

presented at the VI Annual Small Millets Workshop, May 1992, Ranchi, Bihar, India.

Maloo, S.R. 1995. Small millet as an economically alternative poultry food ingredient. Paper presented at the 2nd Agricultural Science Congress, Jan 1995, Andhra Pradesh Agricultural University, Hyderabad, Andhra Pradesh, India.

Sampath, T.V., Razvi, S.M., Singh, D.N., and Bondale, K.V. 1990. Small millets in Indian Agriculture. Page 32 in Small millets in global agriculture. (Seetharam, A., Riley, K.W., and Harinarayana, G., eds.). New Delhi: Oxford and IBH Publishing Co.

Pathology

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

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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 exertion. 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 microscopical 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.

Pearl millet line	Disease incidence (%)			
	Natural pathogen <i>C. fusiformis</i>		Unnatural pathogen <i>C. africana</i>	
	1991	1991	1992	<i>C. africana</i> after passage through pearl millet 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 sphaelial stage (*Sphaelia sorghi*) of *C. africana* microscopically by virtue of their conidial characteristics; the sphaelial 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.

References

Boon-Long, T. 1992. Sorghum diseases of Thailand. Pages 41-43 in Sorghum and millets diseases: a second

world review (de Milliano, W.A.J., Frederiksen, R.A., and Bengston, G.D., eds.). Patancheru 502 324, Andhra Pradesh, India: International Crops Research Institute for the Semi-Arid Tropics.

Chinnadurai, G., and Govindaswamy, C.V. 1971. Host range of the sugary disease pathogen. *Madras Agricultural Journal* 58:600-603.

de Milliano, W.A.J. 1991. Sorghum diseases in Southern Africa. Pages 9-19 in *Sorghum and millets diseases: a second world review* (de Milliano, W.A.J., Frederiksen, R.A., and Bengston, G.D., eds.). Patancheru 502 324, Andhra Pradesh, India: International Crops Research Institute for the Semi-Arid Tropics.

Frederickson, D.E., Mantle, P.G., and de Milliano, W.A.J. 1991. *Claviceps africana* sp. nov.; the distinctive ergot pathogen of sorghum in Africa. *Mycological Research* 95:1101-1107.

Frederickson, D.E., and Leuschner, K.L. (In press.) Potential use of benomyl for the control of ergot (*Claviceps africana*) in sorghum A-lines in Zimbabwe. *Plant Disease*.

Futrell, M.C., and Webster, O.J. 1966. Host range and epidemiology of the sorghum ergot organism. *Plant Disease Reporter* 50:828-831.

McLaren, N.W. 1994. Efficacy of systemic fungicides and timing of preventative sprays in the control of sugary disease of grain sorghum (*Sorghum bicolor*). *South African Journal of Plants and Soil* 11:30-33.

Mughogho, L.K. 1986. Ergot. Pages 38-49 in *Compendium of sorghum diseases* (Frederiksen, R.A., ed.). American Phytopathological Society.

Reis, E.M., Mantle, P.G., and Hassan, H.A.-G. 1996. First report in the Americas of sorghum ergot disease, caused by a pathogen diagnosed as *Claviceps africana*. *Plant Disease* 80:463.

Sundaram, N.V. 1974. Natural occurrence of *Sphacelia sorghi* on *Pennisetum typhoides*. *Indian Phytopathology* 27:124-125.

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

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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 dominant 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 seedlings 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 D₁ (27 Jul), D₂ (7 Aug), D₃ (17 Aug), D₄ (27 Aug), D₅ (7 Sep), N₀ (0 kg N ha⁻¹), N₁ (25 kg N ha⁻¹), N₂ (50 kg N ha⁻¹), N₃ (75 kg N ha⁻¹), N₄ (100 kg N ha⁻¹).

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

T₁-D₁N₀ T₆-D₂N₀ T₁₁-D₃N₀ T₁₆-D₄N₀ T₂₁-D₅N₀
T₂-D₁N₁ T₇-D₂N₁ T₁₂-D₃N₁ T₁₇-D₄N₁ T₂₂-D₅N₁
T₃-D₁N₂ T₈-D₂N₂ T₁₃-D₃N₂ T₁₈-D₄N₂ T₂₃-D₅N₂
T₄-D₁N₃ T₉-D₂N₃ T₁₄-D₃N₃ T₁₉-D₄N₃ T₂₄-D₅N₃
T₅-D₁N₄ T₁₀-D₂N₄ T₁₅-D₃N₄ T₂₀-D₄N₄ T₂₅-D₅N₄

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-