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Development of gray leaf spot from inoculated foci on two maize cultivars in Uganda

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Abstract

The spatio-temporal spread of gray leaf spot on maize caused by *Cercospora zeae-maydis* was studied in two locations in central Uganda. Gray leaf spot development was assessed for three cropping seasons at 0.75 m to 5.25 m from inoculated foci on two open-pollinated maize varieties. In all the seasons, disease spread was adequately depicted by both power and exponential models. Distance from inoculum focus significantly ($P<0.05$) affected leaf damage but the effect of direction from inoculated focus was variable depending on the season, location and cultivar. Thus, depending on the location and seasonal variation, genotype effect greatly affects severity of gray leaf spot. Gradients for the two cultivars differed significantly ($P<0.001$) in intercept (a) but not slope (b) of the linearised power disease gradient for the number of lesion on the ear leaf. Our results indicate that maize genotypes affect both increase and spread of gray leaf spot. The rapid flattening of the disease gradient was indicative of long distance dispersal of *C. zeae-maydis* in tropical unlike in temperate environments.

Key words: *Cercospora zeae-maydis,* disease gradients, *Zea mays*

Introduction

Cercospora zeae-maydis (Tehon and Daniels), causal agent of gray leaf spot is an important pathogen of maize *(Zea mays)* world-wide (Ward *et al.*, 1999). The fungus survives on crop residues that is often instrumental in the development of gray leaf spot epidemics (Payne *et al.,* 1983). When conditions are favourable, sporulation has been reported to occur on the surface of crop residues, producing abundant conidia that constitute primary inocula for nearby maize plants (Summer *et al.*, 1981; Lipps *et al.,* 1983). The characterisation of gray leaf spot spread from an inoculum source and selection of empirical models to describe spatial pattern of disease are necessary to further advance understanding of the disease dynamics (Cambell and Madden, 1990). Parameters estimated with this approach can be employed as tools to study the spatial aspect of the disease inoculum foci (Jeger, 1989; Cambell and Madden, 1990). Despite this, there is limited information on the spatial component of gray leaf spot under Ugandan conditions. Since land fragmentation is common in most parts of Uganda, the same plot of land may be grown to maize season after season. In such cases, crop debris may become an important source of inoculum. Hence, it is important to understand the role of local inoculum source on gray leaf development and associated disease parameters. The description of disease gradients from a source of inoculum allows for estimation of the rate of spatial spread of disease. Moreover, it is possible to determine the relative potential for disease spread within different maize cultvars by comparing slope (b) of the gradient, which describes the rate of disease decrease from inoculum source (Gregory, 1968). The objective of this study was therefore to characterize the spatio-temporal patterns

of gray leaf spot epidemics, and to determine the effect of maize genotype and direction from inoculated foci on spread of *C. zeae-maydis* on maize in a bimodal agroecology of central Uganda.

Materials and methods

Host genotypes

Two open-pollinated maize cultivars, Longe 1, a moderately resistant and popcorn, a susceptible cultivar (Bigirwa *et al.,* 1999) were used to study the spread of gray leaf spot from inoculum foci. Longe 1 is a commercial cultivar grown in Uganda bred at Namulonge Agricultural and Animal Production Research Institute while popcorn is also a commercial local cultivar that was introduced by a local seed company.

Field plots

Field plots were established during the first rains (March – July) of 1999 at two locations, Makerere Agricultural Research Institute, Kabanyolo and Kyambogo. The experiment was repeated during the second rains (September – December) of 1999 and the first rains (March – July) of 2000. These are subsequently referred to as 1999A, 1999B and 2000A, respectively. The plots were laid on land that was previously planted with sweetpotatoes (*Pomoea batatas* L.) at Kabanyolo and fallow for more than five years at Kyambogo. In both locations, the plots were tractor-ploughed and disc-harrowed before planting. The trials were planted on 19th March 1999 and 29th April at Kabanyolo and Kyambogo, respectively. The plots were arranged in a randomised complete block design with three replications. The experiment was repeated in two subsequent seasons with planting done on 25 September 1999 and 13 April 2000 at Kabanyolo and 1 October 1999 and 21 April 2000 at Kyambogo. Each experimental unit measured 16 x 16 m with 16 maize rows planted at a spacing of 75 x 30 cm. All plots were hand planted with two or three seeds per hill to ensure germination in all the plots but the seedling plants were later thinned to one to maintain a plant population of about 44,000 plants per ha. In both cases, plots were hand weeded at V3 and V8 growth stages (Ritchie *et al.,* 1989).

Inoculation and disease assessment

At the centre of each plot, an area of 1.5 x 1.5 m was marked with a coloured stain to establish inoculum foci. All plants within inoculum foci were inoculated by placing approximately 5 – 10 *C. zeae-maydis* colonized sorghum seeds into the whorls of each plant at V6 growth stage (Ritchie *et al.,* 1989). One pair of plants in each compass direction (N, S, W and E) was tagged at approximately 0.75, 1.5, 2.25, 3.0, 3.75, 4.5 and 5.25 m distance from the edge of the inoculated foci. The tagged plants in each direction were assessed for percentage ear leaf area affected (PELAA) as described by Freppon *et al.* (1996). In addition, the number of gray leaf spot lesions on ear leaf of the tagged plants were counted and used for statistical analysis. Disease assessments commenced 40 days after inoculation at both Kabanyolo and Kyambogo, which corresponded to approximately the R1 growth stage (Ritchie *et al.,* 1989). A total of 4 assessments were made at 7 – 10 days intervals.

For each distance from the inoculum foci, the mean PELAA and gray leaf spot lesion counts were calculated for each cultivar, gradient point and direction of disease assessment. When no significant differences were detected between directions, data for the four compass points were pooled for both cultivars and locations. Data collected at different times were used to compute area under disease progress curve (AUDPC), slope of gradient (b), apparent infection rates (r), and intercept (a). The power model, $y = axb$ (Gregory, 1968) and exponential model, $y = a\exp(-bx)$ (Kiyosawa and Shiyomi, 1972) were linearised after log transformation and fitted to the pooled data for each replicate. These

models were selected because of their common usage and because similar studies by Asea (2001) on the spread of gray leaf spot from colonized maize residue indicated their suitability. In these models, y is the proportion of disease severity at x units of distance from the inoculum foci and b is the slope. The appropriateness of each model was evaluated on the basis of coefficients of determination (R^2) and plots of residuals against time and distance. Analysis of variance (ANOVA) was used to determine the effect of genotypes, distance and direction on PELAA and lesion counts. The ANOVA was also conducted on r, b, initial disease (Y_1) and final disease (Y_f) and AUDPC. All the mean comparisons were done using Fisher's protected least significant difference (LSD) at P = 0.05.

Results

The effect of genotype on the severity of gray leaf spot was highly significant ($P<0.001$) during the three maize growing seasons. There was more disease development on the Popcorn than on Longe 1 as indicated by higher lesion counts, PELAA and AUDPC (Tables 1 and 2). Probably due to seasonal variations, there was greater gray leaf spot development during the second season 1999 than the first season of 2000 and 1999. The main effect of direction from inoculum foci on PELAA was not significant in both seasons of 1999, but significant in the season of 2000. Distance from inoculum foci did not significantly affect PELAA in the first season of 1999, but significantly affected PELAA in both 1999B and 2000A and the trend varied by location (Table 2). There was greater disease development at Kyambogo than at Kabanyolo. The interactive effect of cultivar x direction x location was significant in both 1999B and 2000A. Assessment of gray leaf spot lesion counts on the ear leaf indicated that genotype had significant ($P<0.05$) effect on final lesion count and AUDPC. Consistently, more lesions developed on Popcorn than Longe 1. For 1999A, there was significant effect of distance from inoculum source on both lesion development and AUDPC. Similarly location effect was significant on gray leaf spot development in terms of final lesion counts and AUDPC. The interactive effect of cultivar x direction x location were also significant for the same periods.

Both exponential and power models adequately depicted the spread of GLS across locations and seasons. However, power model was chosen to represent the disease spread because of its common usage (Gregory, 1968; Campbell and Madden, 1990). The main cultivar effect was highly significant ($P<0.001$) for intercept, a in both locations being higher on popcorn than on longe 1. Contrastingly, disease gradients, b were not significantly ($P>0.05$) affected by cultivar, direction and location across seasons. The b values ranged from –0.112 and –0.623 in the three seasons (Table 4).

Discussion

Host genotype had a significant effect on progress and spread of gray leaf spot from the inoculated focus. Our results and those reported earlier on another maize foliar disease, northern leaf blight of maize (Pataky *et al.,* 1986; Adipala *et al.,* 1993) suggest that in moderately resistant and resistant genotypes, spread of C. *zeae-maydis* is limited to neighbouring plants. In this study, spread of gray leaf spot from inoculated foci was dependent on the level of resistance, being restricted on resistant than on susceptible cultivars. The early studies by Payne and Waldron (1983) revealed that genotype significantly affected sporulation capacity of C. *zeae-maydis* if only local sources of inoculum are important.

The power model adequately described disease spread within each genotype grown at the two locations. Interestingly, the gradient parameter b was not significantly affected by genotype effect, yet the number of lesions declined with distance from the source plants. The slope rapidly flattened. These results indicate that apart from the focus expansion, subsequent lesions contribute to lesion development. It therefore appears that in the gray leaf spot pathosystem, genotype resistance levels are critical in generation of inoculum and spread of disease particularly if inoculum source is abundant.

Table 1. Main effect of cultivars and distance from inoculum source on initial disease (Y_i), final disease (Y_f), standardized area under disease progress curve (AUDPC) and slope (r) of logit transformed increase in lesion number of gray leaf spot lesions in two maize cultivars at two locations in Uganda, 1999A, 1999B and 2000A.

Distance	Cultivar							
	Popcorn				Longe 1			
	Y_i[a]	Y_f[b]	AUDPC[c]	r[d]	Y_i	Y_f	AUDPC	r[d]
Kabanyolo1999A								
0.75	2.25	28.0	351.7	0.08	0.0	4.86	68.0	0.07
1.5	1.58	25.0	311.1	0.07	0.0	4.16	62.3	0.07
2.25	1.08	24.0	305.1	0.08	0.0	4.2	58.1	0.09
3.0	1.16	24.0	303.8	0.07	0.0	3.62	45.8	0.07
3.75	1.33	23.0	305.6	0.08	0.0	3.11	38.1	0.06
4.5	1.08	22.0	286.9	0.09	0.0	3.20	36.2	0.07
5.25	0.66	21.0	279.9	0.08	0.0	3.00	35.1	0.07
SED	0.19	0.86	8.68	0.003	0.0	0.26	5.13	0.003
Kyambogo								
0.75	6.9	27.6	355.2	0.06	0.4	7.3	89.3	0.06
1.5	5.2	26.5	346.4	0.05	0.5	5.0	69.7	0.06
2.25	4.3	24.0	337.9	0.04	0.2	9.4	67.8	0.09
3.0	4.2	23.5	326.9	0.06	0.3	6.6	58.9	0.04
3.75	4.0	24.4	318.5	0.05	0.3	5.3	57.4	0.07
4.5	3.5	22.1	290.3	0.05	0.2	4.4	45.1	0.05
5.25	2.9	20.1	289.0	0.05	0.3	4.0	45.0	0.06
SED	0.49	0.96	9.84	0.003	0.04	0.72	5.86	0.006
Kabanyolo1999B								
0.75	6.1	64.3	555.0	0.05	0.0	8.2	60.9	0.07
1.5	3.1	44.1	396.1	0.06	0.0	5.3	55.9	0.07
2.25	3.7	39.0	365.1	0.06	0.0	4.7	48.0	0.06
3.0	4.2	37.0	357.1	0.05	0.0	6.5	37.9	0.08
3.75	4.2	35.5	396.9	0.05	0.0	3.0	36.2	0.05
4.5	4.5	29.0	311.1	0.04	0.0	4.9	27.2	0.07
5.25	3.1	20.1	311.1	0.04	0.0	2.9	33.8	0.06
SED	0.39	5.20	32.07	0.003	0.0	0.71	4.68	0.004
Kyambogo								
0.75	8.4	79.7	719.7	0.05	0.0	18.9	200.1	0.11
1.5	8.1	61.0	552.6	0.05	0.0	15.3	143.3	0.11
2.25	6.7	44.6	503.8	0.05	0.0	9.4	79.4	0.09
3.0	7.4	46.8	553.6	0.05	0.0	8.8	94.3	0.09
3.75	5.4	46.2	383.7	0.05	0.0	9.4	82.5	0.08
4.5	5.7	50.4	493.5	0.05	0.0	4.0	33.0	0.03
5.25	6.0	46.8	601.7	0.05	0.0	6.9	60.9	0.08
SED	0.45	4.82	86.93	0.00	0.0	1.92	21.08	0.01
Kabanyolo 2000A								
0.75	4.5	57.3	529.7	0.02	0.0	9.2	55.5	0.08
1.5	2.2	39.5	343.8	0.03	0.0	3.9	27.0	0.10
2.25	2.5	34.6	396.6	0.04	0.0	3.3	24.5	0.11
3.0	3.0	26.6	277.4	0.04	0.0	4.7	32.3	0.08
3.75	3.1	33.7	377.0	0.04	0.0	2.5	20.6	0.11
4.5	3.1	26.1	301.7	0.03	0.0	6.8	44.7	0.10
5.25	2.8	36.0	380.8	0.03	0.0	1.9	22.8	0.10
SED	0.28	3.96	30.96	0.003	0.0	0.98	4.90	0.005
Kyambogo								
0.75	6.0	70.3	619.4	0.02	0.0	15.1	137.3	0.08
1.5	5.6	56.0	477.1	0.03	0.0	12.4	112.7	0.06
2.25	4.8	31.1	373.8	0.03	0.0	7.5	58.4	0.08
3.0	5.7	40.9	459.0	0.03	0.0	7.0	61.5	0.07
3.75	3.9	42.2	323.8	0.03	0.0	7.8	59.3	0.09
4.5	4.1	37.5	387.8	0.04	0.0	2.5	21.5	0.10
5.25	4.2	55.3	496.3	0.03	0.0	5.1	42.6	0.08
SED	0.33	5.10	36.92	0.002	0.0	1.61	15.26	0.005

[a]Number of lesions on ear leaf 39 days after inoculation
[b]Number of lesions on ear leaf 60 days after inoculation
[c]AUDPC values divided by the number of days from the first to the disease assessment times
[d]Apparent infection rate calculated using linearised logistic model as described by Campbell and Madden (1990).

Table 2. Main effect of distance on final percentage ear leaf area affected (PELAA) and area under disease progress curve (AUDPC) during 1999A, 1999B and 2000A seasons[a].

Distance(m)	1999A		1999B		2000A	
	PELAA[b]	AUDPC[c]	PELAA	AUDPC	PELAA	AUDPC
0.75	14.0	204.7	18.7	231.2	16.2	197.7
1.5	15.7	215.6	15.4	198.7	12.8	168.7
2.25	16.7	214.9	15.1	203.1	11.9	163.4
3.0	13.7	190.8	14.6	199.8	13.1	169.3
3.75	13.7	195.1	13.6	178.5	10.8	154.7
4.5	14.3	189.7	16.1	191.3	10.7	146.6
5.25	14.9	206.7	14.2	181.1	9.6	135.0
$LSD_{(0.05)}$	NS	NS	NS	31.1	2.3	26.5

[a] Pooled data for the four directions (N, E, S and W) for both cultivars and locations.

[b] Final percentage ear leaf area affected assessed 60 days after inoculation.

[c] Area under disease progress curve calculated from PLEAA assessed 4 times and standardised by dividing the number of days between first and last assessment times.

Table 3. Main effect of distance from inoculum foci on last lesion count and area under disease progress curve (AUDPC) of gray leaf spot (GLS) during 1999A, 1999B and 2000A seasons[a].

Distance(m)	1999A		1999B		2000A	
	Lesion number[b]	AUDPC[c]	Lesion number	AUDPC	Lesion number	AUDPC
0.75	3.4	46.3	5.4	69.8	5.2	65.3
1.5	3.5	49.5	4.9	62.5	4.7	57.0
2.25	3.8	49.6	4.2	57.6	4.0	52.3
3.0	3.3	44.2	4.4	59.2	4.2	53.6
3.75	3.4	45.1	4.1	53.5	3.9	49.2
4.5	3.3	42.6	3.8	51.9	3.8	50.0
5.25	3.4	46.9	4.4	58.0	4.1	52.3
$LSD_{(0.05)}$	NS	NS	0.7	9.9	0.7	9.5

[a] Pooled data for the four directions (N, E, S and W) for both cultivars and locations.

[b] Mean final lesion count on the ear leaf 60 days after inoculation.

[c] Area under disease progress curve calculated from lesion number assessed 4 times and standardised by dividing the number of days between first and last assessment times.

Table 4. Regression of numbers of gray leaf spot lesion on the ear leaf on log_{10} distance of Cercospora zeae-maydis infested foci on two maize cultivars.

Assessment time	Cultivar					
	Popcorn			Longe 1		
	b	Yo	R^2	B	Yo	R^2
Kabanyolo1999A						
18 June	-0.42	-4.02	0.66	0.00	0.00	0.00
26 June	-0.30	-2.12	0.65	-0.39	-4.42	0.29
3 July	-0.11	-1.02	0.42	-0.25	-2.94	0.71
10 July	-0.18	-1.00	0.95	-0.26	-3.00	0.91
SED	0.07	0.71	0.11	0.08	0.93	0.21
Kyambogo						
21 July	-0.18	-2.94	0.32	0.00	0.00	0.00
30 July	-0.12	-1.96	0.51	-0.40	-4.12	0.74
7 August	-0.11	-1.02	0.42	-0.25	-2.90	0.71
14 August	-0.19	-0.98	0.82	-0.26	-2.50	0.31
SED	0.02	0.46	0.11	0.08	0.87	0.18
Kabanyolo1999B						
11 December	-0.26	-2.90	0.67	0.00	0.00	0.00
18 December	-0.34	-1.68	0.90	-0.17	-4.28	0.40
24 December	-0.16	-1.15	0.32	-0.11	-3.81	0.05
30 December	-0.85	0.27	0.92	-0.44	-2.57	0.59
SED	0.15	0.55	0.14	0.09	0.96	0.14
Kyambogo						
17 December	-0.23	-2.41	0.74	0.00	0.00	0.00
23 December	-0.24	-1.54	0.45	-0.96	-2.75	0.80
29 December	-0.21	-0.61	0.19	-0.79	-2.20	0.73
5 January	-0.72	0.85	0.75	-0.73	-1.58	0.79
SED	0.12	0.40	0.13	0.21	0.59	0.19
Kabanyolo2000A						
18 July	-0.28	-3.23	0.61	0.00	0.00	0.00
25 July	-0.26	-1.93	0.43	-0.32	-4.57	0.62
1 August	-0.33	-0.93	0.52	-0.33	-4.02	0.40
8 August	-0.74	-0.08	0.67	-0.51	-2.68	0.37
SED	0.11	0.68	0.05	0.11	1.02	0.13
Kyambogo						
20 July	-0.22	-2.76	0.68	0.00	0.00	0.00
28 July	-0.21	-1.81	0.29	-0.12	-3.04	0.86
5 August	-0.25	-0.86	0.26	-0.72	-2.72	0.67
12 August	-0.40	0.37	0.37	-0.78	-1.82	0.71
SED	0.04	0.67	0.10	0.20	0.68	0.91

b=rate of disease decrease over distance, Yo=intercept or logit (Y) and R^2=coefficient of determination
Data are averages for four directions (North, South, West and East of disease assessments from inoculated foci).

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