Integrated Effects of Polygenic Resistance and a Protective Fungicide on Development of Potato Late Blight

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Appreciation is expressed to H. D. Thurston for identifying the race of the isolate of *Phytophthora infestans* and to O. E. Schultz, W. A. Sinclair, and H. D. Thurston for suggestions concerning experiments and the manuscript.

Accepted for publication 14 March 1975.

ABSTRACT

The relative polygenic resistance to *Phytophthora infestans* in eight potato (*Solanum tuberosum*) cultivars was determined by calculating the apparent infection rate (r) for each cultivar in field plots. The effects on r of weekly applications of fungicide to Sebago (polygenically resistant) and Russet Rural (susceptible) were determined and related quantitatively to the effect of polygenic resistance. On the basis of reduction in r, the effect of greater polygenic resistance in Sebago than in Russet Rural was equivalent to approximately 0.5 kg fungicide (a.i.)/hectare applied weekly to Russet Rural.

Phytopathology 65:908-911

Additional key words: horizontal resistance, epidemic, pest management.

For certain diseases the use of race-specific resistance governed by one or a few host genes has been ineffective as a control procedure because of the rapid appearance of new races of pathogens which were virulent to cultivars containing such genes. Late blight of potato (*Solanum tuberosum* L.) caused by *Phytophthora infestans* (Mont) dBy. is among these diseases. Virulent [sensu Flor, (4)] biotypes of the pathogen were detected shortly after breeding lines with race-specific resistance were released as commercial cultivars (15). Consequently, plant breeders have concentrated on the development of resistance described variously as field, general, partial, horizontal, polygenic, or multigenic (3, 15, 20). For late blight the effect of this type of resistance is reduction of the sporulation capacity of lesions, reduction of lesion size, and retardation of the rate of sporangiophore growth (6, 10, 16). Collectively these effects reduce the rate of epidemic development (20).

Periodic applications of a protective fungicide for control of a compound-interest disease [sensu van der Plank (18)] also reduce the rate of epidemic development (19). Presumably, polygenic resistance and the periodic application of a protective fungicide should be useful in combination (13). It should be possible to reduce the rate of epidemic development [or apparent infection rate (18)] to a given value with less fungicide on a cultivar with a high level of polygenic resistance than on a cultivar with less resistance.

This hypothesis was tested in 1973 and 1974 using potato cultivars with different levels of resistance to *P. infestans*. Specific objectives were to determine: (i) the range of polygenic resistance in certain potato cultivars grown in northeastern USA, (ii) the extent to which this resistance reduces the apparent infection rate, and (iii) whether polygenic resistance can be quantitated in terms of periodic applications of a fungicide. Methods and results in the two years were similar. Therefore only the 1974 experiments are reported here. A preliminary report has been published (5).

MATERIALS AND METHODS.—*Field plot design.*—Small plots were treated in randomized complete blocks with four or five replications per treatment, depending upon the experiment. Plots each five rows wide (0.9 m between rows) and 4.6 m long, were separated from each other by fallow soil (3.7 m within blocks and 4.6 m between blocks).

Cultural procedures.—Foundation or certified potato "seed" was treated with a zinc ion-maneb dust and planted at approximately 23 cm spacing in the row. The cultivars Russet Rural and Sebago were selected because they have the least and greatest amounts of polygenic resistance, respectively, among commercially grown potatoes in New York State. Additional cultivars with intermediate levels of polygenic resistance were used in 1974 only: Chippewa, Green Mountain, Hudson, Katahdin, Kennebec, and Superior. Fertilizer (10-20-10) was applied at 1,680 kg/hectare (ha) at planting time (5 June). Herbicide (Linuron 50 WP, 3.4 kg/ha) was applied prior to plant emergence. Plants were hilled when 25-35 cm tall. Insecticide (carbaryl, 50 WP at 1.68 kg/ha or Meta-Systox R, 2 EC at 2.5 liters/ha) was applied as needed. Fungicide (Dithane M-45, a coordination product of zinc ion and maneb, 80% WP) was applied at 7-day intervals with a John Bean Spartan Sprayer utilizing 935 liters water/ha (100 gal/acre). Fungicide treatments were 0, 0.22, 0.67, or 1.79 kg a.i./ha. Plots receiving no fungicide were sprayed with water. Vines were killed (3.5 liters dinoseb in 47.5 liters diesel fuel/ha) on 9 September.

Conditions conducive to development of *P. infestans* were maintained by periodic sprinkler irrigation (0.17 cm water/hour). The plots were irrigated from 0800-0830 hour and 2000-2030 hour daily from 24 July to 3 September.

The fungus was established by atomizing an unsprayed plant in the center of each plot on 24 July with 8 ml of a suspension (3,000 sporangia/ml) of *P. infestans* (Race 1, 4) zoospores and sporangia obtained from lesions on potato leaves. Plants not adequately infected were reinoculated.

The proportion of diseased tissue (% disease) was estimated every 3-6 days from date of inoculation until vines were killed, using a modification of a blight assessment key published by the British Mycol. Soc. (1). The modification was that one lesion per 10 plants = 0.01% disease and that one lesion/plant = 0.1% disease. One estimate was made for each of four quadrants per plot and the average value for each plot and date was

computed. The percentage disease was not corrected for plant growth during the season. For analyses, percentage disease (x) was converted (18) to:

$$\log_{10} \frac{x}{1\text{-}x}$$

For each plot, the apparent infection rate (r) was calculated according to the formula:

$$r = \frac{2.3}{t_2 - t_1} \left(\log_{10} \frac{x_2}{1 - x_2} - \log_{10} \frac{x_1}{1 - x_1} \right)$$

where time(t) is days beginning when $x = 0.1\%$ and ending when $x = 90\%$.

RESULTS.—Development of the late blight epidemic on Sebago and Russet Rural was retarded differentially by the fungicide treatments (Table 1, Fig. 1). A plot of r versus fungicide dosage indicated that the two variables were not linearly related. However, a straight line could be calculated for $\log_{10}$ r versus fungicide (Fig. 2). Analysis of covariance (14) indicated that the slope of the regression line for Russet Rural was not significantly different from that for Sebago. However, the elevations of the two lines were significantly different ($P < 0.01$). Adequate control of late blight on Russet Rural was obtained with 1.79 kg fungicide/ha applied weekly, and interpolation on the regression line for Sebago indicates that approximately 1.3 kg fungicide/ha applied weekly to Sebago would have given equivalent control in this environment. Thus, the data indicate that the difference in polygenic resistance between Russet Rural and Sebago, in the environment of this experiment, was equivalent to 0.42 - 0.67 kg fungicide/ha applied weekly.

The effects of fungicide dosage on yields of the two cultivars were compared. For each cultivar, the yield at 1.79 kg fungicide/ha was regarded as maximum. Yields at other levels of fungicide were expressed as percentage of maximum and regressions of relative yield on fungicide dosage were calculated (Fig. 3). For this calculation, the values obtained at 1.79 kg fungicide/ha were not included. Yield response to this high dosage of fungicide was obviously not linear, whereas yield response to the lower dosages of fungicide was linear. Analysis of covariance indicated that the slopes of the lines were not significantly different, but that elevations were different ($P = 0.01$).The separation between the two lines was equivalent to 0.25 - 0.40 kg fungicide/ha applied weekly. Yield data from the experiment in 1973 had indicated that the difference between the two cultivars was equivalent to 0.22 - 0.45 kg fungicide/ha applied weekly.

In another experiment, the relative levels of polygenic resistance in six additional commercial cultivars was assessed by calculating the apparent infection rates in the absence of fungicide. Four replicate plots of each cultivar were included. Chippewa and Superior supported infection rates nearly as rapid as did Russet Rural, whereas the apparent infection rate on Kennebec was nearly as low as that on Sebago (Table 2).

The differences among cultivars can be expressed in terms of the amount of fungicide that might be required to reduce the apparent infection rate on Russet Rural to that on a given cultivar. Figure 4 shows the apparent infection rate of each cultivar in relation to the regression of $\log_{10}$ r

TABLE 1. Effect of different levels of weekly applications of protectant fungicide[a] on the apparent infection rate (r)[b] of *Phytophthora infestans* in plots of Russet Rural and Sebago potatoes in 1974

Fungicide (kg active ingredient/ha)	Apparent infection rate (r) (per unit per day)	
	Russet Rural	Sebago
0.00	0.753 A[c]	0.254 C
0.224	0.326 B	0.219 C
0.672	0.245 C	0.109 D
1.79	0.068 DE	0.043 E

[a]Coordination product of zinc ion and maneb, 80% WP, in 935 liters water/ha.

[b]Infection rate: $r = \frac{2.3}{t_2 - t_1} (\log_{10} \frac{x_2}{1 - x_2} - \log_{10} \frac{x_1}{1 - x_1})$

where x = percent disease $\times 10^{-2}$, and t = days.

[c]Values followed by the same letter are not significantly different at $P = 0.05$.

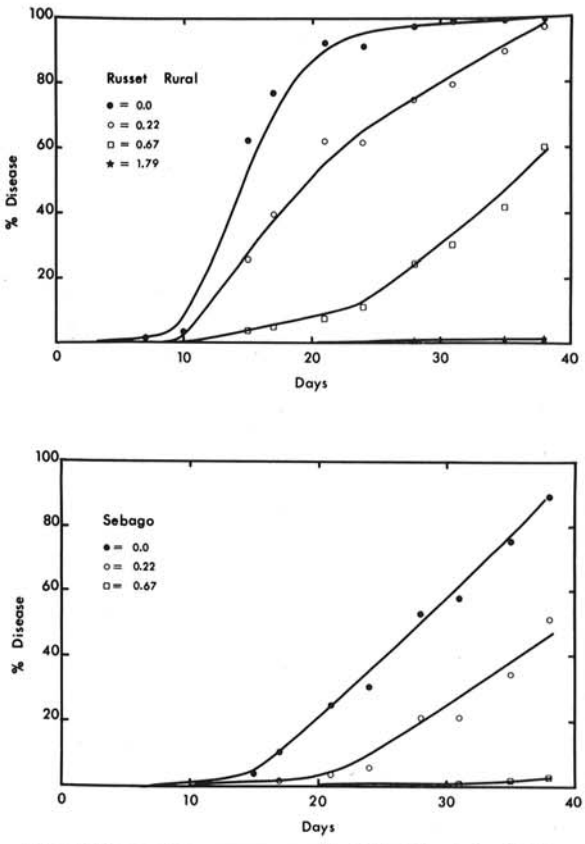

Fig. 1. Disease progress curves of late blight in plots of Sebago and Russet Rural potatoes as influenced by fungicide dosage (0.0, 0.22, 0.67, or 1.79 kg a.i./ha). Observations began 29 July 1974, and percentage disease was estimated using a modification of a blight assessment key published by the British Mycological Society (1). Data points are means of five plots per treatment. Fungicide doses refer to the active ingredient of Dithane M-45 (coordination product of zinc ion and maneb, 80% WP). The disease in plots of Sebago potatoes treated with 1.79 kg fungicide/ha was less than 0.7% and consequently was not plotted.

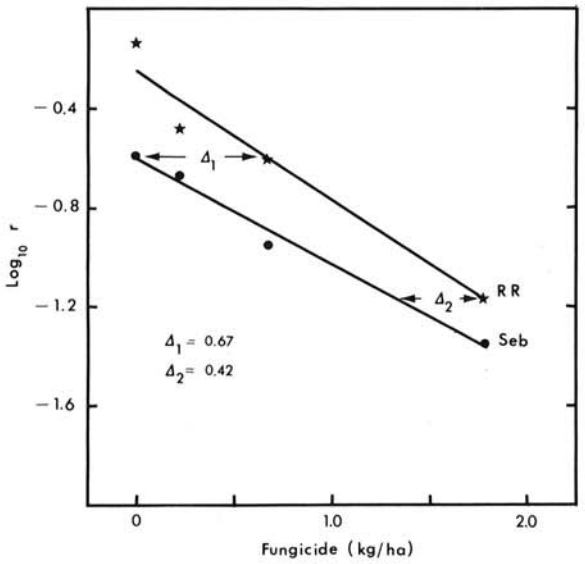

Fig. 2. Effect of fungicide dosage on the apparent infection rate (r) of *Phytophthora infestans* in plots of Sebago (Seb) and Russet Rural (RR) potatoes in 1974. Analysis of covariance indicated that the slopes of the two regressions were not significantly different, but that the elevations were significantly different ($P \leq 0.01$). Fungicide dose is expressed in active ingredient of the coordination product of zinc ion and maneb, 80% WP, applied weekly. The regression equation for Russet Rural was $y = -0.231 - 0.548\ x$ and for Sebago was $y = -0.634 - 0.411\ x$.

versus fungicide applied to Russet Rural. If for each cultivar the slope of a regression of $\log_{10}$ r on fungicide dosage were also −0.584, the differences in r among cultivars could be translated into fungicide doses required to cause equal changes in r in Russet Rural. Thus, the difference in polygenic resistance between Chippewa and Hudson would be equivalent to approximately 0.2 kg fungicide/ha applied weekly, and the difference between Hudson and Sebago equivalent to approximately 0.45 kg fungicide/ha applied weekly.

DISCUSSION.—The epidemiological data are consistent with van der Plank's observations that both polygenic resistance and periodic application of a protective fungicide act to reduce the apparent infection rate (20). Although the differences in polygenic resistance among the cultivars tested were slight, the difference between the extremes was such that if weekly applications of 1.79 kg fungicide/ha effected adequate control on Russet Rural, then weekly applications of 1.3 - 1.4 kg fungicide/ha should effect adequate control on Sebago. Similar results were obtained in 1973 when the difference between Russet Rural and Sebago was found equivalent to 0.4 - 1.0 kg fungicide/ha applied weekly. Some European and Latin American potato cultivars have levels of polygenic resistance much higher than those evaluated in this study (16). Consequently greater reductions in the amount of fungicide required for adequate control should be possible with the introduction of cultivars with high levels of polygenic resistance. Additionally, under environmental conditions less conducive to *P. infestans* than those used in these tests, the apparent infection rate in Sebago might be reduced to an acceptably low value with less than 1.3 kg fungicide/ha. Presumably fungicide efficiency could be further increased if the fungicide were applied according to a forecasting system (2, 7, 11).

In the experiments reported here, different concentrations of fungicide were applied weekly. Further work is necessary to determine whether the same relationship between fungicide and polygenic resistance would prevail if the interval between applications rather than the concentration of fungicide were varied.

The nonlinearity of regressions of r versus fungicide concentration was not expected. Although a straight line could be fitted to a regression of $\log_{10}$ r on fungicide (Fig. 2), this line might not be the best representation of the relationship. Especially for the data obtained with Russet Rural, it could be argued that a curve instead of a straight

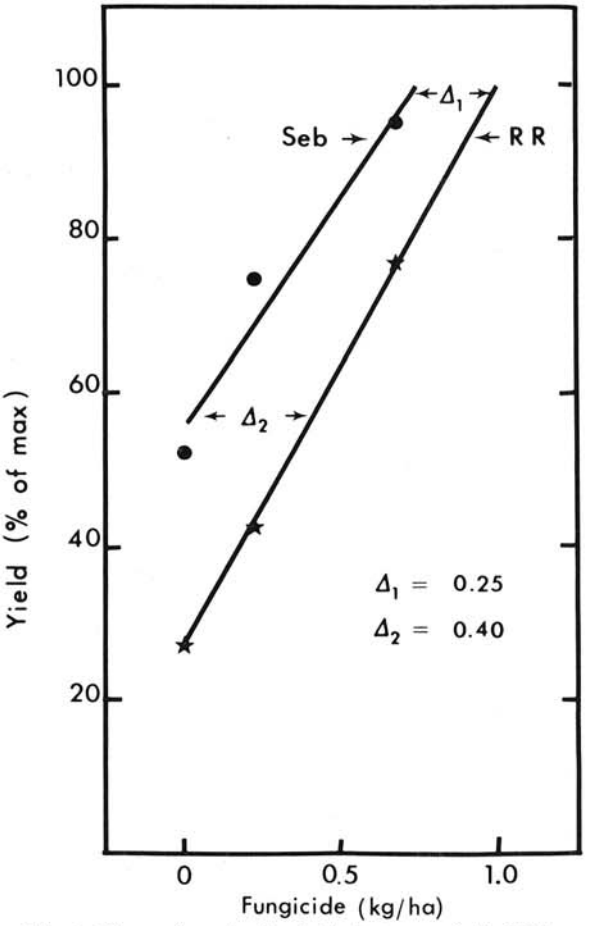

Fig. 3. Effects of varying fungicide dosage on yield of Sebago and Russet Rural potatoes under conditions favorable for increase of *Phytophthora infestans*. Yields are presented as percentage of maxima, which were 38,800 and 32,800 kg/hectare(ha) for Sebago and Russet Rural, respectively, obtained in plots sprayed weekly with 1.79 kg a.i./ha of fungicide (coordination product of zinc ion and maneb, 80% WP). Regressions were calculated on the basis of relative yields at 0, 0.22, and 0.67 kg fungicide/ha. The regression equation for Russet Rural was $y = 27.4 + 65.7\ x$, and for Sebago was $y = 55.7 + 54.9\ x$.

line might best describe the relationship between $\log_{10}$ r and fungicide dosage. But even as described (Fig. 2) the relationship between r and fungicide suggests that a given increment of fungicide will reduce r to a greater extent if applied to a plant receiving no fungicide than if applied to plants receiving some fungicide. This type of relationship should be considered in pest management decisions.

The application of these results is based on the assumption that polygenic resistance is relatively stable. It is generally believed that polygenic resistance is more stable than monogenic resistance, but whether a pathogen can adapt moderately to a cultivar with polygenic resistance is debatable (8, 9, 12, 17, 21). If a biotype of a pathogen could adapt to one cultivar, then differences among cultivars could not be expressed reliably in terms of fungicide equivalents. Because of the emphasis placed on polygenic resistance in breeding programs, and because less fungicide will probably be used to control compound-interest diseases of these cultivars, the stability of polygenic resistance should be tested critically.

TABLE 2. Apparent infection rates of *Phytophthora infestans* in eight potato cultivars

Cultivars	Apparent infection rate (per unit per day)
Russet Rural	0.718 A[a]
Chippewa	0.540 B
Superior	0.512 B
Hudson	0.436 C
Katahdin	0.336 DE
Green Mountain	0.312 DE
Kennebec	0.279 EF
Sebago	0.252 F

[a]Rates followed by any common letter are not significantly different at $P = 0.01$.

[b]Infection rate: $r = \frac{2.3}{t_2 - t_1} \left(\log_{10} \frac{x_2}{1 - x_2} - \log_{10} \frac{x_1}{1 - x_1}\right)$

where x = percent disease $\times 10^{-2}$, and t = days.

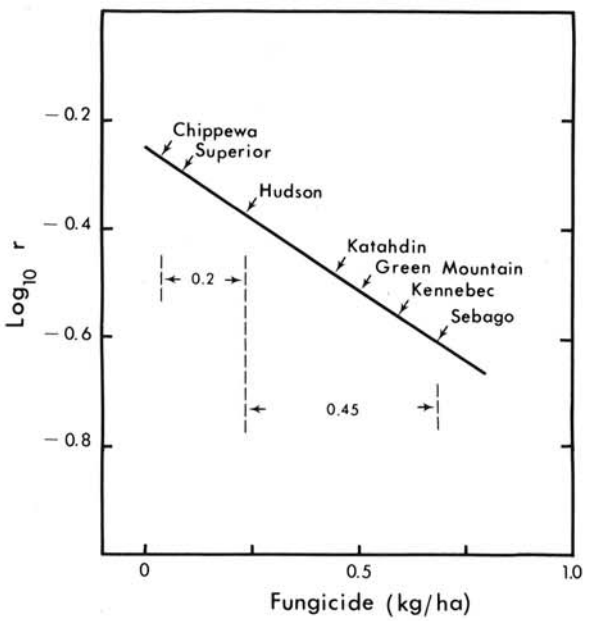

Fig. 4. Predicted relationship of fungicide and polygenic resistance among seven potato cultivars. Apparent infection rates were positioned on the regression of $\log_{10}$ r versus fungicide obtained from Russet Rural (Fig. 2). Regression equation was $y = 0.231 - 0.584$ x.

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