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Windborne spread of ergot disease (*Claviceps africana*) in sorghum A-lines in Zimbabwe

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Ergot disease spread rapidly in Zimbabwe amongst replicated plots of male-sterile sorghum A-lines, from a group of centrally situated and precociously inoculated plants. Prominent secondary conidiation by the pathogen, *Claviceps africana*, on the surface of exuded honeydew provided airborne spores which were trapped in a Burkard continuous spore trap and showed diurnal peaks of concentration in air close to the primary source of inoculum. The rate of disease spread ($r = 0{\cdot}2$; range 0·14–0·58) closely matched that recorded for other plant pathogens such as *Phytophthora infestans* and *Puccinia graminis tritici*, and it is concluded that the characteristic secondary conidia of *C. africana* were the principal epidemiological agents within the experimental area. Ergot spread by windborne secondary conidia has significant epidemiological and economic implications for sorghum hybrid breeding in southern Africa.

INTRODUCTION

The sorghum ergot fungus, *Claviceps africana*, is endemic in eastern and southern Africa where annually, especially in Zimbabwe and South Africa, the disease which it causes becomes a chronic problem amongst the A-lines (male-sterile sorghums) used in F1 hybrid breeding programmes (De Milliano, 1992). Yields of seed from infected A-lines are barely 1000 kg/ha but this could be expected to rise to 2000 kg/ha and 3000 kg/ha on small and large-scale farms, respectively, if *C. africana* can be controlled. Hybrids are the best means of exploiting the agronomic potential of sorghum in the region and projections by the Southern Africa Development Coordination Conference/International Crops Research Institute for the Semi-Arid Tropics (SADCC/ICRISAT) at the research station at Matopos, Zimbabwe for the year 2000 estimate that 680 000 ha will be planted to hybrids.

Sorghum ergot was first recognized as a potential threat to F1 hybrid seed production in Africa in the 1960s (Futrell & Webster, 1965). Since then this potential has been realized so that devastating losses of seed yield and quality have followed the apparently sudden appearance and rapid spread of *C. africana* in sorghum breeders' fields (SADCC/ICRISAT, 1990). The epidemiology of ergot on sorghum in southern Africa is poorly understood, partly because the involvement of windborne propagules has only recently been recognized (Frederickson *et al.*, 1989). Disease spread by secondary conidia of *C. africana* has been demonstrated on a small scale in horticultural tunnels, but the measurement of disease spread over a wider area and involving a wide range of sorghum A-line genotypes is needed. The ergot pathogen of Zimbabwe and other parts of Africa is now seen to be a distinct species (Frederickson *et al.*, 1991), differing from the sorghum ergot pathogen of India, *C. sorghi*, in several phytopathological as well as biochemical aspects. Therefore, knowledge of the epidemiology of this disease and pathogen in Africa needs to come from studies done with the African pathogen in Africa. Surprisingly for cereal pathogens of such historic and economic importance, very little experimental epidemiological study of ergot fungi has been published. Most assumptions are anecdotal and assume airborne transmission of ascospores or spread of sphacelial conidia by head-to-head contact, rainsplash and insects (Mantle, 1988).

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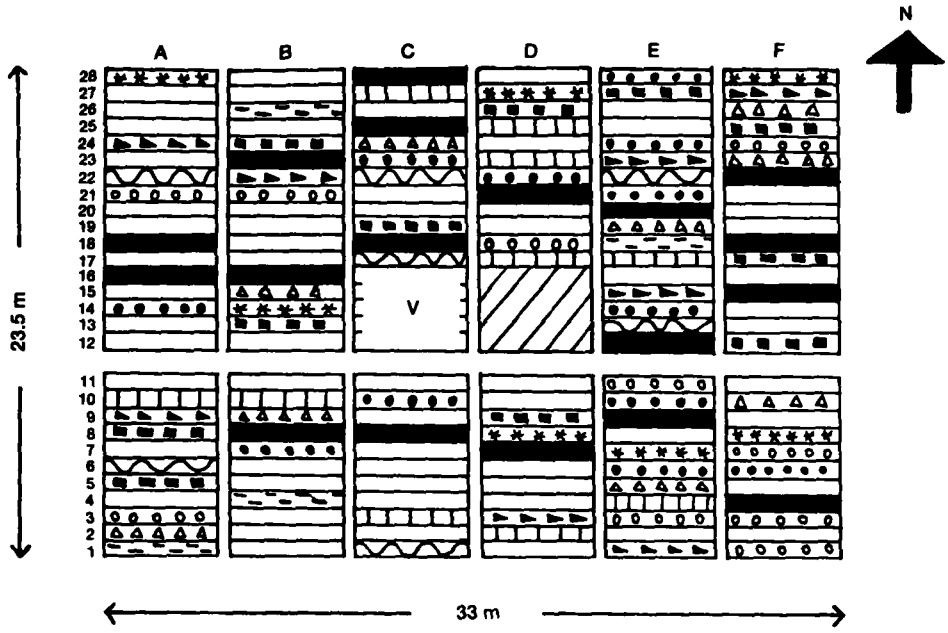

Fig. 1. Area planted as sorghum varieties (V and adjacent diagonally hatched area) surrounded by the A-line plots in the ergot spread experiment. Plots of the 10 A-lines from which replicated data were collected are similarly represented: * * * ATX 623; ■ ■ CK 60A; ▬▬ D2A; ∿∿∿ IS 10638A; • • • • MA6; △ △ △ SPL 32A; - -=-=- SPL 38A; □□□□□ SPL 109A; O O O O SPL 177A; ►►► 2219 A.

MATERIALS AND METHODS

Experimental design

Drought delayed planting of sorghum until late January 1989, when a 23·5 × 33 m area was sown at the SADCC/ICRISAT research station, Matopos, Zimbabwe. The area (Fig. 1) was subdivided into 158 plots which, apart from a slightly off-centre 2·2 × 9·4 m area planted with five plots of a range of sorghum self-fertile varieties of diverse habit (hatched area), each contained one of 19 A-lines. The A-lines were randomly replicated 4–18 times (Fig. 1) according to availability of seed. Each plot contained one 5-m row of plants separated 0·6 m from adjacent plots. Plant stands at heading were very even (25–30 plants per row) except at two points (grid references D28 and F5 of Fig. 1) where there was sporadic seedling emergence in A-lines which were not used for analysis. At emergence from the flag leaf sheath six panicles were enclosed in net of mesh aperture 0·5 mm to exclude flying insects which might be vectors of conidial inoculum, as used in a previous experiment (Frederickson *et al.*, 1989). No other experiments on ergot disease occurred at the research station in that year and no spontaneous precocious source of inoculum was evident, even in sorghum breeding trials 0·5 km distant. A spore trap operating in an adjacent plot of pearl millet showed that no *C. africana* propagules were evident in the air up to the start of the sorghum inoculation.

Inoculation of sorghum varieties

The experiment started when, at anthesis on 29 March 1989 (day 1), 50 panicles of five SADCC ICRISAT sorghum varieties (accessions 2656, 2690, 2690-2, 3487 and 3893 selected to flower shortly in advance of the surrounding A-lines [hatched area in Fig. 1]) were inoculated using a hand atomizer containing a fresh *C. africana* conidial suspension prepared as previously described (Frederickson *et al.*, 1989). Inflorescences were enclosed in a polythene bag within which the inoculum was sprayed to avoid drift, though this was considered to be a somewhat superfluous precaution as no other flowering sorghum was in the vicinity. Each inoculated panicle was retained within the polythene bag, the resultant high relative humidity favouring the pathogen and also minimizing the risk of self-pollination.

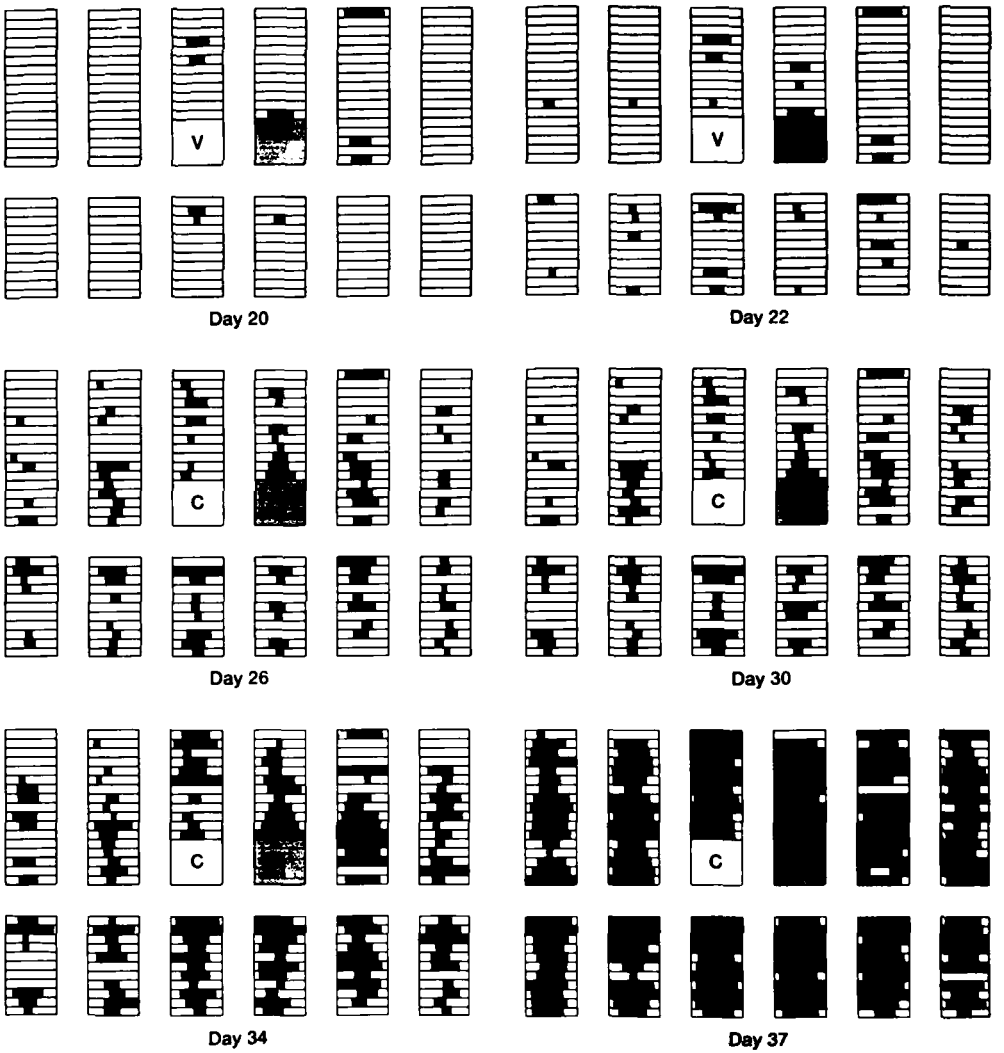

Fig. 2. Progressive spread of ergot disease from the inoculated sorghum varieties (central hatched area that became infectious on day 11) to the A-lines during stages in the epidemic. Black areas represent the proportion of disease incidence in each plot. Varieties not inoculated (V) were cut down, becoming C at day 26.

Panicles were resprayed daily with inoculum after each successive basipetal flush of anthesis to produce maximal disease severity. Bags were removed after the last inoculation. Some varieties in area 'V' of Fig. 1, which flowered obtrusively tall, were not inoculated and at day 26 were cut down (becoming C of Fig. 2).

Evaluation of flowering and disease in A-lines

From 29 March onwards, each plant in each A-line plot was scrutinized daily, noting the days when >5% spikelets of each panicle showed stigma exsertion (flowering) and when the first sphacelial symptoms of *C. africana* appeared. Subsequently the timing of honeydew exudation and its associated secondary conidiation was recorded. Following the appearance of the first infections in the plots, disease incidence (the cumulative frequency of infected plants in each plot) and severity (mean percentage of ergotized florets in each infected panicle per plot) were recorded throughout the A-lines every 2–4 days over a 24-day period. Of the 19 A-lines moni-

Table 1. Overall ergot disease severities, apparent infection rates and rates of flowering in 10 sorghum A-lines. Fast, moderate or slow flowering means that by day 20, 80–90%, 75–79% or 60–70% of plants were flowering, respectively

Sorghum A-line	Mean ergot severity, % (range)	*P*	Ergot disease rating	Apparent infection rate (*r*)	Rate of flowering
SPL 109A	58 (20–90)		Severe	0·28 ± 0·03	Fast
MA6	50 (25–90)	> 5% (NS)	Severe	0·14 ± 0·016	Slow
D2A	37 (1–80)	1–5%	Severe	0·20 ± 0·025	Fast
ATX623	32 (10–50)	1–5%	Moderate	0·29 ± 0·052	Fast
SPL 32A	25 (< 1–75)	< 1%	Moderate	0·28 ± 0·039	Moderate
SPL 177A	17 (< 1–25)	« 1%	Low	0·18 ± 0·018	Fast
SPL 38A	14 (< 1–25)	« 1%	Low	0·27 ± 0·041	Moderate
CK60A	7 (< 1–25)	« 1%	Low	0·20 ± 0·017	Moderate
2219A	3 (< 1–15)	« 1%	Low	0·26 ± 0·05	Slow
IS10638A	1 (< 1–2)	« 1%	Low	0·19 ± 0·017	Slow

tored, the final severities on day 37 for the total number of plants of the 10 A-lines which were most replicated (Fig. 1) are presented here (Table 1). A-line SPL 38A was replicated four times; the other nine were replicated 7–18 times (mean, 10).

Weather records and spore trapping

Concurrent with disease assessment, weather monitoring (temperature, relative humidity, wind direction and the duration of periods of rain) was carried out within the experimental area and a Burkard volumetric spore trap was used to measure *C. africana* secondary conidia concentrations in air. Air was sampled at a rate of 0·6 m^3/h through an orifice 2 × 14 mm and directed at a vaseline/wax trapping surface (Frederickson *et al.*, 1989). The spore trap operated almost continuously but the data presented here show hourly concentrations of secondary conidia during two periods: (1) days 11–16, covering the first 6 days of honeydew production in the varieties and (2) at a later stage in the epidemic (days 24 to 33). The spore trap orifice was situated at 0·6 m above ground and either 2 m or 4 m downwind (south to south-west) from the varieties for (1) and (2) respectively. Use of the term downwind refers only to the general prevailing direction. Light and variable wind direction was, of course, quite common.

Data analysis

The experiment sought to follow the course of epidemic development throughout a heterogeneous group of male-sterile sorghum lines. The design involving natural inoculum from a central source with airborne secondary conidia as a part component potentially allowed observation of new infections arising up to 15 m distant, well beyond head-to-head contact or rainsplash. Assessment of disease incidence in panicles enclosed in fine-mesh net allowed presumed evidence of the efficacy of airborne secondary conidia as inoculum. That the data collected conformed in general to classical patterns of disease spread was of itself remarkable, given the constraints of field experimentation in the semi-arid tropics. Thus it would be unreasonable to subject the results to other than simple analyses, namely calculation of the apparent infection rate (*r*) (Van der Plank, 1963) and analysis of variance (Snedecor & Cochran, 1980) to define significance of differences between ergot disease severities in some sorghum A-lines.

RESULTS

Infection in the sorghum varieties

In the inoculated sorghum varieties, *C. africana* infection was apparent as sphacelia forcing the florets open after a latent period of 8 days. Honeydew exudation followed at 10 days post-inoculation. The typical symptom (Frederickson *et al.*, 1989) of secondary conidiation (non-viscous, colourless honeydew droplets becoming white at the surface) followed 1 day later. This was reflected by finding secondary conidia trapped at that time. The disease cycle from

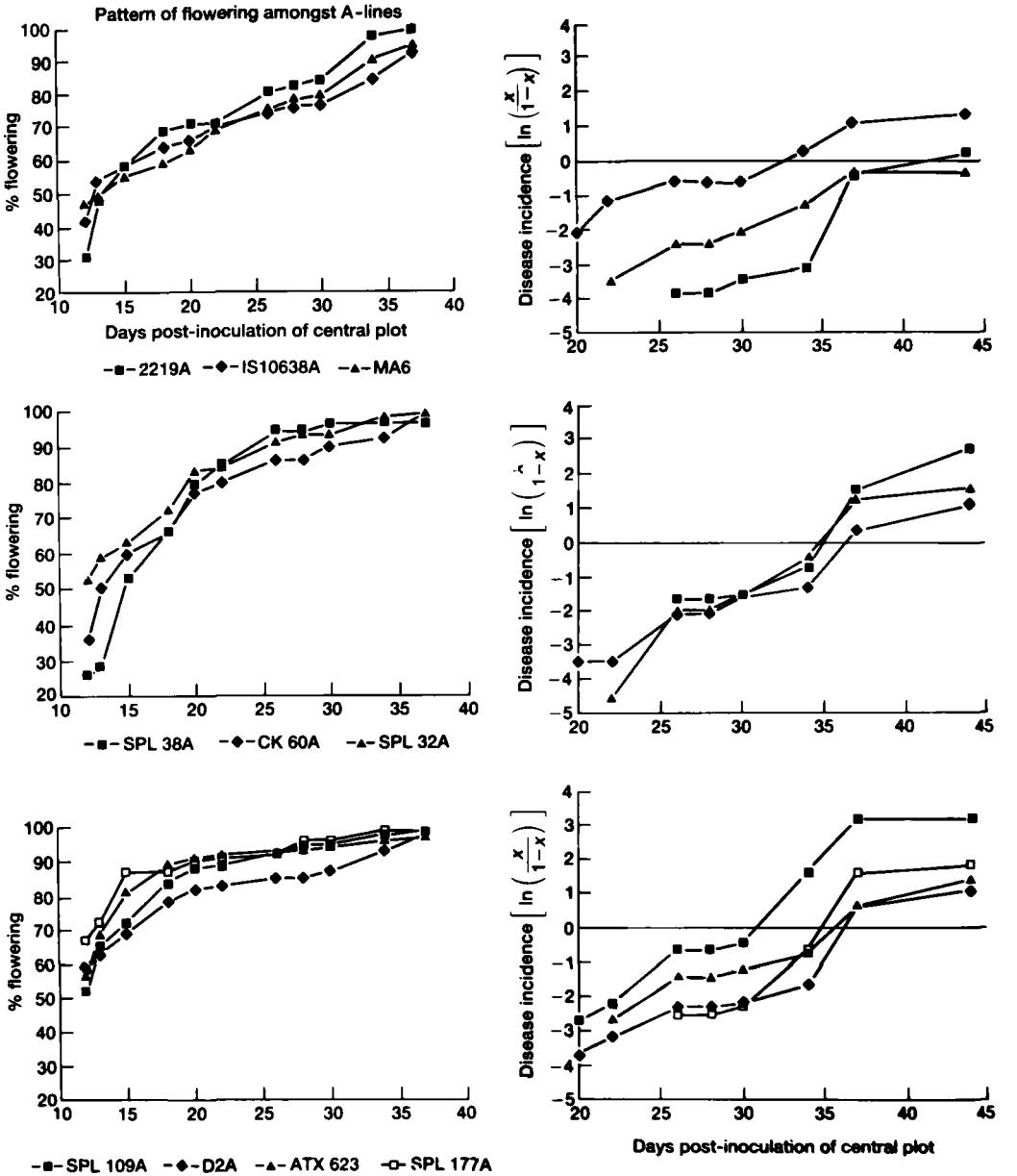

Fig. 3. Progress of flowering (left) and amount of ergot disease on a time scale (right) in 10 sorghum A-lines, grouped according to their fast, moderate or slow rate of flowering.

inoculation to the sporulation stage was, therefore, 11 days. Unfertilized gynoecia remain receptive to ergot pathogen inoculum for at least 7–9 days (Frederickson & Mantle, 1988). Autoinfection could be expected to contribute to build-up of severity in a panicle.

Flowering patterns of sorghum A-lines

Flowering began synchronously amongst the 10 highly replicated A-lines in the plot at day 12 but the flowering patterns could be differentiated into one of three groups (Fig. 3) as fast, moderate or

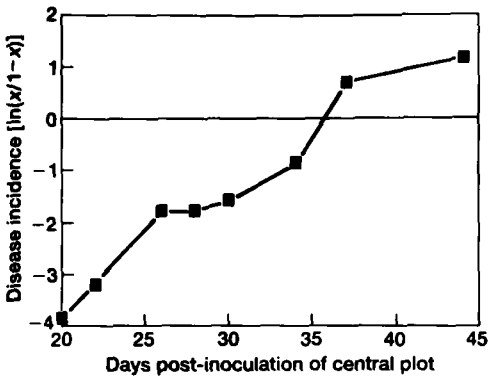

Fig. 4. Overall rate of ergot disease epidemic progression in the 10 sorghum A-lines.

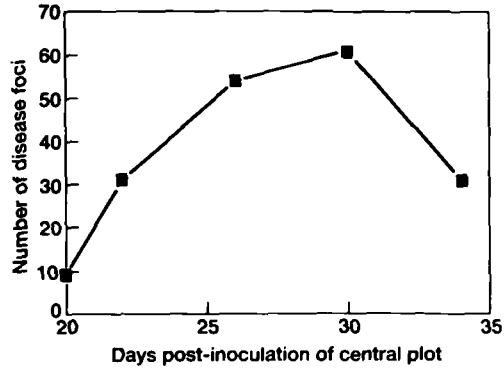

Fig. 5. Changes in the overall number of ergot disease foci in the 10 sorghum A-lines during the first 15 days of epidemic development.

slow. This meant that by the 8th day following the start of flowering (day 20 overall) 80–90% (fast), 75–79% (moderate) or 60–70% (slow) of plants were flowering. In the fast group more than 50% of plants began flowering on the same day. Unevenness of the plant stand did not obviously influence the flowering pattern of the various A-lines.

Ergot disease outbreaks and spread in the A-lines

The first appearance of ergot disease in the A-lines occurred at day 20, only 8 days after the A-lines entered the susceptible flowering stage. Figure 2 shows the progress of disease. The field area is subdivided into rectangular A-line plots, which have been shaded black to illustrate the diseased proportion, and to indicate their positions in relation to the original inoculum source. Clearly, the initial infections in A-lines were not confined to the vicinity of the source but were located downwind and even up to 15 m upwind in a northerly direction. Initial infections were of low (< 1%) severity as might be expected from a low and irregular inoculum concentration in air.

By day 24 more new infections had appeared, predominantly in the south and south-east of the plot. By day 26 disease outbreaks had become more general in the A-lines and epiphytotic in all directions; already in the A-line MA6 all plants in one plot were infected with a mean severity (amount of panicle diseased) of 40%.

The timing of honeydew (sphacelial stage) symptoms of ergot disease during the period day 20 to day 26 conforms to infection originating from the dispersal of the primary inoculum source, since the timing is well within that required for two disease cycles. Figure 4 shows a smooth early progression, consistent with infections during the early phase of the epidemic arising from an increasing amount of inoculum from a discrete source. Generally, the data in Fig. 4 represent an exponential increase in disease incidence over the period. Figure 5 shows a progressive increase in the number of plots or groups of adjacent plots in which infection by *C. africana* had become established up to day 30. These discrete areas are termed disease foci. By day 34 the number of disease foci had halved, suggesting that the disease had moved into an epidemic phase, resulting in merging of previously distinct foci. These results are consistent with the exponential pattern implied in Fig. 4, and in the wider data presented in Fig. 2.

At day 37, after a period equivalent to only two cycles of infection, disease incidence had reached nearly 70%. All A-lines were infected and mean disease severities reached 58% in some panicles (maximum severity recorded, 90%: Table 1). With so much disease, distinction of infection cycles would have become blurred at this stage, as would differentiation between the infective roles of airborne secondary conidia and sphacelial or secondary conidia transmitted by physical contact.

It was notable that all six panicles enclosed in fine-mesh net at emergence and which flowered during the period of secondary spore detection in air contracted ergot disease. The most probable inoculum for this infection would have been airborne inoculum.

Apparent infection rates amongst the A-lines

The graphical representation of the increase in

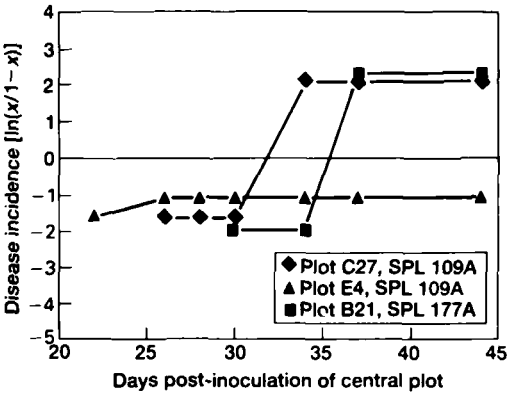

Fig. 6. Ergot disease spread in the A-line plots of sorghum in which apparent infection rates differed significantly from other replicates of the same A-line.

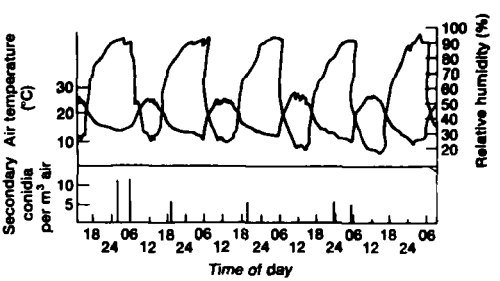

Fig. 7. Concentrations of secondary conidia of *Claviceps africana* in air in the experimental sorghum area (histogram) from day 11 (1 day after the start of honeydew exudation in the inoculated varieties) to day 16. The upper graph shows relative humidity (upper trace) and temperature (lower trace) during the same period. Spore trap located 2 m south of inoculum source.

disease incidence with time in the A-lines (Fig. 4) shows a relatively high apparent infection rate ($r = 0{\cdot}2 \pm 0{\cdot}023$ per unit per day). However, the value for r is the average measure of disease increase for the epidemic. Figure 4 shows that the infection rate was not constant throughout the epidemic. Considering the relatively low concentration of secondary conidia prevailing initially and the design of a single source of disease, the infection rate became remarkably high between days 20 and 26. The abundance of flowering host plants avoided a lag phase; an average of 80% of panicles were flowering by day 26. The highest apparent infection rate was between days 34 and 37, as expected when inoculum pressure was greatest and infected sources were both numerous and evenly distributed.

Mean values of r for each A-line over the days 20–44 period (Table 1; Fig. 3, lower graphs) ranged from 0·29 per unit per day (ATX 623) to 0·14 per unit per day (MA6). Following conventional regression analysis (Zarr, 1974) three plots (B21, C27 and E4; Fig. 1) had individual r-values significantly different from those of others of the same A-line and are shown in Fig. 6. These extreme patterns serve to illustrate the range of epidemiologies which may arise naturally in field experiments and which demand caution in perceiving intrinsic differences between sorghums in their response to *C. africana*.

Aerobiology of secondary conidia

Burkard spore trapping showed that when secondary conidiation began in the primary source at day 11—1 day after honeydew exudation began—secondary conidia were spasmodically present in air and their concentration did not exceed 10/m³ (Fig. 7). No macrospores were even seen on spore-trap slides, as expected. This situation continued until it rained on day 24. On the following day secondary conidia were more regularly present throughout the day, their concentration rising dramatically around nightfall (Fig. 8) to above 200/m³, consistent with the diurnal fluctuation reported previously (Frederickson *et al.*, 1989). The direct effect of rain was to wash spores from the air so that typical maximum concentrations (days 31, 32, 33; Fig. 8) were less than 100 secondary conidia per m³. However, these concentrations were much above the dry weather concentrations recorded during days 11–15. If rain occurred when nightfall was approaching (days 31 and 32), the peak concentration of secondary conidia expected at around 18.00 hours failed to occur (Fig. 8). However, when rainfall ceased in the early morning, both the expected temperature and humidity fluctuation (temperature down and relative humidity up), and the peak of airborne secondary conidia, were restored, for example, to around 250/m³ (day 30, Fig. 8). Therefore, rain appeared to provide the high humidity which is conducive to secondary conidial production, as evidenced by the appearance of classic symptoms of whitened honeydew the day after rain. However, for optimal appearance and persistence of secondary conidia in air, drying-off periods following rain are also necessary.

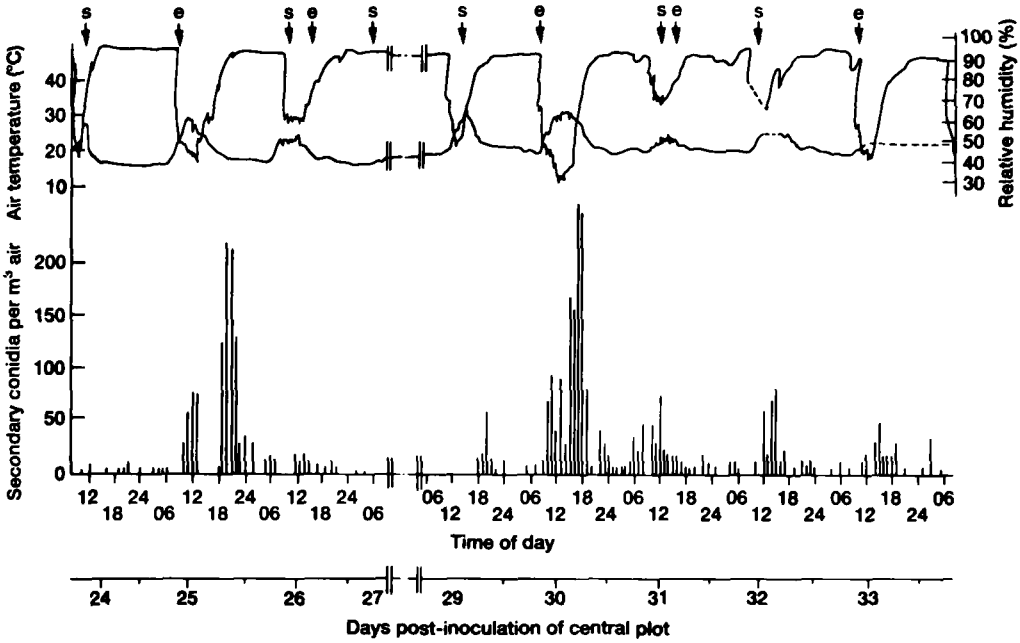

Fig. 8. Concentrations of secondary conidia of *Claviceps africana* in air in the experimental sorghum area (histogram) and changes in relative humidity and temperature (upper and lower traces, respectively) between days 24 and 34. Rainy periods are shown by arrows (s = start of rain; e = end of rain). Discontinuities in data collection are indicated by broken lines. Spore trap located 4 m south-west of inoculum source.

Correlation of production of secondary conidia and disease outbreaks in the plot

At day 20 the pattern of few, sporadic, low severity outbreaks of disease amongst A-lines was consistent with the existence, prior to that time, of the low density of secondary conidia in air (low inoculum pressure; Fig. 7) and a single central source of inoculum. At day 24 the inoculum sources (infection foci) were more numerous and more evenly distributed throughout the plot and secondary conidia were regularly present throughout the day at higher concentrations (Fig. 8). During the period days 26–30 the number of disease foci reached a maximum (Fig. 5) and spore trapping confirmed that concentrations of secondary conidia in air were reaching high values and that their appearance was typically diurnal. All of these considerations, coupled with the increased numbers of susceptible (flowering) hosts available by day 30, ensured that disease outbreaks quickly became more general and uniformly distributed in the plot, the foci increasing in size, and disease severity building up in individual panicles. By day 37, when more infected plants emerged from their latent phase, nearly all the planted area was infected, making the original individual foci obscure.

The experimental design included a wider range of A-lines currently under appraisal for general agronomic performance in the SADCC region in case some striking differences in susceptibility became apparent. None was obvious. However, the heterogeneity in the stand in the experimental area can only give a general demonstration of disease epidemic dynamics.

DISCUSSION

The present findings demonstrate the qualitative expressed potential of airborne secondary conidia in the spread of ergot disease of sorghum in the field in conjunction with the typical role of the sphacelial conidia.

Microcycle or secondary conidiation in a sorghum ergot pathogen was first recognized in the *in vitro* germination of axenically produced conidia of an Indian isolate of *C. sorghi*. At moderate temperature (24–30 °C) most macroconidia germinated in this way, whereas at 37 C spores germinated by forming a hyphal germ tube (Manzarpour, 1985). Secondary conidia *in vivo*

have recently been observed in Indian *C. sorghi* in controlled-environment cabinets and in glasshouse experiments (Bandyopadhyay *et al.*, 1990), but were a particularly striking phenomenon in an African pathogen (*C. africana*) when pathogens from India and Zimbabwe were compared directly on male-sterile sorghum in the UK (Frederickson *et al.*, 1991). Whereas secondary conidiation, causing a whitened surface on honeydew exudate, is the most prominent feature of sorghum ergot disease in the field in southern Africa, it is not unique to sorghum ergot pathogens. The secondary conidiation occurs *in vivo* also in *C. cynodontis* in Africa (Frederickson *et al.*, 1989) and in *C. paspali* in the USA (Luttrell, 1977) but has not been observed in temperate latitudes where *C. purpurea* is the common pathogen of cereals and grasses.

Dissemination of ergot fungi, exemplified by the best-studied pathogen *C. purpurea*, is mainly achieved over only a rather limited distance by conidia in honeydew passing by head-to-head contact, rainsplash and to some extent by insect vectors (Mantle, 1988). While honeydew might be thought to be particularly attractive to insects on account of its sugary composition, such potential vectors are not seen to be particularly abundant on cereal inflorescences exuding honeydew. Thus, even commercial cultivation of pharmaceutical ergot on rye has not presented a significant hazard to nearby cereal crops. In addition, airborne ascospores, providing primary inoculum each year, do not seem to travel far in air through cereal crops, possibly because spore discharge from stromata usually occurs from soil level. The filiform ascospores have to rise 0·5–2 m to impact on a receptive stigma, even when a weed grass has a role in epidemic development in a cereal crop (Mantle *et al.*, 1977). Thus, most ascospores impact ineffectually on vegetation near the site of discharge.

While the rapid epidemic development of *C. africana* in the present study may have been partly due to conventional contact mechanisms, a principal factor—at least in the spread to the furthest boundary of the experimental area traced back to the first gaping florests—must have been secondary conidia which had become airborne from infected inflorescences and which were recorded frequently in air sampled a little below the level of inflorescences. The fact that infection was possible within the confines of a fine-mesh net is a strong presumption of airborne propagules, given that total exclusion of all arthropoda while allowing wind penetration in the field would have been experimentally difficult. However, it should be noted that enclosed panicles were not meant to show whether an airborne factor was involved. The spore trap reconfirmed the common occurrence of secondary airborne conidia (Frederickson *et al.*, 1989) so that the enclosed panicles again demonstrated a probable actual role of such as infectious agents. There is no evidence that ascospores of *C. africana* are significant initiators of infection in sorghum. Indeed, the difficulty of obtaining the sexual stage (Frederickson *et al.*, 1991) probably implies that this is rare in the field. Use of male-sterile sorghum A-lines offered maximum opportunity for infection (unfertilized ovaries remaining receptive to ergot infection for several days after flowering [Frederickson & Mantle, 1988]), but the results are not unrealistic since the economic relevance of ergot disease of sorghum relates specifically to the A-lines in F1 hybrid seed production.

Considering the wide range of A-lines used, there was remarkable synchrony of their flowering, a factor favouring the appearance of the first infections at least 15 m from the primary source provided in the experimental design. This distance confirmed findings in a pilot study (Frederickson, 1990). In addition, ergot disease also occurred in other A-line plots located 300 m downwind from the experimental area. The timing of disease expression fitted airborne inoculum spreading from the experiment but no direct airborne connection with the experimental area could be proved. However, fertile sorghums 2 km away downwind remained free from disease.

The overall rate of spread of *C. africana* in sorghum A-lines ($r = 0{\cdot}2 \pm 0{\cdot}023$) compared favourably with values measured in *Phytophthora infestans* epidemics in the moderately susceptible potato variety Eigenheimer ($r = 0{\cdot}21$) and the susceptible Bintje ($r = 0{\cdot}42$) (Van der Plank, 1963). Even the 0·41 value measured for a wheat stem rust epidemic (Asai, 1960) barely matches the values of 0·58 and 0·48 for plots B21 and C27 in the present experiment.

Variation in r-values between A-lines may result from high inoculum pressure, giving high values as in plots B21 and C27, or by particular flowering characteristics restricting disease development as in the line IS 10638A. Receptivity of stigmas may vary, as may the rate of the basipetal flush of flowering within a panicle. The duration and extent of gaping of florests also varies so that differences in disease severity can be expected. Nevertheless, the presumed significance of secondary conidiation in epidemiology, at least

locally within a crop, has been substantiated. Airborne transmission is, therefore, a key factor in the risk of ergot epidemics in sorghum breeding in southern Africa.

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