Evaluating a Crop Loss Model for Head Smut of Sorghum

D. M. Tuleen and R. A. Frederiksen

Respectively, former research associate and professor, Department of Plant Sciences, Texas A&M University, College Station 77843.
Contribution of the Department of Plant Sciences. Authorized for publication as TA-17012 in the Journal Series of the Texas Agricultural Experiment Station.
This work was supported in part by funds provided by AID/DSAN/XII/G-0149. The authors gratefully acknowledge the technical assistance at each trial location.
Accepted for publication 24 March 1982.

ABSTRACT

Tuleen, D. M., and Frederiksen, R. A. 1982. Evaluating a crop loss model for head smut of sorghum. Phytopathology 72:1278-1280.

A model simulating grain sorghum yields relative to the incidence of head smut was evaluated. Grain sorghum hybrids were inoculated in the seedling stage with *Sphacelotheca reiliana* by the hypodermic injection technique in four field trials at three locations in Texas. Percentages of infection were determined at anthesis; grain crop yields were calculated in grams per panicle and kilograms per hectare. The percentage of grain yield loss relative to uninoculated plots was directly proportional to the percentage of plants with smutted or phylloid panicles with regression coefficients from 0.84 to 1.09 and R^2 from 0.39 to 0.80.

Additional key words: *Sorghum bicolor*.

A disease-loss model is a convenient way to mathematically represent the adverse effects of a disease on crop yield in affected fields. As a subroutine in a crop growth simulation model, it is a valuable tool growers can use to define economic levels of the disease and appraise potential management procedures. A model developed by Arkin et al (1) has reliably simulated grain yield in grain sorghum (*Sorghum bicolor* (L.) Moench); however, the numerous pathogens responsible for yield reductions in this crop were not considered in the initial model. Quantitative information is needed to evaluate disease relative to crop yield.

The objective of our experiment was to test a crop loss model for head smut of sorghum. Head smut, which is caused by *Sphacelotheca reiliana* (Kuehn) Clint., has been responsible for major losses in Texas and other sorghum-growing areas of the world (4). The disease occurs endemically in many of these areas. It is generally controlled by genetic resistance; however, variation in the pathogen has necessitated several shifts in genetic sources of resistance. At present, four races of *S. reiliana* have been reported (4). We studied the sorghum head smut system because a relatively effective method of artificial inoculation was available to induce systemic infection and because we could provide sufficient control plots (uninfected) for statistical comparisons. In the past, inaccurate predictions by disease-loss models have been attributed to the lack of zero disease (control) plots and/or too few observations. Sufficient observations to provide a low experimental error are necessary so that the effects of disease can be adequately defined.

MATERIALS AND METHODS

Cooperative field trials were conducted in Texas near College Station (Texas Agricultural Experiment Station research plots) and Victoria during 1975, and on the Blackland Research Center near Temple in 1971. All experiments were carried out similarly unless otherwise stated. A randomized block design was used. Four treatments, replicated six times at College Station (planted 30 March 1975) and Temple and four times at College Station (planted 19 April 1975) and Victoria resulted in 24 and 16 plots, respectively. Plots 6 m long at Temple and 4 m long at other locations consisted of single rows 1 m apart; plots were rogued to a crop density of 12 plants per meter of row. Cultivars RS610, NK233, and RS671 were used at College Station, Victoria, and Temple, respectively. All three cultivars react similarly to *S. reiliana*. Plots were managed to favor plant growth and yield with wide spacing between plants to avoid competition.

Seedlings at the four- to five-leaf stage were inoculated 1 mo after planting by using the hypodermic injection technique described by Edmunds (3). The four treatments were designed to produce four levels of disease incidence: 100, 50, 25, and 0% of the plants, respectively, were inoculated.

The pathogen was maintained as compatible haploid monosporidial lines on potato-dextrose agar. Cultures were increased in potato-dextrose broth in Erlenmeyer flasks on a reciprocal shaker for 4 days at ambient temperature. We combined equal quantities of broth suspension of each monosporidial line and injected the mixed suspension into the nodal area below each shoot apex by using a hypodermic syringe fitted with a 25-gauge (0.51 mm diameter) needle.

Disease assessments were made prior to harvest. The number of infected panicles, as indicated either by the smut sori or phylloid panicles described by Wilson and Frederiksen (8), was recorded as a ratio to the number of uninfected panicles in the same plot. Uninfected panicles that produced grain were harvested when the grain was mature. The numbers of these healthy panicles per plot were recorded for College Station and Victoria. Grain yields were corrected to 13% moisture and calculated in kilograms per hectare. Yield loss for each diseased plot was computed as a percentage of the mean yield for 'zero disease' control plots at the respective locations. Analysis of variance was performed on yield data and linear regression analysis was used to evaluate the relationship between yield loss and disease incidence. Data were analyzed using the SAS (Statistical Analysis System) computer program (2). The level of statistical significance used for all evaluations was $P=0.05$.

The crop loss model is a first degree equation, in which $Y=$ grain yield and x = proportion of disease, with a y-intercept at zero infection and zero yield at 100% infection.

RESULTS

Grain yields were reduced significantly in all trials following inoculation, on both a per hectare and per panicle basis. When treatment differences were evaluated for each trial (Table 1), losses per hectare presented as a percentage of the disease-free control were consistently related to increased disease incidence with two

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.

0031-949X/82/10127803/$03.00/0

TABLE 1. Mean grain yields and incidence of head smut for four inoculated sorghum trials in 1971 and 1975[a]

Location and planting date	Treatment (% inoculated)	No. of panicles[b]	Panicles with head smut (%)	Yield[b] (kg/ha)	Loss (%)	Yield[b] (g/panicle)	Loss (%)
College Station,	0	33.5 t	0.0	4,853 q	...	59.0 s	...
30 March 1975	25	39.3 u	17.3	4,352 qr	10.3	51.3 t	13.1
	50	41.8 u	24.7	3,617 r	25.5	48.3 t	18.1
	100	57.5 v	30.7	4,188 qr	13.7	39.9 u	32.4
College Station,	0	33.3 w	0.0	3,308 s	...	43.2 vw	...
19 April 1975	25	34.5 w	15.9	3,325 s	−0.5	46.5 v	−7.6
	50	41.8 x	27.3	2,625 st	20.6	37.2 w	13.9
	100	44.8 x	38.4	2,135 t	35.5	28.6 x	38.5
Victoria,	0	37.3 y	0.7	4,644 u	...	38.6 y	...
10 March 1975	25	35.8 y	26.3	3,100 v	33.2	38.3 y	0.8
	50	39.3 y	37.1	2,834 v	39.0	36.3 y	6.0
	100	52.5 y	66.7	1,011 w	78.2	24.0 z	37.8
Temple,	0	45.0 z	0.0	3,850 x	...	...	...
12 April 1971	25	46.2 z	16.6	3,055 y	20.6	...	...
	50	44.5 z	23.6	2,950 y	23.4	...	...
	100	48.8 z	37.2	2,164 z	43.8	...	...

[a] Means of six replications at College Station (early planting) and Temple, four replications at College Station (late planting) and Victoria.
[b] Means followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's new multiple range test.

notable exceptions, both in the College Station trials. Losses at Victoria and Temple correspond relatively well with the disease incidence for each of the four treatments and were greater than the corresponding disease incidences. Although losses were significant in both College Station trials, they were less than their corresponding disease incidences. The slight yield increase with the 25% treatment in the late College Station planting was not statistically significant. With exceptions in the 25% treatment plot for the late College Station planting and the 100% treatment plot for the early College Station planting, yield losses were related to the incidence of head smut. Tillering can occur in sorghum as a consequence of the inoculation procedure used in this test. Puncturing the apical meristem occasionally initiates a lateral shoot. The lateral shoot, when it appears, grows less vigorously than the main shoot, flowering 7–10 days later. Panicles on both primary and lateral shoots usually exhibit the same reaction to inoculation: either both will be symptomless or, in a systemically infected plant, both will be smutted or sterile. Only rarely did a tiller have an infected panicle when the panicle produced by the primary shoot remained uninfected. An increase in the number of panicles (infected plus uninfected), which corresponds to an increase in the percentage of plants inoculated in the College Station trials, can be attributed to tillering. No significant difference in panicle numbers was observed at Victoria or Temple; however, differences in yield per uninfected panicle were observed for treatments at all locations for which data were available. Losses per panicle (uninfected) increased correspondingly with an increase in disease incidence at the three locations for which data were available. The differences in yield potentials (mean yields from disease-free control plots) among the four trials are a consequence of different cultivars and growing seasons. Therefore, yield data were transformed to loss as a percentage of the yield potential for each of the four trials. When these losses were plotted against the incidence of head smut, the relationship was essentially 1:1. Equations for regression lines of loss/head smut incidence with their corresponding R^2 values are shown in Table 2. The lower R^2 values for Temple and the later College Station trial indicate that the model's fit was less satisfactory for these locations. The greatest deviation from a slope of one, which indicates a 1:1 relationship, is seen in the early College Station trial. Slope comparisons were made according to the procedure of Neter and Wasserman (5). Since the statistical analysis indicated that the four regression lines in Table 2 were not significantly different, the data from the four trials were pooled for purposes of estimating a common slope (Fig. 1). The proximity of the y-intercept to zero and slope of one indicates a linear relationship between yield loss and head smut incidence.

TABLE 2. Regression analyses of sorghum grain yield loss/incidence of head smut for four field trials at three locations in Texas in 1971 and 1975[a]

Location and planting date	Cultivar	Regression equation[b]	R-square
College Station, 30 March 1975	RS610	$\hat{Y} = 0.84x - 2.63$	0.671
College Station, 19 April 1975	RS610	$\hat{Y} = 1.09x - 8.30$	0.510
Victoria, 10 March 1975	NK233	$\hat{Y} = 1.04x + 2.26$	0.800
Temple, 12 April 1971	RS671	$\hat{Y} = 0.98x + 3.33$	0.391

[a] Six replications at College Station (early planting) and Temple, four replications at College Station (late planting) and Victoria.
[b] $\hat{Y}$ = percentage yield loss relative to an uninfected plot; x = percentage of smutted or phylloid panicles.

DISCUSSION

Disease-loss models involving pathogens with secondary cycles of infection and the dimension of time (for instance, models describing foliar disease epidemic development) are considerably more complex than the single-point model we have suggested for head smut. Our model assumes a simple linear relationship between yield loss and the percentage of infected plants in a population because the period of invasion and establishment of infection by *S. reiliana* is brief; plants that escape infection as seedlings cannot be infected later. Disease escape is a factor contributing to the reduction of disease at the epidemiological level and therefore is an important consideration in simulating yield with head smut.

S. reiliana survives in the soil as chlamydospores that subsequently germinate and infect seedlings prior to the migration of apical meristem above soil level. The organism colonizes meristematic tissue producing, instead of the floral primordium, a mycelial mat intermixed with vascular bundles and encased in the flag leaf sheath of its host. The life cycle is completed when the peridium of the smutted head bursts and a shower of black chlamydospores fall to the ground. Prediction of disease-loss appears to be academic in the case of head smut. Unlike sorghum downy mildew, in which reliable disease estimates can be obtained early so that therapeutic action may be taken if the predicted losses exceed the economic threshold (7), estimates of head smut incidence are made at anthesis. Predictions at this time are too late for the grower to take any action to reduce disease-loss.

With some diseases, as we have reported for sorghum downy mildew (7), disease loss is not directly related to disease incidence because healthy plants compensate for the loss of diseased seedlings

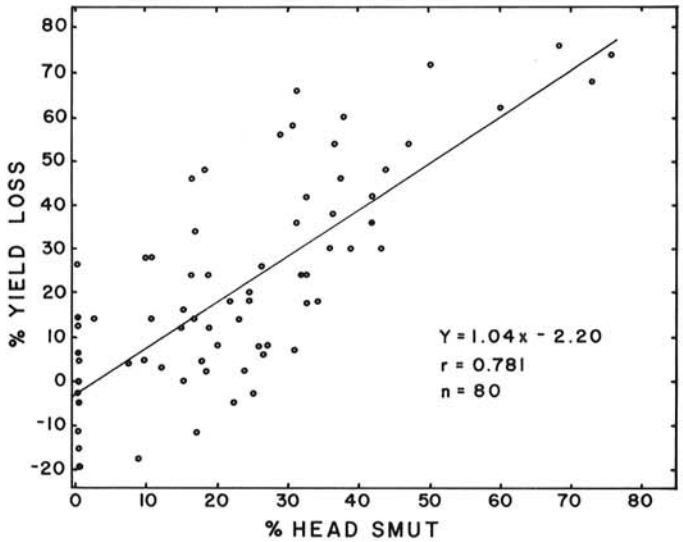

Fig. 1. Regression of sorghum grain yield loss on incidence of head smut (percentage of diseased panicles) for four data sets from field trials in Texas; nine hidden observations occur.

by increased growth and yield in response to the reduced competition. Healthy plants adjacent to diseased or dead plants may produce more grain per panicle or they may tiller, as is the case with some sorghum genotypes, and produce more panicles per plant. We would not expect this type of yield compensation with head smut, because infected plants remain throughout the growing season and the smutted panicles probably compete for light, water, and nutrients the same as those on uninfected plants do.

Our results suggest that yield is essentially reduced as would be expected from arithmetic thinning of the crop. However, two sources of error were possible in these experiments which would counteract each other. First, the tillering phenomenon previously described probably accounts for significant differences in numbers of panicles (infected plus uninfected) for the two College Station trials (Table 1). Tillering in the uninfected plants failed to completely compensate for the loss of yield from diseased plants, since crop yields for these trials decreased with increasing numbers of inoculated plants. Tillering in uninfected plants could account for the decrease in yield per panicle, since tillers appear and mature later and develop less vigorously in competition with the main shoots.

The second possible source of error is in the determination of disease incidence. It is possible that some of the inoculated, symptomless plants were infected and that the infection reduced yield of the plants even though the fungus did not colonize the inflorescence. Using the 100% treatment as a basis for comparing inoculation efficiency, we observed that symptomless panicles accounted for approximately 69, 62, 33, and 63% of the total number of panicles observed at four respective locations. If some of the symptomless, inoculated plants were infected, energy in those plants may have been diverted to biochemical defense reactions as shown by Smedegaard-Petersen and Stølen (6) for a resistant barley cultivar infected with powdery mildew. Losses in yield from asymptomatic plants could be an important consideration in the selection of disease-resistant or disease-tolerant cultivars.

In summary, a single-point model satisfactorily described the relationship between incidence of head smut and measured grain yields with the statistical precision desired in four sorghum field trials. The limitation imposed by regression analysis is evident; since biological processes are rarely linear, some divergence in actual yield from estimated yield can always be expected. With better information about the effects of inoculum density, soil types, and environmental conditions on infection and symptom development, a multivariant model could be constructed to better account for the disease incidence/disease-loss relationship.

LITERATURE CITED

1. Arkin, G. F., Vanderlip, R. L., and Ritchie, J. T. 1975. A dynamic sorghum growth model. Trans. Am. Soc. Agric. Eng. 19:622-626, 630.
2. Barr, A. J., Goodnight, J. H., Sall, J. P., Blair, W. H., and Chilko, D. M. 1979. SAS User's Guide. Statistical Analysis Institute Inc., Raleigh, NC. 494 pp.
3. Edmunds, L. K. 1963. Use of sporidial hypodermic injection to test sorghum for head smut resistance. Plant Dis. Rep. 47:909-913.
4. Frederiksen, R. A., and Reyes, L. 1980. The head smut program at Texas A&M. Pages 267-469 in: Proc. of the Int. Workshop on Sorghum Diseases, Hyderabad, India, 1978. ICRISAT, Patancheru, A. P., India. 469 pp.
5. Neter, J., and Wasserman, W. 1974. Applied Linear Statistical Models. Richard D. Irwin, Inc., Homewood, IL. 842 pp.
6. Smedegaard-Petersen, V., and Stølen, O. 1981. Effect of energy-requiring defense reactions on yield and grain quality in a powdery mildew resistant barley cultivar. Phytopathology 71:396-399.
7. Tuleen, D. M., and Frederiksen, R. A. 1981. Simulating yield losses in grain sorghum due to sorghum downy mildew. Agron. J. 73:983-987.
8. Wilson, J. M., and Frederiksen, R. A. 1970. Histopathology of the interaction of *Sorghum bicolor* and *Sphacelotheca reiliana*. Phytopathology 60:828-832.