An Index for Cultivar Resistance Based on Disease Progress Curves

K. B. McRae and H. W. Platt

Regional statistician (Atlantic), Agriculture Canada, Research Station, Kentville, N.S. B4N 1J5; and research scientist, Agriculture Canada, Research Station, Charlottetown, P.E.I., C1A 7M8, Canada.
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ABSTRACT

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A new index for assessing cultivar resistance to infection compares the rate of disease progress in cultivars relative to that in a standard cultivar. Disease progress curves for cultivars are separated into components due to the disease response of individual cultivars and the underlying disease progression for the particular field conditions, as determined by the standard cultivar. The index has a special meaning if the logarithm of the percentage of healthy tissue in a test cultivar is directly proportional to that in the standard cultivar during the course of the epidemic. The proportionality constant for each test cultivar (relative to the standard) can then be estimated by a simple algorithm, even if the disease progression is monitored only partially. The index and related procedures are illustrated with data collected for assessing resistance of potato cultivars to *Phytophthora infestans* over four growing seasons. Although the proposed index and the index based on area under the disease progress curve gave similar separation among cultivar groups of similar maturities within years, the new index was more stable over years and has a clear meaning. Modifications to the conduct of trials for determining cultivar disease resistance using the proposed index are discussed.

Additional key words: cultivar assessment, late blight resistance, potato cultivar assessment.

Identifying differences in disease resistance among cultivars is a task typically faced by pathologists, plant breeders, and cultivar assessment committees. The examination of disease progress curves (DPC) has become a useful procedure for identifying such differences. Madden (5) provides a review of models describing epidemics and gives specific statistical techniques for analyzing data for plant diseases, including a general epidemic model. Because these models describe disease progress as explicit functions of time, they tacitly assume steady state conditions. When changing environmental conditions drive a growth response, France and Thornley (2) recommend that the variable of time not be included in the model's equations expressing changes in the state variables (e.g., disease level) but enter the model only through the environmental driving function.

Several measures for cultivar resistance have been proposed that do not assume a known functional form of the underlying DPC. Fry (3) compares two such measures with the apparent infection rate for potatoes infected by late blight (caused by *Phytophthora infestans* (Mont.) de Bary) i.e., area under the disease progress curve (ADPC) and final disease rating (proportion of tissue affected). ADPC was found to be more reliable than the other two measures for identifying general cultivar resistance and determining effective rates of fungicides. One other measure not studied by Fry would be time to a fixed proportion of diseased tissue, say 50%. Platt and Tai (6) consider a measure of cultivar resistance for potatoes against late blight based on the method of principal components.

Although ADPC is a useful measure of general cultivar resistance, it does have severe limitations for field trials conducted over different seasons and locations. ADPCs depend on cultivar effects and individual trial effects. Within a single trial even the choice of the observation period can affect the measure, particularly if the initial and/or final portions are included or excluded (Fig. 1). The ranking of the cultivars would not be greatly affected, but the magnitude of differences among them may be. The task of determining general cultivar resistance from multiple trial data from several seasons is reduced if cultivar effects are distinct from the individual trial effects.

Standard cultivars or control treatments are generally included in field trials. Inclusion of such standards takes on special importance when they are used as a reference, from which the effect of an individual cultivar or treatment on the rate of disease progress can be assessed. In such trials, the number of standard plots should be increased within each replicate, as noted by Finney (1), to improve the average precision of the cultivar-standard comparisons.

For the case where diseases develop through most of the range from 0 to 100%, we propose a simple method for assessing differences in disease resistance among cultivars. The method considers a DPC to have a distinct nonrandom cultivar component added to a stochastic component that is common to all cultivars in the same trial. A cultivar's resistance to a pathogen is assumed to be characterized by the degree that the resultant disease progresses faster or slower in it than in a standard cultivar. A single rate

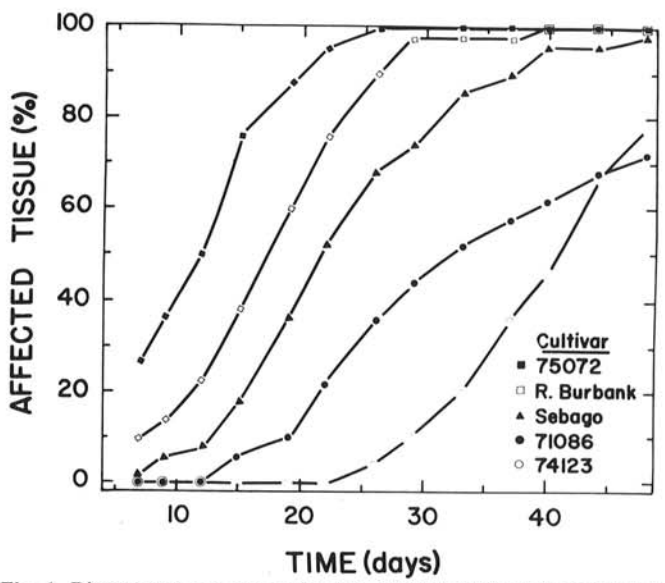

Fig. 1. Disease progress curves for five selected cultivars from Table 1 (71086, 75072, Russet Burbank, 74123, and Sebago).

constant may be sufficient for describing the cultivar component when it is independent of time.

METHODS

Let $Q(t)$, $t > 0$, represent the hypothetical DPC for a standard cultivar (or treatment). Observations can be taken on $Q(t)$ at times $t_1, t_2 \ldots t_n$ and an empirical representation of $Q(t)$ may be constructed by the piecewise linear curve connecting the observed values of $Q(t_1), Q(t_2) \ldots Q(t_n)$, as in Figure 1. Let $P(t)$ represent the DPC for a test cultivar in the same trial and observed at the same times as $Q(t)$. Both $P(t)$ and $Q(t)$ are theoretical DPCs, representing the proportion of diseased tissue at time t, ranging from zero disease to complete disease, given sufficient time. (The method might be adapted for diseases that develop to a maximum less than 1.) The observed values of the DPCs (which may not include 0 and 1), their theoretical values, and their areas are expressed by decimal numbers, in this study, but the visual ratings are expressed as percentages.

When the values of $(P(t_i), Q(t_i))$, $i = 1, 2 \ldots n$, are plotted on a unit square and connected from (0,0) to (1,1), as in Figure 2, the area is divided into two parts. Let the area under the curve be denoted by U, which we name as the area under the standardized DPC, or ASDPC. Then U is a measure of the overall difference in the rate of disease progress for that cultivar compared with the standard; for similar disease progression in both cultivars $U \cong 1/2$, if $U > 1/2$ ($< 1/2$) then the disease progresses generally faster (slower) in the test cultivar than in the standard cultivar. Thus U, suitably transformed, can be used as an index of cultivar resistance relative to the standard.

From the empirical graph of $P(t_i)$ versus $Q(t_i)$ on the unit square, the observed area U under the curve may be calculated from the sum of the trapezoids between points by

$$U = \Sigma_{i=1}^{n+1}[Q(t_i) - Q(t_{i-1})] \times [P(t_i) + P(t_{i-1})]/2 \qquad (1)$$

where $P(t_0) = Q(t_0) = 0$, and $P(t_{n+1}) = Q(t_{n+1}) = 1$.

Monitoring the complete course of a disease is desirable, but because monitoring is often incomplete, an estimation procedure is given when a certain functional relationship is tenable.

Functional form for the standardized DPC. Although the index of cultivar resistance given by U, $\sin^{-1}(\sqrt{U})$ or $\text{logit}(U)$ may be sufficient for some cultivar evaluation committees and plant breeders, the approach can be extended by choosing a functional form for the standardized DPC. For the pathogen-host combination that motivated this study, *P. infestans* and potato, the empirical DPCs could be approximated by the relationship between the proportions of healthy tissue

$$1 - P(t) = (1 - Q(t))^{\theta}, \; 0 < t < \infty \qquad (2)$$

or

$$-\log(1 - P(t)) = -\theta \log(1 - Q(t)) \qquad (3)$$

Once the disease progression $Q(t)$ and the parameter θ are known, then the process $P(t)$ can be approximated. The degree to which $P(t)$ progresses faster than $Q(t)$ depends on the value of θ. For $\theta = 1$ the DPCs are identical; for $\theta > 1$, $P(t)$ progress faster; for $\theta < 1$, $P(t)$ progresses slower than $Q(t)$. Equation 2 defines a series of curves on the unit square (Fig. 3) for various values of θ with the curves for θ and $1/\theta$ being symmetrical about the main diagonal. A consequence of equation 2 is that θ equals the slope of the standardized DPC at $t = 0$; once the process $P(t)$ begins with standardized rate θ, its course depends only on the standard disease progression given by $Q(t)$. Thus equation 2 assumes θ is a constant for each cultivar, and not a function of time during the process.

The area above the theoretical standardized DPC is $1/(1+\theta)$, obtained by calculus:

$$A = \int_0^1 (1 - P)dQ = \int_0^1 (1 - Q)^{\theta} dQ = 1/(1+\theta). \qquad (4)$$

The area U under the curve is $\theta/(1+\theta)$.

Equation 2 may be plausible for many disease progress studies with or without a DPC of known functional form. If the underlying DPCs are Weibull with common shape parameters, e.g., see Madden (4), then equation 3 is satisfied. In general, the relationship may be judged plausible if the scatter plot of the points falls on a line through the origin, which corresponds to the point at $t = 0$. If the resistance characteristics change during the development of the disease, then equation 3 cannot hold. Also, if the disease resistance for a cultivar depends on specific races of pathogen, then the estimated value of θ will depend on the races present.

Estimation of θ (complete monitoring). The observed values of a DPC are highly correlated if they are taken on the same individuals, so that estimation of θ may require more specialized techniques than ordinary least squares (5). Using equation 4, θ may be easily estimated if two conditions are satisifed, namely: *a*) The disease progress curves of all cultivars have been monitored simultaneously at several points over the whole disease progression; and *b*) $Q(t)$ is determined more precisely than the other cultivars on trial.

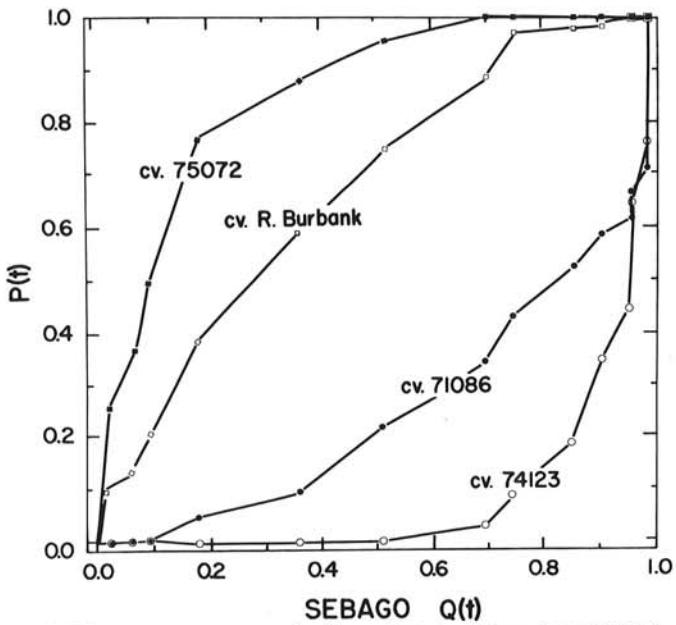

Fig. 2. Disease progress curves for four selected cultivars, from Table 1, standardized against Sebago.

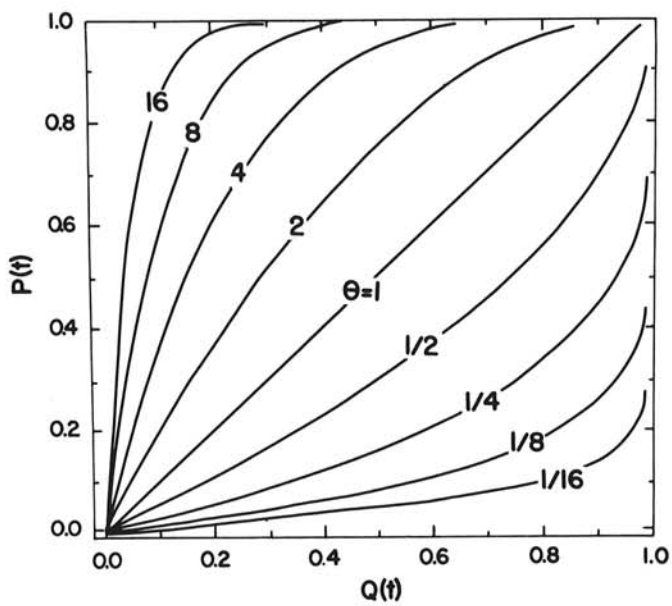

Fig. 3. Graph of relationship $P(t) = 1 - (1 - Q(t))^{\theta}$ for selected values of θ.

The area above or under an empirical (or observed) standardized DPC can be set equal to their theoretical values, analogous to the method of moments. Equation 4 may be rearranged, where $\hat{}$ denotes an estimated value, and written as

$$\hat{\theta} = (1-\hat{A})/\hat{A} = \hat{U}/(1-\hat{U}), \tag{4a}$$

which we expect provides a stable estimator of θ based on areas under and above a standardized curve. Leedow and Tweedie (4) studied the topic of weighted area methods for estimating the parameters of a growth curve, finding them to be 80–90% efficient on simulated data and providing robust estimates on field data. Because a perturbation in the standardized DPC at an individual observation time has little effect on the total area above or below the curve, its influence on the estimate of θ will be small, so that the estimator essentially is independent of the error structure for the repeated measurements on the same experimental units. Condition *b* is important because the DPCs of several cultivars are being compared with the standard and is satisfied when the standard cultivar is represented by multiple plots, Finney (1). It is important, also, for incomplete monitoring.

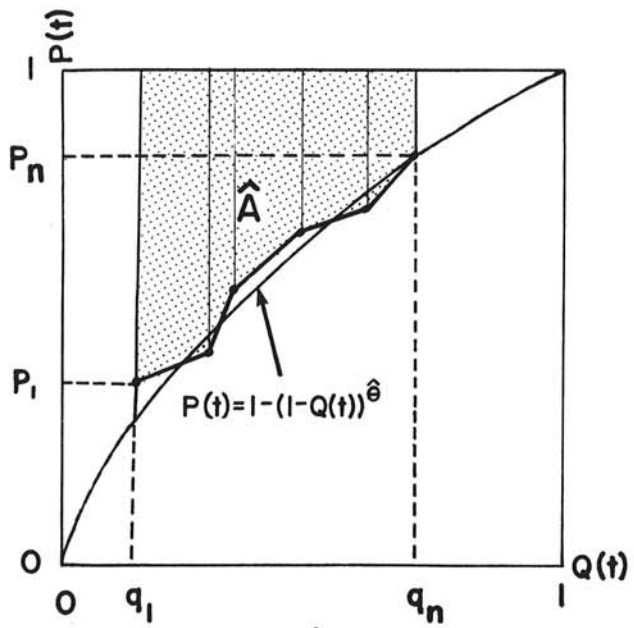

Fig. 4. Diagram illustrating the area $\hat{A}$ above empirical curve and the fitted curve, where the disease progress is monitored between $0 \leq q_1 < q_n \leq 1$ in the standard cultivar and between $0 \leq p_1 < p_n \leq 1$ in the test cultivar.

Estimation of θ (incomplete monitoring). For the case where equation 2 is tenable, condition *a* may be relaxed and incomplete monitoring considered. Estimation of θ is still possible, using the empirical area above the observed portion of the curve, but an iterative solution is required. Suppose the first observation of $Q(t)$ was q_1, and the last observation was q_n, then the area of the trapezoids above the observed curve is equated to the theoretical area (Fig. 4), viz

$$\begin{aligned} A &= \int_{q1}^{qn} (1-Q)^{\theta} dQ \\ &= [(1-q_1)^{1+\theta} - (1-q_n)^{1+\theta}]/(1+\theta). \end{aligned} \tag{5}$$

The unique solution to equation 5 may be obtained by Newton's iterative method. The right-hand side of equation 5 is a strictly decreasing function of θ, equal to $q_n - q_1$ (which is greater than $\hat{A}$) when $\theta = 0$, and equal to 0 when θ approaches infinity. If the disease levels are observed to be zero then $\hat{A}$ would equal $q_n - q_1$ and θ would be taken to be zero. (Fewer than five iterations were needed for convergence in our work for this study.)

Because q_1, q_n or both are not fixed constants, they must be determined from the observed values of $Q(t)$. Consequently, the extra precision on $Q(t)$ is important also for obtaining a precise estimate of θ from equation 5, which depends on the partial area $\hat{A}$ as well as on q_1 and q_n.

Detection of nonproportionality. Although the area under the empirical standardized DPC will equal the area under the fitted curve based on equation 2 when monitoring is complete, its shape may not correspond to those in Figure 3. A delay in the onset of the disease or a change in the rate of disease progression, such as from a rapid breakdown after initial tissue resistance, will shift the empirical curve. We developed a diagnostic procedure for detecting such changes based on areas under portions of the empirical and fitted curves. The procedure is simple to program on a computer and is independent of the error structure of the repeated observations, but its efficiency may be affected by incomplete monitoring of resistant cultivars.

For a given value of $Q(t)$, say q', let $\hat{U}_1$ denote the area under the empirical standardized DPC from 0 to q', and let $\hat{A}_2$ denote the area above the same curve from q' to 1. Let $U_1(\hat{\theta})$ and $A_2(\hat{\theta})$ denote the respective areas about the corresponding fitted curve. If equation 2 is true, then the expected value of $(\hat{U}_1 + \hat{A}_2)$ will equal $U_1(\hat{\theta}) + A_2(\hat{\theta})$. We propose the statistic

TABLE 1. Mean percentage of tissue affected by late blight in potatoes at various dates in 1983

	August									September			
Cultivar	3	5	8	11	15	18	22	25	29	2	5	9	13
73008	0	0	0	0	0	0	0	0	0	1	1	3	4
AM6642	0	0	0	0	1	0	0	0	1	3	3	3	4
7670-7	0	0	0	0	0	0	0	0	1	2	6	9	14
Dorita	0	0	0	0	1	3	6	8	11	14	19	23	28
7703-1	0	0	0	0	1	2	3	7	10	19	24	33	36
Kennebec	0	0	0	1	2	1	2	4	9	19	23	33	40
71086	0	0	0	5	10	23	35	44	53	59	63	68	73
74123	0	0	0	0	0	1	3	9	20	35	45	65	78
Bison	0	0	0	0	0	3	5	14	24	38	48	68	85
Sebago	2	6	9	18	36	51	69	74	85	90	95	95	98
Russet Burbank	10	14	21	39	60	75	90	98	99	99	100	100	100
Green Mountain	10	14	20	34	46	65	80	91	96	100	100	100	100
76079	6	11	18	43	59	78	91	96	99	100	100	100	100
Bintje	11	15	23	40	64	79	94	96	100	100	100	100	100
Yellow Gold	8	9	14	33	69	89	99	99	100	100	100	100	100
Hudson	5	9	16	34	56	76	94	99	100	100	100	100	100
74094	16	21	33	51	68	84	95	99	100	100	100	100	100
76076	2	7	10	28	55	80	100	100	100	100	100	100	100
Oneida	20	24	39	60	76	90	100	100	100	100	100	100	100
75072	25	36	50	76	88	96	100	100	100	100	100	100	100
75040	14	24	38	68	89	99	100	100	100	100	100	100	100
76057	18	24	53	80	99	100	100	100	100	100	100	100	100
76036	34	46	78	90	100	100	100	100	100	100	100	100	100

$$D = \text{logit}\,(\hat{U}_1 + \hat{A}_2) - \text{logit}\,(U_1(\hat{\theta}) + A_2(\hat{\theta})) \quad (6)$$

as a diagnostic measure for nonproportionality in equation 3.

The required areas about the fitted curve may be calculated by integration between the appropriate limits in equation 4 to obtain

$$U_1(\hat{\theta}) = q' + ((1-q')^{1+\hat{\theta}} - 1)/(1+\hat{\theta}) \quad (7a)$$

$$A_2(\hat{\theta}) = (1-q')^{1+\hat{\theta}}/(1+\hat{\theta}). \quad (7b)$$

Although the choice of q' is arbitrary, the method for choosing should exploit the symmetry of the curves for θ and $1/\theta$. We propose using the value of $Q(t)$ corresponding to the intersection of the fitted curve with the off-diagonal line, viz the solution of

$$1 - P(t) = (1 - Q(t))^{\hat{\theta}} \text{ and } 1 - P(t) = Q(t)$$

giving

$$(1-q')^{\hat{\theta}} = q'. \quad (8)$$

Equation 8 can be solved by Newton's method, and $1/(1+\hat{\theta})$ provides a good starting value for q'.

Application of the method for cultivar assessment. The value of $\hat{A}$, $\hat{\theta}$, or $A(\hat{\theta})$—the latter two are based on fitted curves from complete or incomplete monitoring—provides a single index of the relative rates of disease among cultivars. The variance of $\hat{\theta}$ increases as θ deviates from 1, in part because the area above or the area below the empirical curve on the unit square becomes small and is more subject to perturbations. The logarithm of $\hat{\theta}$ has a more stable variance over the range of $\hat{\theta}$ and makes the symmetrical relationship between P and Q and between Q and P evident by a change of sign. Replicated experiments, with estimates in each replicate or block, provide an empirical estimate of the variance of $\hat{\theta}$. Consequently, general disease resistance of cultivars may be compared through an analysis of variance for $\log \hat{\theta}$, provided the range of θ is moderate, followed by a multiple comparison procedure such as the protected LSD, Waller-Duncan multiple range test, or Dunnett's procedure (7). The inverse sine transformation of $A(\hat{\theta})$ is expected to be better for comparing cultivars widely different in their resistance, say with values of θ outside the range of (1/8, 8).

When cultivars are compared in experiments repeated over years, the estimated value of θ should be more stable than ADPC due to its definition, provided that the pathogenic races are similar each year. Instability may indicate host resistance to only some of the races present. Provided that the estimates are similar over years, a combined estimate of general resistance may be provided by the mean.

RESULTS AND DISCUSSION

Field plots with three or four blocks of 22, 29, 35, and 22 potato cultivar/seedling treatments (originating from the Fredericton Potato Breeding Program, N.B., Canada) were established during 1980–1983, inclusive, as described previously by Platt and Tai (6). Plants of a susceptible cultivar (Green Mountain), planted between each treatment, were inoculated on 20–24 July, each year, with a race complex sporangial suspension (races: 1–4, 6, and 9) of *P. infestans* (approximately 5,000 sporangia per milliliter). Visual estimates of the amount of diseased leaf and stem tissue, as a percentage of the total surface area, were recorded at 3–7-day intervals during August and September. The trials did not have multiple plots of the standard nor was the progress of the disease completely monitored; these data do not conform to conditions *a* and *b* for proper estimation of θ. Monitoring was incomplete in 1981 due to severe disease symptoms occurring over a short period (3 or 4 days); the diseased tissue averaged 46% at the first assessment and the maximum was 97%. The data are used here to illustrate the general approach and its robustness under differing experimental conditions, rather than for an evaluation of the cultivars.

Mean percentages of diseased tissue for individual cultivars in 1983 are given in Table 1. Sebago was chosen as the standard cultivar. Cultivars were widely different in their resistance to late blight; the range was beyond that which could be expected to be referenced by a single standard cultivar for all maturity classes, but the example shows the effect of stretching assumptions beyond reasonable limits. ADPCs were calculated for cultivars in all blocks by

$$\Sigma_{i=1}^{n-1}(P(t_{i+1}) + P(t_i)) \times (t_{i+1} - t_i)/2 \quad (9)$$

in which t_i = time in days, $i = 1 \ldots n$, and $P(t_i)$ = percentage of diseased tissue. Mean DPCs are given for five cultivars in Figure 1 (71086, 75072, Russet Burbank, 74123, and Sebago). Equation 3 would require a common starting point and the overall slope of the DPC would be less for small θ. The curves need not be sigmoid, but curves that intersect violate equation 3. The shape of the curve for cultivar 74123 indicates nonproportionality and suggests an initial time lag with a slope similar to Sebago after the onset of disease.

Plots of the logarithms of the proportion of healthy tissue for each cultivar versus Sebago, over time, indicate a linear relationship through the origin except for cultivar 74123 (Fig. 5). Plots of observed $P(t_i)$ for the individual cultivars versus $Q(t_i)$ and the fitted lines based on the geometric mean of $\hat{\theta}$ (i.e., back transformed mean $\log \hat{\theta}$) are given in Figure 6. The fitted line for cultivar 74123 is above the first eight observed values and below the last five, indicating a departure from the assumed functional relationship with Sebago.

The values in Table 1 may be used to illustrate the calculations for the nonproportionality diagnostic. For cultivar 74123 relative to cultivar Sebago, $\hat{\theta} = 0.0939$ from equation 5 and $q' = 0.8413$ from equation 8. From equation 1, $\hat{U}_1 = 0.0220$ and $\hat{A}_2 = 0.0845$, whereas $U_1(\hat{\theta}) = 0.0492$ and $\hat{A}_2(\hat{\theta}) = 0.1220$, from equations 7a and 7b, are derived by splitting the summation (and interpolating about q') or integration into two intervals, $[0, q']$ and $(q', 1]$.

Thus $\hat{U}_1 + \hat{A}_2 = 0.1065$, $U_1(\hat{\theta}) + A_2(\hat{\theta}) = 0.1712$, and

$$D = \log(0.1065/0.8935) - \log(0.1712/0.8288) = -0.550$$

The negative sign indicates slower initial development than given by the fitted curve and the magnitude of D would have to be compared with the standard error of 0.130, from the analysis of variance table, indicating statistical significance ($P < 0.001$).

Mean values for $\log \hat{\theta}$, ASDPC, and relative ADPCs are given in Table 2 for those cultivars that were included in two or more of

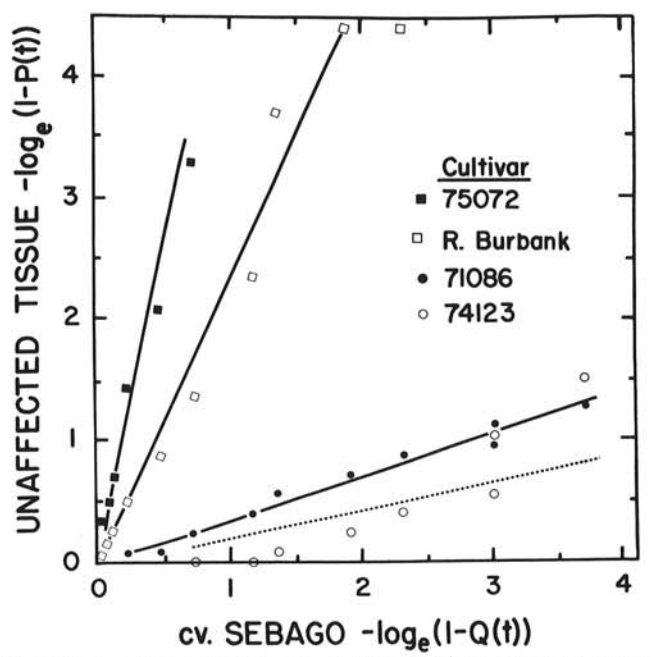

Fig. 5. Plots of the negative of the logarithm of the proportion of healthy tissue in the test cultivars vs. that in the standard cultivar, Sebago.

the yearly trials. Severity of blight was generally greater earlier in the season in 1981 and 1983 than in 1980 and 1982, as indicated by the ADPC of Sebago, except for cultivars 74123, Bison, and Kennebec. Because of the difference in relative ADPCs and

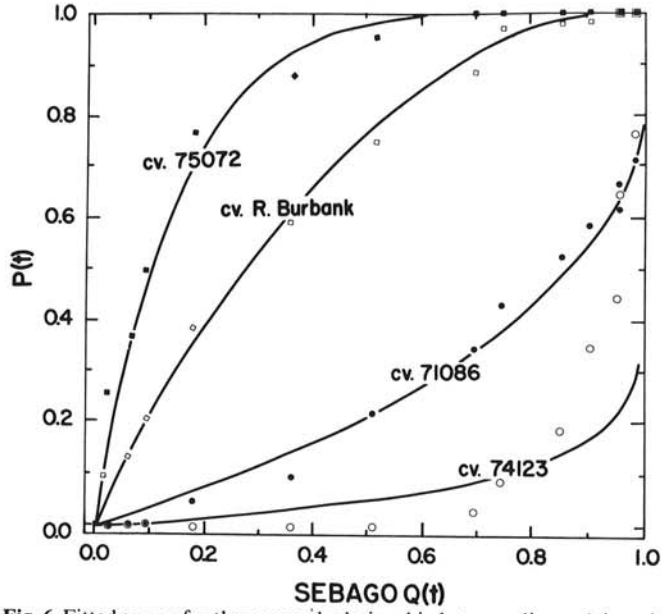

Fig. 6. Fitted curves for the assumed relationship between diseased tissue in the test cultivars and that in the standard cultivar, Sebago.

differing cultivars tested in each year, it was not readily apparent how the individual estimates should be combined into one. Estimates for log $\hat{\theta}$ and ASDPC are generally stable, except for the same three cultivars noted above, and may be averaged. The year-to-year deviations for the three cultivars (noted above) may be due to differences in genetic resistance to the specific races of *P. infestans* present in individual years. This instability of a few cultivars indicates the need for multi-year assessment trials and the necessity for identifying those cultivars that are unstable and determining the cause.

Separation by multiple range procedures between cultivars for general resistance to late blight is similar for both ADPC and ASDPC based on the F values, except in 1981. The SEM for ASDPC was greatest in 1981 due to the severely incomplete monitoring, resulting in a reduced F value, but the estimated values were consistent except for cultivar 74123 and Dorita. The generally greater SEM for the ASDPC than the relative ADPC (approximately by a factor of $\sqrt{2}$) was expected because it is based on differences between two cultivars; multiple plots for the standard cultivar would reduce this differential. But the range of cultivar values, on the angular scale, was expanded with ASDPC over that for the relative ADPC, resulting in similar separation among cultivars. The variance of log $\hat{\theta}$ generally increased with its magnitude, so that with the widely different cultivars in 1982 and 1983 the statistical separation among cultivars was reduced. The inverse sine transformation was more effective in stabilizing the residual variance.

The advantage of log $\hat{\theta}$ and ASDPC over the relative ADPC is due to their clear definition, their greater stability over years, and

TABLE 2. Mean values for $\log_e \hat{\theta}$ and area under the disease progress curves for potatoes affected by late blight in cultivar evaluation trials conducted at Charlottetown, P.E.I. over 4 yr, four, three, three, and four replicates and with 13, 11, 6, and 9 observation times in 1983, 1982, 1981, and 1980, respectively. Cultivars with values in italics show evidence of log (% healthy tissue) not being proportional with that for Sebago

Cultivar	Maturity rating[a]	Log. $\hat{\theta}$ (Sebago as standard)					Standardized ADPC (angles)				Relative ADPC (angles)			
		1983	1982	1981	1980	Mean	1983	1982	1981	1980	1983	1982	1981	1980
76036	E	2.52		2.09		2.31	74.1		70.0		64.6		68.7	
75072	E	1.81	2.01	*2.42*		2.08	67.8	69.9	*72.8*		61.0	47.0	69.6	
72090	E			1.98	2.04	2.01			69.2	70.1			68.3	56.0
76057	E	2.04	1.89	*2.07*		2.00	70.0	68.5	*70.0*		61.0	48.1	68.5	
75040	M	1.66	2.01			1.84	66.3	69.7			59.0	48.1		
75009	E/M			1.65	1.89	1.77			64.9	68.5			65.8	56.1
74064	E/M			1.49	1.54	1.52			64.4	65.1			64.9	51.3
74094	M	1.08	1.32	*1.72*		1.37	59.7	62.5	*67.0*		56.2	40.2	67.1	
74047	M		1.28	1.37		1.33		62.0	63.1			38.2	64.3	
R. Burbank	L	0.77	1.49			1.13	55.7	64.5			53.5	41.7		
Bintje	(M)	0.86	1.25	1.18		1.10	57.0	61.8	60.8		54.2	40.7	63.8	
74103	E			1.13	1.00	1.07			59.9	58.6			62.7	45.8
75111	E		0.81	0.93	1.35	1.03		56.2	57.9	63.0		34.9	60.3	48.6
74060	M/L			0.83	1.20	1.02			56.5	61.2			60.9	48.5
76079	M	0.78	0.95	0.94		0.89	55.8	58.0	57.9		53.3	35.4	61.2	
73099	M			0.65	1.11	0.88			53.8	60.0			52.8	46.6
G. Mountain	L	0.48	1.19	0.85	0.63	0.79	51.8	61.1	56.8	53.7	51.4	40.0	60.0	43.1
74043	M			*0.33*	*1.11*	0.72			*49.4*	*60.0*			56.3	48.6
76076	M	0.71		0.73		0.72	55.0		55.1		52.5		59.5	
75135	M			*0.16*	0.70	0.43			*47.2*	54.8			54.2	43.8
74016	M			0.63	0.20	0.42			53.9	47.8			58.9	37.6
75114	L			0.25	0.35	0.30			48.5	50.0			54.8	37.7
73068	L			0.12	0.08	0.10			46.7	46.2			53.6	37.7
Sebago	L	0.00	0.00	0.00	0.00	0.00	(45.0)	(45.0)	(45.0)	(45.0)	(46.0)	(26.7)	(51.2)	(36.4)
Bison[b]	M	*−2.24*	1.63			−0.31	*18.3*	65.3			25.8	44.3		
Libertas	(M)		−0.86	−0.02		−0.44		33.3	44.7			18.4	49.8	
74123[b]	M	*−2.93*	1.67	*−1.76*	*0.83*	−0.55	*16.4*	66.5	*22.6*	*56.5*	24.3	44.1	34.8	45.8
Kennebec[b]	M	*−3.07*	0.98			−1.05	*12.4*	58.4			17.2	36.3		
71086	M	−1.05	−0.71	−1.54		−1.10	30.6	35.1	25.0		33.4	20.3	31.8	
7703-1	(M)	*−3.04*	*−3.73*			−3.38	*12.5*	*9.0*			17.3	5.2		
Dorita	(M)	−3.14	−3.39	−4.42		−3.65	12.4	10.6	6.3		15.6	5.5	7.7	
73008	L	*−6.88*	*−4.35*	−2.94	*−5.38*	−4.89	*1.9*	*6.7*	13.0	*4.0*	4.0	3.4	17.7	3.3
7670-7	(M)	−5.16	*−5.01*			−5.09	4.5	*4.9*			8.0	2.4		
AM6642	(M)	−5.68	*−4.98*			−5.33	3.7	*5.0*			5.1	2.7		
SEM		0.237	0.270	0.240	0.118		1.48	2.05	2.66	1.48	1.11	1.69	1.75	1.19
df		54	38	50	42		54	38	50	42	54	38	50	42
F value		145	90.0	43.5	219		330	150	43.2	113	387	103	80.5	110

[a] Maturity ratings: E = early, M = mid-season, L = late with presumed ratings in parentheses.
[b] Cultivars with inconsistent responses relative to Sebago over years.

the resulting combined-years' estimate of general resistance to infection. Whereas the proposed method appeared to be useful for indexing general resistance of potato cultivars to late blight, the proportionality assumption was often untenable when the magnitude of log $\hat{\theta}$ was greater than 2.1, i.e., $\theta > 8$ or $\theta < 1/8$, indicating that an additional parameter would be required for an adequate model (Table 2). A time delay parameter, or a change in rate parameter, might be considered. When the proposed index is used with widely differing cultivars for which the proportionality assumption is untenable, such as in our example, $\hat{\theta}$ will represent an averaged rate of disease progress rather than a single constant independent of time. The additional parameter would subdivide the groups further, perhaps grouping the generally resistant cultivars according to the degree that they delayed the onset of disease symptoms or changed the standardized rate of disease progress.

The advantage of ASDPC is expected to be increased with suitably designed experiments. A more refined procedure should be possible, particularly if the proportionality assumption is tenable for all cultivars on trial, and the underlying disease progression can be estimated from all cultivars, rather than from a single standard cultivar as proposed in this paper. More complex models might be considered including those with a lag phase before the onset of disease or changes in the standardized rate of disease progress.

Further application of the technique is required to ascertain its usefulness and its limitations, for both cultivar assessment and for fungicide control. Also, its relationship to the concepts of vertical and horizontal resistance in cultivars should be explored.

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