Cercospora Leaf Spot of Cowpea: Models for Estimating Yield Loss

R. W. Schneider, R. J. Williams, and J. B. Sinclair

Research Assistant, Plant Pathologist, and Professor, respectively. First and third authors, Department of Plant Pathology, University of Illinois, Urbana 61801; second author, International Institute of Tropical Agriculture, Ibadan, Nigeria. Senior author's present address: Department of Plant Pathology, University of California, Davis 95616.

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ABSTRACT

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Yield loss of cowpea was correlated with Cercospora leaf spot disease severity from artificially-simulated, chemically-regulated, and naturally-occurring epidemics at Ibadan, Nigeria. Areas under disease progress curves were correlated with yield loss regardless of the shapes of the curves. The yield-loss-estimation model was not accurate for low disease levels. A critical-point model was constructed and tested that related time of occurrence of specific disease severities to percent yield loss. However, the model could be used only for uninterrupted disease progressions. A third model that integrates these two methods is discussed in relation to chemical control programs.

Additional key words: epidemiology, disease control, benomyl, foliar diseases.

Cowpea [*Vigna unguiculata* (L.) Walp.], is the most widely traded and consumed grain legume in tropical Africa (11). Cercospora leaf spot of cowpea, caused by *Cercospora cruenta* (Sacc.) and *C. canescens* Ell. & Martin, is a major disease of this crop in Africa and may reduce yield by as much as 42 percent (8). A single application of benomyl gave adequate control of this disease in Nigeria (10).

Except for one report (7), there have been no detailed studies on the epidemiology or yield-depressing potential of this disease. This study was undertaken to explore different methods for estimating yield loss, and to develop a method which could be used in an economically judicious disease control program.

MATERIALS AND METHODS

Field operations.—Experiments were conducted during three 1-year growing seasons near Ibadan, Nigeria. All field plots were fertilized with 40 kg nitrogen, 60 kg phosphate, and 40 kg potassium/hectare (ha) in the form of ammonium sulfate, triple superphosphate, and muriate of potash, respectively.

Cowpea seeds were treated with a combination of 1 g benomyl [methyl 1-(butylcarbamoyl)-2-benzimidazole-carbamate], Benlate 50 WP, plus 1 g thiram (tetramethyl-thiuram disulfide, Arasan-50-Red) per kg seed and planted in the field on ridges with 75- to 80-cm centers with three seeds per hill at 20-cm spacing. Ten days after planting stands were thinned to one seedling per hill.

The randomized, complete-block design with four replications per treatment was used in all experiments. Each replicate consisted of five 11-m rows, unless otherwise indicated. Yield data were taken from the center three rows of each plot after two plants were removed from the ends of each harvest row. Harvested pods were dried at about 40 C for 48 hours prior to hand threshing. Yield data were based on 14 percent moisture content after drying.

All plots were sprayed to run-off at weekly intervals with 0.1 percent active ingredient (a.i.) Lindane (1,2,3,4,5,6-hexachlorocyclohexane) and 0.1 percent a.i. Rogor [0,0-dimethyl S-(N-methylcarbamoylmethyl)phosphorodithioate] beginning about 14 days after planting and continuing until first flowers appeared (approximately 35 days after planting) to control leaf chewing and sucking insects, and with 0.1 percent a.i. Gardona [2-chloro-1-(2,4,5,-trichlorophenyl) vinyl dimethyl phosphate] from flowering through harvest for the control of lepedopterous flower- and pod-boring larvae.

Disease severity scale.—A Cercospora leaf spot disease severity scale, based on easily distinguishable quantitative symptomatological characteristics, was developed. The scale consisted of the following divisions: 0 = no lesions; 1 = one spot per leaflet; 2 = two to three spots per leaflet, 10% leaflet area affected; 3 = spots covering 10-12% leaflet area, no chlorosis; 4 = spots covering 20-30% leaflet area, no chlorosis; 5 = spots covering 30-40% of leaflet area, no chlorosis; 6 = spots covering 20-40% leaflet area, each spot surrounded by chlorotic halo; 7 = spots covering 20-40% leaflet area, entire leaflet mildly chlorotic; 8 = entire leaflet severely chlorotic; and 9 = abscission of one, two, or all three leaflets. The scale was not based solely on number of spots per leaflet, since spots were of various diameters and caused various degrees of chlorosis. Ratings were made by visually dividing a plant into two to four horizontal areas (depending on plant size) and rating four to 10 leaves within each area. The disease severity index (DSI) is the average of all ratings for each plot within an experimental replicate for each time the ratings were made. Ten plants per 10 m row were usually rated.

Plant age and disease development.—The cultivars Prima and New Era, that are susceptible to *C. canescens* and *C. cruenta,* respectively, were planted in each of two 6-m rows at weekly intervals for 8 consecutive weeks. Each weekly planting of both cultivars was randomized in each of four 16-row blocks. Spreader rows of both cultivars, which were severely diseased during the course of the experiment, were planted around each block as border rows 4 weeks before the test rows to ensure a constant supply of airborne conidia throughout the experiment. The plots were sprinkler irrigated. One week after the last planting, weekly disease ratings were made in all eight plantings using the disease severity scale. DSI's were calculated as the average of all ratings for specific plant ages in each of the four blocks.

Establishment of epidemics.—Each of four experimental blocks consisted of two subplots, both planted to the same 10 cowpea accessions from the IITA collection. Each accession differed in susceptibility to *C. canescens*. Spreader rows of the susceptible cultivar Prima were planted around each block 4 weeks before the test rows were planted. One subplot of each block remained nonsprayed, whereas the other was sprayed to run-off at 2-week intervals with 0.2 percent a.i. benomyl beginning at flowering. Disease severity indexes were determined twice weekly in the nonsprayed plots. Percent yield loss for each cultivar was calculated as:

$$\frac{(\text{yield of sprayed}) - (\text{yield of nonsprayed})}{\text{yield of sprayed}} \times 100.$$

Because of severe insect attack or susceptibility to anthracnose (*Colletotrichum lindemuthianum*) for four of the original 10 cultivars, only six were used in the final statistical anlaysis. Disease progress curves for each of the six cultivars in each of the four blocks were constructed that related DSI to days after flowering.

Simulated epidemic by defoliation.—Assuming the major effect of the pathogen is to reduce photosynthetic area, various "disease levels" were simulated by removing either one, two, or all three leaflets of each trifoliolate leaf. Leaflet removals approximated DSI's of 4, 7, and 9, respectively.

In the first simulated epidemic, one, two, or all three leaflets were removed at four weekly intervals beginning at first flower. Each removal of one or more leaflets for each time period represented a treatment (12 treatments and a control). One week after each defoliation, any new trifoliolate leaves that had expanded were treated similarly. All treatments in both simulated epidemics were protected against naturally occurring Cercospora leaf spot with benomyl sprays at flowering and 2 weeks after flowering. Plots were harvested at 67 days after planting.

The second simulated epidemic was similar to the first except that progressive increases in disease progress curves were simulated within each treatment. This was accomplished by removing one leaflet from each trifoliolate leaf each week until the desired "disease level" was attained. Simulated disease progress curves were initiated at three weekly stages beginning 1 week after flowering. Treatments consisted of single leaflet removals at the following weekly stages: (i) 1, 2, and 3; (ii) 1 and 2; (iii) 1 only; (iv) 2 and 3; (v) 2 only; and (vi) 3 only. Plots were harvested at 71 days after planting. Percent yield losses were calculated by comparison to nondefoliated controls.

Chemically regulated epidemics also were evaluated. Spreader rows of cultivar Prima, planted around each experimental block 1 month before the test rows were planted, were severely infected throughout the experiment. Treatments consisted of 0.2 percent a.i. benomyl sprays at (i) 1, (ii) 1 and 3, and (iii) 3 weeks after flowering. Controls remained nonsprayed throughout the experiment. Disease severity index ratings were made twice weekly from flowering to harvest. Harvests were taken from all plots at 60 and 67 days after planting and the yield data were combined. Yield data from this experiment were presented in an abstract on chemical control of Cercospora leaf spot (10).

In developing models for estimating yield loss, treatments within experimental blocks were considered as separate epidemics with unique disease progress curves. Yield data from all experiments were expressed as percent yield loss by comparison to the disease-free treatments within each experimental block.

Areas under each of the disease progress curves were calculated by dividing the curves into segments corresponding to the timed intervals when disease ratings were made. Each segment was thus represented by a trapezoid whose unequal heights were measured in terms of DSI's at two times of observation, and the length being the number of days between observations. The total area under each progress curve was the sum of all the areas of the component trapezoids plus the area of the triangular segment between the last observation with a DSI of zero and the first observation with a DSI greater than zero. This relationship was expressed by the following formula:

$$A = T_f \left(\frac{DSI_f}{2}\right) + T_i(DSI - DSI_l) + T_i \left[\frac{(DSI - DSI_f) - (DSI - DSI_l)}{2}\right]$$

in which:

A = area under the disease progress curve,

T_f = number of days between the last observation after flowering with a DSI = 0 and the first observation with DSI > 0,

T_i = average interval (days) between observations,

DSI_f = first DSI > 0, and

DSI_l = last observed DSI at harvest.

RESULTS

The two disease progress curves for cultivars Prima and New Era showed that Cercospora leaf spot symptoms did not appear until after the onset of flowering, even though sufficient inoculum was present and environmental conditions were favorable for disease development (Fig. 1). Symptoms progressed acropetally. The disease then progressed logarithmically until the first harvest (60-70 days after planting). By this time there were very few noninfected leaves at the tops of the plants.

Analysis of epidemics.—All of the Cercospora leaf spot

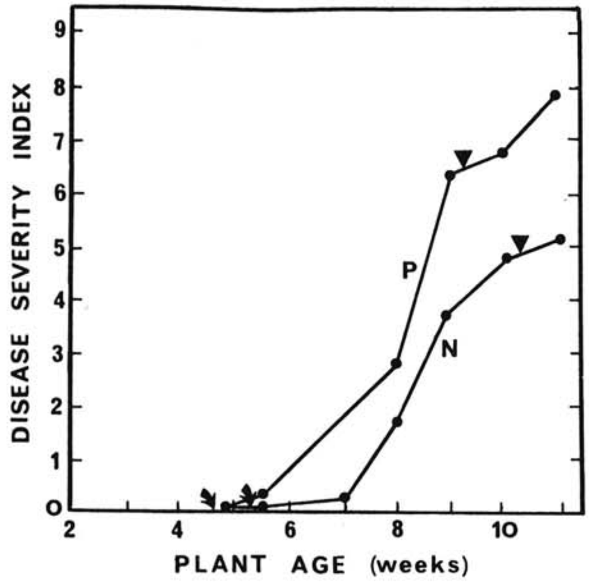

Fig. 1. Disease progress curves for Cercospora leaf spot of the cowpea [*Vigna unguiculata* 'Prima' (P) and 'New Era' (N)]. The curves are based on a disease severity scale of 0-9, where 0 = no disease and 9 = complete defoliation, at given plant ages. Arrows and triangles indicate times of flowering and first harvest, respectively.

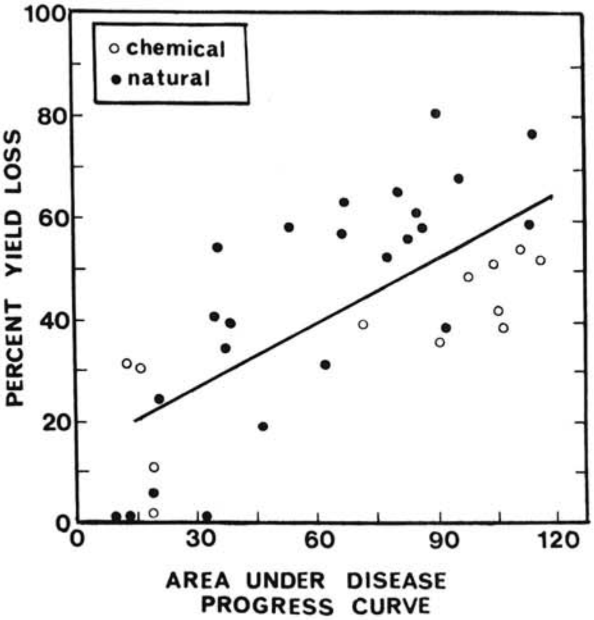

Fig. 3. Relationship of area under disease progress curves and percent yield losses for the combined data of 24 natural and 12 chemically-regulated Cercospora leaf spot epidemics of cowpea (*Vigna unguiculata*). The regression equation is Y = 0.43 X + 14.95 with a coefficient of determination of 0.702 which is significant at $P = 0.01$.

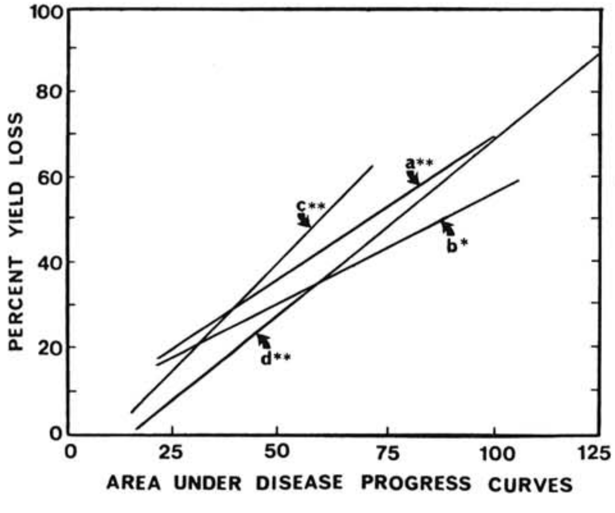

Fig. 2. Relationships of areas under disease progress curves to percent yield losses for: natural (a), chemically regulated (b), and two artifically simulated (c and d) Cercospora leaf spot epidemics of cowpea (*Vigna unguiculata*). Coefficients of determination for a, b, c, and d are 0.826, 0.684, 0.846, and 0.689, respectively, which are significant at $P = 0.01$ (**) and $P = 0.05$ (*).

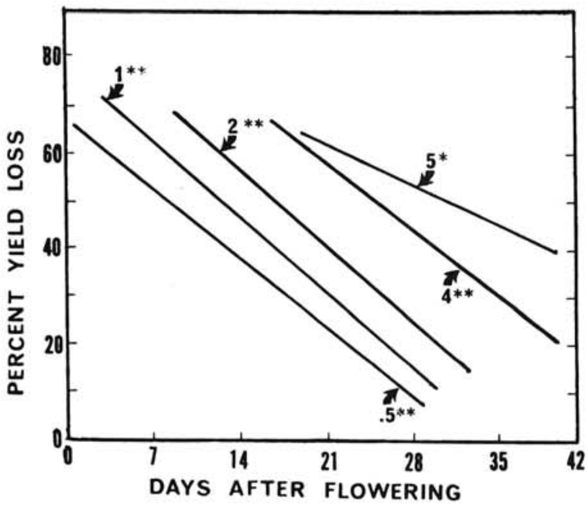

Fig. 4. Relationship of percent yield loss of cowpea (*Vigna unguiculata*) to time of occurrence (days after flowering) of Cercospora leaf spot at five disease severity indexes (DSI). Coefficients of determination for DSI's 0.5, 1, 2, 4, and 5 are −0.680, −0.746, −0.725, −0.697, and −0.485, respectively. Correlations are significant at $P = 0.01$ (**) and $P = 0.05$ (*).

epidemics were analyzed by correlating areas under disease progress curves (AUDPC) with percent yield loss. The coefficients of determination (r^2 values) for the linear regressions of percent yield loss and AUDPC for the combined data of the naturally occurring (differential cultivars), chemically regulated, and two simulated epidemics were 0.826, 0.684, 0.846, and 0.689, respectively (Fig. 2). Regressions A, C, and D were significant at $P = 0.01$; regression B was significant at $P =$

0.05. Results of an analysis of variance of the slopes of these four regression lines indicated that there were no significant differences among the slopes ($P = 0.10$).

Combined data from the natural and chemically regulated epidemics were used to calculate a single regression equation,

$$Y = 0.43\ X + 14.95,$$

where Y = percent yield loss and X = AUDPC (Fig. 3). The variance of the slope (S_b^2) was 0.0051 and the r^2 was 0.702 which was significant at $P = 0.01$.

Data from the 24 natural epidemics were used to develop a model for estimating yield loss based on time of occurrence of specific DSI's (Fig. 4). Regression equations for percent yield loss (Y) and days after onset of flowering (X) for occurrence of DSI's 0.5, 1.0, 2.0, 4.0, and 5.0 were $Y = -2.1\ X + 69.0$; $Y = -2.3\ X + 80.0$; $Y = -2.2\ X + 89.0$; $Y = -2.0\ X + 102.8$; and $Y = -1.2\ X + 87.9$, respectively. The r^2 values were −0.680, −0.746, −0.725, −0.697, and −0.485, respectively. Regressions for DSI's 0.5 - 4.0 were significant at $P = 0.01$, but that for DSI 5.0 was significant only at $P = 0.05$. The S_b^2 values for these regressions were 0.181, 0.116, 0.127, 0.192, and 0.390, respectively.

The relationships between AUDPC's and percent yield loss and time of occurrence of DSI=1.0 (the critical point) and yield loss were tested with the data from the chemically regulated epidemics (Table 1). These data were used, in part, to establish the former relationship but not the latter. These results indicated that the AUDPC:percent yield loss relationship is quite accurate when compared to actual yield loss data. The greatest disparity between estimated and actual yield loss occurred at the lowest disease level with an estimated value of 21 percent and an actual value of 7 percent. The critical point model, which was not constructed with data from this experiment, was also quite accurate in estimating yield loss. It could be used only for the nonsprayed treatments, since the disease progress curves of the chemically regulated epidemics were altered.

DISCUSSION

Cercospora leaf spot of cowpea occurred only after the onset of flowering even though sufficient inoculum was present, and temperature and moisture were favorable for disease development. This was clearly demonstrated in side-by-side comparisons in which older plants were severely diseased and plants in the preflowering stages of growth were disease-free. Schneider and Sinclair (9) showed that one or more preformed fungitoxic compounds were associated with young leaves of a susceptible cultivar and all leaves of a resistant cultivar. After the disease began it progressed logarithmically until just prior to harvest. There were no plateaus in any of the naturally occurring disease progress curves in this study.

The lack of statistically significant differences among slopes for the regressions of percent yield loss on AUDPC for the chemically regulated, naturally occurring, and simulated epidemics is significant for two reasons. This indicated that: (i) most yield loss resulted from defoliation, not from a systemic toxicosis caused by the fungus, and (ii) the disease severity index is a valid measure of the effect of disease on seed yield. The primary criterion for any disease severity scale is its validity (1, 6).

The positive correlation of AUDPC and percent yield loss confirm van der Plank's (12) hypothesis that this is an accurate method for estimating yield loss. James (5) observed that predicting yield losses in potato infected with *Phytophthora infestans* by calculating AUDPC was not as reliable as using a multiple point model since AUDPC did not distinguish between early light infections and late severe infections. This was probably the case for the regression equation derived from the combined data of the chemically regulated and naturally occurring epidemics. An area of 12 would estimate a yield loss of 21 percent, even though only a 7 percent yield loss was actually realized. This probably resulted from the inability of this model to distinguish between an early mild infection and a late severe infection, as postulated by James (5), both having areas less than 45. James (4) stated that for a critical point yield loss estimation model to be effective, the disease should be of short duration and occur while the plant is accumulating yield. Our data show that this may also be the case with AUDPC models for diseases which meet the above criteria. Any interruption during the period of dry weight accumulation, seeds in this case, would be expected to have a detrimental effect, whether it is mild early infection or a severe late infection. Enyi (2) reported that defoliation of cowpea after the early pod-filling stage was most effective in reducing yield, since this is the period when seed dry matter is accumulating. This is also the

TABLE 1. Relation of times of application of benomyl to cowpea yield and areas under Cercospora leaf spot disease progress curves. Actual percent yield losses are compared to estimated losses based on two yield-loss estimation models

Time of benomyl application (weeks after flowering)[a]	Area under disease progress curve	Yield (kg/H)[a]	Actual percent yield reduction	Estimated percent yield reduction (95% confidence interval)[b]: Critical point model	Area under disease progress curve model
1,3	0	1279 x	...	...	...
1	12	1195 xy	7	...	21 (±32)
3	21	985 yz	23	...	24 (±16)
Nonsprayed control	65	748 z	42	45 (±15)	46 (±15)

[a]Data presented in (10). Values followed by the same letter(s) are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

[b]Disease severity index (DSI) = 1.0 occurred at 15 days after flowering.

period of greatest susceptibility to Cercospora leaf spot.

The critical point model (3) for estimating yield loss was also evaluated in this study. Provided that the progress of the disease was uninterrupted (by fungicides) the linear correlations between specific DSI's occurring at various times after the onset of flowering and percent yield loss were statistically significant. This model was tested in a separate experiment which was not used to establish the original relationship. This system also could be adapted to a multiple point (4, 5) approach by determining DSI's of uninterrupted epidemics at various times after flowering. Thus, if the first DSI rating indicated a severe yield loss and subsequent observations indicated moderate losses, corrections could be made in the prognosis during the course of the epidemic. We did not establish such correction factors in this study. The critical point model probably would be adequate for estimating yield loss of cowpea affected with Cercospora leaf spot since it fulfills the criteria established by James (4) for such a model. The disease is of short duration and only occurs during the period of product (seed) accumulation.

The AUDPC and critical point models could be integrated to form a rational decision-making scheme on whether or not to use fungicides at particular times during the growing season. A critical point observation may indicate a given percent yield loss. Even if the disease is controlled at that stage, there will be some yield loss resulting from early infection. This yield loss could not be estimated on the basis of a critical point model since the disease progress curve would be altered by the fungicide. An AUDPC analysis would estimate this yield loss even though a fungicide was applied. Assuming that the disease progress curve reached a plateau after the fungicide was applied, the area under the remaining portion of the curve would be roughly rectangular with an area calculated as follows:

Area = (days from fungicide application to harvest) × (DSI at time of fungicide application).

This area would then be added to the AUDPC for the time previous to the fungicide application; the sum being the total projected AUDPC of the chemically altered disease progress curve. Analysis of the epidemics from this study indicated that late applications of benomyl resulted in plateaus in the disease progress curves, whereas early applications resulted in slight depressions of the progress curves. This resulted from the production of new, noninfected leaves which increased the ratio of healthy to diseased leaves.

An integration of the critical point and AUDPC models would therefore allow one to estimate whether a chemical control program would be economically judicious based on expected yield losses for noncontrolled and controlled disease situations.

Although these studies were conducted during three climatologically distinct growing seasons within 1 year in tropical Africa, the results can be viewed only as models. Spreader rows were always heavily diseased around plots, which were sprinkler irrigated, so that the disease always progressed to its fullest potential under the various environmental conditions. Thus, these models only estimate the maximum percent yield loss which can be expected for a given disease situation. These methods for estimating yield loss should be tested and modified in different areas during several growing seasons before they are used.

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