A Model for Weather-Based Forecasting of Anthracnose on Annual Bluegrass

T. K. Danneberger, J. M. Vargas, Jr., and A. L. Jones

Former graduate research assistant and professors, Department of Botany and Plant Pathology, and the Pesticide Research Center, Michigan State University, East Lansing 48824. Present address of senior author: Department of Agronomy, Ohio State University, Columbus 43210.

Michigan Agricultural Experiment Station Journal Series Article 11076.
Research supported by the Michigan Turfgrass Foundation and the O. J. Noer Foundation.
Accepted for publication 3 November 1983.

ABSTRACT

Danneberger, T. K., Vargas, J. M., Jr., and Jones, A. L. 1984. A model for weather-based forecasting of anthracnose on annual bluegrass. Phytopathology 74:448-451.

A multiple regression equation relating hours of leaf wetness and temperature to the incidence of disease incited in annual bluegrass by *Colletotrichum graminicola* was developed from 2 yr of field data. The model is $ASI = 4.0233 - 0.2283\ LW - 0.5308\ T - 0.0013\ LW^2 + 0.0197\ T^2 + 0.0155\ (LW \times T)$, in which ASI = anthracnose severity index, T = average daily temperature (C) for a 3-day period 10–12 days preceding symptom expression and LW = average hours of leaf wetness per day for the same period. ASI values of 1, 2, 3, 4, 5, and 6 were equal to <10, 11–20, 21–30, 31–40, 41–50 and >51% of the turfgrass area diseased, respectively. The accuracy of the model was tested with data from three locations in 1982. The model successfully predicted 14 of 16 periods of disease increase when an ASI value of 2 was taken as the minimum conditions for infection. Average daily ASI values predicted from temperature and leaf wetness data were related to rate of disease increase according to the Gompertz transformation.

Anthracnose caused in annual bluegrass (*Poa annua* var. *reptans* (Hauskins) Timm.) by the fungus *Colletotrichum graminicola* (Ces.) Wils. causes severe damage to golf course greens and fairways (2,3,8,11). The fungus overwinters in both dead and living plant material (5) and, during the summer, conidia produced in acervuli infect annual bluegrass plants. In Michigan, anthracnose is particularly severe during periods of warm weather in summer. The disease develops as irregularly shaped patches of yellow-bronze turf that range from a few centimeters to several meters across. Infected leaves have elongated reddish brown lesions that expand and encompass the entire leaf blade (8). Results of laboratory studies show that the optimum temperature range for growth of the fungus is 22–31 C (7,8). Inoculation studies (2) under greenhouse conditions show that *C. graminicola* can cause disease on annual bluegrass between 27 and 33 C.

The purpose of this study was to develop a model to predict anthracnose severity on annual bluegrass from leaf wetness and temperature data collected in the field. Many of the approaches used in this study were previously used to develop a prediction model for cherry leaf spot (4).

MATERIALS AND METHODS

At the Michigan State University Soils Research Farm, East Lansing, MI 48824, and at the Glengary Country Club, Sylvania, OH 43560, 4 × 4-m plots were established in four replicated 10 × 10-m areas of annual bluegrass during 1980 and 1981. Anthracnose was severe at both locations in 1979. The grass on the plots at both locations was mowed to a height of 1.25 cm three times a week, irrigated as needed to prevent wilting, and fertilized with 28 kg of nitrogen per hectare in May, June, August, and September (total 112 kg/ha/yr). Levels of phosphorus and potassium were adequate according to soil test results obtained from the Michigan State Soil Testing Laboratory.

Belfort leaf wetness recorders (Belfort Instruments, Co., Baltimore, MD 21224) were used to monitor air temperature and hours of leaf wetness at each location from 1 May to 10 October 1980 and 1981. Leaf wetness measurements included wetting periods resulting from rain, dew, and irrigation. The leaf wetness sensors were located 1.3 cm above the soil surface.

Disease severity as the percentage of area in each plot with anthracnose symptoms was estimated every 3–5 days. The number of infected plants from three 400-cm^2 subplots within a single plot were counted and correlated with corresponding visual ratings. To detect the spread of inoculum at each location, disease-free, 1-mo-old annual bluegrass plants in 700-cm^3 (15.2-cm diameter) clay pots were placed in holes in the ground so that the soil in the pot was flush with the surrounding soil surface in the center of each plot. The potted plants were replaced weekly with new, disease-free, greenhouse-grown plants. During the exposure period, the plants were subjected to the same mowing and moisture regime as the surrounding plot area. The plants in their pots were placed in a mist chamber for 48–60 hr at 25 ± 5 C, moved to a greenhouse bench, and examined periodically for 2 wk for symptoms of anthracnose. Isolations from leaf lesions on these plants were made periodically on potato-dextrose agar (PDA; Gibco Diagnostics, Madison, WI 53713) to verify the presence of *C. graminicola*.

Model development. A model relating daily mean air temperatures and hours of leaf wetness to anthracnose severity was developed by using regression techniques. The model was based on conditions at the time of infection, which were determined by subtracting a given time from the time disease ratings were taken. This period of time from the onset of infection to the development of visual symptoms was determined in a greenhouse experiment.

Determination of maximum symptom expression period. One-month-old annual bluegrass plants were grown in 700-cm^3 clay pots containing a mix of sand, soil, and peat (1:1:1, v/v). Plants were fertilized with 50 kg/ha each of nitrogen, phosphorus, and potassium and were maintained at a height of 2.5 cm by weekly mowing.

The isolate of *C. graminicola* used to inoculate the plants was obtained from an annual bluegrass plant at the Field Crops Laboratory, East Lansing, MI 48824. The isolate was grown on 4% PDA at 22 C. Spores from 18-day-old cultures were suspended in sterile distilled water and a spore concentration of 10^5 spores per milliliter was determined with a hemacytometer. Two milliliters of the spore suspension was applied to the plants from a DeVilbiss hand atomizer. Viability of the spores, determined by atomizing spores onto blocks of PDA in petri plates and incubating at 22 C for 48 hr, was >90%.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

The plants were continuously misted for 48 hr then placed on a bench in a controlled-temperature greenhouse maintained at 22 ± 2, 25 ± 2, or 30 ± 2 C. Initial appressoria formation was noted by microscopic observation. The day maximum disease expression occurred was determined by lesion counts per 20 randomly selected leaf blades. If no increase in lesion number occurred, maximum disease development was considered complete.

Each temperature treatment was replicated four times and the experiment was repeated twice. The results of the two experiments were averaged and are presented in Table 1.

The assumption was made, due to variation in symptom development at the three temperatures, that 10–12 days were required for symptom development. Therefore, temperature and wetness data for 10, 11, and 12 days preceding the date of each disease rating were averaged and the means were correlated with corresponding disease severity values according to Pearson's method as given by Nie et al (6).

Validation of the model. From 1 May to 1 September 1982, the model was tested at three locations: Robert Hancock Turfgrass Research Center, East Lansing, MI 48824; Glengary Country Club, Sylvania, OH 43560; and Meadowbrook Country Club, Livonia, MI 48151. In previous years (1980–1981), anthracnose had been severe at each location. Disease severity was monitored in three 4 × 4-m plots selected randomly within a 10 × 10-m area at each location.

One Belfort leaf wetness recorder per location was set with the sensor 1.3 cm above the soil surface to monitor hourly temperature and duration of leaf wetness. An anthracnose severity index (*ASI*) was calculated daily using the regression model. Disease severity in each plot was rated every 2–4 days.

Anthracnose severity versus rate of disease increase. To determine if the expected anthracnose severity index (*ASI*) values computed with the model were related to rate of disease increase, linear regression analysis was done on the data collected in 1982 from the Hancock Research Center and the Glengary Country Club. The Meadowbrook data were not included since they were almost identical to those from the Hancock Research Center. Disease increases were determined by using the logistic (10) and Gompertz (1) methods. Tests for homogeneity of regression coefficients (9) were performed before the two sets of data were pooled for regression analysis.

RESULTS

Model development. An acceptable second-order model relating temperature and duration of leaf wetness to disease severity took the form:

$$ASI = b_0 + b_1 LW + b_2 T + b_{11} LW^2 + b_{22} T^2 + b_{12}(LW \times T) + e \quad (1)$$

in which ASI = anthracnose severity index, LW = hours of leaf wetness, and T = average daily temperature (C) for a 3 day period, 10-12 days preceding symptom expression. The b values are least-square estimates of the partial regression coefficients and e is a normally distributed random variable with mean zero and variance σ^2. The *ASI* values were 1, 2, 3, 4, 5, and 6, which corresponded to 1–10, 11–20, 21–30, 31–40, 41–50, and >51%, respectively, of the turfgrass area diseased. This model accounted for 84% of the observed variation in disease severity and all estimated coefficients were statistically significant at $P = 0.01$. The actual model was:

$$ASI = 4.0233 - 0.2283 LW - 0.5308 T - 0.0013 LW^2 + 0.0197 T^2 + 0.0155(LW \times T).$$

The relationship of temperature and duration of leaf wetness to disease severity is shown in a computer-generated surface (12) of the original data from the two monitoring sites (Fig. 1A). A comparative surface generated from points predicted with the equation (Fig. 1B) shows a good fit of the model for temperature values from 14 to 28 C and for wetting durations up to 24 hr. Examination of residuals (the difference between the original data points and those predicted by the model) supported the assumption that the error components are independent, have a mean of zero, and a constant variance.

Model validation. To determine if *ASI* values calculated from daily temperature and wetness data accurately predicted disease development, *ASI* values were calculated from weather data for each of the three locations. To account for a latent period of 10–12 days, three consecutive *ASI* values were averaged and related to visual estimations of disease made 10 days later. Visual estimates were related to actual counts made from three 10 × 10-cm subplots within a single plot. The model predicted 14 of 16 periods of disease increase correctly (88%). Twice in mid-May, the model predicted disease when none appeared. Potted plants exposed at the Michigan State University Soil Research Laboratory in 1980 and 1981 failed to develop disease during May. Exposed plants did not exhibit disease until the week of 16 June in 1980 and 10 June in 1981.

Predicted *ASI* values were directly related to the actual

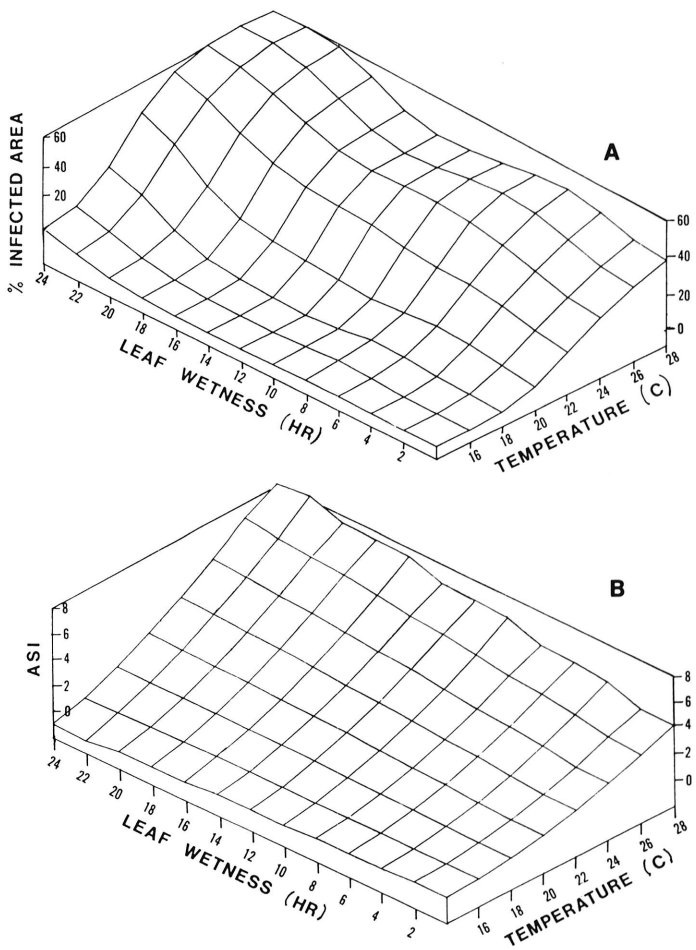

Fig. 1. Relationship of temperature and duration of leaf wetness to infection of annual bluegrass by *Colletotrichum graminicola*. **A,** From actual field data. **B,** Predicted from regression equation. *ASI* = anthracnose severity index.

TABLE 1. Stages of anthracnose development on annual bluegrass in days from initial inoculation

Average temperature (± 2C)	Formation of appressoria (day)	Maximum disease development (day)
22	2[a]	12.5
25	2	11.9
30	2	9.2
Average	2.0	11.20

percentage of diseased area observed 10 days later (Fig. 2). However, low *ASI* values (1.0 to 1.8) estimated disease when none was present. Because 14 of 16 wetting periods suitable for infection had *ASI* values >2, the assumption was made that an *ASI* value of 2 was the threshold value for infection (Fig. 3). Also, 90% of the wetting periods with no disease development fell on or below the line for *ASI* = 2.

Relationship of anthracnose severity index to rate of disease increase. Anthracnose severity index (*ASI*) values were plotted from data collected at the Robert Hancock Turfgrass Research Center (representative of the three sites) (Fig. 4). Disease was first predicted on 14 and 15 May and should have appeared in late May. *ASI* values >2 were detected on 29–30 June and were followed about 10 days later by an increase in the area of annual bluegrass infected. Favorable periods on 6–12 July and 14–19 July were both followed by periods of disease increase.

Rates of disease increase and daily *ASI* values were calculated for two locations (Table 2). The resultant *F*-statistics for homogeneity of regression coefficients before pooling the data from Glengary Country Club and Hancock Research Center were not significant ($P = 0.05$). Therefore, the data from the two locations were combined. When rates of disease increase were compared to daily *ASI* values, the Gompertz model resulted in a better fit ($r^2 = 0.912$) than the logistic model ($r^2 = 0.646$). The resultant regression showed that the predicted daily *ASI* values were related to the rates of disease increase (Fig. 5).

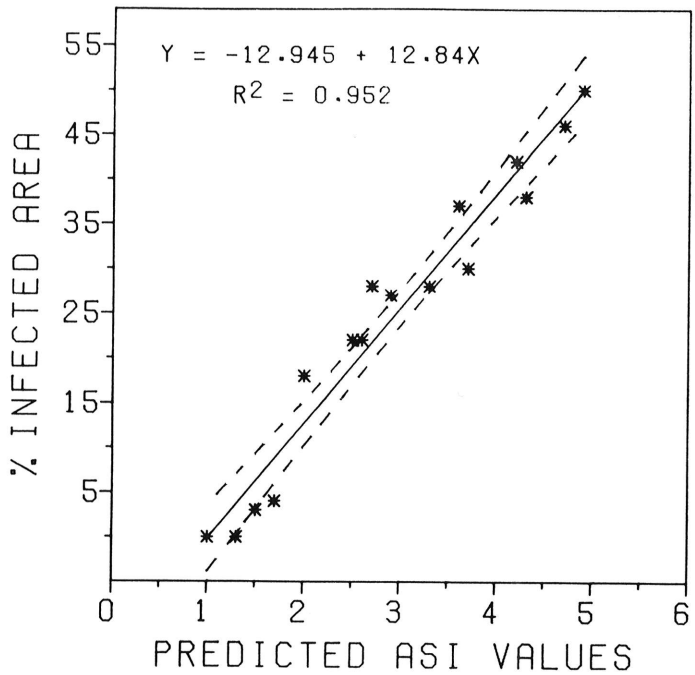

Fig. 2. Regression and 95% confidence limits of anthracnose severity index (*ASI*) values predicted from mean temperature and leaf wetness duration data collected in the field on actual estimate of infection at three locations used for validating the predictive model in 1982.

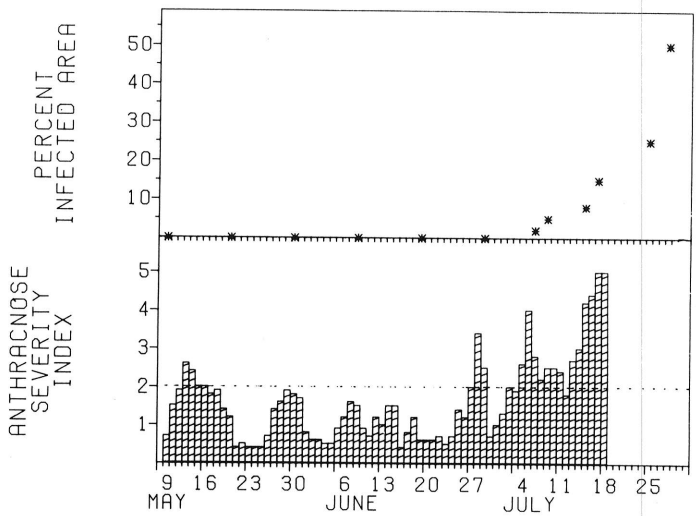

Fig. 4. Development of anthracnose on annual bluegrass in East Lansing, MI, during 1982 and the favorability of temperature and leaf wetness as expressed from predicted values by the anthracnose severity index (*ASI*) model. Dotted line represents predicted threshold value for infection.

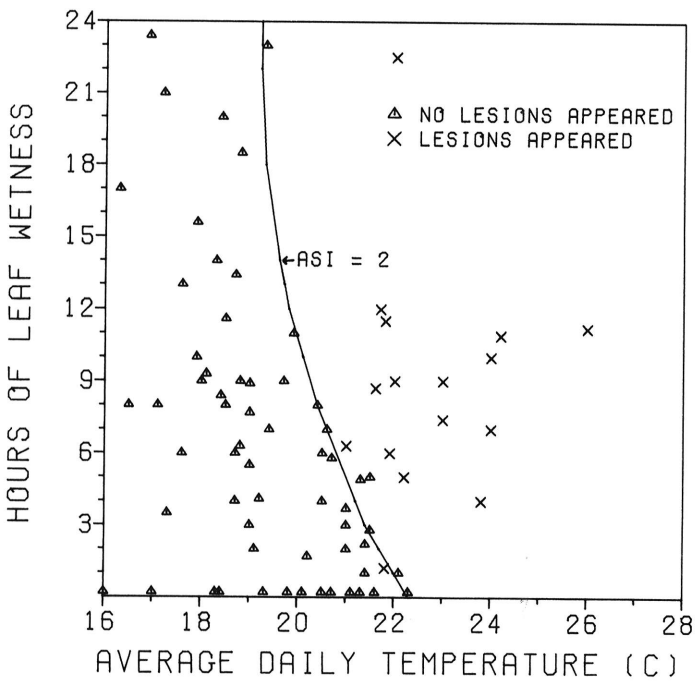

Fig. 3. Comparison of leaf wetness periods and average daily temperatures monitored in the field to subsequent infection. An anthracnose severity index (*ASI*) of 2 was the minimum value needed for infection.

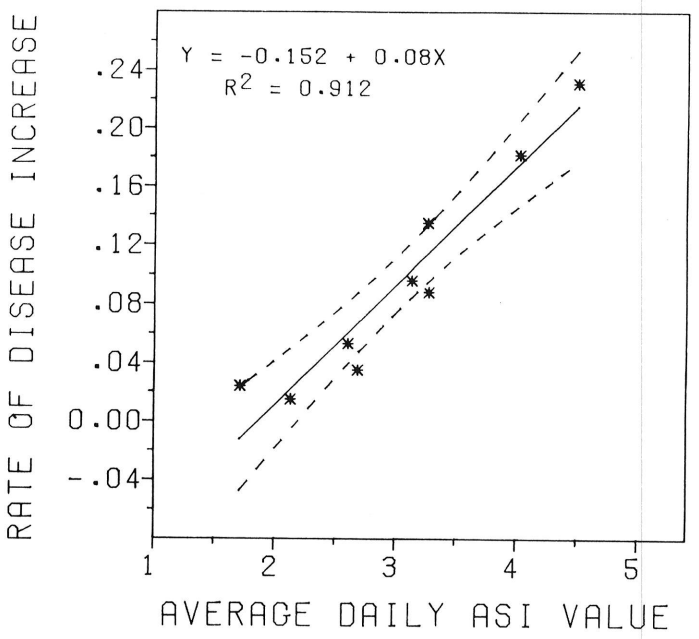

Fig. 5. Fitted regression line and 95% confidence limits relating the proportional rate of change in disease (as computed with a Gompertz transformation) to an average daily anthracnose severity index (*ASI*) computed from mean temperature and wetness duration data (see Table 2).

TABLE 2. Rates of change in anthracnose severity and average daily anthracnose severity index (*ASI*) calculated with an infection model from temperature and leaf wetness data collected from two locations in Michigan

Date (t_1–t_2)	Infection area (%)		Infection rate		Average
	D_1[w]	D_2	Logistic[x]	Gompertz[y]	*ASI*[z]
Glengary					
22–24 July	5	10	0.231	0.088	3.27
24 June–6 July	10	15	0.027	0.015	2.13
6–9 July	15	40	0.245	0.182	4.02
9–15 July	40	70	0.082	0.135	3.26
Hancock					
27–29 June	3	5	0.170	0.053	2.60
29 June–5 July	7	8	0.019	0.024	1.71
5–7 July	8	15	0.209	0.096	3.13
7–15 July	15	25	0.057	0.035	2.68
15–17 July	25	50	0.231	0.231	4.50

[w] D = percent infected area for the first (t_1) and second (t_2) dates of disease assessment.
[x] Logistic = $\ln(D_2) - \ln(D_1)/(t_2 - t_1)$.
[y] Gompertz = $-\ln(-\ln D_2) - (-\ln(-\ln D_1)/(t_2 - t_1))$.
[z] Sum of *ASI* values from $t_1 - 12$ to $t_2 - 12$ divided by $t_2 - t_1$.

DISCUSSION

A multiple regression model was developed for predicting the severity of anthracnose of annual bluegrass from daily mean temperatures and leaf wetness periods. The design of the model assumes the presence of adequate inoculum and a susceptible host.

The model accurately predicted that disease would increase when *ASI* values were 2 or above. When *ASI* values were below this value, predictions were not followed by probable outbreaks of disease. Index values in early May sometimes predicted disease development, but none occurred. This apparent failure of the model resulted from a lack of inoculum as determined by using live plants as spore traps.

Development and testing of the model has been limited to average temperatures ranging from 16 to 28 C. Extrapolations from the model outside these bounds will produce erroneous results. Although higher and lower temperatures should be studied, temperatures between 16 and 28 C fit the range within which the fungus is damaging in the field. The assumption we made in regard to incubation period may not be appropriate under all conditions and further work is needed. Also, the model does not account for the varying degree of susceptibility of different biotypes of annual bluegrass to anthracnose (2) or the effect of nitrogen fertilization on disease development (3). To account for these factors and as more information becomes available, future adjustments in the model will be needed.

The model may allow for the development of new strategies for managing anthracnose of annual bluegrass. Turf managers could reduce the severity of disease by changing irrigation timing to reduce periods of leaf wetness at critical times. It may also be possible to time fungicides more accurately with this model. However, further research on the timing of application and postinfection effectiveness of fungicides is needed. This is because spray treatments based on the model will be delayed until after the onset of infection. Work is currently being done to utilize these predictions to schedule the use of fungicides.

LITERATURE CITED

1. Berger, R. D. 1981. Comparison of the Gompertz and logistic equations to describe plant disease progress. Phytopathology 71:716-719.
2. Bolton, A. J., and Cordukes, W. E. 1981. Resistance to *Colletotrichum graminicola* in strains of *Poa annua* and reaction of other turfgrasses. Can. J. Plant Pathol. 3:94-96.
3. Danneberger, T. K., Vargas, J. M., Jr., Rieke, P. E., and Street, J. R. 1983. Anthracnose development on annual bluegrass in response to nitrogen carriers and fungicide application. Agron. J. 75:35-38.
4. Eisensmith, S. P., and Jones, A. L. 1981. A model for detecting infection periods of *Coccomyces hiemalis* on sour cherry. Phytopathology 71:728-732.
5. Naylor, V. D., and Leonard, K. J. 1977. Survival of *Colletotrichum graminicola* in infested corn stalks in North Carolina. Plant Dis. Rep. 61:382-383.
6. Nie, H. C., Hull, H., Jenkins, J. G., Steinbrenner, K., and Bent, D. H. 1975. Statistical Package for the Social Sciences. (Pages 279-288) McGraw-Hill, New York.
7. Smith, J. D. 1954. A disease of *Poa annua*. J. Sports Turf Res. Inst. 9:344-353.
8. Sprague, H. B., and Eval, E. E. 1928. Experiments with turf grasses. N.J. Agric. Exp. Stn. Bull. 497. pp. 55.
9. Steel, R. G. D., and Torrie, J. H. 1960. Principles and Procedures of Statistics. McGraw-Hill, New York. 481 pp.
10. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
11. Vargas, J. M. 1981. Management of Turfgrass Diseases. Burgess Publ. Co., Minneapolis, MN. 204 pp.
12. Wittick, R. I. 1980. GEOSYS: A computer system for the description and analysis of spatial data. Michigan State University (East Lansing) Center for Cartographic Research and Spatial Analysis. Tech. Rep. 5. Department of Geography, Michigan State University, East Lansing. 48 pp.