistent, this might be because of the different dispersal gradients for the two systems or because of the great importance of lesion expansion for *P. infestans*. But, rather than concluding that there should be a greater host-diversity effect in settings with what appears to be a general spatial inoculum, it is also important to consider the total amount of inoculum coming into a field. Continual inflow of spores would likely decrease host-diversity effects more than the arrangement of initial inoculum could.

There was evidence for greater levels of infection under general inoculation when compared with focal inoculation for single-genotype plots of susceptible cv. Red LaSoda. This might be anticipated because, for focal inoculation, there is a lag time before infection occurs in areas further from the primary infection. Also, for general inoculation, lesion expansion can continue longer before the limits to uninfected tissue are reached. The result seems

intuitive and corroborates studies with rusts (14), but there has been surprisingly little work done on the influence of inoculum pattern on epidemic progression. As both mating types of *P. infestans* occur together in fields in more potato growing areas, soilborne oospores may become more important (1) and form a frequent source of a general inoculum pattern.

The effect of host diversity on yield is more difficult to interpret in our study, because we were not working with isolines that differed only in late blight resistance. A host-diversity effect for increased yield was found in 1997 not only for susceptible cv. Red LaSoda, as might be predicted by its decreased disease levels in mixtures, but also for the resistant genotype. We can speculate that benefits to the resistant genotype occurred because of lowered competition when growing with susceptible cv. Red LaSoda. The test for a host-diversity effect on yield for cv. Red LaSoda is not as straightforward as it might be, because the outer two rows of each experimental plot were harvested. This was done in an attempt to capture as much of the potential effects of inoculum pattern as possible in the small experimental plots, for which all rows were close to the corner that received inoculation for focal inoculum treatments. Because the outer two rows were sampled, two-fifths of cv. Red LaSoda plants in mixtures were planted on the corners of the experimental plots (Fig. 1), and thus might be expected to be larger than plants in the middle of rows because of lower interspecific competition. This is in contrast to the single-genotype plots used for estimating host-diversity effects; in these, only 4 of 18 plants in the outer two rows were corner plants. Thus, yield per plant in mixtures might have been greater simply because a higher percentage of cv. Red LaSoda individuals were corner plants. But we were able to test whether this mechanism was at work by comparing cv. Red LaSoda in the first and last rows from plots of mixtures receiving general inoculum. If yield for corner plants was greater, then the row with two cv. Red LaSoda individuals on the corners should have a higher cv. Red LaSoda yield per cv. Red LaSoda plant than the row with both cv. Red LaSoda individuals in the middle of the row (Fig. 1). In 1997, the average yield per cv. Red LaSoda plant in the row with cv. Red LaSoda individuals on the corners was 0.75 kg, and the average yield per cv. Red LaSoda plant in the row with no cv. Red LaSoda individuals on the corners was 1.7 kg. Apparently, increased yield in mixtures in 1997 was not an artifact of susceptible plant position within the experimental plots. In 1998, there was no significant effect of host diversity on yield, but the trend was toward decreased yield in mixtures.

For tuber infection as well, the 2 years were very different. In 1997, no infection was detected in the resistant genotype. In that year, there was a statistically significant reduction in infection on susceptible cv. Red LaSoda. In the second year there were no significant effects, but there was infection in the resistant genotype. In the second year, the epidemic was protracted by hot, dry conditions so that the epidemic seemed to come to a standstill and tubers were probably exposed to lower levels of sporangia for a longer period of time. The analysis is further complicated by the fact that some tubers with very high levels of soft rot were noted in the field but could not be counted with confidence. Thus, our

TABLE 4. Analysis of the effects of potato genotype, genotype mixture, and initial inoculum pattern of *Phytophthora infestans* on potato yield and percent soft rot-infected tubers

| | P values from ANOVA[a] | | | | |
| | Yield | | % Infected tubers | | |
Source	1997	1998	1997	1998	Form of contrast (**c'**)[b]
Both genotypes combined					
Host-diversity effect	<u>0.009</u>	0.619	…	…	(10/36, 26/36, –1, 10/36, 26/36, –1)
Focal inoculum pattern	<u>0.002</u>	…	…	…	(0, 0, 0, 10/36, 26/36, –1)
General inoculum pattern	0.570	…	…	…	(10/36, 26/36, –1, 0, 0, 0)
Inoculum pattern	0.418	0.563	…	…	(1, 1, 0, –1, –1, 0)
Host genotype	<u>0.000</u>	<u>0.000</u>	…	…	(1, –1, 0, 1, –1, 0)
Host diversity–inoculum pattern interaction	<u>0.041</u>	0.521	…	…	(10/36, 26/36, –1, –10/36, –26/36, 1)
Inoculum pattern–host genotype interaction	0.200	0.207	…	…	(1, –1, 0, –1, 1, 0)
Red LaSoda (susceptible)					
Host-diversity effect	<u>0.003</u>	0.147	<u>0.004</u>	0.683	(1, –1, 1, –1)
Focal inoculum pattern	<u>0.017</u>	…	<u>0.016</u>	…	(0, 0, 1, –1)
General inoculum pattern	<u>0.020</u>	…	0.028	…	(1, –1, 0, 0)
Host diversity–inoculum pattern interaction	0.805	0.658	0.791	0.318	(1, –1, –1, 1)
Inoculum pattern in pure stands	0.937	0.658	0.704	0.925	(1, 0, –1, 0)
A90586-11 (resistant)					
Host-diversity effect	<u>0.059</u>	0.972	…	0.971	(1, –1, 1, –1)
Focal inoculum pattern	<u>0.020</u>	…	…	…	(0, 0, 1, –1)
General inoculum pattern	0.842	…	…	…	(1, –1, 0, 0)
Host diversity–inoculum pattern interaction	<u>0.093</u>	0.613	…	0.820	(1, –1, –1, 1)
Inoculum pattern in pure stands	0.256	0.279	…	0.414	(1, 0, –1, 0)

[a] Analysis of variance. P values for tests that indicate significance at the $\alpha = 0.1$ level are underlined.

[b] The order of treatments for contrasts is as in Table 1. For analyses of cv. Red LaSoda alone, treatments 1, 3, 4, and 6 are present; for analyses of A90586-11 alone, only treatments 2, 3, 5, and 6 are present.

measures of percent infection are based on tubers with intermediate levels of soft rot, those that may have had large soft areas but still maintained some structural integrity, and the number of tubers with high levels is not known. Our analysis is also dependent on the assumption that soft rotting occurred because tubers had previously been infected by *P. infestans*.

As a first step in evaluating the usefulness of potato mixtures for late blight management, we have learned that there can be a host-diversity effect for reduced late blight, corroborating Andrivon's (data not shown) results. We have also demonstrated the potential for increased yield in simple potato mixtures. The next step is to learn how to manipulate the mechanisms that produce the effect to try for optimal disease management. Although the sort of simple mixture described here may be of direct use for growers who might use a less valuable resistant cultivar to protect a more valuable susceptible cultivar, it functions mainly as an experimental system for probing the presence of host-diversity effects (7). One obvious problem is that use of this mixture did not slow epidemic progress enough to be a sufficient form of management if used alone. The speed of the epidemic probably resulted in part because of the small plot size and heavy initial inoculum load, but even in settings more conducive to host-diversity effects this mixture probably would not have been adequately protected. Use of fungicides to slow epidemics in combination with mixtures would allow both a viable period of foliage for tuber-filling and a longer epidemic with more generations and thus, potentially, a larger benefit from mixing (6). A more useful mixture would probably contain more genotypes, where each cultivar has a high enough level of quantitative resistance that it will not rapidly be defoliated by compatible pathogen races. Although potato breeders currently de-emphasize use of qualitative resistance, potato mixtures may be one means of using qualitative resistance that still has some value for extant pathogen populations. Even if a sizable proportion of the pathogen population has genes for overcoming the particular forms of qualitative resistance, deployment of genes for qualitative resistance in mixtures may still partition the pathogen population so that overall disease levels are reduced. High levels of tuber blight resistance would also be desirable in all mixture components.

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