The Influence of Host and Pathogen Genotypes on the Apparent Infection Rates of Potato Late Blight Epidemics

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ABSTRACT

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The capacity of potato genotypes to reduce the apparent infection rate of potato late blight epidemics was investigated by studying different isolates of *Phytophthora infestans*. In 1977, the rate-reducing capacity of seven host genotypes was assessed with two pathogen isolates and, in 1978, five host genotypes were tested with four isolates. The isolates were selected only for ability to overcome vertical resistance conditioned by the R_1 gene. Epidemics were monitored in the field and apparent infection rates were calculated for each of the 14 cultivar/isolate combinations in 1977 and the 20 cultivar/isolate combinations in 1978. The apparent infection rates were subjected to analysis of variance. An orthogonal comparison procedure revealed cultivar × isolate interactions with probability levels of $P \leqslant 0.07$ and $P \leqslant 0.01$ for 1977 and 1978, respectively. The interactions imply that the level of rate-reducing capacity of host genotypes differs for different isolates of the pathogen. It is suggested that several different isolates of the pathogen be employed in screening host genotypes in order to assess their rate-reducing capacity.

Attempts to achieve a stable resistance in potato (*Solanum tuberosum* L.) to *Phytophthora infestans* (Mont.) de Bary by the incorporation of R genes (2) from *S. demissum* into *S. tuberosum* have failed repeatedly (12). Consequently the disease is controlled by frequent applications of protective fungicides, which is both expensive and time-consuming. A search for potato genotypes which characteristically reduce the apparent infection rate (12) of the epidemic currently is receiving greater attention. Potato cultivars possessing such an attribute have been shown to require less fungicide to achieve a desired level of disease control (4).

The term "rate-reducing capacity" used herein is based on the

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fact that in the same environment, epidemics involving some potato cultivars have lower apparent infection rates than others. Fry (4) referred to this phenomenon as an expression of polygenic resistance. It qualifies as horizontal resistance as defined by Nelson (7).

The stability of this rate-reducing capacity remains to be determined. It can be argued that its stability is dependent upon the number of genes conditioning the trait, assuming that the number of genetic changes that must occur in the pathogen for it to overcome the resistance is proportional to the number of genes conditioning the resistance in the host. Stability also is likely to be affected by the variability and the relative parasitic fitness (8) of the pathogen population. The chance that pathogen genotypes will appear that seemingly are not affected by host resistance genes will be increased for a pathogen population which produces more offspring with a broad range of variability.

Major questions remain to be answered if rate-reducing genotypes are to be selected for potato breeding programs. Of primary importance is whether one isolate of the pathogen is sufficient to identify rate-reducing capacities that can withstand the variability of the pathogen population. Potentially, sufficient pathogen variation could exist to preclude the effectiveness of single-isolate screening.

The purpose of this study was to statistically test interactions among selected commercial potato cultivars and naturally occurring isolates of the pathogen population chosen only for their capacity to overcome vertical resistance.

MATERIALS AND METHODS

1977 Experiment. Seven potato cultivars (Bintje [B], Green Moutain [GM], Katahdin [Ka], Kennebec [Ke], Russet Rural [RR], Sebago [Se], and Superior [Su]) were evaluated at The Pennsylvania State University Plant Science Research Center at Rock Springs for relative rate-reducing capacity against two pathogenic isolates of *P. infestans*. The isolates were designated as I and II, and were identified as race 1, 2, 3, 4, and race 1, 4, respectively (2). The fact that Ke possesses the R_1 gene for blight resistance should not affect the rate-reducing capacity since both isolates are pathogenic in the presence of the R_1 gene.

The experimental design was a split-plot with whole plots represented by isolates and subplots represented by cultivars. The whole plots were separated by a distance of approximately 100 m and were not replicated. Subplots of each of the seven cultivars were replicated three times in each whole plot, and consisted of six rows 6.0 m in length. Spacing between rows and between plants within rows measured 91 cm and 25 cm, respectively. Each subplot was surrounded by six rows of field corn planted 45 cm apart. The corn served as a barrier to confine the inoculum produced by different epidemics within their respective subplots. The experiment was analyzed as a variety trial with isolates treated as locations (3).

A single plant near one end of one of the middle rows of each subplot was inoculated with the appropriate isolate of *P. infestans* at about 1950 hours on 1 July 1977. Inoculum consisted of a zoospore suspension from sporulating cultures grown on lima bean agar. Inoculum was standardized at 10^5 zoospores per milliliter of glass-distilled water. A manually operated sprayer was used to apply 25 ml of inoculum suspension to each inoculated plant.

Disease severity in each subplot was assessed from a single area approximately 1 m^2, surrounding the inoculated plant. The Horsfall-Barratt (6) rating system was used in making assessments which were recorded at 7, 9, 11, and 13 days after inoculation. Disease severity proportions were transformed to logits and regressed on time to obtain the apparent infection rate of each epidemic.

Analysis of variance was performed on the calculated apparent infection rates. Data were analyzed by using the Minitab (10) and SAS (1) statistical programs on The Pennsylvania State University Computation Center IBM System 3033 computer. An orthogonal comparison procedure was employed to identify sources of significant variation within the interaction (11). By this method the interaction sum of squares was partitioned into contrasts with one degree of freedom. These contrasts were based on independent comparisons of the cultivar rate-reducing capacity among and within isolates. The orthogonal arrangements were selected in light of a previous report (4) concerning the expression of polygenic resistance in potato cultivars and first-hand observations made at the Rock Springs Farm.

1978 Experiment. The rate-reducing capacity of five potato cultivars (GM, Ka, Ke, RR, and Se) was assessed in a 0.4-ha (1-acre) field by using four pathogenic isolates of *P. infestans* at the Rock Springs Farm. The isolates were designated as isolates I, II, III, and IV. Isolates I and II were the same ones used in 1977 and isolates III and IV were identified as race 1, 2, 3, 4 and race 1, 4, respectively.

The experimental design was a split-plot with whole plots represented by the four isolates and subplots represented by the five cultivars. Thus, there were 20 different cultivar/isolate combinations. Each subplot consisted of three 4-m-long rows of a single cultivar planted 91 cm apart. Individual subplots were replicated three times in each whole plot. Each subplot was surrounded by four rows of field corn planted 47 cm apart. Alleyways 4.5-m wide existed between replications and were used for insecticide spraying equipment. The experiment was analyzed as a cultivar trial with isolates treated as locations (3).

Inoculum was obtained from sporulating lesions on detached leaflets of infected plants of potato cultivar Abnaki. Sporangia of each of the four isolates were washed from the leaflets with cold (10 C) glass distilled water. The recovered spore suspensions were then stored in glass beakers at 10 C. After 3 hr the inoculum suspensions were standardized to 10^5 zoospores per milliliter. The center plant of each subplot was sprayed with approximately 25 ml of the appropriate isolate suspension using a manual sprayer at 2000 hours on 7 August 1978.

At 5 days after inoculation, lesions were first observed only on the center plant. Thereafter, blight severity was assessed by using the Horsfall-Barratt rating system (6) at 2- or 3-day intervals for 21 days after the primary lesions appeared. Five plants, including the center plant and four plants surrounding the center plant at 1.5 m from it, were assessed for blight severity. Recorded disease severity proportions were transformed into logits and regressed on time (days) to obtain the apparent infection rate of each epidemic.

TABLE 1. Analysis of variance for apparent infection rates of epidemics involving two isolates of *Phytophthora infestans* on seven potato cultivars in field plots in 1977

Source	D.F.	M.S.	F
Isolates	1	0.0822	16.78**
Reps in Isolates	4	0.0049	0.77
Cultivars	6	0.0279	4.36**
(B vs RR)[a]	1	0.0083	1.30
(Ka vs Su)[b]	1	0.0034	0.53
(Ke vs GM)[c]	1	0.0013	0.21
(B/RR vs Ka/Su)[d]	1	0.0509	7.95**
(B/RR/Ka/Su vs Ke/GM)[e]	1	0.0404	6.31**
(Se vs others)[f]	1	0.0633	9.89**
Cultivars × Isolates	6	0.0144	2.25
(B vs RR) × Isolates	1	0.0062	0.99
(Ka vs Su) × Isolates	1	0.0604	9.43**
(Ke vs GM) × Isolatese	1	0.0001	0.01
(B/RR vs Ka/Su) × Isolates	1	0.0025	0.39
(B/RR/Ka/Su vs Ke/GM) × Isolates	1	0.0106	1.67
(Se vs others) × Isolates	1	0.0061	0.96
Reps × Cultivars in Isolates (error)	24	0.0064	

[a] Contrast between cultivars Bintje and Russet Rural.
[b] Contrast between cultivars Katahdin and Superior.
[c] Contrast between cultivars Kennebec and Green Mountain.
[d] Contrasts between the Bintje/Russet Rural combination and the Katahdin/Superior combination.
[e] Contrast between the Bintje/Russet Rural/Katahdin/Superior combination and the Kennebec/Green Mountain combination.
[f] Contrast between Sebago and all other cultivars; **highly significant F-test, $P \leq 0.01$.

Analysis of variance was performed on apparent infection rates and the same orthogonal comparison procedure was used to identify sources of significant variation within the interaction as was used in 1977. The contrasts were based on independent hypotheses regarding the cultivar rate-reducing capacity among and within isolates and isolate fitness among and within cultivars. The hypotheses were tested with an F-test.

RESULTS

1977 Experiment. The analysis of variance performed on the apparent infection rates for the 1977 data show highly significant ($P \leq 0.01$) effects due to cultivars and isolates (Table 1). The probability level of the cultivar × isolate interaction was $P \leq 0.07$. The partitioning of the interaction sum of squares into independent contrasts allowed the major source of variation within the interaction to be identified. The (Ka vs Su × Isolates) contrast was highly significant and accounted for most of the interaction variation. The apparent infection rates of Isolates I and II on Ka were 0.30 and 0.54, respectively, while those of Su were 0.48 and 0.43, respectively (Table 2). In terms of the expression of their rate-reducing capacities, Ka was more resistant than Su when inoculated with Isolate I, but the order was reversed for Isolate II (Table 2).

1978 Experiment. Analysis of variance for the 1978 experiment showed highly significant ($P \leq 0.01$) contributions to observed variation by both cultivars and isolates (Table 3). The contribution of the cultivar × isolate interaction was also significant ($P \leq 0.05$). The interaction sum of squares was partitioned into four contrasts, each with three degrees of freedom. Results of F-tests from these contrasts suggested that the variation in the difference between RR and others over isolates was the major source of variation involving the cultivar × isolate interaction, and that this variation was responsible for the expression of a significant interaction. However, an F-test with more than one degree of freedom for the numerator mean square represents an average test of as many independent comparisons as there are degrees of freedom. It is possible for this average test to fail to detect significant variation among independent comparisons. For this reason, the ([Ke vs Se] × [II vs IV]) contrast with three degrees of freedom was partitioned into three independent contrasts (Table 3). As a result, the ([Ke vs Se] × [II vs IV]) contrast was found to have a probability level of $P \leq 0.06$.

Examination of the mean apparent infection rates for the 20 cultivar/isolate combinations presents another perspective of the interaction between Ke and Se and Isolates II and IV (Table 2). For epidemics involving Isolate II, the rate-reducing capacity of Ke ($r = 0.24$) exceeded that of Se ($r = 0.27$). The order was reversed for inoculations made with Isolate IV ($r = 0.19$) and $r = 0.16$ for Ke and Se, respectively).

TABLE 2. Apparent infection rates of epidemics involving different isolates of *Phytophthora infestans* on potato cultivars in field plots in 1977 and 1978

	Isolate					
	1977		1978			
Cultivar	I	II	I	II	III	IV
Russet Rural	.55[a]	.56	.40	.48	.33	.34
Bintje	.46	.55	...	...	...	...
Katahdin	.30	.54	.33	.32	.28	.21
Superior	.48	.43	...	...	...	...
Kennebec	.35	.49	.26	.24	.24	.19
Green Mountain	.33	.48	.32	.32	.29	.23
Sebago	.34	.36	.27	.27	.24	.16

[a] All values represent the mean of three replications.

TABLE 3. Analysis of variance for apparent infection rates of epidemics involving four isolates of *Phytophthora infestans* on five potato cultivars in field plots in 1978

Source	D.F.	M.S.	F
Isolates	3	0.0319	35.44**
Reps in Isolates	8	0.0009	1.50
Cultivars	4	0.0444	74.00**
(Ke vs Se)[a]	1	0.0001	0.17
(Ka vs GM)[b]	1	0.0005	0.83
(Ke/Se vs Ka/GM)[c]	1	0.0324	54.00**
(RR vs others)[d]	1	0.1448	241.33**
Cultivars × Isolates	12	0.0021	3.50*
(Ke vs Se) × Isolates	3	0.0008	1.33
(Ke vs Se) × (I vs III)[e]	1	0.0001	0.17
(Ke vs Se) × (II vs IV)[f]	1	0.0024	4.00
(Ke vs Se) × (I/III vs II/IV)[g]	1	0.0000	0.00
(Ka vs GM) × Isolates	3	0.0001	0.17
(Ke/Se vs Ka/GM) × Isolates	3	0.0003	0.50
(RR vs others) × Isolates	3	0.0071	11.83**
Reps × Cultivars in Isolates (error)	12	0.0006	

[a] Contrast between cultivars Kennebec and Sebago.
[b] Contrast between cultivars Katahdin and Green Mountain.
[c] Contrast between Kennebec/Sebago combination and Katahdin/Green Mountain combination.
[d] Contrast between Russet Rural and all other cultivars.
[e] Contrast between *P. infestans* Isolates I and III.
[f] Contrast between *P. infestans* Isolates II and IV.
[g] Contrast between isolate combinations I/III and II/IV: *significant F-test, $P \leq 0.05$; **highly significant F-test, $P \leq 0.01$.

DISCUSSION

The presence of cultivar × isolate interactions has important implications that must be considered in any proposed use of rate-reducing resistance. Based on the results of the present research, we believe it is not possible to discuss the expected level of rate-reducing capacity of a host genotype without referring to the pathogen genotype used to make the test. Conversely, reference to relative parasitic fitness of the isolates should be made only with regard to the particular host genotype on which the comparison was made.

The expression of the interaction has practical significance in breeding and screening for potato genotypes for the rate-reducing capacity. Exposure of host genotypes to only one pathogen isolate may lead to the selection of a genotype or genotypes which may not express a rate-reducing capacity when challenged by the natural field population of *P. infestans*. The procedure to identify this attribute must, therefore, involve a number of different pathogen isolates. The choice and the number of isolates to be used will likely depend upon the size of the experimental facility for screening the host genotypes and/or the number of available isolates.

The implications of the interaction go beyond the immediate problem of screening for the rate-reducing phenomenon. Widespread use of cultivars with rate-reducing capacity is likely to cause natural selection in the pathogen population for increased fitness. This selection for pathogen genotypes less affected by the resistance will thereby limit its expected stability. The response to selection may not be nearly as dramataic as that for vertical resistance (12,13) but, although increases in parasitic fitness may be subtle, in time the cumulative response could be large enough to greatly decrease a cultivar's rate-reducing capacity. The "erosion" of resistance has already been alluded to by Gallegly (5) and Niederhauser (9). Our results support this "erosion" concept.

LITERATURE CITED

1. BARR, A. J., J. H. GOODNIGHT, J. P. SALL, and J. T. HELWIG. 1976. A User's Guide to SAS. Sparks Press. Raleigh, NC. 329 pp.
2. BLACK, W., C. MASTENBROEK, W. R. MILLS, and L. C. PETERSEN. 1953. A proposal for an international nomenclature of races of *Phytopathora infestans* and of genes controlling immunity in *Solanum demissum* derivatives. Euphytica 2:173-178.
3. COMSTOCK, R. E., and R. H. MOLL. 1963. Genotype-environment interactions. Pages 164–196 in: W. D. Hanson and H. F. Robinson, eds. Statistical Genetics and Plant Breeding. Publication 982, National Academy of Sciences — National Research Council, Washington, DC. 623 pp.
4. FRY, W. E. 1975. Integrated effects of polygenic resistance and a

protective fungicide on development of potato late blight. Phytopathology 65:908-911.
5. GALLEGLY, M. E. 1968. Genetics of pathogenicity of *Phytophthora infestans*. Annu. Rev. Phytopathol. 6:375-396.
6. HORSFALL, J. G., and R. W. BARRATT. 1945. An improved grading system for measuring plant diseases. (Abstr.) Phytopathology 35:655.
7. NELSON, R. R. 1978. Genetics of horizontal resistance. Annu. Rev. Phytopathol. 16:359-378.
8. NELSON, R. R. 1979. The evolution of parasitic fitness. Pages 23–46 in: Horsfall and Cowling, eds. Plant Pathology, An Advanced Treatise. Academic Press, New York. 466 pp.
9. NIEDERHAUSER, J. S. 1962. Evaluation of multigenic 'field resistance' of the potato to *Phytophthora infestans* in 10 years of trials at Toluca, Mexico. (Abstr.) Phytopathology 52:746.
10. RYAN, T. A., B. F. RYAN, and B. L. JOINER. 1975. Student Handbook for the Minitab Statistical Computing System. The Pennsylvania State University, University Park. 200 pp.
11. SNEDECOR, G. W., and W. G. COCHRAN. 1967. Statistical Methods. Iowa State University Press, Ames. 593 pp.
12. VANDERPLANK, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
13. VANDERPLANK, J. E. 1968. Disease Resistance in Plants. Academic Press, New York. 206 pp.