Potential for *Phytophthora infestans* Populations to Adapt to Potato Cultivars with Rate-Reducing Resistance

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ABSTRACT

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We quantified the rate and extent of adaptation of four populations of *Phytophthora infestans* to selected potato cultivars that differed in levels of rate-reducing resistance. The initial populations had different degrees of heterogeneity: single isolates, mixtures of isolates, and mutagenized isolates. Infection efficiency, sporulation, and rate of epidemic development in field plots were used as criteria to measure adaptation. Two subpopulations were derived from each initial population as a result of repeated asexual generations on either of two cultivars. Two pairs of subpopulations were developed in the greenhouse and two in the field. Each subpopulation was tested for infection efficiency and sporulation on its "own" cultivar (the one on which it had been cultured repeatedly) and on the "other" cultivar (on which it had not grown previously). Subpopulations differed in infection efficiency, but adaptation was not indicated because the changes were not differential for the "own" vs. the "other" cultivar. In the field, disease progressed as rapidly in plots composed of two cultivars planted alternately as it did in plots of either cultivar alone. We interpret the results of these studies and previous ones, as well as the constancy of the relative resistance of cultivars over time (disregarding R genes) to indicate that rapid adaptation of *P. infestans* populations to cultivars with rate-reducing resistance is unlikely.

Late blight, induced by *Phytophthora infestans* (Mont.) de Bary, is one of the most important diseases of potato (*Solanum tuberosum* L.) worldwide (15). As individual resistance genes (R genes) were discovered and introduced into commercial potato cultivars, new races of *P. infestans*, able to overcome the genes, appeared quickly (5,15). Due to the ability of *P. infestans* populations to overcome host genes for differential resistance, such genes have contributed little to controlling late blight. Plant breeders have therefore concentrated on developing cultivars with rate-reducing resistance (14,16). This resistance may be expressed as fewer infections from a given number of sporangia, decreased sporulation, or reduction of lesion size (6). This type of resistance, which slows but does not stop epidemic development, is generally considered nonrace-specific and has been reported to remain stable over time (16).

Some researchers have suggested that with time a pathogen population may increase in ability to cause disease on a particular cultivar with rate-reducing resistance. This increased capacity of a particular pathogen population to cause disease on a given cultivar has been termed adaptation (2). In this context, adaptation refers to a process of gradual increase of aggressiveness in the fungus in response to growth on a specific cultivar, not the change (probably a single step mutation) that permits the pathogen to overcome a single major gene for resistance. Adaptation as defined here has been suggested to be due either to selection of preexisting variants best able to grow on a specific cultivar or to mutations or nonheritable changes triggered by growth conditions on a specific cultivar (7). Parlevliet (10), working with barley (*Hordeum vulgare* L.) and leaf rust incited by *Puccinia hordei* Otth., found a differential interaction among fungal isolates and host varieties with partial resistance. One isolate appeared to have adapted to a specific cultivar, resulting in greater disease severity than the other combinations of isolate and cultivar observed. This isolate-host combination showed a shorter latent period, higher infection frequency, and higher spore production than would have been expected if all cultivars had ranked all isolates in the same order (10). Work with *Puccinia striiformis* West. and wheat, *Triticum aestivum* L., also showed differential interactions between two wheat cultivars and two pathogen isolates, apparently of the same race, indicating that the pathogen isolates were adapted to their particular cultivars (8).

Adaptation of *P. infestans* to potato cultivars was suggested in two studies where radial growth of the fungus on tuber slices was used as the test criterion and also by a field study in which effect of different isolates and cultivars on epidemic development was observed. Jeffrey, Jinks, and Grindle (7) found that *P. infestans* isolates, newly isolated from the field, grew better on tubers of the cultivar from which they had been isolated than on other cultivars. Caten (2) also found that isolates of a given race of *P. infestans* grew faster on tuber slices of the cultivar on which they had grown in the field than on other cultivars. Isolate × cultivar interactions observed in late blight epidemics in the field plots were interpreted to mean that the effective level of rate-reducing resistance of host genotypes differed for different isolates of the pathogen (9). If isolates of *P. infestans* adapt significantly to potato cultivars, the long-term use of these cultivars would be endangered.

Previous studies have not assessed the potential of *P. infestans* populations to adapt to particular potato cultivars within a single growing season. Radial growth of the fungus on tuber pieces (2,7) is not indicative of fungal growth in the field, and there is little correlation between polygenic resistance in the foliage and resistance in tubers (5). Isolate × cultivar interactions were reported in field tests (9). However, because these tests involved random pairing of isolates and cultivars that did not evolve in long-term association with each other, observations of isolate × cultivar interactions were not evidence that *P. infestans* populations adapted in response to growth on specific cultivars (9). Whether an isolate of *P. infestans* could adapt enough during a growing season to leaf and stem tissue of a potato cultivar to render host resistance ineffective remains unanswered.

If significant adaptation of *P. infestans* populations to potato cultivars occurs, a population grown repeatedly on foliage of a given cultivar should gain in ability to induce disease on that cultivar, compared with other populations. We sought to quantify the rate and extent of *P. infestans* adaptation to foliage of selected cultivars by measuring infection efficiency and sporulation in the greenhouse and by measuring the rate of epidemic development in small field plots.

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MATERIALS AND METHODS

Creation of subpopulations of *P. infestans* on different cultivars. For each experiment to measure adaptation, two subpopulations originating from an initial population of *P. infestans* were needed. The initial population was inoculated to each of two cultivars. Sporangia produced on each cultivar were repeatedly inoculated to that same cultivar, resulting, after several cycles, in subpopulations, potentially adapted to the different cultivars. Subpopulations were derived from a different initial population for each of four experiments (Table 1).

One pair of subpopulations developed in the field originated from an initial population of one isolate of *P. infestans* race 0. One subpopulation, termed "Sebago (field)," was obtained from an isolated plot of cultivar Sebago in which an epidemic had developed after artificial inoculation. The other subpopulation, termed "multiple cultivar (field)," resulted from reisolations from plants of the cultivar Hudson in a field where the initial population had been inoculated to several different cultivars. Dispersal of sporangia among plots of different cultivars during epidemic development minimized the opportunity for the fungus to adapt to one cultivar. After reisolation, the Sebago (field) subpopulation was maintained in the greenhouse on Sebago and the multiple cultivar (field) subpopulation on the susceptible cultivar Norchip.

In addition, a separate sample of sporangia of the same isolate of *P. infestans* race 0 was treated with a chemical mutagen. A zoospore suspension (6×10^4/ml) was shaken for 45 min in a 50 μg/ml solution of *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine (NTG). This treatment resulted in 80% kill of viable zoospores. The zoospores were centrifuged and resuspended in water three times to remove the NTG and then inoculated to an isolated plot of Sebago. Following epidemic development, the fungus was reisolated and maintained in the greenhouse on Sebago plants.

Members of the other pair of subpopulations developed in the field were derived from one isolate of *P. infestans* race 1,3,4. The initial population was inoculated to plots of cultivars Rosa and Kennebec located on different experimental farms, isolated from each other and from other potato fields. At the terminal stage of epidemics in these plots, the fungus was reisolated from each cultivar and maintained in the greenhouse on its respective host cultivar.

A mutagenized subpopulation was developed from race 1,3,4, as described for race 0. Following epidemic development in an isolated plot of cultivar Kennebec, the fungus was reisolated and maintained in the greenhouse on Kennebec plants.

The other two pairs of subpopulations were developed in the greenhouse. One pair was derived from a single isolate of *P. infestans* race 1,2,3,4. One subpopulation of this pair was maintained on Norchip (very susceptible) and the other on Sebago (moderately resistant). Tests of adaptation began after 23 serial passages through the cultivars. Similarly, subpopulations were developed on Rosa and Kennebec, two moderately resistant cultivars, from an initial population consisting of a mixture of race 1,2,4 (one isolate), race 1,3,4 (one isolate), and race 1,2,3,4 (three isolates). Adaptation was tested at intervals, beginning after the second passage and continuing through the 29th passage.

Infection efficiency and sporulation experiments. Potato plants of cultivars Norchip, Sebago, Kennebec, or Rosa were grown from certified seed tuber pieces or from cuttings. Plants were grown in a greenhouse (16–26 C) in clay pots of about 1-L capacity containing a peat and vermiculite mixture (1:1, v/v) with 0.4 kg each of nitrogen, phosphorus, and potassium per cubic meter of mixture. Natural sunlight was augmented with continuous illumination from 40-W cool white fluorescent tubes. Plants 5- to 8-wk-old with at least six fully expanded leaves were used in all experiments.

Infection efficiency was one criterion used to measure adaptation. Infection efficiency was:

$$\frac{\text{number of lesions per leaflet} \times 100}{\text{number of sporangia applied.}}$$

Each isolate was tested on its "own" cultivar, on which it had been cultured repeatedly and on the "other" cultivar, on which it had not grown previously.

Sporangia, washed from sporulating lesions on leaves, in 21 C deionized water, were applied to plants of the appropriate cultivar in a growth chamber at 100% RH. Sporulation had been induced by maintaining infected plants 24–48 hr at 100% RH. Sporangial suspensions were centrifuged and concentration was estimated from the average of three counts on a hemacytometer. Standard deviations on three counts averaged 15% of the mean. Suspensions were adjusted to 5×10^4 sporangia per milliliter (on Norchip and Sebago) or 1×10^5 sporangia per milliliter (on Kennebec and Rosa). A 20-μl droplet was spread on the upper surface of the leaflet with a finger. On each leaf, one subpopulation was placed on leaflets on one side and the other was placed on leaflets directly opposite. Four leaflets on each of the top five expanded leaves on plants of two cultivars were inoculated. Inoculated plants were maintained in a growth chamber at 100% RH for 18 hr (Norchip and Sebago) or for 40 hr (Kennebec and Rosa). Lesions on each leaflet were counted 3–4 days after inoculation.

Sporulation from single lesions was the other criterion used to quantify adaptation. Lesions resulted from inoculations with sporangia ($1–3 \times 10^5$/ml) in a small droplet (2–5 μl) placed on the upper surface of a leaflet. In a given experiment, concentration and volume for each subpopulation were the same. As before, one subpopulation was placed on leaflets on one side of a given leaf, and the other was placed on leaflets directly opposite. Four leaflets on each of the top six expanded leaves on plants of two cultivars were inoculated. Inoculated plants were maintained in a growth chamber at 100% RH for 18 hr (Norchip and Sebago) or for 40 hr (Rosa and Kennebec). Four days after inoculation, plants were placed at 100% RH to induce sporulation. Sporulation of one subpopulation on a leaflet was compared to that of the other subpopulation on the opposite leaflet of the same leaf. After 48 hr, pairs of opposite leaflets with sporulating lesions were chosen randomly and placed into separate vials containing 10% ethanol. Each replication consisted of six leaflets, and there were three to nine replications for each treatment. Sporangia were removed from leaves by shaking vials vigorously for 1 min on a Vortex mixer. Vials were shaken 10 sec to resuspend sporangia immediately before counting. The number of sporangia per lesion was estimated from the average of three counts with a hemacytometer.

Epidemic development. A field experiment was conducted to test whether adaptation could be detected in terms of the rate or extent of epidemic development. Small plots in a completely randomized design were planted with three types of composition: Rosa only, Kennebec only, or Rosa and Kennebec mixed (1:1, in a checkerboard arrangement). Plots were four rows wide (0.9 m between rows) and 3.7 m long with 4.6 m of fallow soil between plots. There were four replicates of each treatment. Certified seed tubers were planted at approximately 23-cm spacing in the row on

TABLE 1. Derivation of subpopulations of *Phytophthora infestans*

Source Population	Repeated culture on	Resulting subpopulations designated
Race 1,2,3,4 (one isolate)	Norchip	Norchip
	Sebago	Sebago
Race 1,2,4, race 1,3,4 (one isolate of each), and race 1,2,3,4 (three isolates)	Rosa	Rosa
	Kennebec	Kennebec
Race 0 (one isolate)	Sebago (field plot)	Sebago (field)
	Multiple cultivars (field plots)	Multiple cultivars (field)
Race 0 (mutagenized)	Sebago (field plot)	Sebago (NTG)[a]
Race 1,3,4, (one isolate)	Rosa (field plot)	Rosa (field)
	Kennebec (field plot)	Kennebec (field)
Race 1,3,4 (mutagenized)	Kennebec (field plot)	Kennebec (NTG)

[a] NTG = *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine.

28 May 1981. Herbicide, fertilizer, and insecticide were applied as previously described (3).

The fungus was established by spraying one potato shoot in the center of each plot on 20, 21, and 22 July with 25 ml of a suspension (500 sporangia per milliliter) of *P. infestans* race 1,3,4 obtained from cultures grown on V-8 and lima bean agar medium (13) 10–14 days at 18 C in darkness. Conditions conducive to development of *P. infestans* were maintained by sprinkler irrigation (0.17 cm of water per hour) from 0730 to 0800 hours daily from 28 July to 31 August.

The proportion of diseased tissue was estimated every 3–6 days from the date of inoculation until the death of vines, using a modification of a blight assessment key published by the British Mycological Society (4). The proportion of diseased tissue was not corrected for plant growth during the season. At the time of inoculation, the total leaf area was not changing dramatically.

RESULTS

If adaptation occurred, higher infection efficiency or sporulation values would be expected for the "own" classes (the two cases where a subpopulation was tested on the same cultivar on which it had been cultured repeatedly) than for the "other" classes (the cases where the fungus was tested on the cultivar on which it had not grown previously). Data from experiments with each of the four pairs of *P. infestans* subpopulations were analyzed to determine if there were significant differences between the pooled "own" class and the pooled "other" class, between subpopulations (disregarding cultivar), and between cultivars. Experiments done after different cycles were treated as replications in the analysis.

Each subpopulation resulting from mutagenesis was compared to the subpopulation derived on the same cultivar from nonmutagenized sporangia of the same source population. In each case the mutagenized population was no more aggressive than the natural population (as measured by infection efficiency) (Table 2). Thus, mutagenesis did not increase the potential for adaptation.

TABLE 2. Infection efficiency[a] for natural and mutagenized subpopulations of *Phytophthora infestans*

Fungal subpopulation	Host cultivar	Mean[b]
Sebago (field)	Sebago	2.9
Sebago (NTG)[c]	Sebago	2.0
Kennebec (field)	Kennebec	0.8
Kennebec (NTG)	Kennebec	0.6

[a] Infection efficiency = (number of lesions per leaflet × 100)/number of sporangia applied.
[b] Values are means of three tests on Sebago and two on Kennebec. Lesions were counted on 15 leaflets in each test.
[c] NTG = *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine.

TABLE 3. Infection efficiency[a] for subpopulations of *Phytophthora infestans* from Rosa (field) and Kennebec (field)

	Subpopulation				
Cycle[b]	Rosa on Rosa (own)[c]	Kennebec on Rosa (other)[c]	Rosa on Kennebec (other)	Kennebec on Kennebec (own)	Mean
10	0.54	0.70	0.74	1.1	0.77
11	0.19	0.21	0.38	0.54	0.33
13-1	0.13	0.26	0.07	0.21	0.17
13-2	0.19	0.33	0.08	0.33	0.23
Mean	0.26	0.38	0.32	0.54	

[a] Infection efficiency = (number of lesions per leaflet × 100)/number of sporangia applied. Each value is the mean of lesion counts on 15 leaflets.
[b] Number of serial passages the fungus was maintained on its "own" host in the greenhouse following reisolation from field plots at the end of the growing season. Two infection efficiency tests were done after cycle 13.
[c] "Own" is the cultivar on which the subpopulation had been cultured repeatedly; "other" is the cultivar on which it had not grown previously.

Further comparisons were done between pairs of natural, nonmutagenized subpopulations only.

Infection efficiency. After repeated cycles of pathogenesis on different cultivars, changes in subpopulations had occurred. For each pair of subpopulations, one subpopulation showed slightly higher mean infection efficiency on both its "own" and the "other" host (Tables 3 and 4). One of the pairs of subpopulations differed significantly in infection efficiency, Kennebec (field) vs. Rosa (field) (Tables 3 and 4). There were no significant differences between the "own" and "other" classes for any of the pairs of subpopulations when data for both cultivars were combined (Table

TABLE 4. Infection efficiency[a] for subpopulations of *Phytophthora infestans*

Fungal subpopulation	Host cultivar		Mean
	Norchip[b]	Sebago[b]	
Multiple cultivars (field)	1.1	0.14	0.6
Sebago (field)	1.9	0.36	1.2
Mean	1.5	0.25	
"Own"[c]	0.7		
"Other"[c]	1.0		
Source of variation	Mean square		
Treatments	1.93**		
Subpopulations	0.73		
Cultivars	4.84**		
Own vs. other	0.22		
	Rosa[d]	Kennebec[d]	
Rosa (field)	0.26	0.32	0.29
Kennebec (field)	0.38	0.54	0.46
Mean	0.32	0.43	
"Own"	0.40		
"Other"	0.35		
Source of variation	Mean square		
Treatments	0.06*		
Subpopulations	0.12*		
Cultivars	0.05		
Own vs. other	0.01		
	Norchip[d]	Sebago[d]	
Norchip	2.4	0.4	1.4
Sebago	2.8	0.7	1.8
Mean	2.6	0.5	
"Own"	1.5		
"Other"	1.6		
Source of variation	Mean square		
Treatments	5.6**		
Subpopulations	0.3		
Cultivars	16.4**		
Own vs. other	0.01		
	Rosa[e]	Kennebec[e]	
Rosa	0.15	0.36	0.26
Kennebec	0.27	0.42	0.34
Mean	0.21	0.39	
"Own"	0.28		
"Other"	0.32		
Source of variation	Mean square		
Treatments	0.08**		
Subpopulations	0.05		
Cultivars	0.20**		
Own vs. other	0.005		

[a] Infection efficiency = (number of lesions per leaflet × 100)/number of sporangia applied.
[b] Values are means of three tests. Lesions were counted on 15 leaflets in each test.
[c] "Own" is the cultivar on which the subpopulation had been cultured repeatedly; "other" is the cultivar on which it had not grown previously.
[d] Values are means of four tests. Lesions were counted on 15 leaflets in each test.
[e] Values are means of six tests. Lesions were counted on 15 leaflets in each test.

4). For the Rosa (field) and Kennebec (field) subpopulations, the infection efficiency of the "own" class was slightly higher but not significantly different from the "other" class (Table 4). For the other three pairs of subpopulations, the infection efficiency for the pooled "own" category was slightly lower than that of the "other" class; thus adaptation was not indicated (Table 4).

Sporulation. The number of sporangia per lesion did not differ between subpopulations on a given cultivar in any experiment, although the number of sporangia per lesion varied among experiments (Table 5). Sporulation of the "own" class, for both cultivars combined, did not differ significantly from that of the combined "other" class (Table 5).

Field test of adaptation. If adaptation occurred during one season, the average of the rates of epidemic development in single-cultivar plots would be expected to be faster than that in mixed plots. Disease progress was assessed for three treatments, Rosa alone, Kennebec alone, or Rosa and Kennebec planted alternately so each tuber was adjacent to tubers of the other cultivar. Rosa and Kennebec have similar levels of rate-reducing resistance as measured by rate of epidemic development in small field plots (Fry, *unpublished*) but were developed from different backgrounds (1,12). Adaptation was not indicated because there were no differences in the rate of disease development for the three treatments (Fig. 1).

DISCUSSION

Evidence concerning the ability of *P. infestans* to adapt to potato cultivars is inconsistent. Jeffrey et al (7) and Caten (2) found that *P. infestans* isolates grew better on tubers of the cultivar from which they had been isolated in the field than on tubers of other cultivars. They did not speculate whether this effect would be sufficient to endanger continued use of these cultivars. Paxman (11) collected *P. infestans* isolates from tubers of three cultivars and, after more than 90 cycles of growth of each isolate on tubers of its own cultivar, found no evidence for adaptation. In observations of epidemic development in the field, differential interactions between *P. infestans* isolates and potato cultivars with rate-reducing resistance were postulated (9). It was suggested that this was evidence for the ability of *P. infestans* populations to adapt to potato cultivars with rate-reducing resistance. In contrast, we found that populations of *P. infestans* changed in pathogenic ability when cultured separately on different cultivars but that these changes did not indicate adaptation because the expression of the changes was not limited to the cultivar on which the subpopulation was developed.

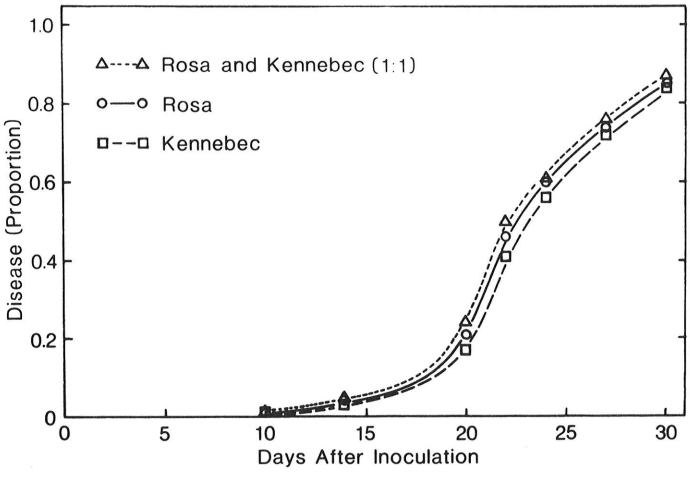

Fig. 1. Disease progress curves of late blight development in plots of potato cultivars Rosa, Kennebec, and Rosa and Kennebec mixed. Plots were inoculated 20, 21, and 22 July with sporangia of *Phytophthora infestans* race 1,3,4. Observations began 31 July 1981. Data points are means of four plots per treatment. Standard deviations of the means were similar for all three treatments and increased from 0.01 (day 14) to 0.04 (days 22–30) as proportion of diseased tissue increased.

Subpopulations cultured on Sebago and Kennebec showed greater infection efficiency than their counterparts on Norchip and Rosa. We did not detect adaptation during the single season of our field studies. Differences in methods could account, in part, for inconsistent evidence about adaptation. In the two laboratory studies that indicated adaptation, radial growth on tuber slices was the measure of an isolate's ability to grow on a particular cultivar (2,7). Factors necessary for growth on tuber slices could be considerably different than those necessary for foliar infection and for epidemic development. The expression of polygenic resistance in the foliage has little correlation with its expression in the tubers

TABLE 5. Mean number of sporangia per lesion (and standard deviation of the mean), in thousands, for subpopulations of *Phytophthora infestans*

Cycle[a]	Subpopulations[b]				Mean
	Norchip on Norchip (own)	Sebago on Norchip (other)	Norchip on Sebago (other)	Sebago on Sebago (own)	
27	150[c] (36)	130 (44)	45 (13)	18 (8)	86
28	140 (22)	160 (18)	49 (13)	64 (7)	103
30	310 (32)	270 (36)	150 (26)	130 (15)	215
Mean	200	187	81	71	
Means of subpopulations					
Norchip	140		Sebago	129	
Both own	136		Both other	134	
Source of variation				Mean square	
Treatments				1.39×10^{10}**	
Subpopulations				4.32×10^{8}	
Cultivars				4.13×10^{10}*	
Own vs. other				5.33×10^{6}	

	Rosa on Rosa (own)	Kennebec on Rosa (other)	Rosa on Kennebec (other)	Kennebec on Kennebec (own)	
6	31[d] (13)	22 (0.3)	180 (19)	160 (20)	98
25	100 (17)	100 (22)	130 (11)	100 (9)	105
29	96 (8)	100 (10)	140 (17)	200 (12)	134
Mean	76	74	147	153	
Means of subpopulations					
Rosa	112		Kennebec	114	
Both own	114		Both other	110	
Source of variation				Mean square	
Treatments				5.70×10^{9}	
Subpopulations				1.88×10^{7}	
Cultivars				1.70×10^{10}*	
Own vs. other				5.21×10^{7}	

	Rosa on Rosa (own)	Kennebec on Rosa (other)	Rosa on Kennebec (other)	Kennebec on Kennebec (own)	
11	4[e] (1)	4.3 (2)	14 (3)	13 (4)	9
13	21 (3)	21 (1)	21 (2)	18 (3)	20
14	63 (1)	99 (1)	46 (1)	82 (1)	72
Mean	29	41	27	38	
Means of subpopulations					
Rosa (field)	28		Kennebec (field)	40	
Both own	33		Both other	34	
Source of variation				Mean square	
Treatments				1.38×10^{8}	
Subpopulations				3.89×10^{8}	
Cultivars				2.79×10^{7}	
Own vs. other				1.54×10^{6}	

[a] Number of serial passages of the fungus through its "own" host before experiment was done.

[b] "Own" is the cultivar on which the subpopulation had been cultured repeatedly; "other" is the cultivar on which it had not grown previously.

[c] Means based on three replications (cycle 27), six replications on Norchip plants and nine on Sebago (cycle 28), and four replications (cycle 30).

[d] Means based on five replications on Kennebec plants and three replications on Rosa (cycle 6), five replications on Kennebec and four on Rosa (cycle 25), and five replications (cycle 29).

[e] Means based on five replications on Kennebec and three replications on Rosa (cycle 11) and four replications (cycles 13 and 14).

(5).

Infection efficiency and sporulation in our system varied over time. For two pairs of subpopulations (Rosa-field and Kennebec-field, Norchip and Sebago), overall infection efficiency decreased with successive experiments. Differences in condition of inoculum, plants, and incubation conditions could have contributed to this variability. The experiments done were sensitive enough to detect differences between cultivars and between subpopulations and thus would have detected adaptation if the magnitude had been large enough to be an important effect.

Data from individual experiments could suggest cultivar-specific adaptation. In our first test of the Rosa and Kennebec field subpopulations, the Kennebec subpopulation on Kennebec showed much higher infection efficiency than any of the other three combinations of cultivar and subpopulation (Table 3). In subsequent trials this difference was much less pronounced. Latin et al (9) found that one isolate of *P. infestans* (isolate II) ranked Kennebec as more resistant than Sebago, whereas isolate IV reversed the order, with Sebago appearing slightly more resistant. They suggested this cultivar × isolate interaction was evidence for cultivar-specific adaptation and "erosion" of rate-reducing resistance (9). These results may be due only to variability in the system because in 1977 isolate II ranked Kennebec as less resistant than Sebago, the opposite of the results of the 1978 trial (9). Thus with few replications, cultivar-specific adaptation could be indicated that, in the course of more experiments, would appear to be due only to random variation in the system. The studies of Latin et al (9) were conducted with random combinations of isolates and cultivars. Where consistent differences in aggressiveness among populations of *P. infestans* do occur, they do not necessarily result from cultivar-specific adaptation. Differences are more likely to be due to selection, over long periods, of characteristics optimum for growth of the fungus, unrelated to individual host cultivars. An ability to adapt in response to new cultivars cannot be assumed.

Our field experiment that began with a single race of *P. infestans* gave no evidence of adaptation during a single growing season. Rates of epidemic development for plots of one cultivar were similar to those in plots of mixed cultivars. Long-term observation also provided no evidence of adaptation occurring in the field. Vanderplank (16) found that 10 cultivars used in the Netherlands from 1938 to 1968 maintained their relative ranking in resistance during this period. Sebago, the moderately resistant cultivar used in our experiments, has been known as moderately resistant during 30 yr of commercial production.

Improved growth of fungal isolates on a given cultivar may be due to selection of variants already present in the fungal population rather than change in response to growth on the cultivar (7). Expression of adaptation would thus depend on the diversity in the population. Diversity could be increased by using different single isolates, populations derived from a mixture of isolates, or populations resulting from mutagenesis. In our studies, subpopulations on different cultivars were derived from different initial populations, varying in their level of diversity. Three pairs of subpopulations were derived from initial populations, each consisting of a different single isolate. Greater diversity was obtained by combining five isolates to make the initial population from which the Rosa and Kennebec subpopulations were derived. Two other subpopulations resulted from treatment of *P. infestans* zoospores with a chemical mutagen (NTG) and inoculation to large isolated field plots of Sebago and Kennebec. We did not detect change in any of these populations that would indicate either selection or adaptive change.

Adaptation of *P. infestans* to potato cultivars with rate-reducing resistance should be evaluated carefully. A number of factors, in addition to the organism's variability, affect the extent to which adaptation may be expressed. Rate-reducing resistance is expected to be stable and durable. For potato, this form of resistance has shown little specificity, is usually polygenic, and should require a number of mutations for a fungus to adapt to it (17). It may involve host and pathogen processes that have coevolved for long periods, so further significant adaptation of the pathogen to the host is unlikely (17). Populations with greater pathogenic ability on a given cultivar might not be best adapted for survival between seasons in tubers. Changes in cultivar use patterns minimize continual exposure of a given *P. infestans* population to the same cultivar. Control practices, including seed certification, destruction of inoculum sources such as cull piles, and fungicide sprays, limit the size of pathogen populations. These factors combine to limit the potential for rapid adaptation of *P. infestans* populations to potato cultivars with polygenic, rate-reducing resistance.

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