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Comparing different epidemiological models in field evaluations of selected genotypes from *Solanum tuberosum* CIP population A for resistance to *Phytophthora infestans* (Mont.) De Bary in Kenya

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Summary

Late blight caused by *Phytophthora infestans* was monitored in field plots of potato genotypes selected from population A of the International Potato Center (CIP) germplasm collection. Disease severity was measured as percent blighted leaf area and used to compute area under disease progress curves (AUDPC), apparent infection rates (r) and severity at epidemic onset (Yo). AUDPCs revealed more distinct differences among the genotypes than any other disease assessment parameter. Percent disease severity measured 67–77 days after planting (D_{67-77}) explained more variation in AUDPCs than measurements made on any other single day. Increase in percent diseased leaves fit the monomolecular model more closely than the Gompertz, logistic or exponential model. All disease assessment parameters varied among the genotypes and were significantly ($p < 0.01$) correlated with each other. Genotypes with larger AUDPCs generally had higher DS_{67-77} and faster rates of disease increase (r). Clones 386191.7 and 381403.23 were more susceptible to late blight than all other entries tested. The lowest disease levels were observed on clone 382155.2. Frequency distribution of AUDPCs among genotypes appeared continuous and did not differ significantly ($p < 0.05$) from normal distribution suggesting the observed resistance may be attributable to minor genes.

Introduction

Late blight caused by *Phytophthora infestans* (Mont.) De Bary is one of the most devastating diseases of potato (*Solanum tuberosum* L.) world-wide (Hooker, 1981). It occurs wherever potato is grown and can cause tuber yield losses of up to 100% in susceptible germplasm if left uncontrolled (Henfling, 1987). Use of fungicides is the most common method employed for late blight control (Vanbruggen et al., 1986). However, the major limitations of this strategy include the high expenses and serious environmental and health risks associated, particularly in developing countries where potato is grown mostly by economically challenged and poorly educated small-scale producers. Moreover, recent changes in the population structure of the late blight fungus in many countries have led to the advent of new races that are more aggressive and resistant to previously effective fungicides (Fry et al., 1993). Thus, alternative strategies such as use of disease resistance need to receive greater attention for future integrated disease management programs for late blight.

Resistance to late blight varies both qualitatively and quantitatively (Gees & Hohl, 1988). Although qualitative (race specific, vertical or monogenic) resistance to late blight can be easy to identify and relatively simple to transfer through breeding, only temporary exploitation of this resistance is possible because it is readily overcome by evolution of new physiologic pathotypes that are virulent and more aggressive to the existing qualitative genes (Tooley et

al., 1986). Quantitative (horizontal, partial or polygenic) resistance on the other hand tends to be more durable and stable (Forbes & Jarvis, 1994). Many potato genotypes with quantitative resistance to late blight do not posses sufficient resistance to preclude use of fungicides. However, such germplasm may be used to initiate breeding programs targeted at increasing the level of resistance in agronomically acceptable cultivars. Also, they may allow modification of conventional fungicide application regimes to reduce amounts and frequency of fungicide use. This partially explains why the International Potato Center (CIP) has undertaken research that has led to the development of population A which has more than 169,000 potato genotypes. This population which segregates for quantitative resistance to late blight is composed of genotypes that express horizontal resistance (Anon., 1995). Because quantitative resistance is greatly affected by environment and host physiology variations (Umaerus, 1970), population A genotypes are presently being evaluated in several environments world-wide to test their stability over diverse ecological zones. At the Kenya regional center of CIP, some genotypes from the population A have been selected for adaptability to the local conditions as well as superior tuber yield and quality. This study assessed a sample of the selected genotypes and our objectives were: 1) To identify genotypes with acceptable levels of resistance to late blight, 2) to determine the critical period for taking late blight severity measurements and 3) to select a suitable model for transforming late blight severity data when estimating rates of disease progress for epidemiological studies.

Materials and methods

Experiments were conducted at the Kenya Breweries Experimental Station at Mau Narok, Kenya in the first (April-August) and second (September-December) rain seasons of 1995. Trials were conducted in fields planted with barley *(Hordeum vulgare* L.) in the previous year as part of a 2-year barley-rapeseed (*Brassica* spp.) rotation. Potato is often severely attacked by late blight throughout the year in this region so no artificial inoculation was necessary. Plots consisted of 3 m rows spaced 0.75 m apart and at a planting distance of 0.25 m within rows. Each row received 100g of Diammonium phosphate (DAP) fertilizer and 5 g of furadan (nematicide) at planting. Standard cultural practices for cultivation of potato were followed but no fungicides were applied for late blight control. A total of 31 genotypes from population A germplasm were evaluated in this study.

Plots were arranged in a randomized complete block design with 3 replications. Initial infection was allowed to start from natural inoculum. Entire plots were assessed for percent damaged (i.e. blighted or defoliated) leaf area beginning from the time when 10% leaf damage was noticed on the most severely attacked clone. Subsequent disease severity measurements were taken at 10 days intervals to give a total of 5 readings. Area under disease progress curve (AUDPC) values were calculated from percent disease severity values using the following equation (Shaner & Finney, 1977).

$$\text{AUDPC} = \sum_{i=1}^{n} [(Y_i + Y_{i+1})/2](X_{i+1} - X_i)$$

in which Y_i is percent blighted leaf area on the ith observation, X_i is date of observation in days after planting and n is the number of disease severity readings taken. A stepwise regression procedure was used to determine which disease severity observations were most influential in the development of AUDPCs. Percent disease severity data were also fitted to linearized forms of the Gompertz, logistic, monomolecular and exponential models using the following equations (Campbell & Madden, 1990):

Gompertz	: -ln [-ln (Y/Y_{max})]
Logistic	: ln [$Y/(Y_{max}$-$Y)$]
Monomolecular	: ln [Y_{max} / (Y_{max} - Y)]
Exponential	: ln (Y)

where Y_{max} is the maximum percent disease severity (assumed to be 100% for all genotypes in this study) and Y is observed percent disease severity. Rates of disease progress expressed as apparent infection rates (r) and epidemic at disease onset (Y_o) were estimated from the slope and intercept of simple linear regression of transformed disease severity data against time in days after planting. The most suitable model for assessing infection rates was determined using standardized residual plots, coefficient of determination (R^2) and additional statistics as described by Neter et al. (1983). It is not possible to compare the values of R^2 obtained from the models directly and so the transformed values of disease were detransformed to calculate new values of R^2 (Hau & Kranz, 1977). After choosing the most suitable model, autocorrela-

Table 1. Area under disease progress curve (AUDPC), percent disease severity measured 67 (DS_{67}) and 77 (DS_{77}) days after planting, slope (r), intercept (Y_o) for regression of data transformed by monomolecular model on time, among potato genotypes from CIP population A affected by late blight in Kenya during season 1 (April-August) and season 2 (September-December), 1995

	Season 1[b]				Season 2			
Clone	AUDPC	DS_{67}[c]	r	Y_o	AUDPC	DS_{77}[c]	r	Y_o
385283.12	...	...	...	...	3638*a*	99*a*	0.080*d-f*	–1.933*a-e*
387561.10	...	...	...	...	3592*a*	75*c-f*	0.087*c-f*	–2.667*b-e*
386191.7	3355*a*	87*a-c*	0.082*b-e*	–3.293*a-e*	3385*a-c*	85*a-e*	0.105*a-d*	–4.579*f-h*
381403.23	3327*a*	90*ab*	0.081*b-f*	–3.307*b-e*	3203*b-d*	85*a-e*	0.106*a-d*	–5.111*g-i*
384651.2	3308*ab*	85*a-d*	0.097*a-c*	–4.165*c-f*	3417*ab*	77*b-f*	0.109*a-c*	–4.374*f-h*
387867.4	3262*a-c*	83*a-d*	0.079*b-g*	–3.296*a-d*	3198*e*	72*d-g*	0.080*d-f*	–3.456*d-f*
385261.9	3252*a-d*	87*a-c*	0.104*ab*	–4.904*ef*	3080*b-e*	87*a-d*	0.128*ab*	–6.379*i*
382171.4	3208*a-d*	87*a-c*	0.062*b-h*	–2.323*a-d*	3069*c-e*	85*a-e*	0.106*a-d*	–5.226*g-i*
389576.12	3193*a-d*	85*a-d*	0.085*b-e*	–3.707*b-e*	3014*de*	90*a-c*	0.096*c-e*	–4.688*f-h*
387711.5	3191*a-d*	85*a-d*	0.097*a-c*	–4.165*d-f*	2975*de*	92*ab*	0.132*a*	–0.970*ab*
387967.2	3183*a-d*	85*a-d*	0.071*b-h*	–2.976*a-e*	2771*ef*	85*a-e*	0.066*fg*	–3.109*c-f*
389561.12	3142*a-e*	83*a-d*	0.079*b-g*	–2.940*a-e*	2990*ed*	70*e-g*	0.103*b-d*	–5.140*g-i*
385261.3	3098*a-f*	85*a-d*	0.051*d-h*	–1.798*a-c*	2806*ef*	73*d-f*	0.108*a-c*	–5.571*hi*
383028.2	3080*a-f*	92*a*	0.128*a*	–6.379*f*	2525*fg*	77*c-f*	0.069*e-g*	–3.456*e-g*
385191.13	3033*a-f*	77*a-e*	0.069*b-h*	–3.02*a-e*	2500*fg*	87*a-d*	0.031*h*	–1.115*ab*
381382.34	2996*a-f*	78*a-e*	0.063*b-h*	–2.672*a-e*	2328*g-i*	87*a-d*	0.044*gh*	–2.081*a-e*
381390.20	2990*a-f*	75*c-f*	0.070*b-h*	–3.080*a-e*	2367*gh*	85*a-e*	0.050*gh*	–2.423*a-e*
387382.21	2973*a-f*	85*a-d*	0.068*b-h*	–3.037*a-e*	2301*g-j*	78*b-f*	0.048*gh*	–2.328*a-e*
389582.25	2926*b-g*	73*c-f*	0.056*c-h*	–2.311*a-d*	2245*g-k*	72*d-g*	0.031*h*	–1.306*ab*
386191.15	2906*c-g*	72*c-f*	0.062*b-g*	–2.719*a-e*	2101*h-k*	83*a-e*	0.035*h*	–1.608*a-c*
382127.14	2868*d-g*	77*a-e*	0.058*c-h*	–2.445*a-e*	2154*h-k*	47*ij*	0.0327*h*	–1.419*a-c*
382119.13	2771*e-g*	70*d-f*	0.066*b-h*	–3.109*a-e*	2052*h-l*	53*hi*	0.022*h*	–0.865*a*
390013.1	2756*gf*	72*c-f*	0.035*h*	–1.115*a*	2078*h-l*	55*hi*	0.033*h*	–1.544*a-c*
385261.13	2605*gh*	63*e-f*	0.034*h*	–1.167*ab*	2005*i-m*	53*hi*	0.035*h*	–1.710*a-c*
386295.1	2399*hi*	58*f-h*	0.042*e-h*	–1.929*a-d*	1921*k-n*	35*j*	0.045g*h*	–2.379*a-e*
375080.43	2278*h-j*	55*gh*	0.038*f-h*	–1.710*a-c*	1951*j-n*	63*f-h*	0.036*h*	–1.703*a-c*
389497.1	2269*h-j*	53*gh*	0.036*hg*	–1.597*ab*	1903*k-n*	58*g-i*	0.029*h*	–1.371*abc*
KP90101.1	2165*ij*	53*gh*	0.031*h*	–1.294*ab*	1967*j-m*	68*d-f*	0.037*h*	–1.801*a-d*
387973.23	2155*ij*	47*hi*	0.033*h*	–1.439*ab*	1678*mn*	52*hi*	0.022*h*	–6.677*i*
720097	2154*ij*	57*gh*	0.033*h*	–1.419*ab*	1729*l-n*	65*ef*	0.033*h*	–1.685*a-c*
382155.2	1997*j*	35*i*	0.044*e-h*	–2.250*a-d*	1609*n*	91*ab*	0.027*h*	–1.314*ab*

Genotypes are ranked in order of decreasing AUDPC values in season 1.

[a] Each value is the mean over 3 replications. Values in a column followed by a common letter are not statistically different ($p = 0.05$) as determined by Duncan's multiple range test.

[b] Two genotypes were not included in the analysis in season 1 because the plants were killed by blight less than 3 weeks after planting.

[c] DS_{67} and DS_{77} explained by the greatest amount of variation in AUDPCs in season 1 and season 2 respectively.

tion of residuals as described by Madden (1986) and students *t*-test were used to determine whether the autocorrelation was different from zero. Disease severity parameters were examined by analysis of variance and significant differences among genotypes discerned by Duncan's multiple range test. Correlation coefficients were calculated to evaluate relationships among parameters. Chi-square analysis was conducted on observed AUDPC data to determine conformity to normal distribution. Frequency histograms for AUDPCs were examined to determine whether distributions were continuous or formed discrete groups. Combined analysis of AUDPCs over seasons was conducted to examine the effect of season by clone interaction. As an essential prerequisite for this combined analysis, a

Table 2. Spearman's rank correlation coefficients among disease assessment parameters used to quantify late blight reactions in CIP population A potato genotypes naturally infected by *Phytophthora infestans* in Kenya in season 1 (April-August) and season 2 (September-December) of 1995[a]

	Season 1				Season 2			
Clone	AUDPC[b]	DS_{67}[b]	r[b]	Y_o[b]	AUDPC	DS_{77}[b]	r	Y_o
Season 1								
AUDPC		0.91***	0.85***	–0.77***				
DS_{67}			0.82***	–0.75***				
r				–0.97***				
Season 2								
AUDPC	0.99***					0.53*	0.84***	–0.52*
DS_{77}		0.53*					0.48**	–0.11[ns]
r			0.81***					–0.61**
Y_o				0.24[ns]				

[a] Analysis performed using only the 29 genotypes for which data was available in both seasons.
[b] Area under disease progress curve (AUDPC), percent disease severity measured 67 days (DS_{67}) and 77 days (DS_{77}) after planting, slope (r), and intercept (Y_o) obtained by regression of data transformed by monomolecular model on time in days after planting.
[c] Values followed by ns are not statistically significant and those followed by asterisks (*,** and ***) indicate significant test at p = 0.05, 0.01 and 0.001 respectively.

F-test was conducted and it revealed no heterogeneity of plot errors.

Results

Epidemics of late blight occurred in both seasons of the study but disease was more severe in the first season than in the second season (Table 1). There were significant ($p < 0.05$) differences among genotypes for all disease measurement parameters studied (Tables 1 and 3). The monomolecular model generally produced slightly higher coefficient of determination (R^2 = 0.97), smaller error mean squares (EMS = 0.13) and more random plots of standardized residuals versus predicted values than the Gompertz (R^2 = 0.92, EMS = 0.29), logistic (R^2 = 0.89, EMS = 0.21) and exponential (R^2 = 0.87, EMS = 0.24) models. Goodness of fit of the models to disease progress data, however, appeared to vary from one clone to another. Coefficients of determination were generally higher, error mean squares smaller and residual plots more random for susceptible than for resistant genotypes.

Correlations among all disease assessment parameters were highly significant ($p < 0.01$) (Table 2). With the exception of Y_o, ranking of genotypes for resistance was fairly consistent for all disease parameters (Table 1). In some genotypes, a high AUDPC was not associated with a high r, or a high DS_{67} or DS_{77}. However, AUDPCs discerned the greatest number of differences among the genotypes and explained the greatest amount of variation among the genotypes in the two seasons (Tables 1 and 3). Therefore, only AUDPCs were used for further examination of the data. Clone 382155.2 exhibited the smallest AUDPC while 386191.7 and 381403.23 had the largest AUDPC. The individual estimates of percent disease severity that explained the greatest proportion of the variation in AUDPC were those made 67 (DS_{67}) and 77 (DS_{77}) days after planting in the first and second season, respectively.

Frequency histograms of AUDPC values appeared continuous in the two seasons. Chi-square comparison of each AUDPC distribution with a bimodal normal distribution did not indicate statistically significant deviation from normality although there appeared to be two peaks in the frequency distributions in both seasons (Figures 1 and 2). Combined analysis of AUDPCs over the seasons revealed significant effect of season by clone interaction (Table 3). In the first season the frequency distribution appeared to be skewed towards susceptibility whereas in the second season the skew was more towards resistance (Figures 1 and 2).

Discussion

Reactions of genotypes in population A to late blight have been previously documented elsewhere (Landeo

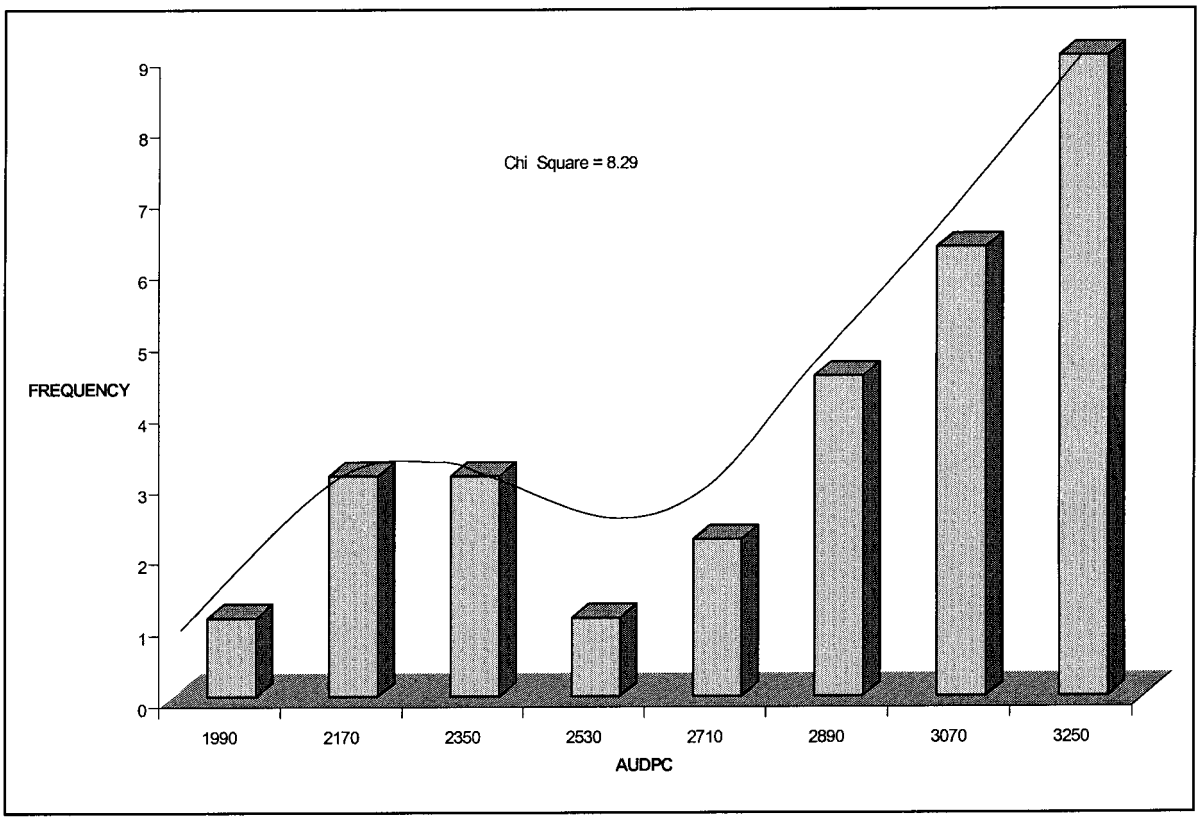

Figure 1. Frequency histograms and distribution of AUDPC values for assessment of resistance to *P. infestans* in CIP population A potato genotypes in Kenya in season 1 of 1995. The Chi square value for an attempted fit to a bimodal normal distribution did not indicate a significant deviation from normality.

et al., 1997) but no published information was available on the response of these genotypes under Kenyan conditions. In addition, no standard checks were available for comparing field reactions of these genotypes in Kenya. This study identified genotypes 381403.23 and 386191.7 as the most susceptible and clone 382155.2 as the most resistant. We recommend these genotypes for use as standard checks for future evaluation of potato genotypes for resistance to late blight in this region. Results obtained from this study could also be used to select genotypes for further studies on the genetics of the observed resistance.

Resistance was generally expressed as smaller AUDPC, lower DS_{67-77}, and smaller r values. However, ranking of genotypes into discrete resistance groups using r, Y_o or DS_{67-77} was not as effective as AUDPCs. This may be due to the lesser precision of r and Y_o as these parameters were computed from averages of transformed percent disease severity values over the entire disease assessment duration. Alternatively, it may be because r and Y_o are under the control of independent genetic factors from AUDPC. In contrast, AUDPC values were computed from averages of percent disease data over 10 day intervals and were therefore more accurate descriptors of disease severity (Johnson et al., 1986). The imperfect fit problems observed in this study may be avoided by using more mathematically explicit models such as the Weibull model or Richard's model (Campbell & Madden, 1990). However, these models may not be useful for evaluating very many genotypes because of the elaborate computational procedures they involve. AUDPC values on the other hand avoid problems associated with imperfect fit of data to statistical models and are relatively easy to calculate. Studies conducted elsewhere have also found AUDPC to be a more practical tool for measuring plant disease resistance (Zeiders & Hill, 1988; Wang et al., 1989).

Visually observing disease severity on any single day was not as effective as AUDPCs but DS_{67-77}

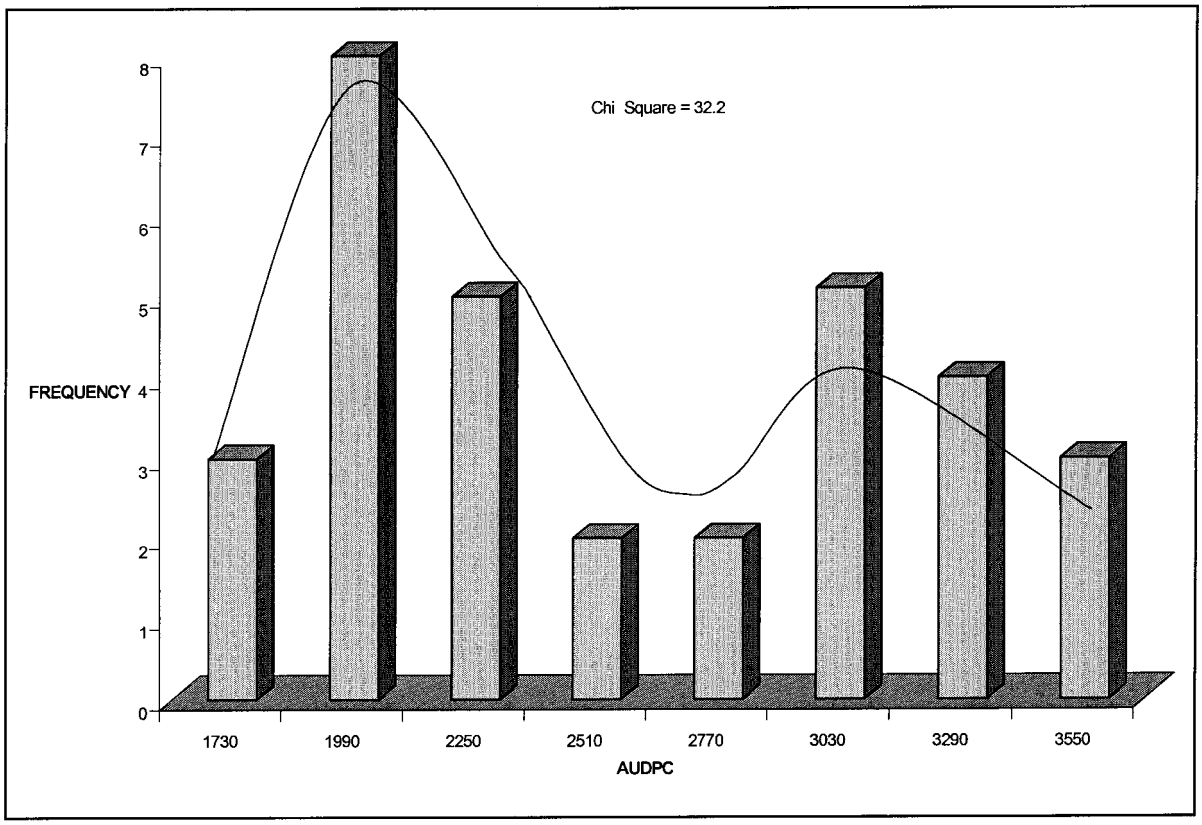

Figure 2. Frequency histograms and distribution of AUDPC values for assessment of resistance to *P. infestans* in CIP population A potato genotypes in Kenya in season 2 of 1995. The Chi square value for an attempted fit to a bimodal normal distribution did not indicate a significant deviation from normality.

ranked genotypes fairly consistently with AUDPCs. There was also high and significant correlation between DS_{67-77} and AUDPC. Due to the great amount of labor and time required for determination of AUDPCs, we recommend visual scoring for a large number of entries during the preliminary stages of evaluation and use of AUDPC only when detection of small differences among the genotypes under evaluation is important.

The apparent continuity in the distribution of AUDPC data suggests that the observed resistance maybe attributable to minor genes. Canizares & Forbes (1995) made similar conclusions in their study of the reaction of *Solanum phureja* sub-species *phureja* Juz and Buk to *Phytophthora infestans*. Evidence for minor genes resistance in our results is further supported by the lack of significant deviation from normality of an attempted fit of disease severity data to normal distribution and the significant effects of season by genotype (i.e. genotype × environment) interaction. Nonetheless, because we did not investigate the nature of inheritance of resistance in this study and since the genotypes used in the study were not derived from single crosses, we recommend genetic experiments to confirm the absence of R-genes for minor gene resistance to late blight among these genotypes. It is, in fact, highly likely that both major and minor genes for resistance to late blight are present in the genotypes tested, as could be inferred from the formation of two distinct peaks in the frequency distribution of AUDPC values.

The monomolecular model gave the best fit to disease severity data out of the four transformation models tested. This was unexpected considering that late blight is a polycyclic ('compound interest') disease. One of the implicit assumptions for application of the monomolecular model in plant disease epidemiology is that the disease has a monocyclic ('simple interest') pattern of development (Vanderplank, 1963). However, the shapes of disease progress curves are af-

Table 3. Analysis of variance for area under disease progress curve (AUDPC), percent disease severity measured 67 days (DS_{67}) and 77 days (DS_{77}) after planting, slope (r), and intercept (Y_o) obtained by regression of data transformed by monomolecular model on time in days after planting

		AUDPC		DS_{67} or DS_{77}[a]		r		Y_o	
	DF	MS	F	MS	F	MS	F	MS	F
Season 1									
Clone	28	524920	13.67**[b]	679.5	9.87**	0.001765	3.69**	4.3121	2.69**
Block	2	90121	2.37[ns]	146.8	2.13[ns]	0.000181	0.38[ns]	1.5610	0.97[ns]
Error	56	38402		68.9		0.000478		1.6022	
R^2		0.87				0.83		0.65	0.58
CV (%)		6.85				11.31		34.46	–46.02
Season 2									
Clone	30	1101072	31.48**	658.2	25.04**	0.0037067	16.35**	8.9946	11.20**
Block	2	55475	1.59[ns]	9.7	0.37[ns]	0.0006300	2.78[ns]	2.2665	2.82[ns]
Error	60	34973		26.2		0.0002267		0.8030	
R^2		0.94		0.90		0.93		0.89	0.85
CV (%)		7.38		8.99		6.44		23.73	–30.83
Combined[c]									
Season (S)	1	6981617	95.90[d]						
Blocks within season	4	12798							
Clone (C)	28	1330269	35.06**						
C × S	28	93010	2.45**						
Pooled error	112	37937							

[a] Disease severity 67 (DS_{67}) and 77 (DS_{77}) days after planting for season 1 and 2, respectively.
[b] F values followed by ns indicate non significant tests and asterisks (* and ** indicate significant test at p = 0.05 and 0.01, respectively.
[c] Combined analysis conducted on only AUDPC data because it explained more of the variation and revealed more differences among genotypes than the other disease assessment parameters.

fected not only by the number of infection cycles and inoculum production per season's epidemic but also by changes in weather conditions during the course of disease development. There may have been weather effects in our study to sufficiently alter the shape of the disease progress curves from the expected sigmoid shape typical of polycyclic diseases. Thus, it may be unreliable to choose a disease progress transformation model based on the intuitive biological meaning of the curves they represent. In some pathosystems, the Gompertz model has been found to be most suitable for disease severity transformation (Johnson et al., 1986; Neher & Campbell (1992)) while in others, the logistic model is more appropriate (Adipala et al., 1994).

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