Field Assessment of Fitness of Isolates of *Phytophthora infestans*

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ABSTRACT

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The fitness of isolates of *Phytophthora infestans* was estimated on potato cultivar Norchip in two different field environments. Fitness differences among isolates were evident in an environment marginally favorable for pathogen increase, while no differences were detected in a more favorable environment. Absolute fitness estimates expressed as early-epidemic growth rates based on the logistic model were significantly higher than those based on the exponential model. Fitness estimates based on visual disease assessments were significantly higher than those based on assessments of pathogen population size (expressed as areas or numbers of late blight lesions). Furthermore, differences in estimated fitness of isolates varied with the method of estimation. Our results suggest that fitness estimates based on different models or methods of assessment should not be used interchangeably.

The measurement of fitness in plant pathogen populations has received limited attention by plant pathologists (3,8,14). Accurate estimates of fitness are required for determining the pathogenic potential of exotic and indigenous pathogens, as well as for evaluating the efficacy of various control strategies.

The fitness of an individual is the contribution it makes to the gene pool of the next generation (19, page 28). Fitness has proven to be an elusive parameter to measure because it is a function of both the environment and of the varied phenotypic effects of the genes that condition fitness (7; and12, page 205).

Fitness of different genotypes is expressed as a function of population size (6). Plant pathologists, however, have substituted disease proportion for population size in pathogen growth models (3,8). The relationship between disease proportion and population size is complex and likely to vary over the course of an epidemic. Thus, the suitability of the above substitution is uncertain. Fitness estimates based on rates of increase of disease proportions may differ from those based on population growth rates.

In addition, the intrinsic rate of population increase (m) often used to estimate fitness of asexually reproducing microorganisms (13,15, and 19, page 54) is based on a model of exponential population growth. The apparent infection rate (22) based on the logistic model, however, is more familiar to plant pathologists, and MacKenzie (14) has advocated its use as a fitness estimate. The two estimates should be compared to determine whether plant pathologists might use apparent infection rates calculated from disease intensity data to predict isolate fitness based on intrinsic rates of population increase.

Our objective was to develop and compare methods of estimating fitness of isolates of an asexually reproducing fungal plant pathogen, *Phytophthora infestans*. We wished to compare fitness estimates calculated from different models (logistic and exponential) and from pathogen population and disease assessments. We also wished to determine how fitness estimates varied with environment. To accomplish these objectives, we determined the relationship between disease proportion and population size for *P. infestans*.

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MATERIALS AND METHODS

Sources of *Phytophthora infestans*. Six isolates of *P. infestans* were used in these studies. Isolate 101 (ATCC 48824) was originally obtained from R. J. Young at West Virginia University. Isolates 102 (ATCC 48716), 106 (ATCC 48719), 111 (ATCC 48720), 127 (ATCC 48723), and 128 (ATCC 48724) were isolated from diseased potatoes in central New York State. All isolates were race 0 except isolates 101 and 127 which are, respectively, race 1, 3, 4, 5, 7 and race 2, 3, 10. Differentials used to assess race were plants containing the following resistance genes: R0, R1, R2, R3, R4, R5, R7, and R10.

Field experiments. Certified seed tubers of potato cultivar Norchip were planted at two locations (designated A and B). Location A was at the Homer C. Thompson Research Farm at Freeville, NY. Location B was 9 km east of Ithaca, NY (about 12 km from location A). Plots four rows wide (0.9 m between rows) and 3.7 m long were planted at about 23-cm spacing in the row on 23 May 1983 (location A) and 3 June 1983 (location B). There were 4.6 m of fallow ground between plots. Herbicide, fertilizer, and insecticide were applied as described previously (9).

Cultures of *P. infestans* were grown on V8-lima bean agar medium (17) in 9-cm-diameter petri dishes for 10–14 days at 18 C in darkness. Sporangial suspensions were prepared by using a cotton swab and washing cultures with two 10-ml washes of sterile distilled water. The suspensions were filtered through four layers of cheesecloth to remove mycelial fragments, and were adjusted to 1.25×10^4 sporangia per milliliter by using a hemacytometer.

Potato plots were inoculated by placing about 500 sporangia in a 40 μl drop of water on each of the four subterminal leaflets of three leaves in the center of each plot on 27, 28, and 29 July 1983 (location A) and 8, 9, and 10 August 1983 (location B). This procedure established a single-isolate population of *P. infestans* in each plot. Conditions conducive to infection by *P. infestans* were created at both locations by inoculating in the evening (1800–2100 hours) and by using sprinkler irrigation just before and after inoculation to maintain high relative humidity in the plots. Subsequent sprinkler irrigation was applied as necessary to facilitate rapid epidemic progress only at location A, thus creating diverse environments at the two locations.

At location A, the experiment was replicated five times in a split-plot arrangement of a randomized complete block design in which the two methods of assessing disease and/or populations were main plots and the six isolates were subplots. The split-plot arrangement was used to determine whether entering plots to assess

population size of *P. infestans* affected the amount of disease relative to plots in which only whole-plot visual disease assessments were taken.

For level one of the main plot factor, visual assessment of disease in whole plots was the only technique employed, resulting in minimal disturbance of the plots. The proportion of diseased tissue was estimated every 3–6 days from the date of inoculation until the death of all foliage; a modification of a blight assessment key published by the British Mycological Society was used (9). The proportion of diseased tissue was not corrected for plant growth during the assessment period because the leaf area was no longer increasing dramatically by the time of inoculation.

For level two of the main plot factor, visual disease assessments in whole plots as described above were performed; in addition, these plots were used to assess population growth of *P. infestans*. Flags were placed in the center of each plot and at 70-cm diagonals from the center. A wooden device was constructed which circumscribed an area of 0.25 m^2. This device was placed over the flags and the pathogen population size was estimated by assessing the total area of undried lesion tissue on all leaves in each sampling quadrat. We reasoned that the area of hydrated lesion tissue should be proportional to the number of individuals in that area. Sporulating, expanding lesions (excluding dead and/or dried tissue on which no sporulation was visible) were compared with circular diagrams having radii of 0.5, 1.0, 1.5, and 2.0 cm and assigned to a size class. The total number of lesions assigned to each size class was recorded for each quadrat. Areas of individual lesions were summed to yield a population estimate for each quadrat.

To obtain overall population estimates for the center 2.25 m^2 of each plot (at the center and corners of which the quadrat assessments had been made), the total population estimate summed over all four outer quadrats was doubled (to compensate for the four outer quadrats not sampled), and added to that of the central quadrat. Fig. 1 presents a diagram showing the dimensions of the whole plots and sampling quadrats for level two of the main plot factor at location A.

Visual assessments were also made on the sampling quadrats in each plot at the same time that population size was assessed. To obtain overall estimates of the proportion of diseased tissue for the central 2.25 m^2 area of each plot, the combined assessments for all four outer quadrats was doubled (to account for the four unassessed quadrats) and added to that of the central quadrat, and this sum was divided by nine.

At location B, we used a randomized complete block design with five replications. The six isolates of *P. infestans* were the treatments. Disease and population assessments were obtained in a manner identical to that used for level two of the main plot factor at location A.

Estimation of host leaf area. Leaves comprising five different size classes were collected from the plots. Leaves were traced and their areas were determined with a planimeter. From these measurements, a series of five known-area leaf size class diagrams were constructed.

The wooden device which circumscribed an area of 0.25 m^2 was randomly placed within plots at both locations just before the onset of disease. The frequency distribution of each leaf area size class was recorded for 20 randomly selected quadrats at each location. Leaf areas were summed to yield an estimate of host leaf tissue for each 0.25 m^2 quadrat. The mean value of host leaf area for the 20 quadrats at each location was multiplied by nine to provide an estimate of the total host leaf tissue in the central 2.25-m^2 area of the plots.

Calculation of pathogen growth rates, areas under disease progress curves, and areas under population growth curves. Growth rates based on the exponential and logistic models were calculated by using proportional disease and population data. Population assessments based on lesion areas and lesion numbers were converted to proportions of total host leaf tissue in the sampled area. The sum of the areas of sporulating lesions in each sampled area was divided by the total area of host tissue in the sampled area. The total area of host tissue in the sampled area was divided by the area of a circular lesion of 1-cm radius (the most frequent lesion size) to yield total host tissue expressed in terms of potential lesion numbers. Actual lesion number assessments were divided by this estimated total lesion number to yield proportions of leaf tissue occupied by lesions.

Logistic and exponential growth rates were expressed as the regression coefficient (slope) obtained by regressing the transformed value of ln $[X/(1-X)]$ (logistic model) or ln $[X]$ (exponential model) on time, in which X represents the appropriate proportional disease or population assessment. For cases in which X represents a proportional disease assessment, we consider the logistic growth rate to be equivalent to the apparent infection rate (r) (22). A value of 0.0001 was substituted for zeros so that log-transformed values could be obtained.

The area under the disease progress curve ($AUDPC$) was calculated according to the formula described by Tooley and Grau (21) except that we expressed disease as a percentage rather than as a proportion. The area under the population growth curve ($AUPGC$) was calculated by using the same formula, except that lesion areas or lesion numbers were substituted for percent disease in the formula.

Statistical comparison of growth rates. Analyses of variance were performed on the differences between growth rates based on different models or assessment types at each location. A significant F value for the overall mean (1) indicated a difference between the two types of growth rates being compared, while a significant F value for isolate was analogous to a significant interaction between isolate and the factor for which the rates were being compared (i.e., model or assessment type).

RESULTS

The epidemics produced at locations A and B differed in intensity. At location A, the use of irrigation permitted plants to grow vigorously all season, resulting in a closed canopy by late July when inoculations were made. The epidemics at location A progressed rapidly up to about 65% disease (based on whole-plot disease assessments 26 days postinoculation), and then gradually approached 100% disease.

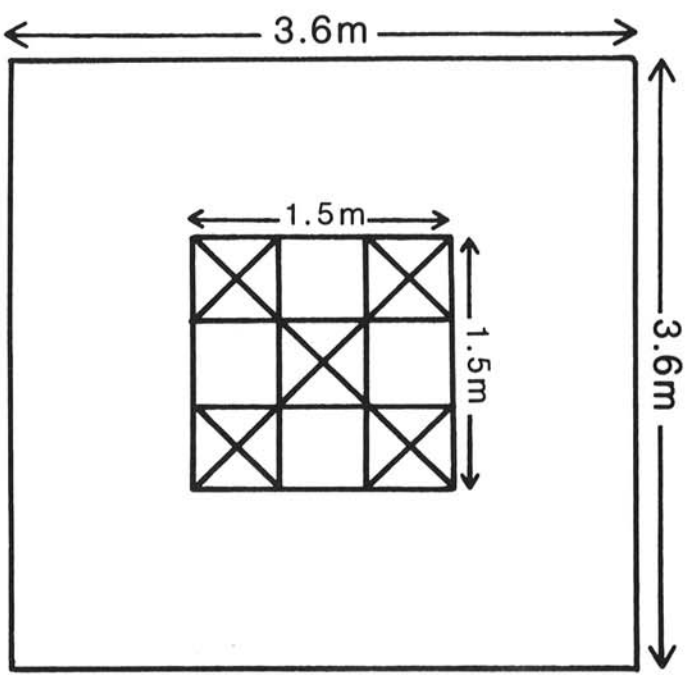

Fig. 1. Diagram of an individual potato plot showing dimensions (to scale) of whole 12.96 m^2 plot area on which visual disease assessments were made, and central 2.25 m^2 area in which disease assessments and population assessments of *Phytophthora infestans* were made. The squares containing crossed diagonals represent the 0.25 m^2 quadrats in which disease and population assessments were made, while the empty squares represent unsampled quadrats.

At location B, there was little rainfall all season, and plants were irrigated only once prior to the time of inoculation. The canopy was less dense at location B than at location A and disease progressed more slowly at B than at A. When about 20% disease had been reached at location B (based on whole-plot assessments), the severity of early blight caused by *Alternaria solani* increased to high levels, precluding further late blight assessments.

Isolate comparisons based on whole-plot disease assessments. When late blight was assessed visually for the whole plots, significant differences ($P < 0.05$) among isolates were observed at both locations. At location A (Table 1), differences in percent disease became apparent at 12 days postinoculation and remained so until 36 days postinoculation. Isolate differences were also found for measurements of areas under the disease progress curve (*AUDPC*) and apparent infection rates (r) (Table 1). The method of assessment (population plus disease assessments versus disease assessments only) used as the main plot factor in the split-plot design had no significant effect on percent disease, *AUDPC*, or r values at any dates.

At location B (Table 2), differences among isolates in percent disease were apparent at 11, 14, 16, and 19 days postinoculation. *AUDPC* and r values calculated on the basis of the restricted epidemic also revealed isolate differences (Table 2).

Isolate comparisons based on sampling quadrat disease and population assessments. Fig. 2 presents the disease progress and population growth curves for each isolate of *P. infestans* at location A. Isolate differences ($P < 0.05$) were found for percent disease at 16, 19, and 22 days postinoculation and for population size at 19

TABLE 1. Percentage late blight, area under the disease progress curve (*AUDPC*), and apparent infection rate (r) for six isolates of *Phytophthora infestans* at location A

Isolate	Disease[a] (%) at postinoculation day: 8	12	15	18	21	26	32	36	46	*AUDPC*[b]	r[b]
101	0	2	4	23	50	62	72	86	98	441	0.3118
102	0	4	8	36	66	72	76	87	98	588	0.3372
106	0	3	7	33	58	66	72	83	98	526	0.3181
111	0	3	7	28	57	65	66	80	97	502	0.3150
127	0	3	6	24	50	59	66	83	97	449	0.3025
128	0	2	5	26	55	64	71	86	98	480	0.3186
BLSD[c]	ns[d]	1	2	5	4	5	5	5	ns[d]	41	0.0128

[a] Based on whole-plot visual disease assessments.
[b] *AUDPC* and r were calculated from data collected from 8 to 26 days postinoculation.
[c] Bayes least significant difference, $k = 100$ (20).
[d] Analysis of variance F value not significant, $P = 0.05$.

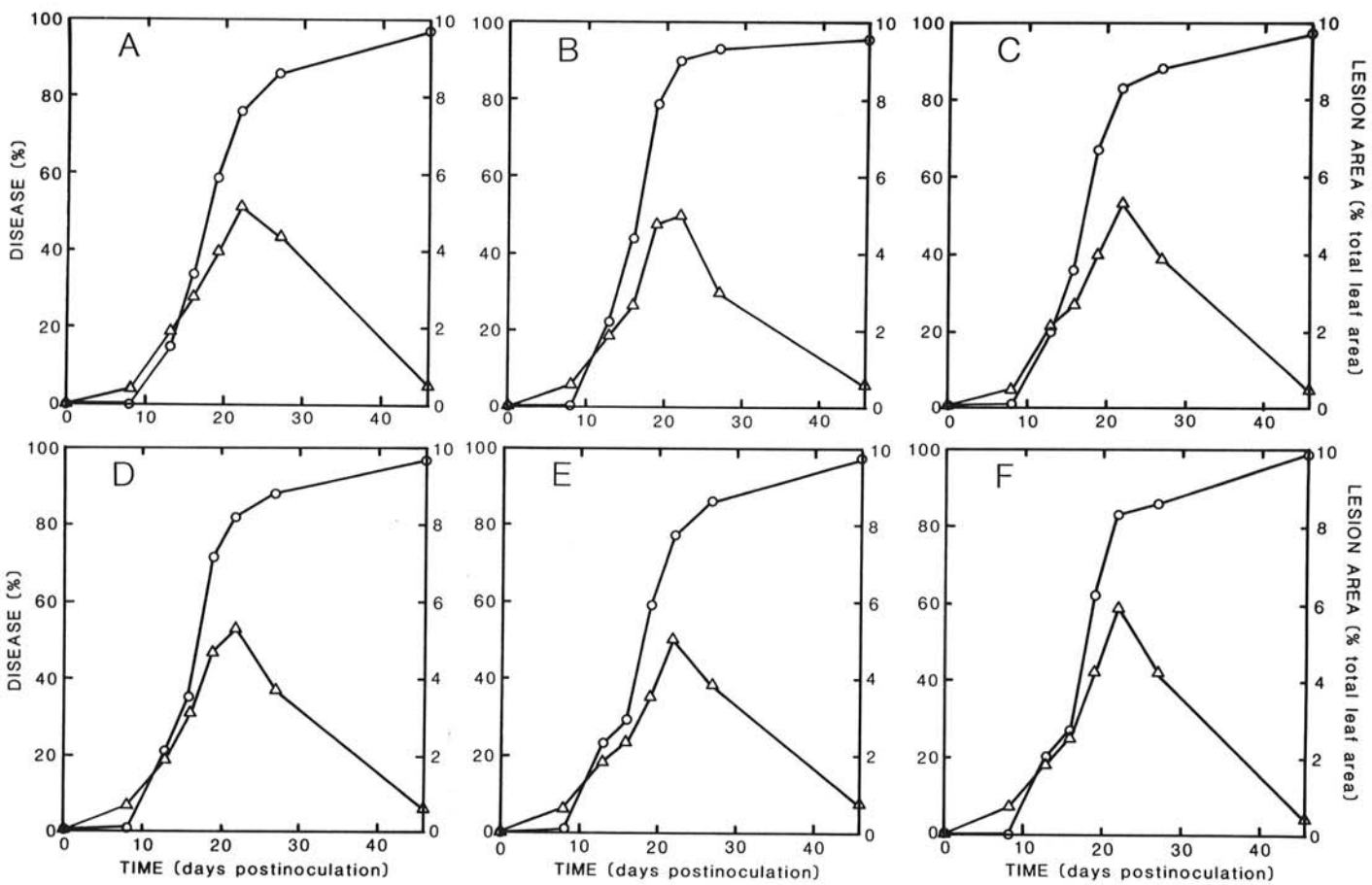

Fig. 2. Disease progress curves and population growth curves for six isolates of *Phytophthora infestans* based on sampling quadrat assessments at location A. **A,** Isolate 101; **B,** isolate 102; **C,** isolate 106; **D,** isolate 111; **E,** isolate 127; and **F,** isolate 128. Circles represent disease progress curves; triangles represent population growth curves.

and 27 days postinoculation. Peak population size occurred for all isolates at 22 days postinoculation.

After reaching their peak, populations of isolate 101 (the least aggressive in terms of whole-plot *AUDPC*) declined at a slower rate than did those of isolate 102 (the most aggressive isolate) (Fig. 2).

Fig. 3 presents the disease progress and population growth curves for each isolate of *P. infestans* at location B. Isolate differences ($P < 0.01$) in percent disease were found at 8, 13, 16, and 19 days postinoculation and in population size at 8, 13, and 16 days postinoculation. Population peaks were reached at 16 days postinoculation, probably due more to the marginal environment than to host tissue limitation.

Lesion numbers were compared to lesion areas as estimates of population size. Fig. 4 shows plots of mean lesion numbers and areas versus time for the six isolates at location A. In contrast to the other isolates, populations of isolates 102 and 111 expressed in terms of lesion numbers peaked earlier than those expressed in terms of lesion areas. At 16 days postinoculation, a highly significant isolate effect ($P < 0.01$) was detected for lesion numbers, but not lesion areas. This appeared largely due to isolate 127 which was distinctly lower in lesion numbers than other isolates at this date. Also, at 27 days postinoculation, larger differences among isolates were found for lesion numbers than for lesion areas. This was largely due to isolate 101 (the least aggressive isolate) which retained more lesions than other isolates at this date.

Estimation and comparison of isolate fitnesses. We estimated the absolute fitness (10) of the six isolates of *P. infestans* at locations A and B as rates of exponential and logistic growth based on proportional disease and population assessment data collected from 0 to 13 and from 0 to 16 days postinoculation. Because results based on data collected over these two time intervals were largely similar, we only present those based on data collected from 0 to 13 days postinoculation (Table 3). At location A, no significant differences ($P < 0.05$) among isolates were found. At location B, isolate differences were found for all types of growth rates calculated (Table 3).

Regardless of the type of assessment on which growth rate calculations were based, those calculated from the logistic model were significantly higher ($P < 0.01$) than those based on the exponential model at both locations. Also, regardless of the model on which growth rate calculations were based, rates calculated from disease assessment data were significantly higher ($P < 0.01$) than those calculated from population assessment data at both locations. Rates based on lesion numbers were significantly higher ($P < 0.01$) than those based on lesion areas.

When we analyzed differences in fitness estimates calculated from the two models or from the two types of assessments at location B, we found a significant ($P < 0.05$) effect due to isolate.

TABLE 2. Percentage late blight, area under the disease progress curve (*AUDPC*), and apparent infection rate (*r*) for six isolates of *Phytophthora infestans* at location B

Isolate	Disease[a] (%) at postinoculation day: 11	14	16	19	*AUDPC*[b]	*r*
101	0	1	3	6	20.2	0.5076
102	0	3	9	13	48.8	0.7616
106	1	3	7	14	47.6	0.4382
111	0	2	5	7	29.3	0.6487
127	0	2	9	13	47.3	0.9141
128	0	1	3	5	16.2	0.7625
BLSD[b]	0.4	1	4	4	15.6	0.3815

[a] Based on whole-plot visual disease assessments.
[b] Bayes least significant difference, $k = 100$ (20).

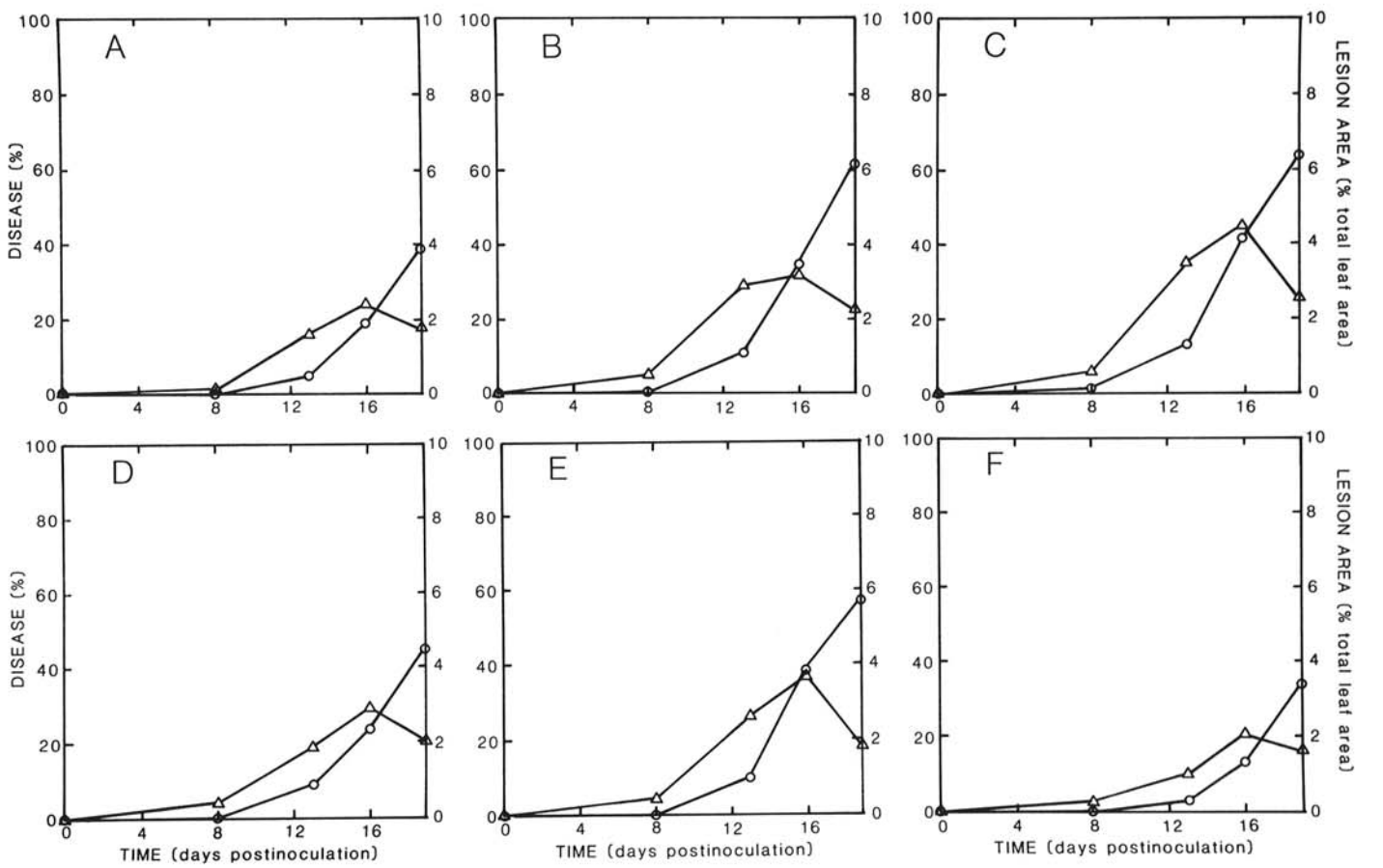

Fig. 3. Disease progress curves and population growth curves for six isolates of *Phytophthora infestans* based on sampling quadrat assessments at location B. **A,** Isolate 101; **B,** isolate 102; **C,** isolate 106; **D,** isolate 111; **E,** isolate 127; and **F,** isolate 128. Circles represent disease progress curves; triangles represent population growth curves.

We found the same result when we compared intrinsic growth rates and apparent infection rates at location B.

Separate analyses of variance for each type of growth rate fitness estimate were performed using location as a factor. Significant ($P < 0.05$) or highly significant ($P < 0.01$) location-isolate interactions were noted for all types of growth rate fitness estimates.

Additional fitness measures reflecting the rates of increase of the six isolates were calculated from lesion area population assessment data. These included the population size attained by the isolates at

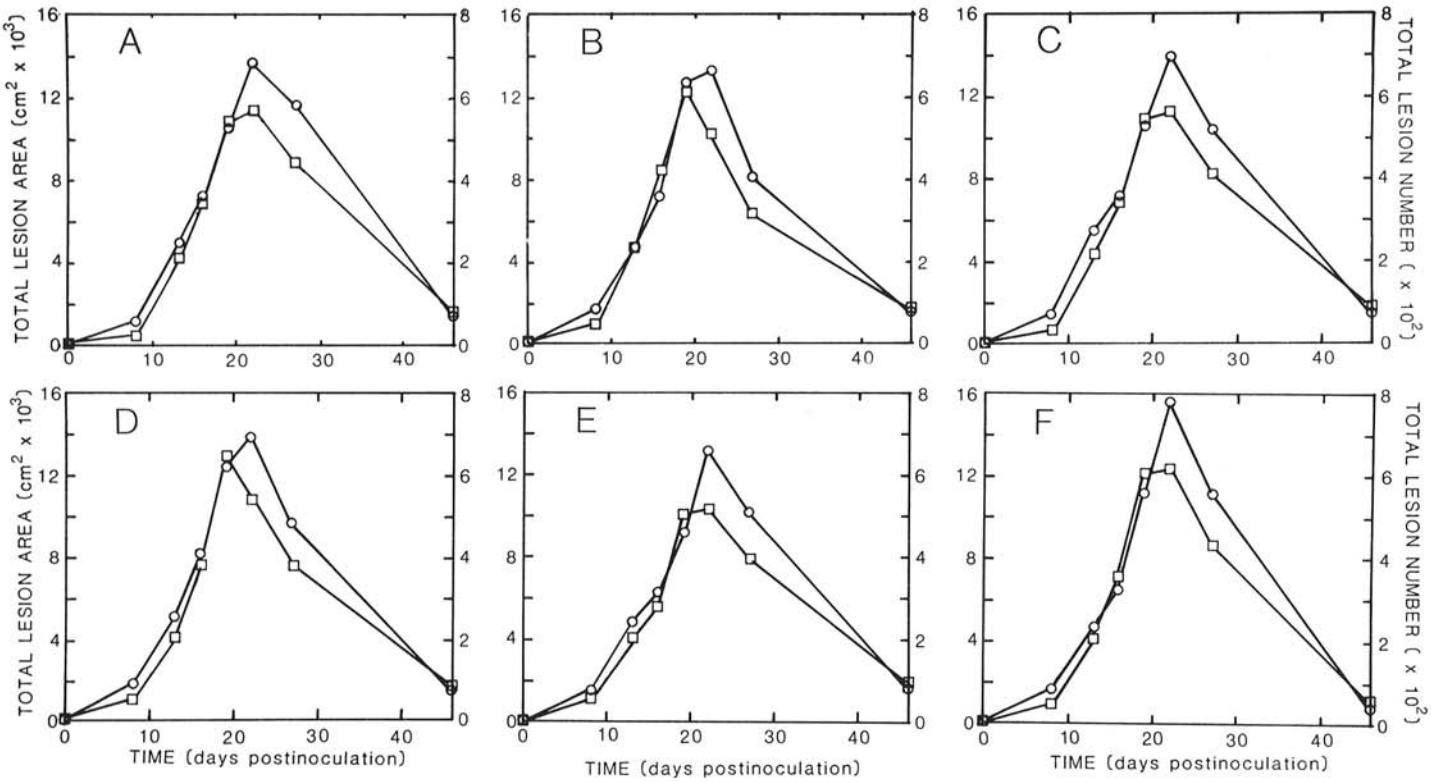

Fig. 4. Comparison of population growth curves based on lesion area (circles) and lesion number (squares) population assessments for six isolates of *Phytophthora infestans* at location A. **A,** Isolate 101; **B,** isolate 102; **C,** isolate 106; **D,** isolate 111; **E,** isolate 127; and **F,** isolate 128.

TABLE 3. Logistic (*r13*) and exponential (*m13*) growth rates calculated from population and disease assessment data collected up to 13 days postinoculation at locations A and B for six isolates of *Phytophthora infestans*[v]

Location A		Location B		Location A		Location B	
Isolate	*r13*	Isolate	*r13*	Isolate	*m13*	Isolate	*m13*
Lesion area[w]:							
106	0.4157 a[z]	106	0.4566 a	106	0.4142 a	106	0.4541 a
111	0.4138 a	102	0.4392 a	111	0.4123 a	102	0.4371 ab
128	0.4084 a	127	0.4301 ab	128	0.4070 a	127	0.4282 ab
127	0.4077 a	111	0.4009 bc	127	0.4064 a	111	0.3995 bc
101	0.4071 a	101	0.3564 cd	101	0.4059 a	101	0.3552 cd
102	0.4065 a	128	0.3512 d	102	0.4051 a	128	0.3505 d
Lesion number[x]:							
102	0.4366 a[z]	106	0.4800 a	102	0.4346 a	106	0.4765 a
111	0.4307 a	102	0.4752 a	111	0.4289 a	102	0.4719 a
128	0.4256 a	127	0.4687 a	128	0.4239 a	127	0.4656 a
127	0.4253 a	111	0.4491 ab	127	0.4236 a	111	0.4466 ab
106	0.4250 a	128	0.4103 bc	106	0.4231 a	128	0.4087 bc
101	0.4206 a	101	0.4026 c	101	0.4188 a	101	0.4007 c
Disease[y]:							
127	0.5994 a[z]	106	0.5468 a	127	0.5806 a	106	0.5373 a
102	0.5982 a	127	0.5286 a	102	0.5801 a	127	0.5210 a
111	0.5940 a	102	0.5276 a	111	0.5770 a	102	0.5191 a
128	0.5772 a	111	0.4938 a	128	0.5612 a	111	0.4873 ab
106	0.5724 a	128	0.4287 bc	106	0.5558 a	128	0.4264 bc
101	0.5492 a	101	0.4031 c	101	0.5374 a	101	0.3995 c

[v] Growth rates were calculated from data collected from the central 2.25 m^2 area of each plot. Data are means of five replications and are presented in descending order of magnitude.
[w] Growth rates calculated from lesion area population assessment data.
[x] Growth rates calculated from lesion number population assessment data.
[y] Growth rates calculated from visual disease assessment data.
[z] Means within a column followed by the same letter do not differ significantly according to the Bayes least significant difference procedure, $k = 100$ (20).

13 days postinoculation (*P13*), and the area under the population growth curve up to 13 days postinoculation (*AUPGC13*) (Table 4). Significant differences among isolates in *P13* and *AUPGC13* were found at location B, but not location A. When analyzed over locations, a significant ($P < 0.05$) location-isolate interaction was observed for *P13* and a highly significant ($P < 0.01$) location-isolate interaction was observed for *AUPGC13*.

Comparison of fitness and disease measures. The fitness measures chosen for these comparisons were the exponential growth rate based on lesion area population assessments collected up to 13 days postinoculation (designated *m13*, Table 3), the area under the population growth curve from 0 to 13 days postinoculation (*AUPGC13*, Table 4), and the population size at 13 days postinoculation (*P13*, Table 4). The disease measures chosen included the logistic growth rate (apparent infection rate) based on proportional disease assessments collected up to 13 days postinoculation (*r13*, locations A and B, Table 3) and up to 46 days postinoculation (*r46*, location A only, Table 5), the area under the disease progress curve calculated from data collected up to 13 days postinoculation (*AUDPC13*, locations A and B, Table 5) and up to 46 days postinoculation (*AUDPC46*, location A only, Table 5) and percent disease at 13 days postinoculation (*D13*, locations A and B, Table 5).

At location A, most correlations within and among fitness and disease measures were nonsignificant ($P < 0.05$) except that *m13* and *P13* were significantly correlated, as were *AUDPC13* and *D13*. At location B, correlations within and among fitness and disease measures were all highly significant ($P < 0.01$).

Finally, we investigated whether fitness and disease measures based on sampling quadrat data were correlated with disease measures based on data collected from whole plots. At location A, the three fitness measures *m13*, *AUPGC13*, and *P13* were not significantly correlated with any whole-plot disease measures.

At location B, the three fitness measures showed significant ($P < 0.05$) or highly significant ($P < 0.01$) correlation with whole-plot percent disease assessed at 14, 16, and 19 days postinoculation and with whole-plot *AUDPC*. The fitness measures were not correlated with whole-plot apparent infection rate.

DISCUSSION

Our results reaffirm the important role played by the environment in conditioning the fitness of individual genotypes (7,12). No fitness differences among isolates were found at location A, whereas marked differences existed at location B. In addition, fitness measures were highly correlated with disease measures at location B, indicating that the most fit isolates caused the most disease. The lack of significant correlations at location A was not surprising since no significant differences among isolates were observed at location A for any fitness and disease measures except *AUDPC46*.

We found that different methods of estimating fitness gave different results. Fitness estimates calculated from disease assessment data were significantly higher than those calculated from measures of population size. The logistic model yielded estimates significantly higher than those of the exponential model.

More importantly, isolate differences in fitness varied depending on the method of estimation. Isolate rankings did not vary with different estimation methods; however, the magnitude of isolate differences did vary. Since isolate comparisons will vary with the method used to estimate fitness, estimates such as apparent infection rates based on the logistic model should not be used interchangeably with those such as intrinsic growth rates based on the exponential model.

We found parameters other than growth rates to be desirable for use as fitness estimates. Population size, unlike growth rates, can be obtained by a single assessment and has been used as a fitness measure by other workers (2,4,23). The area under the population growth curve (*AUPGC13*) allowed the detection of more isolate differences and was more highly correlated with most disease measures than was the exponential population growth rate (*m13*). At location B, where significant fitness differences among isolates existed, the fitness estimates were all highly correlated and yielded identical isolate rankings. Such agreement among the variables will benefit workers who wish to use a single variable to estimate fitness in future studies.

TABLE 4. Area under the population growth curve (*AUPGC13*) and population size (*P13*) obtained from lesion area population assessment data collected up to 13 days postinoculation at locations A and B for six isolates of *Phytophthora infestans*[y]

Location A		Location B		Location A		Location B	
Isolate	*AUPGC13*	Isolate	*AUPGC13*	Isolate	*P13* (cm^2)	Isolate	*P13* (cm^2)
111	2,546 a[z]	106	2,489 a	106	556 a	106	682 a
102	2,329 a	102	2,061 ab	111	515 a	102	553 ab
128	2,311 a	127	1,764 bc	101	495 a	127	513 ab
106	2,292 a	111	1,453 cd	102	493 a	111	369 bc
127	2,226 a	101	1,009 de	127	488 a	101	314 c
101	1,944 a	128	819 e	128	476 a	128	194 c

[y] Calculations were based on data collected from the central 2.25 m^2 area of each plot. Data are means of five replications and are presented in descending order of magnitude.
[z] Means within a column followed by the same letter do not differ significantly according to the Bayes least significant difference procedure, $k = 100$ (20).

TABLE 5. Disease measures[u] for potato late blight caused by six isolates of *Phytophthora infestans* at two locations

	Location A				Location B	
Isolate	*AUDPC13*[v]	*D13*[w]	*AUDPC46*[x]	*r46*[y]	*AUDPC13*[v]	*D13*[w]
101	41 a[z]	15 a	2,584 c	0.2895 a	13 bc	5 bc
102	60 a	22 a	2,850 a	0.2731 a	30 a	11 a
106	54 a	20 a	2,698 bc	0.2910 a	35 a	13 a
111	57 a	21 a	2,713 b	0.2654 a	24 ab	9 ab
127	61 a	23 a	2,615 bc	0.2704 a	28 a	10 a
128	54 a	20 a	2,649 bc	0.3008 a	10 c	3 c

[u] Based on visual disease assessments made on the central 2.25 m^2 area of each plot. Data are means of five replications.
[v] The area under the disease progress curve calculated from 0 to 13 days postinoculation.
[w] Percent disease at 13 days postinoculation.
[x] The area under the disease progress curve calculated over the entire epidemic (0 to 46 days postinoculation).
[y] Logistic growth rate calculated from proportional disease assessments obtained over the entire epidemic (0 to 46 days postinoculation).
[z] Means within a column followed by the same letter do not differ significantly according to the Bayes least significant difference procedure, $k = 100$ (20).

We found at location B that *AUDPC* values and disease assessments made on whole plots were highly correlated with fitness measures based on the sampled areas. Thus, isolate fitnesses may be estimated from certain whole-plot disease assessments in future studies.

Although our population estimates may not be directly comparable to those of other workers who have quantified fitness in microbial populations (5,16) (because filamentous fungi such as *Phytophthora* do not exist solely as unicellular units), we feel they represented valid measures of population size. Of the two types of population assessments which we used, the lesion area assessments were probably more accurate because the area of hydrated lesion tissue should be proportional to the number of individuals in that area. Rotem et al (18) also felt that assessments of lesion areas accurately reflected patterns of multiplication and inoculum potential of *P. infestans*. Our observation that populations of some isolates reached their maximum earlier in terms of lesion numbers than lesion areas can be explained by lesion coalescence.

The fitness estimates we made were noncompetitive (3,11), and as such are different from estimates used in studies on competing genotypes. Methodology to assess competitive fitness of *P. infestans* is not fully developed. However, once competitive estimates are available, relative fitness (7; 12, page 121) can be calculated for *P. infestans*.

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