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Pathology

Pearl Millet as an Alternate Host of the Sorghum Ergot Pathogen, *Claviceps africana*

D E Frederick son[1] and P G Mantle[2]

(1. Biochemistry Department, Imperial College, London SW7 2AY, UK; Present address: Biological Sciences, University of Zimbabwe, Harare, Zimbabwe; and 2. ICRISAT Southern and Eastern Africa Region, Matopos Research Station, PO Box 776, Bulawayo, Zimbabwe).

Introduction

The ergot pathogen, *Claviceps africana* Frederickson, Mantle and de Milliano, has been the major cause of severely depleted yields in sorghum (*Sorghum bicolor* (L.) Moench) hybrid seed production in southern Africa (de Milliano 1992). In Zimbabwe the pathogen causes annual grain losses of up to 25% following the colonization of male-sterile A-line ovaries, and additional losses of up to 12% also occur when contamination of seeds with honeydew causes mold growth, spoilage, and quality reduction (Frederickson and Leuschner, in press, McLaren 1994).

The host range of *C. africana* is poorly defined. In some of the earlier experiments with *C. africana* by Futrell and Webster in Nigeria in 1966, natural inoculum from *Panicum maximum* infected sorghum. Similarly, ergot from the same grass in Thailand was transferred experimentally to sorghum, as was inoculum from *Dicanthum annulatum, Brachiaria mutica,* and three sorghum species, *S. sudanensis, S. almum,* and *S. halcpanse* (Boon-Long 1992). It is assumed, from the evidence on sorghum ergot in Thailand (Frederickson et al. 1991), that the findings of Boon-Long all refer to *C. africana.* However, *C. africana* has not been found on any alternate host outside the genus *Sorghum* in Zimbabwe. Further, that the Indian sorghum ergot pathogen (*C. sorghi,* anamorph *Sphacelia sorghi)* can also infect *Jschaemum pilosum, Cenchrus setigerous,* and *C. ciliaris* (Chinnadurai and Govindaswamy 1971, Mughogho 1986) is not necessarily relevant to vegetative hyphal parasitism by *C. africana* since the two sorghum pathogens are taxonomically distinct, and *C. ciliaris* could not be infected by sorghum ergot in Zimbabwe, even using a high concentration of inoculum.

In Zimbabwe, pearl millet (*Pennisetum glaucum* (L.) R. Br.) is grown alongside sorghum as a communal crop. Although it matures earlier than sorghum, extensive tillering causes temporal overlap of the two crops. Since Sundaram (1974) found sphacelial infections of *C. sorghi* on pearl millet adjacent to sorghum in India, we thought that infectivity of *C. africana* on pearl millet should be studied experimentally in Africa.

Materials and Methods

At Matopos Research Station, near Bulawayo, Zimbabwe, pearl millet lines were sown in a 4 m x 6 m block in six replications in 1991, and two replications in 1992. Due to seed availability only four lines were common to both years. Upon emergence from the boot in April, spikes were bagged and inspected daily for the onset of stigma emergence. At approximately 20% stigma emergence they were inoculated with either the natural pathogen, *C. fusiformis,* or the sorghum ergot pathogen *C. africana,* Conidial inoculum (106 conidia mL^{-1}) was directed at inflorescences until run-off. Spikes were re-bagged, and the inoculation procedure repeated over the next 2-3 days until the completion of stigma exsertion. Concurrent inoculations of A-line sorghum with *C. africana* were performed to verify the infectivity of *C. africana* inoculum.

Ergot incidence and severity were assessed on pearl millet spikes 3-4 weeks post-inoculation. Infected spikes were removed to encourage further tillering of the millet and to allow microscopal verification of the identity of the pathogen based on conidial characteristics. In 1991, *C. africana* inoculum from successful pearl millet infection was re-inoculated onto pearl millet to see if its infectivity changed after one passage through the host.

Results

Claviceps africana consistently gave 100% disease incidence on male-sterile sorghum, and in the absence of a

Table 1. Comparative experimental ergot disease incidence in pearl millet genotypes by the natural pathogen, *Claviceps fusiformis,* and the unnatural pathogen, *Claviceps africana,* in Zimbabwe, 1991 and 1992.

	Disease incidence (%)			
	Natural pathogen *C.* fusiformis	Unnatural pathogen C. africana		*C.* africana after passage through pearl millet
Pearl millet line	1991	1991	1992	1991
ICMPES 25	-[1]	-	3	-
ICMPES 35	-	-	11.5(70)	-
ICMPES 39	0(12)[2]	15(138)	10	2(47)
ICMPES 45	23(13)	16(124)	4	3(54)
ICMSR 221	14(14)	17(218)	-	2(74)
ICMSR 260	43 (15)	23 (167)	-	7(77)
ICMH 451	0(10)	7 (62)	-	4(23)
PMV 1	36(11)	20(183)	18(114)	11 (65)
852 B	7(67)	2.5(78)	7 (14)	-

1. - = not tested.
2. Number of inoculated inflorescences shown in parentheses.

pollinator, achieved approximately 95% disease severity within infected inflorescences.

The natural ergot pathogen of pearl millet (*Claviceps fusiformis*) established disease with moderate incidence on most, but not on all, of the lines tested (Table 1); but disease severity was relatively low, not exceeding 15%.

In contrast, *C. africana* established a parasitic association with all the pearl millet lines tested, with incidence as high as 23% in ICMSR 260, the genotype that had also supported the highest incidence when inoculated with *C. fusiformis.* However, severities were always low, at 1-5%. Consistent findings were obtained in the pearl millet lines that were common to the 2 consecutive years' experiments. Arc-sine transformed data for experiments in 1992 showed the mean comparative incidence of *Claviceps africana* in a sorghum A-line (100 %) versus three pearl millet lines (2.5,11.5, and 18%) were significantly different at $P = 0.01$. All infections on pearl millet lines were verified as the sphacelial stage (*Sphacelia sorghi*) of *C. africana* microscopically by virtue of their conidial characteristics; the sphacelial fructification of *C. fusiformis* is quite different from that of *S. sorghi.*

After one passage through a pearl millet host, *C. africana,* did not apparently become more infectious on this host. Rather, the results suggest that infectivity may have declined. Indeed, although *C. africana* inoculum passaged through pearl millet still infected male-sterile sorghum, it did so with reduced (49%) severity.

Discussion

The experimental observations confirm pearl millet as an additional host of endemic *C. africana* under high disease pressure in Zimbabwe, even in the more difficult conditions for infection during years of severe drought in southern Africa. While this may not be of local practical importance, the recent global extension of ergot disease in sorghum for the first time in South America east of the Andes in 1995 (Reis et al. 1996) and in Queensland, Australia in 1996, has greatly heightened interest in and concern for the economic consequences of the disease. Relatively little firm evidence on reservoirs of inoculum exists in the literature and the present short report, coupled with similar recent findings in Brazil (E M Reis, personal communication) and Queensland (M Ryley, personal communication) formalizes a role for pearl millet that may at least be helpful in phytosanitary considerations to reduce further adverse impact of *C. africana* disease in sorghum. Whereas ergot disease principally influences F_1 hybrid seed production, the effects on grain sorghum production could become significant if widespread reservoirs of infection become established.

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Effect of Different Levels of Nitrogen and Time of Sowing on Incidence of Grain Mold in Pearl Millet

Padma Sri M, and A V Ramana Reddy
(Sri Venkateswara Agricultural College, Andhra Pradesh Agricultural University, Tirupati, Andhra Pradesh, India)

Pearl millet, *Pennisetum glaucum* (L.) R. Br., the fourth most important food crop in India, it is a drought-tolerant crop widely distributed in the semi-arid tropics. Among several factors that restrict the production potential of pearl millet, head molds play a dominent role in reducing production, and the market value of grain in Chittoor and Cuddapah districts of Andhra Pradesh.

In preliminary studies conducted in the laboratory, several fungi, e.g., *Curvularia* spp, *Alternaria* spp, *Fusarium* spp, *Drechslera* spp, and *Penicillium* spp were found associated with head mold of pearl millet. Similar results were also reported by Luttrell (1954) in USA, and Girisham and Reddy (1985). Therefore, this study was made to determine the effect of different levels of nitrogen, and different sowings dates on the incidence of mold in pearl millet.

The seeds were sown at 10-day intervals, starting 27 Jul 1989 at a seed rate of 4 kg ha^{-1}, in a nursery that was maintained weed-free for 21 days. After fertilizers had been applied to the main field, 21-day-old healthy seedings were transplanted into it at the rate of one seedling $hill^{-1}$, with 45 cm between rows and 15 cm within the rows.

The experiment was conducted using a split-plot design with sowing dates as the main plots (18 x 13.5 m), and N levels as subplots (3.6 x 2.7 m). All the treatments were replicated thrice. The five sowing dates and five nitrogen levels used in 1989 as treatments were D_1 (27 Jul), D_2 (7 Aug), D_3 (17 Aug), D_4 (27 Aug), D_5 (7 Sep), N_0 (0 kg N ha^{-1}), N, (25 kg N ha^{-1}), N_2 (50 kg N ha^{-1}), N_3 (75 kg N ha^{-1}), N_4 (100 kg N ha^{-1}).

There were 25 treatments for various combinations of sowing dates and nitrogen levels:

T_1-D_1N_o	T_6-D_2N_0	T_{11}-D_3N_0	T_{16}-D_4N_0	T_{21} - D_5N_0
T_2-D_1N_1	T_7-D_2N_1	T_{12}-D_3N_1	T_{17}-D_4N_1	T_{22} - D_5N_1
T_3-D_1N_2	T_8-D_2N_2	T_{13}-D_3N_2	T_{18}-D_4N_2	T_{23} - D_5N_2
T_4-D_1N_3	T_9-D_2N_3	T_{14}-D_3N_3	T_{19}-D_4N_3	T_{24} - D_5N_3
T_5-D_1N_4	T_{10}-D_2N_4	T_{15}-D_3N_4	T_{20} - D_4N_4	T_{25} - D_5N_4

The results indicated that there was minimum (12.9%) mold incidence in the crop sown on 17 Aug with 0 kg N ha^{-1}. Such low incidence may be due to the prevailing high temperature (36.4°C) coupled with low (68.4%) rel-