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The distribution and spread of sorghum downy mildew in sorghum and maize fields in Nigeria and Zimbabwe

C.H. Bock[1,*] and M.J. Jeger[2]
[1]*Natural Resources Institute, Central Avenue, Chatham Maritime, Kent ME4 4TB, UK (Fax: +15614625986; E-mail: cbock@ushrl.ars.usda.gov);* [2]*Horticulture Section, Department of Agriculture and Horticulture, Wye College, University of London, Wye, Ashford, Kent TN5 5AH, UK;* [*]*Current address: Department of Plant Pathology, USDA-ARS-USHRL, 2001 South Rock Rd., Fort Pierce, FL 34945, USA*

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Abstract

Sorghum downy mildew (*Peronosclerospora sorghi*, SDM) is a damaging disease of sorghum and maize crops in Africa. Runs analysis was used to study the distribution of systemically infected sorghum and maize plants in Nigeria and Zimbabwe. The temporal and spatial development of local lesions of SDM on sorghum in Zimbabwe was investigated by assessing the local lesion symptoms caused by conidia in plots with a single point source of inoculum. With ordinary runs analysis, there was evidence of clustering of disease in some fields in the humid areas of Nigeria and the semi-arid areas of Nigeria and Zimbabwe. Clustering was found in two of the eight runs analyses performed on maize in the humid south of Nigeria, and in only one of the eight runs in Zimbabwe, which was interpreted as a predominance of random infection at the time of assessment and at the spatial scales assessed. Symptoms of local lesions of SDM developed rapidly across plots from an introduced point source of infection. After 9 days-exposure to the source of inoculum, the incidence of diseased leaves was 1.2%, and after 50 days it was 74.5%. A disease gradient which initially developed flattened as the plot became uniformly diseased. The predominant wind direction was NNE, and most rapid spread of disease was towards the SSW and WSW. In conclusion, local lesions can spread rapidly in sorghum crops, suggesting that they may be an important source of conidial inoculum for further local and systemic infections during the growing season.

Introduction

Sorghum downy mildew (SDM) caused by *Peronosclerospora sorghi* [(Weston & Uppal) C.G. Shaw] is a destructive disease of sorghum and maize, and can cause severe yield loss (Williams, 1984). Although there is substantial knowledge of the epidemiology of the disease (Jeger et al., 1998), relatively little is known about disease patterns in fields and the spread of disease between plants. A random pattern of diseased plants could mean the pathogen was not spreading from plant to neighbouring plant within the field, while a clustering of diseased plants may indicate spread from plant to neighbouring plant. Assessment of the disease pattern and disease spread is critical to epidemiological investigations, and may provide knowledge of value when control methods such as sowing date and planting strategy are chosen.

Peronosclerospora sorghi produces ephemeral, asexual conidia and long-lived, sexual oospores. The production of oospores follows a monocyclic pattern, while conidia may be polycyclic. Infection with both oospores and conidia causes systemic disease. Systemic disease is particularly serious as it causes sterility. Conidia may also cause local lesions. In the USA, soil-borne oospores act as the principal source of inoculum (Frederiksen, 1980), and result in a typical clustered pattern of diseased plants (Schuh et al., 1986; 1988). In other areas conidia are the principal

causes of systemic disease (Cohen and Sherman, 1977; Rajasab et al., 1979; Ramalingham and Rajasab, 1981).

Clustering of diseased plants has been observed in situations where conidial inoculum was artificially introduced into the field in Thailand (Hau et al., 1995). Where natural infection from conidia has been observed in southern Nigeria, the distribution of systemically infected maize plants was generally random (Olanya et al., 1993). No information exists for the patterns of diseased sorghum plants in Africa.

Two strains of *P. sorghi* occur in Africa. A maize-infecting strain occurs in southern Nigeria (Anaso et al., 1987), and a more widespread sorghum/maize-infecting strain occurs in the semi-arid areas of western, eastern and southern Africa. Oospores are produced abundantly by the sorghum/maize strain (Bock et al., 1997), but rarely by the maize strain (Adenle and Cardwell, 2000). In many areas of Africa where epidemics of the sorghum/maize strain have been reported on sorghum (de Milliano, 1992; van der Westhuizen, 1977; King and Webster, 1970), cropping is seasonal with most planting taking place early in the season. Infections are often caused by soil-borne oospores (Adipala et al., 1999), and these systemically infected plants produce conidia. As the season progresses, conidia are produced in increasing quantities from greater numbers of diseased plants (Bock et al., 1998a). Much of this inoculum comes from local lesions (Rajasab et al., 1980). The short latent period (3–5 days) of local lesions allows them to contribute to the rapid build up of both systemically infected plants and further local lesions of surrounding plants (Jones, 1970; Schmitt and Freytag, 1974). In humid zones of southern Nigeria where the maize strain occurs, local lesions are not found and oospores are only rarely formed (Adenle and Cardwell, 2000). In this area, disease is perpetuated by conidia. Thus, conidia are crucial to perpetuation of the maize strain, and probably play an important role in semi-arid regions of Africa and India when weather conditions are suitable (Bock et al., 1998a; Ramalingam and Rajasab, 1981). Conidia of the sorghum/maize strain can come from systemically or locally diseased plants. Local lesions can be prevalent in a crop even when systemic disease is at low incidence (Bock et al., 1998a,b), and probably produce much of the asexual inoculum that can lead to further local or systemic infection of surrounding tillers or plants.

Moving fronts of disease have been described (Berger and Luke, 1979; Jeger, 1983; Hau et al., 1995; Drepper et al., 1993), but not applied to the spread of local lesions of SDM. Hau et al. (1995) have shown the importance of within-season, plant to neigbouring plant spread for systemic infection of maize caused by conidia from systemically infected plant. Despite their potential importance as a source of inoculum, the temporal and spatial dynamics of local lesion development in sorghum have not been investigated.

The following experiments were undertaken to assess the pattern of systemic SDM in maize and sorghum fields in (1) the continuously cropped humid regions of southern Nigeria, and (2) the semi-arid, seasonally cropped parts of northern Nigeria and Zimbabwe. Further experiments were performed to ascertain the temporal and spatial development of local lesions, and their role in the epidemiology of SDM.

Materials and methods

Runs analysis of the distribution of systemic disease in sorghum and maize fields

Runs analysis was performed as described by Campbell and Madden (1990). In southern Nigeria, three maize fields were assessed for the distribution of systemically infected plants, and in northern Nigeria and Zimbabwe three fields of sorghum were assessed, taking four runs of at least 50 plants each in each field. In southern Nigeria (1991), four runs were taken in one field, and two in each of two other fields, with at least 25 plants in each run. When taken in the same field, the runs were taken at different sites and did not overlap. In Nigeria (1991), the fields were sown to an unknown local variety. In Zimbabwe (1992), the three areas of sorghum were grown at the SADC/ICRISAT/SMIP farm at Matopos. The maize and sorghum fields chosen were all at growth stages from mid-vegetative phase to flower. In each maize field, a run of 100 plants was taken, each plant being assessed consecutively for the presence or absence of disease. However, in northern Nigeria and Zimbabwe, sorghum field size was such that runs (100 plants in Nigeria, 200 plants in Zimbabwe) were composed of adjacent rows of plants, which were summed as described by Campbell and Madden (1990). The method detects whether diseased plants in a homogeneous field occur randomly or non-randomly within rows. Random distribution suggests all arrangements are equally possible, while a non-random distribution is indicated by clustering. Thus the expected number of runs, $E(U)$ is given by:

$$E(U) = 1 + \frac{2m(N-m)}{N}$$

where m is the number of diseased plants in a row, and N is the total number of plants in the row. The standard deviation of $U(s(U))$ is calculated by:

$$s(U) = \left(\frac{2m(N-m)[2m(N-m)-N]}{N^2(N-1)} \right)^{1/2}$$

A normal test using the continuity correction (0.5) was then used to determine clustering:

$$Z = \frac{[(U+0.5) - E(U)]}{s(U)}$$

If the value of Z is less than -1.645, clustering is indicated ($P = 0.05$).

Spread of local lesion sorghum downy mildew from a point source of inoculum

This experiment was undertaken in 1992/3. The experiment had a randomised block design with three blocks (Figure 1). The six plots (8×8 m) were planted to the SDM susceptible sorghum variety Marupantse (25 February 1993), and were each surrounded by a 1 m wide border row of the SDM resistant variety SV-1. The between row distance was 0.5 m, and plants were thinned a week after emergence to 0.1 m within-row spacing, resulting in a plant density of 20 plants per m^2. To assess disease spread from a point source of inoculum, a single 20-day-old, systemically diseased, container-grown sorghum plant was placed in the centre of three of the six plots. The remaining three plots were controls to monitor background disease, and did not receive an inoculum source. The diseased plant acted as a source of inoculum in the treated plots, and the first assessment of disease incidence and severity in all plots was made when the infected plants were placed in the plots. The disease assessments were made on individual plants at 25, 75, 125, 175, 225, 275, 325 and 375 cm along the vertical and horizontal transects of the square plot (all at 90° to each other), and at 40, 120, 200, 280, 360, 440, 520 and 600 cm along the diagonal transects (also at 90° to each other) from the plot centre to the edge. Overhead irrigation was applied every four days to provide moisture suitable for asexual spore production and infection. As we were interested primarily in the development and spread of local lesions from the point source within the plot, main stems or tillers that became systemically infected during the period of the experiment were rogued before they produced

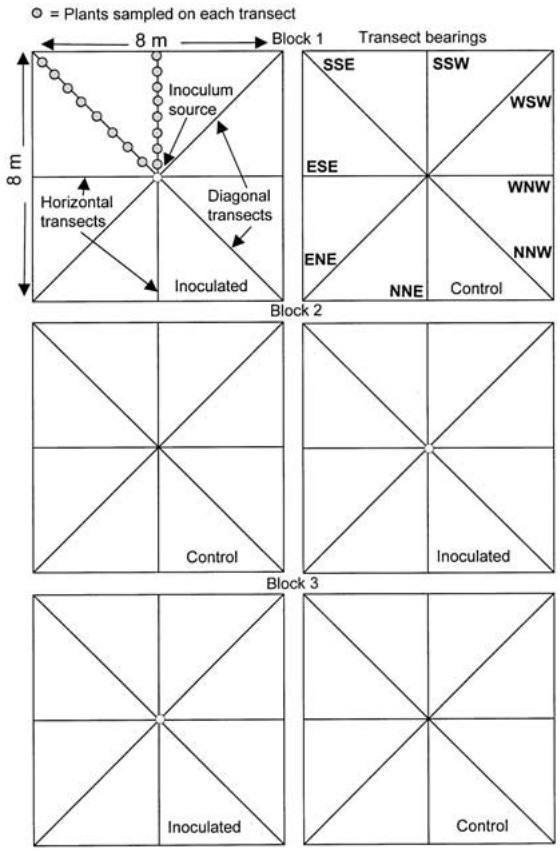

Figure 1. The layout of plots showing blocks, treatments, plant sampling procedures and transect directions in an experiment to investigate the spread of downy mildew (*P. sorghi*) local lesions in sorghum at Matopos, Zimbabwe (further details described in the Materials and methods).

inoculum. Only seven plants developed systemic disease during the assessment period, none were plants in transects being assessed for local lesions. The plots were assessed over 50 days on 2, 10 and 23 March and 5 and 21 April 1993. Assessments of both disease incidence and severity were taken. The incidence of local lesion-diseased leaves per plant was measured by counting total leaf number per plant, and the number of diseased leaves. The severity of disease was assessed on a 0–9 scale where 0 = no symptoms, 1 = <1%, 2 = 1–10%, 3 = 11–25%, 4 = 26–35%, 5 = 36–50%, 6 = 51–65%, 7 = 66–75%, 8 = 76–90% and 9 = >90% leaf area diseased. Wind direction was obtained from an automated weather station at SADC/ICRISAT/SMIP from 0000 to 0800, which is the time at which most conidia are produced and dispersed (Bock et al., 1998a).

Data analysis

The data on distribution of systemically diseased plants were analysed by ordinary runs analysis (Madden et al., 1982; Campbell and Madden, 1990) with Microsoft Excel V5.0. The spread of disease from local lesions was analysed using Genstat V5.3 (Genstat, 1993). Regression analysis was performed to compare development of disease on inoculated and control plots. Regression analysis was further used to investigate the relationship between distance from inoculum source and the number of days to first disease symptoms for the control and treated plots. A regression solution was developed to describe the temporal and spatial movement of the epidemic front, taking into account transect direction and time for development of first symptoms. Initially, an analysis of variance was performed for each sample date to ascertain the effect of transect type and bearing. To investigate the effect of transect type, distance from the source of inoculum and timing of assessment, a repeated measures analysis of variance was performed on the epidemic development for mean disease incidence for the inoculated plots. Time was treated as a sub-plot in the repeated measures analysis and the degrees of freedom in the time stratum were multiplied by the factor 0.7893 to deal with any correlation between disease measurements and time.

Results

Runs analysis of the distribution of systemic disease in sorghum and maize fields

Results of the runs analysis of the distribution of systemic disease in maize and sorghum fields are shown in Table 1. Clusters of diseased plants were found in two (25%) of the eight runs in the humid forest. For the sorghum crops, only one (12.5%) of the fields in Zimbabwe had a clustered distribution of diseased plants.

Spread of local lesion sorghum downy mildew from a point source of inoculum

The inoculated plots progressed through the epidemic earlier and had a higher incidence of disease (Figure 2). The epidemic development was described by a second-order polynomial-regression solution on both treated and control plots. Analysis of variance showed no significant differences between disease incidence or severity on inoculated and control plots on 2 March (no disease observed, Table 2), but on 10 March ($F = 8.35$, $P = 0.0042$), 23 March ($F = 257.36$, $P < 0.0001$), 5 April ($F = 89.86$, $P < 0.0001$), and 21 April ($F = 33.40$, $P < 0.0001$) the differences were significant.

Table 1. Runs analysis of maize fields in southern Nigeria and sorghum fields in northern Nigeria and Zimbabwe assessed for distribution of systemic SDM (*P. sorghi*)

Crop type	Sample field and variety	Field location	Mean incidence of SDM	Number of runs		St. Dev.	Z[a]	P[b]
				Obs	Exp			
Maize	1-Local	Akure	42	40	50	4.85	−2.004	0.0228*
	1-Local	Akure	46	51	51	4.94	0.065	0.5199
	1-Local	Akure	35	47	47	4.52	0.111	0.5398
	1-Local	Akure	40	47	49	4.77	−0.419	0.3446
	2-Local	Edo-Ekiti	42	44	50	4.84	−1.182	0.1251
	2-Local	Edo-Ekiti	47	44	51	4.96	−1.375	0.0885
	3-Local	Edo-Ekiti	53	47	51	4.96	−0.770	0.2266
	3-Local	Edo-Ekiti	67	28	45	4.39	−3.923	0.0002**
Sorghum	1-Marupantse	Matopos	39	89	96	6.71	−1.067	0.1587
	1-Marupantse	Matopos	27	75	79	5.49	−0.712	0.2420
	1-Marupantse	Matopos	16	45	53	3.68	−2.280	0.0122*
	2-DC-75	Matopos	16	54	53	3.68	0.117	0.4404
	2-DC-75	Matopos	15	55	51	3.48	1.267	0.9032
	2-DC-75	Matopos	8	33	30	2.05	1.249	0.8944
	3-Local	Zaria	6	13	12	1.08	0.574	0.7257
	3-Local	Zaria	4	9	9	0.72	0.444	0.6736

[a] Z = Standardized variable: large negative values indicate clustering, i.e. <-1.64.
[b] P = Significance level, * = 0.05, ** = 0.001.

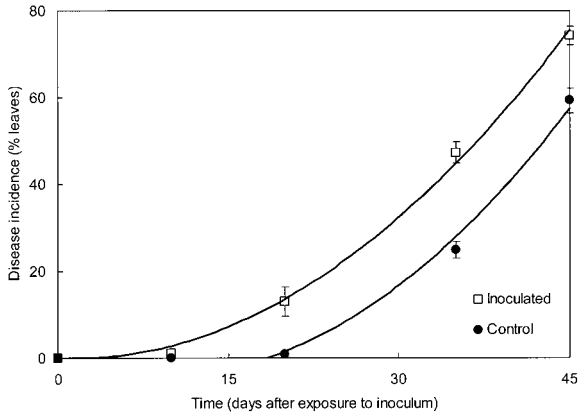

Figure 2. The temporal progress of development of local lesions of SDM (*P. sorghi*) on control plots and plots receiving inoculum from a point source. Regression solution for plots with an inoculum source, $y = -0.1444x + 0.0407x^2$, $R^2 = 0.97$; and for control plots $y = -1.1403x + 0.0548x^2$, $R^2 = 0.94$ (regression lines shown for values >0). Standard errors of the means are shown, and data points are means of 192 plants.

Table 2. Mean incidence and severity of SDM (*P. sorghi*) local lesions in sorghum plots assessed for disease spread at Matopos, Zimbabwe, 1993

Date of assessment (day of the year)	Control		Inoculated[a]	
	Incidence[b]	Severity	Incidence	Severity
61	0 (0)	0 (0)	0 (0)	0 (0)
69	0 (0)	0 (0)	1.2 (0.2)	0.12 (0.05)
82	2.0 (0.6)	0.24 (0.09)	22.7 (1.2)	1.68 (0.01)
95	25.0 (0.2)	1.43 (0.07)	47.2 (3.7)	1.86 (0.06)
111	58.7 (5.4)	1.80 (0.10)	74.5 (2.3)	2.19 (0.05)

[a]Inoculated plots had a point source of inoculum as a source of conidia.
[b]Parentheses contain standard deviations.

By the second assessment, disease was found on 1.2% of leaves on the inoculated plots. By the final assessment date, a mean of 74.5% of leaves were diseased with local lesion SDM in the inoculated plots. No disease was found in the control plots until the third assessment date (2.0% leaves diseased). By the final assessment date, 58.7% of leaves were diseased. The mean severity followed a similar increase as the incidence. Thus, plots provided with a source of inoculum had a greater incidence and severity of disease from local lesions throughout the period of assessment.

Plants in inoculated plots had symptoms of disease earlier than control plots at all distances assessed (Figure 3). Disease in the inoculated plots showed a

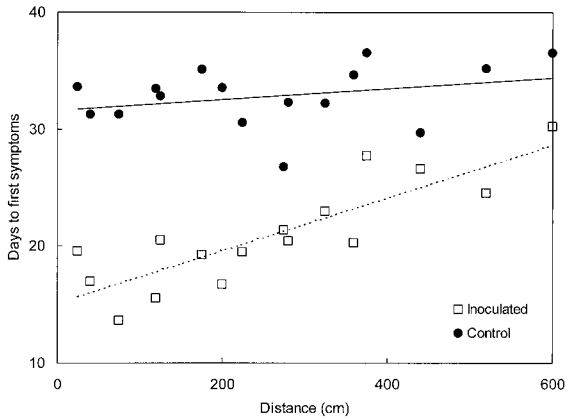

Figure 3. The time to first appearance of symptoms of local lesions of SDM (*P. sorghi*) different distances from a point source of inoculum. Regression solution with inoculum source, $y = 15.2 + 0.029x$, $R^2 = 0.72$, control plots, $y = 31.634 + 0.0046x$, $R^2 = 0.09$. Data points are means of 12 plants.

linear relationship between days to first symptoms and distance from the point source of inoculum. The control plots showed no relationship with distance and the whole plot became infected at about the same time. However, the further away from the source of inoculum in the treated plots, the number of days to first symptoms becomes similar to the control plots. The point at which the extrapolated regression lines cross occurred at 9.04 m from the centre of the plot, after 36.5 days. At this time and distance, the effect of the inoculum source on days to first symptoms would not be discernable from background disease levels. The number of days to first symptoms was influenced by compass bearing relative to the predominant wind direction (Figure 4). The longest period to first symptoms was for the NNE transect (17.1 days), and the least amount of time was for transects towards the WSW and SSW (each 13.4 days). The wind during 0000–0800 h blew from the NNE for 66.6% of the time, and comparatively small proportions of time from the other directions (<10%), so disease was spread more rapidly in the predominantly downwind direction. The results of the repeated measures analysis of variance (Table 3) did not show overall a significant difference in disease incidence between transect types (T, perpendicular or diagonal). However, there were significant effects of the transect compass bearing (T*P, *F.Pr.* = 0.01 and T*D, *F.Pr.* = 0.05), distance from the source of inoculum and transect type (T*Dist_p, *F.Pr.* = 0.01 and T*Dist_d, *F.Pr.* = 0.001), and the linear trend of distance (T*PL_p, *F.Pr.* = 0.001 and T*DL_d, *F.Pr.* = 0.01). Time of assessment also

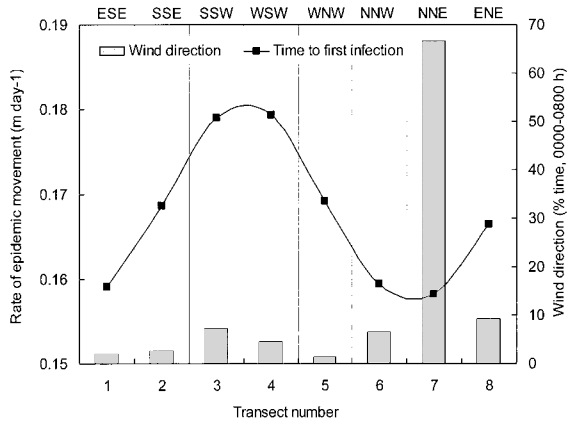

Figure 4. The rate of development of local lesion symptoms of SDM (*P. sorghi*) relative to wind direction. Regression solution, $y = 15.2 + 0.029x - 1.110\sin(a) + 1.731\cos(a)$, where a is the bearing of the transect from north in degrees.

had a significant effect (TA, *F.Pr.* = 0.001), and the interaction of assessment date, transect type and distance was significant ($TA^*T^*Dist_p$, *F.Pr.* = 0.01 and $TA^*T^*Dist_d$, *F.Pr.* = 0.05). There was also a significant linear trend of distance with sample date on different transects ($TA^*T^*P^*L_p$, *F.Pr.* = 0.05 and $TA^*T^*D^*L_d$, *F.Pr.* = 0.01). The incidence of local lesion symptoms along horizontal and diagonal transects of inoculated and control plots at different assessment dates is shown in Figure 5(A)–(D). In the inoculated plots, the plants closer to the centre of the plot (and therefore closer to the source of infection) initially had a greater incidence of disease than the plants towards the edge of the plots. However, by the final assessment date, the disease gradient was less steep. The control plots did not show a consistent gradient of disease, as symptoms developed equally over the whole plot.

Discussion

In the fields assessed in this study, the distribution of plants systemically diseased with SDM in both the humid zone of southern Nigeria and the drier areas of northern Nigeria and Zimbabwe was similar. At the time of the runs analysis (middle vegetative phase-flower) some clustering existed in both the maize fields from the humid zones and the sorghum fields from the semi-arid zones. We did not find as strong evidence for disease clustering as Olanya et al. (1993), who found greater evidence for clusters of diseased maize in the arid north of Nigeria compared to the south. Clustering occurred in only 12.5% of samples of sorghum in the arid zones of Zimbabwe and Nigeria, and only 25% of maize samples in the humid zones of Nigeria. We interpret this as random infection of plants in most instances, although the growth stage at which we performed the runs may have influenced our interpretation. In Thailand, Hau et al. (1995) found that diseased plants initially had a clustered pattern, which became random as the incidence of systemic disease approached 100%. However, they used doublet analysis to assess disease distribution, while Olanya et al. (1993), and our study performed runs analysis. Madden et al. (1982) undertook a comparison of runs and doublet analysis and found that at high disease incidence, doublet analysis mis-indicated randomness. Nevertheless, at the spatial scale of these runs analyses there was little evidence of clustered contagion, at larger scales (e.g. fields) clustering may occur.

External sources of inoculum (conidia) may have caused the bulk of infections in Nigeria, rather than near neighbour plant spread. This is in contrast to what has been observed in the USA, where Schuh et al. (1986; 1988) used Morisitas index of dispersion to show that systemic SDM was clumped in distribution, and was related to the oospore distribution in the soil. Oospores are the principal source of infection in the USA, and thus disease distribution may be related to oospore patterns. However, in southern Nigeria, air-borne conidia of the maize-infecting strain appear to be the sole source of infection (Anaso, 1989) and a random pattern of systemic disease may result (Olanya et al., 1993). In other parts of Africa where the sorghum/maize strain occurs, conidia may also be produced in large quantities (Bock et al., 1998a). However, oospores are likely to be important at least for primary infections in the semi-arid areas of Africa to initiate epidemics (Adipala et al., 1999; Bock and Jeger, 1999). Where oospores are the primary source of inoculum, the disease will tend to be monocyclic, with polyetic spread. However, production of conidia and often-prodigious development of local lesions suggest that the pathogen can also have a polycyclic phase.

Drepper et al. (1993) and Hau et al. (1995) previously studied the spatial and temporal spread and development of systemic disease of maize in Thailand. Systemic epidemic development approximated to logistic or monomolecular functions depending on whether the inoculum arrived as a single event or continuously, as in fields that contained systemically diseased plants. Hau et al. (1995) also found that the incidence of systemic disease decreased with distance

Table 3. Repeated measures analysis of variance of the effect of distance from inoculum source, transect direction (diagonal or perpendicular), distance along transects and assessment date on incidence of local lesion downy mildew in square plots of sorghum containing an inoculum source of *P. sorghi* placed in the plot centre at Matopos, Zimbabwe in 1993

Source of variation			Df	SS	MSS	Vr	*F. Pr.*
Block			2	6033	3017	2.6	
Block*	T		1	104	104	0.1	
Subject	T*P		3	16773	5591	4.8	0.01
Stratum	T*$Dist_p$		7	25920	3702	3.2	0.01
		T*L_p	1	21644	21644	18.7	0.001
		R	6	4276	713	0.6	
	T*D		3	13397	4466	3.9	0.05
	T*$Dist_d$		7	38079	5440	4.7	0.001
		T*L_d	1	29400	29400	25.5	0.001
		R	6	8679	1447	1.3	
	T*H*$Dist_p$		21	23136	1102	1.0	
		T*P*L_p	3	5369	1790	1.6	
		R	18	17767	987	0.9	
	T*D*$Dist_d$		21	21052	1002	0.9	
		T*D*L_d	3	4703	1568	1.4	
		R	18	16349	908	0.8	
	R		125	144368	1155	4.7	
Block*	TA		4 (3)	421739	105435	429	0.001
Subject*	TA*T		4 (3)	1560	390	1.6	
Time stratum	TA*T*P		12 (10)	3245	271	1.1	
	TA*T*$Dist_p$		28 (22)	15317	547	2.2	0.01
		TA*T*L_p	4 (3)	5049	1262	5.1	0.001
		R	24 (18)	10268	427	1.7	0.05
	TA*T*D		12 (10)	1900	158	0.6	
	TA*T*$Dist_d$		28 (22)	13095	468	1.9	0.05
		TA*T*L_d	4 (3)	2392	598	2.4	
		R	24 (19)	10703	446	1.8	0.05
	TA*T*P*$Dist_p$		84 (66)	25003	298	1.2	
		TA*T*P*L_p	12 (10)	7272	606	2.5	0.05
		R	72 (57)	17735	246	1.0	
	TA*T*D*$Dist_d$		84 (66)	27291	325	1.3	
		TA*T*D*L_d	12 (10)	8960	747	3.0	0.01
		R	72 (57)	18330	255	1.0	
	R		498 (393)	122335	245		
Total			944	900189			

[a]Source of variation: T = transect type in plot (diagonal or perpendicular); P = direction of perpendicular transect (NNE, ESE, SSW or WNW); D = direction of diagonal transect (ENE, SSE, WSW or NNW); $Dist_p$ and $Dist_d$ = distance along the transect from the inoculum source for perpendicular and diagonal transects, respectively; L_p and L_d = linear trend of distance along perpendicular and diagonal transects, respectively; R = residual variation; TA = Time of assessment (date).

[b]Amended degrees of freedom for the Block*Subject*Time stratum are shown in parentheses (see Data Analysis Section).

[c]*F. Pr.* values are indicated when significant.

from the source of inoculum. The slope of the gradient flattened as the epidemic progressed and wind direction affected the spread of disease. Our observations of spread of local lesion symptoms in a sorghum field agree with these observations; a disease gradient develops and then flattens as the disease becomes uniformly distributed through successive cycles of infection and sporulation. Wind direction had an effect on the development of disease. We found local lesions developed earliest on the WSW and SSW side when the predominant wind direction was NNE.

Although the temporal and spatial spread of local lesions had not previously been reported, it is known that the latent period of this phase is short (3–5 days;

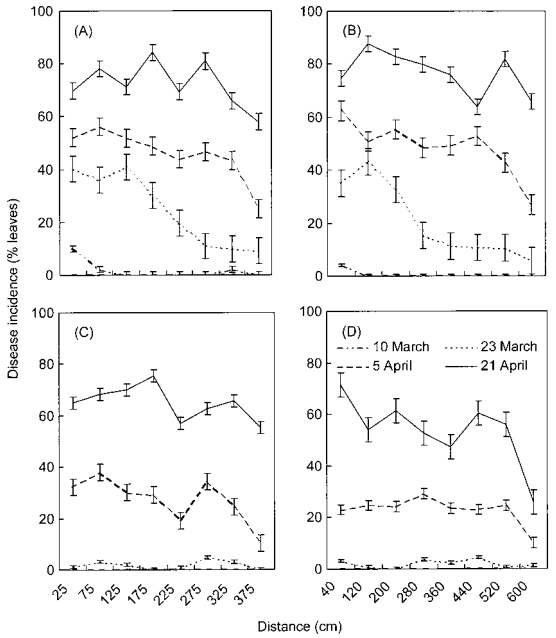

Figure 5. Local lesion infection of sorghum with downy mildew (*P. sorghi*) different distances from plot centres in plots with an inoculum source in the centre (A and B) and control (C and D) plots. A and C show the disease incidence in the perpendicular transects and B and D show disease incidence in the diagonal transects, respectively (standard errors of the means are indicated).

Jones, 1970; Schmitt and Freytag, 1974). Local lesions may be a source of conidia for systemic infection of susceptible plants. Indeed, Bock et al. (1998a,b) found that the incidence of systemic disease could be low (<8% plants diseased), while incidence of local lesions was high (>70% leaves diseased), with a large quantity of conidia being sampled in the air. The short latent period of local lesions allows repeated cycles of spore production and infection. On the other hand, systemic infection to sporulation can take 1–3 weeks (Schmitt and Freytag, 1974), and both sorghum and maize plants become resistant to systemic infection in 3–4 weeks (Cardwell et al., 1997; Bock et al., 1998a). The short latent period of local lesions allows the potential to contribute to the rapid build up of both systemically and locally infected plants.

Our observations are that while the sorghum/maize strain of SDM readily causes local lesions on sorghum plants of all ages, only young maize plants are susceptible to this type of infection (pers. obs.). Drepper (1986) also found that local lesions do not produce as many conidia as systemic infection on maize. However, we found that local lesions on field-grown sorghum plants produced as many conidia per unit area as infected leaf material on adjacent, systemically diseased plants (Bock et al., 1998a). In India, local lesions are reported to produce similar quantities of conidia to systemically diseased sorghum plants (Rajasab et al., 1980; Shetty and Saffeeulla, 1981). Thus, the contribution of local lesions to inoculum in sorghum crops may be equivalent to an equal area of systemically diseased leaf.

Systemic disease is the major cause of yield loss in epidemics of SDM. The distribution of systemically diseased plants can provide information on the source of inoculum, which may be used when considering control strategies. Local lesions can develop and spread rapidly causing disease over a large leaf area as an inoculum source can contribute to further systemic or local lesion disease development.

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