Effect of Disease Assessment Method on Ranking Potato Cultivars for Resistance to Early Blight

B. J. CHRIST, Assistant Professor, Department of Plant Pathology, The Pennsylvania State University, University Park 16802

ABSTRACT

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Significant differences among several potato cultivars (*Solanum tuberosum*) were observed for disease reaction to early blight, caused by *Alternaria solani*, in 1985, 1986, and 1988 field trials and in a greenhouse in 1986. Over the 3 yr, several assessment methods were used to characterize and categorize the disease reaction. Percentage of area of leaves from the middle third of the plant canopy that was covered with early blight lesions, assessed over several dates, provided the most information as well as a feasible method of assessing disease reaction. Cultivar ranking varied according to the assessment method. Regardless of assessment method, the late-maturing cultivars Katahdin and Kennebec were more resistant to *A. solani* than the early-maturing cultivars Norland and Superior but not necessarily any more resistant than the midseason-maturing cultivars Atlantic and Chieftain.

Early blight of potato (*Solanum tuberosum* L.) caused by *Alternaria solani* Sorauer is the major foliar disease on potatoes in Pennsylvania. Fungicides are routinely applied initially when the plants are in bloom and are continued every 7–10 days during the growing season to control early blight (7). The number of fungicide applications could be reduced by growing resistant cultivars (4,5), but there is little information on resistance of potatoes to early blight.

Several researchers have screened potato genotypes or cultivars for resistance to early blight (1–3,6,8–13). Most of these researchers used different methods of assessing the disease reaction, and cultivars were compared for disease reaction on the basis of field assessments at the end of the growing season. Holley et al (12) made field assessments several times after the start of the early blight epidemic and described disease reaction to several cultivars based on apparent infection rates. They observed differences among cultivars and determined that there is rate-reducing resistance in potatoes to *A. solani*. More recently, Pelletier and Fry (14) characterized reactions of three cultivars based on components of resistance: incubation period, lesion expansion rate, and spore production. The ranking of these cultivars was explained by the differences in components of resistance, particularly incubation period and lesion expansion rate.

The objective of this study was to rank several potato cultivars under field and greenhouse conditions for resistance to *A. solani*.

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MATERIALS AND METHODS

Production of inoculum. An isolate of *A. solani* was obtained from naturally infected potatoes, maintained on V-8 juice agar, and grown at 21 C under cool-white fluorescent diurnal light with a 12-hr photoperiod. Six plugs of 7-day-old cultures were transferred into 10 ml of sterile distilled water, agitated, and poured onto water agar to induce sporulation. After 6 days of growth, the conidia were dislodged from the agar surface by repeatedly flooding the plates with sterile distilled water containing five drops of Tween 20 per 100 ml. The resultant suspension was poured through double layers of cheesecloth into a flask. The concentration of the conidial suspension was determined with a hemacytometer.

Cultural practices. Cultivars Belchip, Buckskin, Chieftain, Katahdin, Kennebec, Norchip, Norland, Penn 71, PA 1, Rosa, and Superior were evaluated in 1985. In 1986, cultivars Hampton and Monona replaced Belchip and PA 1, and in 1988, Buckskin, Penn 71, and Rosa were not tested because of limited supply of seed. Certified seed of each cultivar was cut into 57-g pieces and suberized by storing the cut seed at 18 C for 5 days. Seed was placed at 21 C 24 hr before planting.

Plots were established in fields previously planted in alfalfa. Fertilizer (10-10-10 N-P-K, 89 kg/ha) and aldicarb insecticide (Temik 15G, 5.0 kg a.i./ha) were applied in the furrow at planting. Seed pieces were planted by hand. The herbicides metribuzin (Sencor 50W, 0.56 kg a.i./ha) and metolachlor (Dual 8E, 2.02 L a.i./ha) were applied after planting but before plant emergence. During the growing season, the insecticide azinphos-methyl (Guthion 25, 0.77 L a.i./ha), oxamyl (Vydate 2L, 1.12 L a.i./ha), permethrin (Ambush 2E, 0.15 L a.i./ha), or fenvalerate (Pydrin 2.4 EC, 0.28 L a.i./ha) was applied when necessary. Plants were cultivated and hilled as necessary.

Field studies, 1985. Field plots were planted on 14 May. Each consisted of three rows 6.9 m long with 1.5-m breaks between plots within rows. Each row contained 30 seed pieces spaced 0.23 m apart, with 0.91 m between rows. The experimental design was a randomized complete block with each block replicated three times. On 28 July, the foliage of a single plant in the center row of each plot was sprayed with 10 ml of water containing 5×10^4 conidia per milliliter of *A. solani*.

The number of lesions on the inoculated plant was counted 8, 11, and 13 days after inoculation. Disease severity on the inoculated plant was also recorded on day 13, using a scale of 0–7, with 0 = no lesions, 1 = trace to 1%, 2 = 1–5%, 3 = 6–10%, 4 = 11–25%, 5 = 26–50%, 6 = 51–75%, and 7 = 76–100% of foliage covered with lesions. Disease severity among all plants in the plot was recorded 20 days after inoculation, using the 0–7 scale. Lesion count data were subjected to analysis of variance and mean separation tests (LSD). Severity data were subjected to nonparametric analysis of variance in NPARIWAY procedure (SAS, Cary, NC) and mean separation (LSD).

Field studies, 1986. Field plots were planted on 15 May. Each contained three rows 3.4 m long with 1.5-m breaks between plots within rows and 0.91 m between rows. Each row contained 15 seed pieces spaced 0.23 m apart. The experimental design was a randomized complete block with four replications. On 9 July, the foliage of a single plant in the center row was sprayed with 10 ml of a suspension of 1×10^5 conidia per milliliter.

Early blight was assessed four times during the growing season after inoculation. Each leaf on each of 10 randomly tagged stems surrounding the inoculated plant was assessed for percentage of leaf area covered by necrotic lesions and chlorosis by comparison with an illustrated key (15). Disease assessment for each stem started with the lowest leaf and proceeded up the stem.

Disease severity on an individual stem was calculated by averaging severities of either lesions or chlorosis over all leaves on a stem. Severity over all 10 stems per plot was calculated by averaging severities of lesions or chlorosis over the 10 stems. Mean disease severity for individual leaves based on location within the canopy was calculated by averaging severity of lesions or chlorosis over the 10 stems for each leaf location. From the severity data, percentage of infected leaves per stem and percentage of stems per plot were calculated. Area under the disease progress curve (AUDPC) (16) was calculated using the mean severity values per plot.

Analysis of variance was performed, and tests that were significant were subjected to mean separation (Waller-Duncan k-ratio t test, $k = 100$). Data of severity per leaf averaged over the 10 stems per plot and over replicates for each cultivar were fit to least-squares linear regression models. Early blight severity was regressed on leaf location, with the first leaf representing the oldest leaf located on the bottom of the plant and leaf 20 representing the youngest leaf with blight infection on the top of the stem. Comparisons were made among slopes and intercepts using Bonferroni t statistic.

Field studies, 1988. Field plots were planted on 31 May. Planting and experimental design followed those of 1986, except there were six replications and all plots were surrounded by one row of the cultivar Norland. On 26 July, a single plant of Norland adjacent to the center of an experimental plot was inoculated as described above.

Disease severity was assessed four times starting on 23 August and ending on 14 September. Ten main stems were randomly selected from the center row, and five leaves in the middle third of each stem were assessed for percentage of leaf area covered by early blight lesions as described above. Analysis of variance tests were performed on the data of individual assessments as well as AUDPC. Treatments were subjected to a mean separation test (Waller-Duncan k-ratio t test, $k = 100$).

Greenhouse studies. Seed pieces of the cultivars Atlantic, Belchip, Chieftain, Katahdin, Kennebec, Norchip, Norland, Rosa, Superior, Penn 71, PA 1, and Buckskin were grown in 13-cm-diameter plastic pots containing a steam-treated mixture of peat, perlite, and soil (1:1:1, v/v). Plants were grown at 24 ± 5 C under 12 hr of supplemental cool-white fluorescent lamps.

Three plants of each cultivar were inoculated by misting approximately 5 ml of a 1×10^6 conidia per milliliter suspension onto a single plant. Each plant was placed in a plastic bag in the dark for 24 hr to maintain a relative humidity of 100%. After 24 hr, the plants were placed in a walk-in polyvinyl chamber in the greenhouse to maintain a minimum of 80% relative humidity at all times.

The locations of lesions and lesion area were recorded for five lesions per plant four times over a 28-day period. The experiment was conducted three times. Lesion area was regressed on time. Comparisons among slopes were made by the Bonferroni t statistic.

Table 1. Number of early blight lesions and disease severity for 12 potato cultivars in field plots in 1985

	Number of lesions[y]			Disease severity[z]	
Cultivar	Day 8	Day 11	Day 13	On plant	In plot
Norland	48	106	106	7	7
Norchip	66	94	39	5	5
Buckskin	40	38	29	4	3
Chieftain	14	30	22	4	2
Penn 71	9	17	15	4	1
Superior	8	9	17	4	4
Atlantic	8	14	14	4	3
Kennebec	5	12	11	3	3
Katahdin	3	13	5	2	2
Belchip	9	27	8	2	2
PA 1	4	6	4	2	1
Rosa	3	8	7	2	1
LSD ($P = 0.05$)	36	30	22	1	2

[y] Days after inoculation on 28 July.
[z] Severity scale: 0 = no lesions, 1 = trace to 1%, 2 = 1–5%, 3 = 6–10%, 4 = 11–25%, 5 = 26–50%, 6 = 51–75%, and 7 = 76–100%.

Table 2. Early blight severity and area under disease progress curve (AUDPC) values for 12 potato cultivars in field plots in 1986

	Mean severity (%)[x]						
	Day 10		Day 16		Day 23		
Cultivar	Necrosis	Chlorosis	Necrosis	Chlorosis	Necrosis	Chlorosis	AUDPC[y]
Norland	3 b[z]	6 ab	22 a	24 a	78 a	73 a	670 a
Norchip	4 a	9 a	18 ab	21 ab	67 b	67 a	575 b
Superior	2 c	6 ab	16 bc	18 b–d	49 cd	46 bc	445 c
Hampton	1 d	3 b–e	13 cd	18 b–d	53 c	55 b	438 c
Monona	1 d	4 b–d	16 bc	19 a–c	44 cd	43 c	407 cd
Katahdin	1 d	5 bc	14 bc	18 b–d	45 cd	48 bc	399 cd
Penn 71	2 c	3 b–e	13 cd	15 cd	43 d	42 c	381 cd
Buckskin	1 d	3 b–e	13 cd	15 cd	43 d	43 c	372 cd
Atlantic	T e	2 c–e	12 cd	14 cd	41 d	41 c	349 d
Kennebec	1 d	4 b	9 de	13 cd	42 d	45 c	331 d
Chieftain	1 d	T e	4 e	4 e	22 e	22 d	174 e
Rosa	T e	1 de	4 e	6 e	21 e	22 d	164 e

[x] Days after inoculation on 9 July.
[y] Based only on ncecrotic area (16).
[z] Means followed by the same letter within columns are not significantly different according to Waller-Duncan average risk test ($k = 100$); T = trace (less than 1%).

RESULTS AND DISCUSSION

Field studies. In 1985, lesions were first observed 6 days after inoculation but were less than 5 mm in diameter. By day 8, lesion size ranged from 5 to 20 mm, depending on the cultivar; lesions on Norland were the largest. By day 11, large expanded lesions (>15 mm) were observed on Norland, Norchip, Atlantic, Kennebec, Superior, and Buckskin, whereas lesions on Rosa and Belchip remained small and unexpanded. There were significant differences among cultivars for the number of lesions (Table 1). Lesion number increased between day 8 and day 11, which could have been due to variation in length of time for germinating spores to successfully col-

onize and produce symptoms. The symptoms observed were on inoculated plants only. No early blight lesions from natural infections were observed before inoculation, and potatoes had not been planted in this area for at least 5 yr. By day 13, lesions were coalescing and the number of lesions usually had decreased. The differences in number of lesions among cultivars may indicate that infection efficiency differs among cultivars, and this may be one component of resistance to *A. solani*.

Disease severity on inoculated plants differed significantly among cultivars (Table 1). This was expected because of the differences in number of lesions. There were also significant differences in overall disease severity of the plots among cultivars. Buckskin had a large number of lesions and a high severity on the inoculated plant but had a low severity for the overall plot (Table 1). Microclimatic differences within the foliage of the different cultivars could be an important factor in the observed differences between plant and plot disease severity, but the differences observed in infection efficiency could account for these variations. There were five groupings of cultivars based on disease severity in overall plots. In order of decreasing severity, these were: 1) Norland; 2) Norchip; 3) Buckskin, Superior, Atlantic, and Kennebec; 4) Chieftain, Katahdin, and Belchip; and 5) Penn 71, Rosa, and PA 1. Ranking of the cultivars differed according to the method of assessment.

Counting lesions was time-consuming and not feasible for screening large numbers of genotypes to *A. solani*. The scale used to assess severity on the plant and severity of the plot was simple but did not account for the variation observed within a single cultivar. Because more information was needed on how to characterize the disease reaction, more detailed assessments were made in 1986.

In 1986, there were significant differences among cultivars for severity over all assessments and for AUDPC values (Table 2). Norland had the highest severity of early blight lesions on the last two assessments and had the highest AUDPC value. Norchip was significantly different from other cultivars for AUDPC value but was not always different from Superior for lesion severity. On the basis of AUDPC values, the cultivars could be grouped into six categories: 1) Norland; 2) Norchip; 3) Superior and Hampton; 4) Penn 71, Monona, Katahdin, and Buckskin; 5) Atlantic and Kennebec; and 6) Chieftain and Rosa. AUDPC values were highly correlated with severity at the third assessment period ($r = 0.96$).

The amount of chlorosis observed on leaves with early blight lesions differed significantly among cultivars. Not all chlorosis was associated with early blight, but there was a high positive correlation of necrotic lesion severity with severity of chlorosis at the second and third assessment ($r = 0.96$ and 0.99, respectively). Chlorosis was least severe in Chieftain and Rosa, which also had the lowest lesion severity.

The percentage of stems per plot and leaves per stem with visible early blight lesions varied significantly among cultivars (Table 3). By 16 days after inoculation, most cultivars had 100% stems infected per plot, and percentage of leaves infected per stem provided a better measurement for ranking cultivars. Percentage of stems infected was not a good indicator of the amount of early blight because all stems had leaves with necrotic lesions, except early in the epidemic, e.g., at 10 days after inoculation.

When severity was regressed on leaf location, there was a significant difference among cultivars for both slope and

Table 3. Percentage of stems and of leaves with early blight symptoms for 12 potato cultivars in field plots in 1986

	Percentage with early blight symptoms[y]					
	Stems per plot			Leaves per stem		
Cultivar	Day 10	Day 16	Day 23	Day 10	Day 16	Day 23
Norland	70 ab[z]	100 a	100	21 a	72 a	97 a
Norchip	78 a	100 a	100	15 a–d	60 b	97 a
Superior	73 ab	100 a	100	18 ab	52 c	95 ab
Hampton	45 de	100 a	100	9 c–f	33 ef	81 ef
Monona	68 a–c	97 a	100	14 a–d	48 cd	86 c–e
Katahdin	63 a–d	100 a	100	13 b–e	36 e	82 d–f
Penn 71	73 ab	100 a	100	16 a–c	45 d	86 c–e
Buckskin	55 b–e	95 a	100	10 c–f	36 e	88 cd
Atlantic	38 e	97 a	100	6 ef	36 e	90 bc
Kennebec	39 e	97 a	100	8 d–f	28 f	73 g
Chieftain	50 c–e	92 a	97	13 b–e	28 f	79 f
Rosa	40 e	80 b	97	5 f	17 g	54 h

[y] Days after inoculation on 9 July.
[z] Means followed by the same letter within columns are not significantly different according to Waller-Duncan average risk test ($k = 100$).

Table 4. Least squares linear regression data for average early blight severity against leaf location for 12 potato cultivars in field plots in 1986

Cultivar	Intercept[w]	Slope[x]	R^{2y}
Norland	121.4 a[z]	−5.8 a	0.95
Norchip	115.0 b	−5.2 a	0.97
Superior	104.3 b	−6.1 a	0.94
Hampton	103.9 b	−6.1 a	0.94
Monona	92.9 b	−5.7 a	0.91
Katahdin	100.2 b	−5.5 a	0.96
Penn 71	91.2 b	−5.4 a	0.88
Buckskin	95.0 b	−5.4 a	0.95
Atlantic	92.1 b	−5.5 a	0.87
Kennebec	95.4 b	−5.7 a	0.91
Chieftain	52.2 c	−3.3 b	0.79
Rosa	55.4 c	−3.5 b	0.78

[w] Intercept of regression line.
[x] Regression coefficient, or slope, of regression line.
[y] Coefficient of determination, an estimate of the amount of variation explained by the model.
[z] Intercepts or slopes followed by the same letter are not significantly different ($P = 0.05$) according to Bonferroni t statistic.

Table 5. Early blight severity and area under disease progress curve (AUDPC) values for nine potato cultivars in field plots in 1988

	Mean severity (%)[x]			
Cultivar	Day 36	Day 42	Day 50	AUDPC[y]
Norland	13 a[z]	60 a	100 a	919 a
Norchip	6 b	10 b	31 c	236 b
Monona	5 bc	10 b	30 c	233 bc
Superior	3 cd	7 b	39 b	228 b–d
Atlantic	2 d	6 b	24 cd	152 c–e
Chieftain	3 cd	6 b	19 ef	145 de
Hampton	1 d	4 b	17 ef	105 e
Katahdin	2 d	3 b	17 ef	99 e
Kennebec	2 d	3 b	15 f	98 e

[x] Days after inoculation on 26 July.
[y] After Tooley and Grau (16).
[z] Means followed by the same letter within columns are not significantly different according to Waller-Duncan k-ratio test ($k = 100$).

intercept of the regression lines (Table 4). Cultivars were separated into three groups on the basis of intercept and into two groups on the basis of slope. The best regression models were for severity data on leaves five to 15 only. Over cultivars, the leaves with severity closest to the regression line were those in the middle third section of the stem, i.e., leaves five to 15.

Assessment of severity or percentage of leaf area covered by early blight lesions required less time than counting lesions or infected leaves and provided a method for assessing differences among cultivars and ranking cultivars. Assessment of early blight severity several times over the disease epidemic is required to calculate AUDPC values. Comparison of cultivars based on the differences in epidemics calculated by AUDPC provided more information for ranking cultivars. A single assessment at the end of the season may be confounded by cultivars that require a long growing season or by other pest problems. To simplify the assessment and amount of data collected, only leaves located in the middle third of the plant canopy should be evaluated. In order to evaluate this approach, only five leaves in the middle third of the plant canopy on each of 10 main stems were assessed in 1988.

In 1988, there were significant differences among the cultivars (Table 5). On the first assessment date, 28 days after inoculation, only Norland had severity of 1%; all other cultivars had less than 1%. By the second assessment, 36 days after inoculation, all cultivars except Hampton had early blight severity greater than 1% (Table 5). Severities and AUDPC values were lower than in 1986, partially because of an unusually dry growing season with high temperatures during June through August. The epidemic did not start until the end of August after a period of heavy rainfall and low temperatures, even though early blight lesions were observed in early August on the inoculated plants of Norland. The rankings of Hampton and Chieftain were changed. Severities and AUDPC values of Hampton were lower than expected on the basis of cultivar rankings in 1986, and those of Chieftain were higher. These changes may have resulted from plant maturity affecting early blight severity, especially since the epidemic was delayed and did not start until 12 wk after planting. In 1988, planting was delayed because of a wet period in May. Drought, combined with abnormally high temperatures, delayed the early blight epidemic.

Greenhouse studies. Rate of lesion development differed significantly among cultivars (Table 6). Lesions developed faster on Norland, Norchip, and Atlantic than on other cultivars, but these differences were not always significant. The rate of lesion development varied for some cultivars among the three trials, possibly because the trials were conducted at different times of the year and temperatures and day lengths may have influenced the results. There were significant differences among cultivars for early blight severity, which was not always associated with the rate of lesion development.

Norland was the most susceptible cultivar over all field and greenhouse experiments. Norland matures in a shorter growing period than the other cultivars. Its leaves divert nutrients to tubers and senesce earlier than those of any other cultivar, providing an environment conducive to *A. solani*.

The cultivars examined in these trials fall into the following maturity classifications: Norland, very early maturing; Monona, Norchip, and Superior, medium early maturing; Atlantic and Chieftain, medium maturing; Katahdin and Kennebec, medium late maturing; Belchip, Hampton, PA 1, Penn 71, and Rosa, late maturing; and Buckskin, very late maturing. Maturity classification is important. Cultivars that require a long growing season were not always the least susceptible to *A. solani*. For example, Buckskin is very late maturing but was not the most resistant cultivar. Hampton, also a late-maturing cultivar from 1986 data, was susceptible and ranked with cultivars that are early to medium in maturity. Hampton was less susceptible in the 1988 test and was comparable to the late-maturing Katahdin and Kennebec. It may be best to group genotypes of like maturity when evaluating early blight reaction. These cultivars also varied in resistance to late blight, but there was no apparent relationship between late blight resistance and early blight reaction.

Further research is needed to characterize disease reaction of potatoes to *A. solani*. The next step would be to examine early blight severity on foliage of several cultivars and its effect on size, number, dry matter content, and dry rot of tubers. Some cultivars may be more tolerant to early blight than others. An index could be developed that would take both disease severity and apparent tolerance into account in evaluating reaction to early blight.

Table 6. Rates of early blight lesion development and severity for 12 potato cultivars in the greenhouse

Cultivar	Slope of regression of lesion size on time (mm^2 per day)		
	Expt. 1	Expt. 2	Expt. 3
Atlantic	0.63 a[x]	0.81 a	NT[y]
Norland	0.71 a	0.36 b	...[z]
Norchip	0.43 ab	1.35 a	0.28 b
Chieftain	0.36 ab	NT	0.15 b
Kennebec	NT	0.25 b	0.28 b
Belchip	0.25 bc	0.29 b	1.21 a
Superior	0.25 bc	0.12 c	0.17 b
Katahdin	0.22 bc	0.38 b	NT
Buckskin	NT	NT	0.06 b
Penn 71	NT	0.10 c	NT
PA 1	0.06 c	0.08 c	NT
Rosa	0.02 c	0.04 c	0.10 b

[x] Values are means of three replications. Means followed by the same letter within columns are not significantly different according to Bonferroni *t* statistic.
[y] Not tested.
[z] Regression not performed.

LITERATURE CITED

1. Boiteux, L. S., and Reifschneider, F. J. B. 1989. Evaluation of potato germplasm for early blight resistance, 1987. Biol. Cult. Tests Control Plant Dis. 4:19.
2. Christ, B. J. 1987. Reaction of potato cultivars to early blight, 1985. Biol. Cult. Tests Control Plant Dis. 2:17.
3. Christ, B. J. 1988. Reaction of potato cultivars to early blight, 1986. Biol. Cult. Tests Control Plant Dis. 3:19.
4. Christ, B. J. 1988. Effect of fungicide spray schedules controlling early blight on three cultivars, 1987. Fungic. Nematicide Tests 43:132.
5. Christ, B. J. 1990. Influence of potato cultivars on the effectiveness of fungicide control of early blight. Am. Potato J. 67:419-425.
6. Christ, B. J., and Cole, R. H. 1988. Evaluation of potatoes for resistance to early blight, 1987. Biol. Cult. Tests Control Plant Dis. 3:18.
7. Christ, B. J., and Maczuga, S. A. 1989. The effect of fungicide schedules and inoculum levels on early blight severity and yield of potato. Plant Dis. 73:695-698.
8. Corsini, D. L., and Pavek, J. J. 1988. Evaluation of potato breeding selections and cultivars for resistance to field and storage diseases, 1987. Biol. Cult. Tests Control Plant Dis. 3:25.
9. Corsini, D. L., and Pavek, J. J. 1989. Evaluation of potato breeding selections and cultivars for resistance to field and storage diseases for 1988. Biol. Cult. Tests Control Plant Dis. 4:22.
10. Douglas, D. R., and Pavek, J. J. 1972. Screening potatoes for field resistance to early blight. Am. Potato J. 49:1-6.
11. Frank, J. A., Webb, R. E., and Douglas, D. R. 1979. Evaluation of several USDA potato clones for resistance to early blight. Plant Dis. Rep. 63:392-394.
12. Holley, J. E., Hall, R., and Hofstra, G. 1983. Identification of rate-reducing resistance to early blight in potato. Can. J. Plant Pathol. 5:111-114.
13. LeClerg, E. L. 1946. Breeding for resistance to early blight in the Irish potato. Phytopathology 36:1011-1015.
14. Pelletier, J. R., and Fry, W. E. 1989. Characterization of resistance to early blight in three potato cultivars: Incubation period, lesion expansion rate, and spore production. Phytopathology 79:511-517.
15. Reifschneider, F. J., Furumoto, O., and Filgueira, F. A. R. 1984. Illustrated key for the evaluation of early blight of potatoes. FAO Plant Prot. Bull. 32:91-94.
16. Tooley, P. W., and Grau, C. R. 1984. Field characterization of rate-reducing resistance to *Phytophthora megasperma* f. sp. *glycinea* in soybean. Phytopathology 74:1201-1208.