Modeling the Relationship Between Alternaria Leaf Blight and Yield Loss in Muskmelon

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ABSTRACT

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Empirical models were developed to describe an observed relationship between Alternaria leaf blight and yield loss in muskmelon. Data used to develop and validate the models were obtained from replicated experimental field plots at two locations from 1988 to 1991. Area under disease progress curve, critical-point, and multiple-point models were derived and evaluated. All models provided a reasonably good fit to the data. However, a single area under disease progress curve model described losses at both locations, whereas separate critical-point and multiple-point models were necessary for each location. The critical-point models have potential for use in evaluating disease management decisions.

Alternaria leaf blight, caused by *Alternaria cucumerina* (Ellis and Everh.) J.A. Elliot, is a major foliar disease of muskmelon (*Cucumis melo* L. var. *reticulatus* Naudin) in Indiana. The disease occurs in almost all commercial muskmelon fields every year, but the severity of Alternaria leaf blight epidemics varies widely among fields. In lieu of horticulturally acceptable cultivars with adequate resistance to Alternaria leaf blight, growers must rely on repeated applications of protective fungicides to control the disease.

Environmental and economic concerns regarding agricultural chemicals have resulted in a reexamination of disease control programs that require intensive fungicide use and have led to renewed efforts to minimize fungicide use without increasing the risk of serious losses from disease. In order to determine how much disease can be tolerated, the amount of loss attributed to disease must be accurately defined (6,12). Previous reports described losses associated with Alternaria leaf blight (1,3,4,7,10,13) but did not quantitatively define the effect of the disease on yield. The objective of this research was to investigate several empirical models to describe the relationship between muskmelon yield loss and Alternaria leaf blight epidemics.

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MATERIALS AND METHODS

Field research. Data used for development of the models were obtained from field plots at two experimental farms in 1989 and 1990. The Purdue O'Neall Horticultural Research Farm (ONF) is located in Lafayette, Indiana, approximately 250 km north of the Southwest Purdue Agricultural Center (SWP) in Vincennes, Indiana. At each location, experimental plots consisted of single 10.67-m rows with 10 plants per row. Plants within rows were spaced 107 cm apart; rows were spaced 244 cm apart.

Plots were randomized within each of four blocks. Each plot was bordered on one side by an inoculated spreader row. All rows were mulched with 4-mil black polyethylene plastic. Each experimental site was prepared and maintained according to standard commercial practices for muskmelon production. Weeds were controlled by incorporation of bensulide (Prefar 4E) (9.4 L/ha, 4 qt/acre) and naptalam (Alanap L) (14.0 L/ha, 6 qt/acre) prior to transplanting. Insects were controlled with weekly applications of carbaryl (Sevin 80S) (0.4 L/ha, 1.25 pt/acre) or endosulfan (Thiodan 3EC) (0.3 L/ha, 1 pt/acre) from transplanting through the second week of harvest.

Seed of the muskmelon cultivar Allstar (Harris-Moran Seed Co., Rochester, New York) was planted in a commercially prepared potting medium (Terra-Lite Vegetable Plug Mix, W. R. Grace and Co., Cambridge, Massachusetts) in plastic growing trays (50 cells per tray) approximately 4 wk before transplanting in the field. Seedlings with two true leaves were planted on 25 May 1989 and 27 May 1990 at SWP and on 28 May 1989 and 26 May 1990 at ONF. Spreader rows were inoculated on 22 June 1989 and 25 June 1990 at SWP and on 10 July 1989 and 14 July 1990 at ONF.

Inoculum was increased according to a method described by Zhu et al (14). A conidial suspension of *A. cucumerina* was applied to spreader rows shortly before dusk on each inoculation date with a manually operated pressurized sprayer. Approximately 150 ml of a conidial suspension of 5×10^4 conidia/ml was applied evenly over the length of each spreader row. Epidemics of various intensity were created by treating plots with assorted fungicides at several different application schedules. Data from 71 replicated experimental plots (39 at SWP and 32 at ONF) were used to develop the yield loss models described below. Data from 27 replicated experimental plots (15 at SWP and 12 at ONF) that involved different fungicide treatments and spray schedules in 1988 and 1991 were used to validate the models.

Disease severity was assessed visually at 4- to 7-day intervals using the Horsfall-Barratt scale (2). Plots were harvested two or three times each week for at least 4 wk. Yield loss was expressed as the percentage of reduction in weight compared with an estimated maximum yield for each site each year. Maximum yields were represented by the intercept of the linear equation that resulted from regression of plot yields on the final disease percentage estimates for each plot. Disease severity assessments were converted to proportions using Horsfall-Barratt conversion tables. The proportions were then transformed to logits and regressed on a measure of time to obtain a linear description of each of the 71 epidemics.

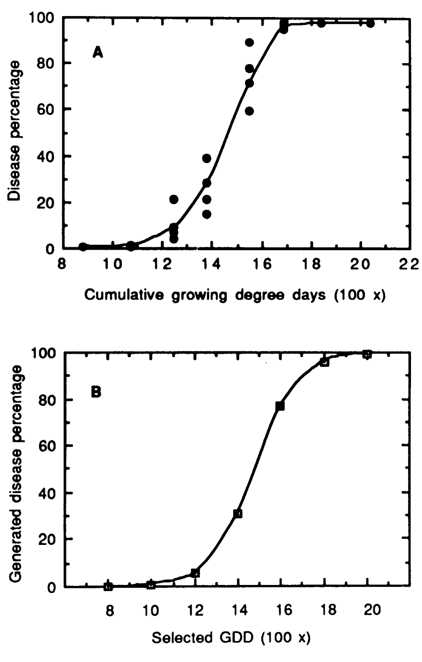

Fig. 1. (A) Example of a disease progress curve representing one of the 71 epidemics created in replicated field plots. **(B)** Generated disease progress curve derived by substituting selected growing degree day (GDD) values into the linear expression ($Y = -14.82 + 0.01\ X$) of the epidemic represented above.

Model development. For model development, time was expressed physiologically (growing degree days, base 10 C) rather than chronologically (days after transplanting), because melon ripening is highly dependent on environmental conditions, and the time from transplanting to melon maturity (date of first harvest) often varies among years and locations. Daily growing degree days (GDD) were calculated according to the equation $\text{GDD} = [(\text{MAX} + \text{MIN})/2] - 10$, where MAX and MIN represent maximum and minimum temperatures. The GDD values were summed beginning on the day of transplanting. Observations made during the 4 yr of this research (including validation studies) indicated that empirically determined GDD threshold values provided more reliable estimates of melon maturity at each location than chronological time (R. Latin, *unpublished*). GDD values were calculated from weather data recorded daily at each experimental farm.

Linear equations describing the relationship between logit disease and time (GDD) for each of the 39 SWP epidemics and 32 ONF epidemics were used to develop the multiple-point (MP) yield loss models. For each location, selected GDD values were substituted into the equations to generate seven disease proportions describing the progress of each epidemic. Selected values at the SWP location for GDD were 600, 800, 1,000, 1,200, 1,400, 1,600, and 1,800. Selected values at the ONF location for GDD were 1,200, 1,400, 1,600, 1,800, 2,000, 2,200, and 2,400. For example, after disease proportion was transformed to logits and regressed on GDD, the disease progress curve in Figure 1A was described by the equation $Y = -14.82 + 0.01\ X$ (where Y is logit disease and X is GDD). Then, selected GDD values were substituted into the equation to calculate a new set of disease logits. The logits were transformed back to disease proportions to generate a new disease progress curve for the epidemic (Fig. 1B). To develop the MP model, the generated disease proportions at the selected GDD values for all the 1989 and 1990 epidemics at each location served as predictor variables in a multiple regression equation to estimate the percentage of yield loss associated with Alternaria leaf blight epidemics.

The linear equations describing the 71 epidemics also were used to develop the critical-point (CP) yield loss model. The critical point, hereafter designated as DD1, is defined as the time (GDD) during the growth of the crop by which disease severity is approximately 1%. The selection of 1% as the disease level for determining the critical point was influenced by practical considerations. Alternaria leaf blight severity of 1% is estimated to be the lowest amount of disease an observer would notice through casual inspection of a muskmelon crop. It also is an amount that is considered manageable with current protective fungicides (R. X. Latin, *unpublished*). The DD1 value for each epidemic was determined by substituting −4.59 (logit 0.01) for the response variable in the linear equations. For example, in the epidemic described in Figure 1, the DD1 value was calculated to be 1,023. The CP model was derived by regressing yield loss percentages on DD1 values.

The areas under the disease progress curves (AUDPC) were calculated from untransformed disease proportions at the time (GDD) of assessment by the method of trapezoidal integration (11). The AUDPC model was developed by regressing yield loss percentages against AUDPC values.

RESULTS

MP models. MP models were derived for each of the two locations. The SWP model was $L = 8.2 - 5{,}465.8\ X_1 + 3{,}863.6\ X_2 - 1{,}284.2\ X_3 + 418.6\ X_4 - 125.5\ X_5 + 52.0\ X_6 - 13.8\ X_7$, where L is yield loss percentage, and X_1, X_2, X_3, X_4, X_5, X_6, and X_7 are disease proportions at GDD values of 800, 1,000, 1,200, 1,400, 1,600, 1,800, and 2,000. The coefficient of determination was $r^2 = 0.79$ (significant at $P = 0.05$), and residual analysis did not suggest a nonconstant variance, indicating that the model provided an acceptable fit to the data. Regression of yield loss percentages observed in validation trials against percentages predicted by the model resulted in a significant linear equation whose intercept value did not differ from 0 and slope was not different from 1.0 ($P = 0.05$), indicating that the model was unbiased in estimating losses (Fig. 2A).

The ONF MP model was $L = 0.6 - 3{,}521.9\ X_1 + 1{,}967.4\ X_2 - 78.7\ X_3 - 73.9\ X_4 + 115.4\ X_5 - 109.8\ X_6 + 51.9\ X_7$, where L is yield loss percentage, and X_1, X_2, X_3, X_4, X_5, X_6, and X_7 are disease proportions at GDD values of 1,200, 1,400, 1,600, 1,800, 2,000, 2,200, and 2,400. A significant ($P = 0.05$) coefficient of determination ($r^2 = 0.86$) and a random residual pattern confirmed the appropriateness of the model. The regression line describing the relationship between yield loss percentages predicted by the model and those observed had an intercept not different from 0 and a slope less than 1.0 but significantly greater than 0. Inspection of Figure 2B shows that the model tends to overestimate losses.

CP models. A linear relationship was established between yield loss percentage and DD1 at both experimental sites (Fig. 3A and B). Losses due to Alternaria leaf blight at SWP were described by the equation $L = 71.8 - 0.054\ X$, where L is yield loss percentage and X is the DD1 value. The model derived at ONF was $L = 110.6 - 0.068\ X$. A test for linear equality to determine the similarity between the two lines showed that the slopes were not different, but the intercept values differed significantly ($P = 0.05$). For both CP models, the coefficients of determination were statistically significant ($r^2 = 0.68$ for SWP and 0.51 for ONF), and residual patterns did not suggest a nonconstant variance. Application of the validation data to the CP models showed that the models were reasonably accurate in estimating losses due to Alternaria leaf blight (Fig. 3C and D).

AUDPC model. Regression of yield loss percentages on AUDPC at each location resulted in an acceptable fit to a linear model (Fig. 4A and B). A test for linear equality was performed to investigate the possibility of pooling the data and deriving a single model describing the relationship of yield loss to AUDPC (8). The test showed that the lines had a common slope, but the intercept values were different ($P = 0.05$). The degree of difference in the intercepts appeared slight, and there were no obvious intuitive arguments that the AUDPC loss models should differ with location. Therefore, the combined model, $L = 2.43 + 0.055\ W$, where L is yield loss percentage and W is AUDPC (Fig. 4C), was validated with test data. Regression of the observed yield loss percentages for the validation trials on those predicted by the AUDPC model resulted in a linear equation with an intercept of 1.39, a slope of 0.78, and a coefficient of determination of $r^2 = 0.86$ (significant at $P = 0.05$) (Fig. 4D). Confidence interval estimation of the regression parameters showed that the intercept was not significantly different from 0, and the slope was significantly different from 0 but less than 1.0 ($P = 0.05$). As a result, the AUDPC model was slightly biased in overestimation of losses, particularly where disease levels were high.

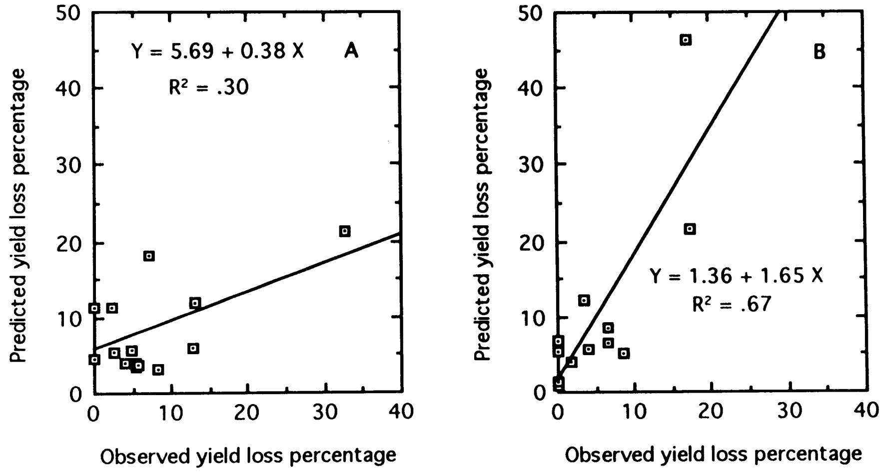

Fig. 2. Regression of observed yield loss percentages from field experiments conducted in 1988 and 1991 against losses predicted by the multiple-point models at the Southwest Purdue Agricultural Center (**A**) and Purdue O'Neall Horticultural Research Farm (**B**).

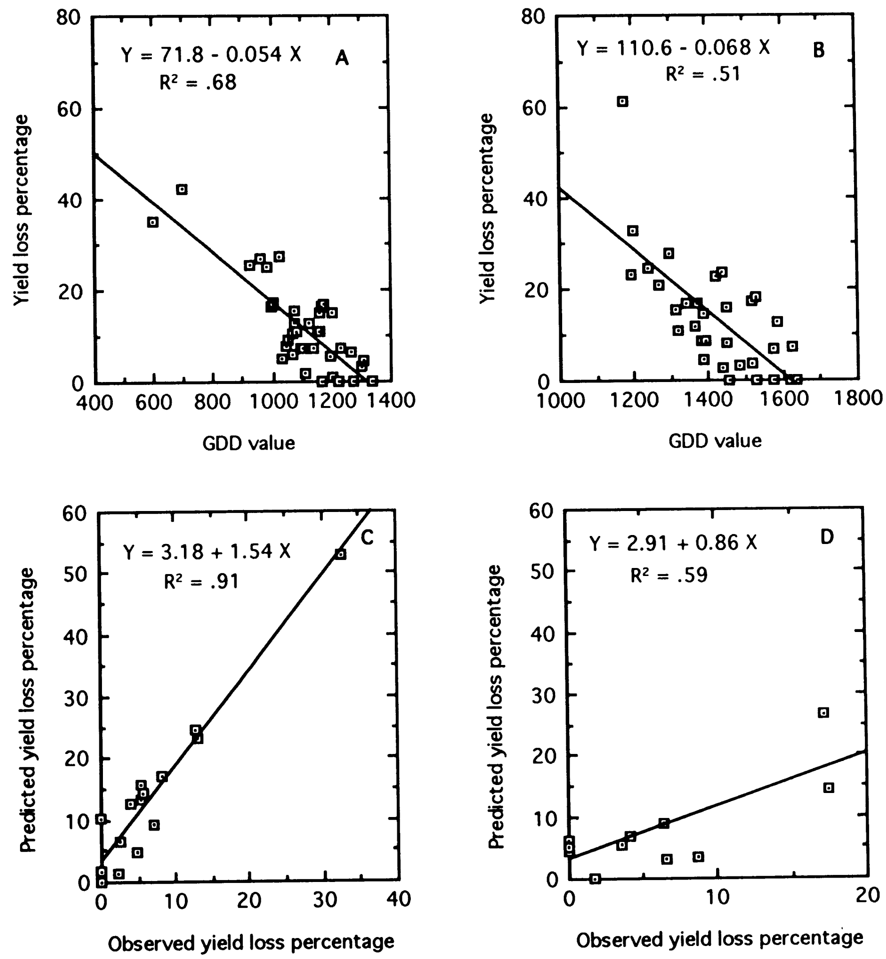

Fig. 3. Linear critical-point models developed at the Southwest Purdue Agricultural Center (SWP) (**A**) and Purdue O'Neall Horticultural Research Farm (ONF) (**B**) describing the relationship between yield loss percentage and degree day values at which 1% disease is expected to occur. Predicted yield loss percentages were regressed against those observed in validation plots for SWP (**C**) and ONF (**D**).

DISCUSSION

Disease yield loss models can be used in a descriptive sense to describe or explain the consequences of disease epidemics or in a predictive sense during the season to project possible losses and justify the need for fungicide protection. Although all three types of models were reasonably accurate in representing the relationship between disease and yield loss, the AUDPC model appeared most appropriate as a descriptive standard because of its flexibility for use at various locations. The CP and MP models employ disease estimates at specific GDD values to determine disease-related losses. Since this investigation showed that different GDD values for maturation of muskmelons occurred at SWP and ONF, separate models were developed for the two experimental locations. Determination of AUDPC, however, employs the increment in GDD during disease progress rather than the specific GDD values. Hence, differences in maturation due to location do not affect the model. A recognized weakness of the AUDPC model is its inability to distinguish between epidemics of different severities with similar AUDPC values, i.e., mild epidemics with a very early onset versus severe epidemics that begin late in the season. Such a limitation should be considered when using the AUDPC model to describe muskmelon losses attributed to Alternaria leaf blight.

Most CP models relate yield loss to disease severity at a critical point in time or growth stage (5). The type of CP model developed through this research is different in that loss is related to the time (as determined by GDD) at which a critical amount of disease occurs. It is similar to the disease-free period model developed by Olofsson for determining yield losses caused by potato late blight (9).

This type of CP model has potential application in providing support for disease control decisions. For example, suppose one is asked to provide disease management recommendations to a grower in southwestern Indiana who is prepared to accept no more than 2% yield loss due to Alternaria leaf blight. By substituting that value (2%) into the equation in Figure 3A, one can determine how long (in GDD) the grower must maintain disease levels no greater than 1% in order to achieve his management objective. The model therefore may be used to help growers determine when fungicide sprays for Alternaria leaf blight control can be terminated without risking serious yield losses to the disease. The CP model also serves an educational purpose in that it can be used to reinforce recommendations for early season protection and show why more disease risk can be accepted toward the end of the season. The fact that separate models were developed for the northern and southern parts of the state is not viewed as a severe limitation. Although both models were reasonably accurate, only the SWP CP model is likely to be used in commercial disease management situations, because 90% of the commercial muskmelon production in Indiana is located within 50 km of the SWP site.

A suspected weakness in all of the models is that they do not account for inherent variability in fruit maturation. Differences in earliness among cultivars is considerable, and a yield loss model developed with a late-season cultivar may not accurately describe losses occurring on a very early-maturing muskmelon, and vice versa. The Allstar muskmelon selected for model development is generally considered a mid-season type. This research also does not account for losses in fruit quality caused by Alternaria leaf blight. Investigations are currently underway to address the effect of disease on sugar content in muskmelon.

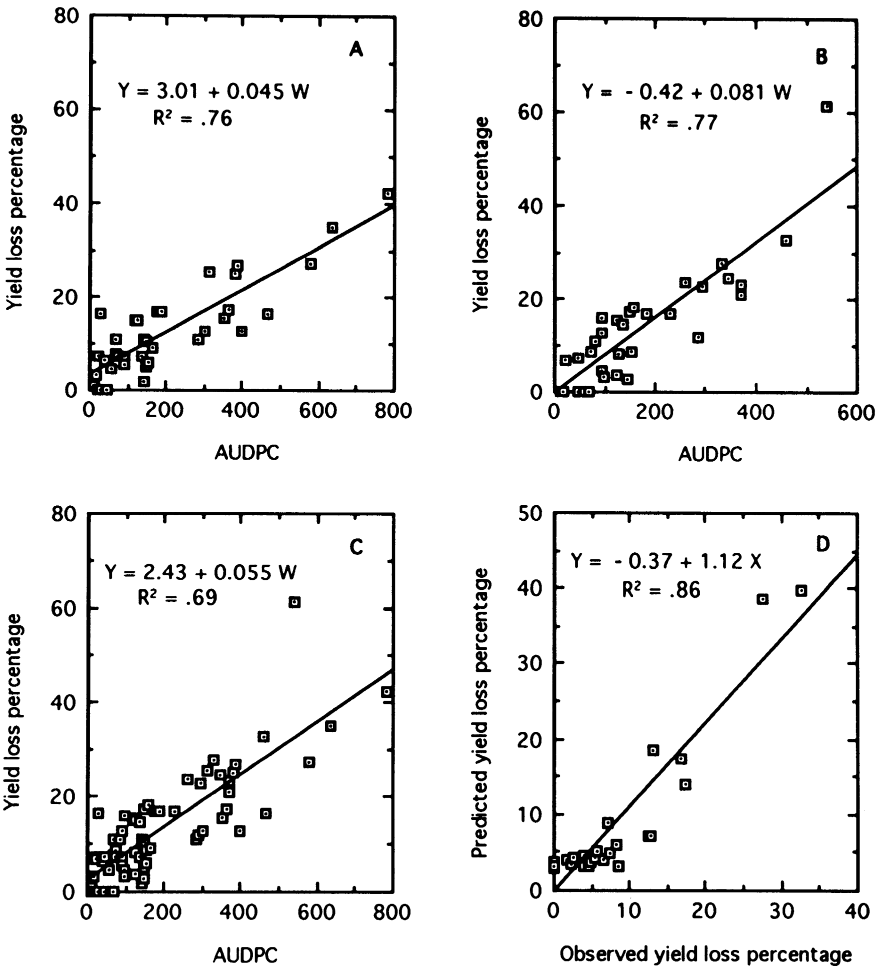

Fig. 4. The relationship between yield loss percentage and area under disease progress curve (AUDPC) was linear at both Southwest Purdue Agricultural Center **(A)** and Purdue O'Neall Horticultural Research Farm **(B)**. The data were pooled to develop a general AUDPC model for both locations **(C)**. A significant linear relationship resulted from the regression of observed loss percentages on those predicted by the pooled model **(D)**.

ACKNOWLEDGMENTS

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LITERATURE CITED

1. Brisley, H. R. 1923. Studies on the blight of cucurbits caused by *Macrosporium cucumerinum* E. & E. Phytopathology 13:199-204.
2. Horsfall, J. G., and Barratt, R. W. 1945. An improved grading system for measuring plant diseases. (Abstr.) Phytopathology 35:655.
3. Ibrahim, A. N., Adel-Hak, T. M., and Mahrous, M. M. 1975. Certain morphological and pathological characters of *Alternaria cucumerina*, the cause of watermelon leaf spot disease in Egypt. Acta Phytopathol. Acad. Sci. Hung. 10:301-307.
4. Jackson, C. R. 1958. Taxonomy and host range of *Alternaria cucumerina*. (Abstr.) Phytopathology 48:343-344.
5. James, W. C. 1974. Assessment of plant diseases and losses. Ann. Rev. Phytopathol. 12:27-48.
6. Madden, L. V. 1983. Measuring and modeling crop losses at the field level. Phytopathology 73:1591-1596.
7. Middleton, J. T., and Whitaker, T. W. 1946. Some diseases of cantaloupes and honeydew melons observed in southern California. Plant Dis. Rep. 30:374.
8. Neter, J., and Wasserman, W. 1974. Applied Linear Statistical Models. Richard D. Irwin, Homewood, IL. 842 pp.
9. Olofsson, B. 1968. Determination of the critical injury threshold for potato blight (*Phytophthora infestans*). Natl. Swed. Inst. Plant Prot. Contrib. 14(119) :85-93.
10. Prasada, R., Khandelwal, G. L., and Jain, P. 1972. Epidemiology, forecasting and control of Alternaria blight of cucurbits. Proc. Indian Natl. Sci. Acad. Part B 37:301-308.
11. Shaner, G., and Finney, R. E. 1977. Effect of

nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. Phytopathology 67:1051-1056.
12. Teng, P. S., and Johnson, K. B. 1988. Analysis of epidemiological components in yield loss assessment. Pages 179-188 in: Experimental Techniques in Plant Disease Epidemiology. J. Kranz and J. Rotem, eds. Springer-Verlag, New York. 299 pp.
13. Thomas, C. E. 1983. Fungicide applications based on duration of leaf wetness periods to control Alternaria leaf blight of cantaloup in South Texas. Plant Dis. 67:145-147.
14. Zhu, Z. Y., Huang, X. M., and Li, Y. H. 1985. An efficient technique for inducing sporulation of *Alternaria solani* in pure culture. Acta Mycol. Sin. 4:180-184. (Abstr.) Rev. Plant Pathol. 65:1096.