

**AN ANNOTATED BIBLIOGRAPHY ON ERGOT OF PEARL MILLET
1920-1987**

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Patancheru, A.P. 502 324, India.
1988**

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Correct citation: Chahal, S.S., Thakur, R.P., and King, S.B. 1988. An annotated bibliography on ergot of pearl millet, 1920-1987. Patancheru, A.P. 502 324, India: International Crops Research Institute for the Semi-Arid Tropics.

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I. PREFACE

Ergot, caused by Claviceps fusiformis Lov., is an important disease of pearl millet [(Pennisetum glaucum (L.) R.Br.)] in the semi-arid tropical parts of the Indian subcontinent and Africa. In recent years the disease has drawn considerable attention, especially because of its widespread occurrence on high yielding F₁ hybrids in India. Research on pearl millet ergot has been limited and published information is scattered. Information problems faced by researchers, because of limited library facilities and difficulties in obtaining relevant literature, prompted us to annotate and list the published literature on this disease.

The bibliography includes published research information from 1920 to 1987. Annotations have been written from the original papers, abstracts, summaries, and the Review of Plant Pathology with the objective of emphasizing important findings. We have excluded information reported in annual reports, theses/dissertations, papers presented in workshops/conferences which are not abstracted, and other media not readily available.

A total of 165 references have been annotated. These are arranged numerically in alphabetical order. Nomenclature of host and pathogen appearing in this bibliography are listed, and author and subject indices are provided. The subject index has been divided into 12 major topics against which relevant reference numbers are indicated.

We hope the bibliography will prove useful to pearl millet researchers, extension workers, and students.

ACKNOWLEDGEMENT

We thank J.M.J. de Wet, Program Director, Cereals, ICRISAT, and K.S. Nandpuri, Director Research and K.S. Aulakh, Head, Department of Plant Pathology, Punjab Agricultural University, Ludhiana, for approving the visit of S.S. Chahal to complete this work. We appreciate the cooperation of the ICRISAT library staff in procuring references and N.K. Ganapathy (Millet Pathology) for his Secretarial assistance.

II. ~~TERMINOLOGY~~ LITERATURE IN THE BIBLIOGRAPHY

The Host:

Scientific names:

Pennisetum glaucum (L.) R. Br.

Pennisetum americanum (L.) Leeke

Pennisetum typhoides (Burm.) Stapf and Hubbard

Pennisetum typhoideum L.C. Rich.

English names:

Bulrush millet

African millet

Indian millet

Pearl millet

Vernacular names:

Bajra

Bajri

Cumbu

Kumbu

Munga

The Pathogen:

C. ~~avicens~~ fusiformis Loveless

C. ~~avicens~~ microcephala (Wallr.) Tul.

III. ANNOTATIONS

1. Agarell, S., and Ramstad, E. 1962. Analysis of *Pennisetum ergot*. *Lloydia* 25(2):67-77.
2. Anand Kumar, K., and Andrews, D.J. 1984. Cytoplasmic male sterility in pearl millet [*Pennisetum americanum* (L.) Leeke] - A review. *Advances in Biology* 10:113-143.

All existing cytoplasmic male-sterile (CMS) lines and other breeding lines of pearl millet were susceptible to ergot (*Claviceps fusiformis*). ICRISAT millet pathologists have been able to identify and buildup high levels of ergot resistance through selection in crosses between lines with low levels of resistance. The progress on incorporation of ergot resistance into CMS lines using backcross breeding was described.

3. Andrews, D.J., King, S.B., Witcombe, J.R., Singh, S.D., Rai, K.N., Thakur, R.P., Talukdar, B.S., Chavan, S.B., and Singh, P. 1985. Breeding for disease resistance and yield in pearl Millet. *Field Crops Research* 11: 241-258.

A detailed account of the pearl millet breeding program for disease resistance and high grain yield carried out at ICRISAT since 1973 is presented. Field screening techniques developed and improved for identification of resistance to ergot, smut, and downy mildew were described. The screening technique for ergot eliminated escape from infection due to extraneous pollen. Ergot inoculum is sprayed onto previously covered protogynous inflorescences developed in a pollen- and inoculum-free environment and immediately rebagged after inoculation. Resistant lines developed by crossing least ergot-susceptible plants are being used in breeding programs.

Several lines have been identified having combined resistance to ergot, smut and downy mildew. The international disease nurseries have been operated to test the stability of resistance. Resistance to ergot is reported to be recessive and multigenic. A backcross breeding program is being followed to transfer resistance into both B and R

lines to produce ergot-resistant hybrids. Exploitation of African material for variability in different characters and development of open-pollinated varieties following recurrent selection and conventional pedigree breeding are discussed.

4. Anonymous. 1973. Controlling ergot disease of bajra. Intensive Agriculture 11(10):8.

Control of ergot by spray of 0.0025% Aureofungin or 0.15% Cuman or Duter thrice at 5-day intervals beginning with earhead emergence was suggested.

5. Anonymous. 1978. Mycotoxicosis in humans: Ergotism. ICMR Bulletin 8(2):2

An account is given of research observations of different workers on the sporadic occurrence of ergot (*Claviceps microcephala*) of pearl millet in India, clinical manifestations of symptoms in humans if the level of contamination of ergot exceeds 1.5% (w/w), nature of alkaloids belonging to the clavine group, results of feeding trials with ergot infested grains of pearl millet on guinea pigs and monkeys, and effects of intraperitoneal injections of alkaloids were described.

6. Anonymous. 1979. Downy mildews and ergot. Farm Fare 1(2):36-37.

Practices to minimize ergot (*C. avicena fusiformis*) in pearl millet included: brine water treatment (5% salt solution) for removal of sclerotia from seed, two to three sprays with 0.2% Ziarn, Miltox or Blitane during flowering, removal and destruction of ergot-infected earheads immediately after their appearance in the crop and after harvesting from the field, deep plowing, and crop rotation. Control measures for downy mildew were also described.

7. Appadurai, R., Subramanian, R., Paramaramani, C. and Purushothaman, S. 1978. Development of disease resistant pearl millet (*Pennisetum typhoides* (Burm.f.) Stapf and C.E. Hubb.). Madras

A pearl millet bulk population, UCV 1, primarily developed for downy mildew resistance, showed a uniformly low incidence of ergot compared to a high incidence (65%) in F₁ hybrids in experimental trials. This was probably due to high pollen productivity which helped escape ergot infection. This variety was released for general cultivation as CO 6 in Tamil Nadu, India.

8. Arya, H.C., and Kumar, A. 1976. Diseases of bajra - a serious problem of Rajasthan desert economy. Transactions of Indian Society of Desert Technology and University Centre of Desert Studies 1(2): 177-182.

Ergot recorded from almost all the Indian states could be identified by appearance of a viscous, sugary exudate emanating from infected florets where sclerotia form in place of grains. Macroconidia (18.9 x 3.1µm) and microconidia (4.9 x 2.8µm) of *Cl. viciae microcephala* spread the disease initiated by the ascospores produced by the germinated sclerotia. The fungus grew readily on Leonian's medium at 25° C at pH 5.0 and 6.0. None of the varieties or hybrids tested were immune. Besides a few cultural practices, like early sowing, seed sanitation, and deep plowing after harvest, fungicidal sprays with Ziram (0.1-0.15%) or a mixture of copper oxichloride and Zineb (500-600 g/ha) twice or thrice prior to ear emergence, were reported for ergot control. Results of research on ergot and downy mildew of pearl millet have been reviewed in light of their importance in Rajasthan. The seriousness of diseases increased after the introduction of F₁ hybrids.

9. Arya, H.C., and Kumar, A. 1982. Ergot epidemic of pearl millet in Rajasthan. Pages 439-451 in Recent Advances in the Biology of Microorganisms. Vol.II. (Bilgrami, K.S., Vyas, K.M., Singh, Bishan, and Singh, M.P. eds.). Dehradun, India: Bishen Singh Mahendra Pal Singh.

Results of investigations of pearl millet ergot with special reference to Rajasthan, India, were reviewed and discussed. Ergot appeared in

an epidemic form in Rajasthan in 1967-1968, 1973, 1975, and 1976. Disease syndrome and etiology of the pathogen were described. The authors also presented a diagrammatic representation of the life cycle of the fungus based on the findings of other workers. The pathogen produces ergotoxins that on consumption cause drowsiness followed by hyperexcitation and redness of face in human beings. Ergot was reported as endemic with primary infection by the sclerotial inoculum and secondary spread by means of conidia. Environmental factors greatly influence disease development. Optimum growth of the fungus on Kirchoff's and Leonian's media occurred at 25° C in light between 2000 and 3000 lux. Several graminaceous plants have been reported as collateral hosts by different workers. Fungicidal control of this disease was suggested by spraying Ziram (0.1-0.15%) or a mixture of copper oxychloride and Zineb (500-600 g/ha), 2-3 times just before flowering. Some other chemicals were also found effective but these reduced seed set.

10. Bajaj, Y.P.S., Phul, P.S., and Sharma S.K. 1980. Differential tolerance of tissue culture of pearl millet to ergot extract. Indian Journal of Experimental Biology 18:429-432.

Preliminary studies revealed differential growth inhibition of pearl millet callus cultures of cultivars PHB 10 and PHB 14 by the extract of Claviceps fusiformis sclerotia. The germination of seeds and excised embryos was not affected by the extract of ergot sclerotia but rooting was inhibited. The medium containing ergot extract, when used at 48 g sclerotia/L concentration, completely inhibited cell proliferation initially; however, a few cells survived and resumed their growth. Certain cells could survive and after a lag period of 2 or more weeks started dividing. The possibility of selecting potentially tolerant or resistant cell lines and regeneration of plants from them is discussed.

11. Bajpai, Nisha. 1976. Bajra causes ergot poisoning. Science Reporter 13:494-495.

A general account of the disease and poisoning

due to consumption of ergot infected pearl millet was reported from Maharashtra and Rajasthan. A survey report by the National Institute of Nutrition, Hyderabad, revealed that out of 14 households in 21 villages, 80% had suffered ergot poisoning with symptoms like nausea, vomiting, and giddiness due to alkaloids such as elymoclavine, chemoclavine, penniclavine, setoclavine, etc. The use of resistant varieties was suggested as a control measure.

12. Balasubramanian, G., Sivaprakasam, K., Robinson, J.G., and Ramaswamy, N.M. 1980. Varietal reaction of pearl millet to the shootfly and ergot disease. *Pestology* 4(2):16-18.
13. Banerjee, S.N., and Joshi, N.C. 1971. Occurrence of some serious diseases on hybrid bajra in Delhi during 1970 and methods of their control. *Pesticides* 5:20-21.

A survey report of pearl millet diseases in the Union Territory of Delhi during Kharif 1970 revealed that out of a total area of 33 585 acres, about 15 661 acres were affected by different diseases. Downy mildew, ergot, smut, and *Pyricularia* blast were recorded and the incidence varied from 5 to 70%, 5 to 90%, 5 to 10% and 2 to 12% for these diseases, respectively. Symptoms of these diseases were also described.

14. Bangar, S.G., Patil, B.C., and Rane, M.S. 1983. Anthesis in relation to ergot of pearl millet. *MILWAI Newsletter* 2:10.

It was observed on seven pearl millet varieties that the longer the time between initiation of stigmas and anthers, the higher the incidence of ergot (*Claviceps fusiformis*).

15. Banks, G.T., Mantle, P.G., and Szczyrbak, Christine A. 1974. Large scale production of clavine alkaloids by *Claviceps fusiformis*. *Journal of General Microbiology* 82: 345-361.

The optimum conditions for large-scale alkaloid production by a strain of *Claviceps fusiformis* was studied. The assessment of 25 sucrose/ammonium

sulfate combinations showed that 200 g/L sucrose and 11.8 g/L ammonium sulfate gave a maximum alkaloid yield of 3.7 mg/mL within 9 days when the fungus was grown in sucrose-ammonium-sulfate inorganic salts medium in 400 L, stirred fermentors. The alkaloid production was maximum when the fungal mass became plectenchymatous. Large-scale production of agroclavine by batch or half-replacement fermentation facilitated both production of high yields of alkaloids and subsequent extraction from the culture filtrate. Throughout the fermentation process oxygen supply was ensured. The alkaloid was extracted efficiently from culture filtrate by a solvent-extraction procedure that involved sequential transfer of product into N-butanol, aqueous tartaric acid, and chloroform followed by crystallization from acetone. The increase in concentration of magnesium sulfate, zinc sulfate, and potassium dihydrogen orthophosphate in multistage fermentation also improved the agroclavine concentration to 6 mg/mL. With this procedure large quantities of pure agroclavine for pharmacological studies is possible.

16. Bhat, R.V. 1977. Identity of the pathogen causing ergot of pearl millet in India. Current Science 46:184-185.

The sclerotia of pearl millet ergot pathogen were obpyriform, up to 7 mm long and 4 mm wide, generally curved, with maximum width at the base. The conidia were hyaline, fusiform to broadly falcate and 28.0-40.0 μ m long, 8.0 μ m wide. The alkaloids from honeydew consisted of ergoclavine, elymoclavine, chanoclavine, setoclavine, and penniclavine, which are different from those produced by C. purpurea. Based on these morphological and chemotaxonomic criteria, the pathogen causing ergot of pearl millet in India was identified as Claviceps fusiformis.

17. Bhat, R.V., and Roy, D.N. 1976. Toxicity study of ergoty bajra (pearl millet) in rhesus monkeys. Indian Journal of Medical Research 64(11):1629-1633.

Sclerotia of Claviceps fusiformis from pearl

millet and their alkaloids administered to male rhesus monkeys by different ways. Sclerotia caused pronounced hyperexcitation when ingested at a rate equivalent to 10 mg alkaloids/kg body weight. Following injection of alkaloids intraperitoneally, toxicity was manifested in the form of hyperexcitation, restlessness, ataxicness, muscle twitching, redness of face, biting of the ground, and loss of response to thermal and tactile stimuli in the hind limbs and tail. The symptoms appeared within 10 min and there was complete, spontaneous recovery in about an hour. These symptoms differed from the toxicity symptoms in humans. The safe limits of the alkaloid dose for humans could not be determined; however, based on circumstantial evidence it appeared that humans are more sensitive to ergot alkaloids than monkeys.

18. Bhat, Ramesh V., Roy, D.N., and Tulpule, P.G. 1975. Ergot contamination of bajra. Proceedings of the Nutrition Society of India 19:7-11.

Consumption of ergoty (Claviceps fusiformis) bajra grains by humans caused gastrointestinal disturbances like nausea, vomiting, giddiness, and diarrhea, and in extreme cases death, in Maharashtra, Gujarat, and Rajasthan. The amount of total alkaloids present in ergot sclerotia was 32 mg/100 g (as against 70 mg/100 g in ergot of rye). However, the alkaloids of pearl millet were different from those of ergot of rye. The safe limit could not be determined due to variable symptom development in man and monkey; however, it was reported that the Central Committee of Food Standards of the Directorate General of Health Services arbitrarily fixed a safe limit of 0.05% of contaminated grains on the basis of safe limits fixed in Western countries for ergot of rye.

19. Bhat, R.V., Roy, D.N., and Tulpule, P.G. 1976. The nature of alkaloids of ergoty pearl millet or bajra and its comparison with alkaloids of ergoty rye and ergoty wheat. Toxicology and Applied Pharmacology 36:11-17.

The alkaloids present in ergot (Claviceps microcephala) sclerotia from pearl millet mainly contain agroclavine and elymoclavine. They did not

respond to the Cock's comb test even in high doses and they did not show any amino acid residues in the acid hydrolysis test. In ergot sclerotia from wheat and rye, the main alkaloid present was ergotamine. When administered separately to chicks, either by injection or feeding, alkaloids from sclerotia of all the three crops caused common symptoms such as listlessness, gasping, drooping of wings, leg weakness, and occasional vomiting. Death occurred more rapidly with ergoty wheat and rye than with ergoty bajra.

20. Bhide, V.P., and Hegde, R.K. 1957. Ergot of bajri [(Pennisetum typhoides (Burm.) Stapf and Hubbard)] in Bombay state. Current Science 26(4):116.

Ergot on bajri (Pennisetum typhoides) was recorded for the first time at a high level (up to 25% in some fields) in India in 1956 in south Satara and Kolhapur districts of Bombay, and in Belgaum and Bijapur districts of Mysore states. It was observed at a low level in Banakskantha district in October 1955. The conidial size of the unidentified pathogen was reported as 20.7 x 6.1 um.

21. Bhora, C.P., and Bansal, V. 1981. Design and development of bajra separator. Transactions of Indian Society of Desert Technology and University Centre of Desert Studies 6(1):37-40.
22. Bindu, K.J., and Narhede, S.T. 1979. Role of pollen and conidia in the occurrence of ergot disease on pearl millet. Research Bulletin of the Marathwada Agricultural University 3(9):123-124.
23. Brack, A., Bruner, R., and Kobel, H. 1962. The course of alkaloid synthesis in saprophytic cultures by a strain of Claviceps from Pennisetum typhoides. Archiv der Pharmazie und Berichte der Deutschen Pharmazeutischen Gesellschaft 295(7):510-515.
24. Brar, G.S., Chand, J.N, and Thakur, D.P. 1976. Fungicidal control of ergot of bajra. Haryana Agricultural University Journal of Research 7:1-5.

Spore germination of Claviceps fusiformis was

completely inhibited in vitro by Difolatan at 100 ppm and by Benlate, Miltox, Dithane M-45, Kasumin, Ziram, and Vitavax each at 500 ppm concentration. Difolatan completely prevented the infection of ergot on the inflorescences of hybrids HB 1 and HB 3 when sprayed at the boot stage. Dithane M-45, Ziram, and Miltox also reduced the infection.

25. Chaha, S.S. 1984. Role of sclerotia in epidemiology of pearl millet ergot caused by *Claviceps fusiformis* Loveless. Indian Journal of Mycology and Plant Pathology 14 Supplementary (1): 12-13. (Abstract).

The sclerotia of *C. fusiformis* germinated after 40 days at 28 ± 1 °C with continuous high level of moisture, producing 1-12 stromata per sclerotium in a pot experiment. Stipes measured 4-6 x 1-2 mm. Capitula were globose with broadly pyriform perithecia and cylindrical asci and ascospores (80-145 x 0.4-0.6 µm). The germination was more in partially and 1 cm deep buried sclerotia than those buried at 4 cm depth. The disease developed when protogynous pearl millet inflorescences were exposed to ascospores indicating their positive role as primary source of inoculum.

26. Chahal, S.S., and Dhindsa, H.S. 1985. Relationship between ergot severity and rainfall distribution during flowering in pearl millet. Indian Phytopathology. 38:329-331.

Ergot (*Claviceps fusiformis*) severity on three pearl millet cultivars, NHB-3, PSB-8 and 7042 was highest during 1983 (with 12 rainy days during flowering) followed by that in 1979 (with 5 rainy days) and 1982 (with 3 rainy days). It was least in 1981 when there were no rains during flowering. There were large differences between ratios of airborne ascospores + conidia: pollen grains which were 1:13 and 1:3 in a non-rainy year (1981) and a rainy year (1983), respectively. The significant reduction in pollen grains in relation to ergot spores increased the ergot severity. The early sown varieties escaped the disease possibly because of effective pollination before the availability of inoculum.

27. **Chahal, S.S., Gill, G.S. and Sharma, S.B. 1987.** Effect of culture filtrate of Fusarium chlamydosporum on growth of Claviceps fusiformis and ergot development in pearl millet. *Plant Disease Research* 2(1):32-36.

The in vitro growth of C. fusiformis was inhibited by the culture filtrate of Fusarium chlamydosporum isolated from ergot sclerotia. The culture filtrate was non-phytotoxic to pearl millet seedlings. In field experiments, ergot development was inhibited most when undiluted filtrate was applied simultaneously with the inoculum at the protogyny stage on genotypes PHB 10 and PCB 15. Significant inhibition was caused by culture filtrate applied 24 h before and after inoculation with ergot conidia. The F. chlamydosporum filtrate was more effective than the spore suspension.

28. **Chahal, S.S., Gill, K.S., Phul, P.S., and Singh, N.B. 1981.** Effectiveness of successive intermating and selection for generating ergot resistance in pearl millet. Page 24 in Abstracts of the Third International Symposium on Plant Pathology 14-18 December 1981. Indian Agricultural Research Institute, New Delhi, India.

29. **Chahal, S.S., Gill, K.S., Phul, P.S., and Singh, N. B. 1981.** Effectiveness of recurrent selection for generating ergot resistance in pearl millet. *SABRAO Journal* 13:184-186.

Screening of a large number of Pennisetum typhoides (P. americanum) inbred lines against Claviceps fusiformis under artificial epiphytotic conditions revealed a lack of major gene resistance, and a new strategy involving recurrent selection to concentrate minor genes controlling polygenic resistance for intrapopulation improvement was developed and pursued. Inbred lines with less than 5% ergot severity were selected and intermated to produce two diallel populations. The progenies were tested for resistance and recurrent intermating and selection was followed separately in these populations. After 3 and 4 cycles of recurrent selection in these populations, the proportions of plants with 0-5% ergot severity

increased considerably indicating the possibility of achieving an appreciable level of ergot resistance by concentrating genes for resistance by recurrent selection.

30. Chahal, S.S., Kumar, R., Sidhu, J.S., and Minocha, J.L. 1986. Peroxidase isozyme pattern in pearl millet lines resistant and susceptible to ergot. *Indian Phytopathology* 39(1):65-69.

Considerable differences were found in the number and concentration of peroxidase isozymes among pearl millet genotypes susceptible (PIB 1231-1, PIB 2231, PIB 1223, PIB 1016 and BIL 3B) and resistant (ICMPES 2, ICMPES 8, ICMPES 17, ICMPES 34, and ICMPES 37) to ergot (*Claviceps fusiformis*). The results suggested the possibility of using peroxidase isozyme pattern analysis as a technique to rapidly screen pearl millet lines for resistance to ergot at the seedling stage. Need for drawing definite conclusions based on a large number of lines and samples from different growth stages was discussed.

31. Chahal, S.S., Mangat, B.K., and Virk, D.S. 1987. Morphological differences between spikelets of some ergot resistant and susceptible pearl millet lines. Page 41 in Abstracts of the First Symposium on Crop Improvement, 23-27 February, 1987. Crop Improvement Society of India, Punjab Agricultural University, Ludhiana, India.

Five ergot (*Claviceps fusiformis*) resistant pearl millet lines showed significantly less time span between emergence of stigmas and anthers as compared with lines moderately susceptible or susceptible to ergot. The length of style was also relatively shorter in the resistant lines.

32. Chahal, S.S., Rao, V.P., and Thakur, R.P. 1985. Variation in morphology and pathogenicity in *Claviceps fusiformis*, the causal agent of pearl millet ergot. *Transactions of British Mycological Society* 84(2):325-332.

Sclerotia of *C. fusiformis* from Aurangabad and ICRISAT Center were the largest (5 x 2.5 mm) and heaviest (1.4 g/100 sclerotia) and from Mysore the

smallest (3 x 2 mm) and lightest (0.4 g/100 sclerotia), with minimum number of furrows in the Mysore collection and maximum in Kovilpatti. Size of macro- and microconidia residual on sclerotia from different locations and ratios of their numbers varied considerably, and time for initiation of germination of macroconidia also varied from 24 h in Aurangabad and Jobner collections to 50 h in Kovilpatti collection. The isolates from these collections differed in colony characters in culture and in virulence on six pearl millet genotypes. Basic information on the extent of morphological variation in *C. fusiformis* in India are given, and lines for further investigations are discussed.

33. Chand, J.N., and Thakur, D.P. 1974. Ergot of bajra and its control. Haryana Farming 3(3):4.
34. Dhindsa, H.S., and Chahal, S.S. 1986. Development of ergot sclerotia on in vitro cultured spikelets of pearl millet. Annals of Biology 2(1):67-71.

Sclerotia of *Claviceps fusiformis* developed in excised spikelets of a pearl millet variety, PSB 8, maintained in vitro on Murashige and Skoog's medium. The stigmas appeared 7 days after placement of the immature spikelets on the medium. When inoculated with *C. fusiformis* conidia, they withered within 2.5 days. On an average, honeydew appeared 5.5 days and sclerotia appeared 14 days after inoculation in 72% and 71.2% of the inoculated spikelets, respectively. The characteristics of axenic cultures from these sclerotia were similar to those obtained from sclerotia collected from the field. The possibility of a laboratory screening technique was discussed.

35. Esvarasurthy, S., Nagarajan, C., Prasad, M.N., Raveendran, T.S., and Shanmugam, N. 1983. Varietal reaction to ergot among awned and awnless varieties of pearl millet. Page 27 in Abstracts of the National Seminar on Breeding Crop Plants for Resistance to Pests and Diseases. 25-27 May, 1983. Tamil Nadu Agricultural University, Coimbatore, India.

Eighteen awned entries of Pennisetum americanum exhibited less than 5% infection by Claviceps fusiformis compared with awnless entries, I4 (33.0%), IBH 428 (27.8%), MS 5141 A (60%), and ICH 440 (75%) under artificial inoculation in the field.

36. Esvaramurthy, S., Nagarajan, C., Prasad, M.M., Raveendran, T.S., and Shanmugam, N. 1983. Varietal reaction to ergot of pearl millet. Page 28 in Abstracts of the National Seminar on Breeding Crop Plants for Resistance to Pests and Diseases, 25-27 May, 1983. Tamil Nadu Agricultural University, Coimbatore, India.

Of 287 entries of Pennisetum americanum, 30 exhibited less than 5% ergot infection and 9 showed less than 2.1% infection when inoculated artificially with conidia of Claviceps fusiformis, compared with 60% in MS 5141A and 75% in ICH 440. Among the nine low susceptible lines, D 111-19 and D 763-10 were pollinators and MS 5540-361 was a male-sterile line.

37. Gill, K.S., Chahal, S.S., and Phul, P.S. 1980. Strategy to develop ergot resistance in pearl millet. Pages 159-161 in Trends in Genetical Research on Pennisetums (Gupta, V.P., and Minocha, J. L. eds.) Punjab Agricultural University, Ludhiana.

Screening of germplasm available at the Punjab Agricultural University, Ludhiana, under artificial epiphytotic conditions resulted in the identification of a few lines as moderately resistant to Claviceps fusiformis. A scheme to generate resistance to ergot is suggested. It includes repeated intermating of moderately susceptible genotypes to allow for new recombinations and then selection for resistant genotypes.

38. Gill, K.S., Phul, P.S., Chahal, S.S., Singh, N.B., Bhardwaj, B.L., and Jindla, L.N. 1979. Studies on disease resistance for the improvement of bajra (Pennisetum typhoides (Burm.) S. & H. Pages 1-80 in Project Report. Punjab Agricultural University, Ludhiana.

Among the large number of inbred lines of pearl millet tested, none was found with an appreciable level of resistance to Claviceps fusiformis. A scheme involving concentration of minor genes through repeated cycles of intermating and selection of the low susceptible lines was devised and pursued resulting in 395 plants with less than 5% ergot severity. The report also includes identification of some lines with resistance to downy mildew, smut, and rust, and survival of Pyricularia oryzae. Also, information on downy mildew with respect to inheritance of resistance, effect of sowing dates, and biochemical basis of resistance is given.

39. Gour, H.N., Verma, O.P., and Pathak, V.N. 1985. Loss of ions in pearl millet seeds treated with honeydew of Claviceps fusiformis. *Gov. Current Science* 54(12):581-582.

The ergot toxins collected from ergot honeydew changed the permeability of seeds of pearl millet. The toxin-treated seeds showed a loss of ions detectable at 30 min and reaching maximum level at 2 h after the treatment. Leaf tissues did not show any loss of ions. This leads the authors to suggest the possible involvement of permeability alterations in ergot pathogenesis.

40. Gour, S.C., Goyal, J. P., and Pathak, V.N. 1975. Forecasting ergot of bajra. Page 52 in *Proceedings of Symposium on Plant Disease Problems*. 18-20 September, 1975. Udaipur, India.

High relative humidity of 85-90% and cloudy atmosphere with 1-5 hr daily sunshine during flowering period were reported favorable for epidemic occurrence of ergot (Claviceps fusiformis) on pearl millet in Rajasthan.

41. Gupta, G.K., Subbarao, G.V., and Saxena, M.B.L. 1981. Meteorological factors in relation to occurrence of ergot disease of pearl millet in an arid climate. Pages 26-27 in *Abstracts of the Third International Symposium on Plant Pathology*, 14-18 December, 1981. Indian Agricultural Research Institute, New Delhi, India.

42. Gupta, G.K., Subbarao, G.V., and Saxena, M.B.L. 1983. Relationship between meteorological factors and the occurrence of ergot disease (Claviceps microcephala) of pearl millet. *Tropical Pest Management* 29:321-324.

Analysis of data from 11 sowing dates in the rainy seasons of 1976 and 1980 using curvilinear regression procedure showed a high positive association of relative humidity with the infection index of ergot on pearl millet in Rajasthan. Multiple correlations (R) increased with increases in rainfall, atmospheric temperature, and sunshine. Relative humidity contributed 35%, rainfall 5%, atmospheric temperature 45%, and sunshine 10% of the total variation in the ergot infection index, indicating from the curvilinear regression curves that the optimum values for occurrence and spread of the disease were 12 mm mean rainfall, 6 h per day mean sunshine, 75% mean relative humidity and 20°C mean atmospheric temperature from stigma emergence to flowering of the crop.

43. Jaubert, P. 1952. A propos du miellat du petit mil. (concerning honeydew of the small millet). *Bulletin of Recherche Agronomie Bambe* 6:67-69.

A form of ergot on Pennisetum typhoides was reported.

44. Jindia, L.N., and Gill, K.S. 1984. Sample size in estimation of genetic parameters for ergot and smut of Pennisetum typhoides (Burm. f.) S. and H. *Madras Agricultural Journal* 71(4):214-216.

For estimation of genetic parameters for resistance to ergot (Claviceps fusiformis) and smut (Tolyposporium penicillariae) in pearl millet populations, at least 76 male parents are necessary to construct the North Carolina Design-1.

45. Joun, B., and Delassus, M. 1971. Main diseases of millet and sorghum in Niger (French). *Agronomie Tropicale* 26(8):830-860.

Claviceps microcephala (Waller) Tul. is described as one of the principal pathogens of

Pennisetum typhoides. Distribution of ergot in Niger, symptoms of the disease, microscopic characteristics and biology of the pathogen, and important control measures were described.

46. Kale, K.O., and More, B.B. 1980. Important diseases of bajra and how to control them. Macro Agriculture Digest 4(12):3-4.

The control of ergot (Claviceps fusiformis), a serious disease of pearl millet in Maharashtra, has been suggested by separation of sclerotia from planting seed with brine water treatment followed by seed treatment with 1% organomercurial compound (Thiram or Captan) at the rate of 2 to 3 g/kg seed. Symptoms of ergot, downy mildew, smut, and rust are also described with suggestions on control of these diseases through host resistance, cultural practices, and use of chemicals.

47. Kannayian, J. 1974. Ergot and ergot alkaloids. Agriculture and Agro-Industries Journal. November issue:1-3.

The percentage of total alkaloids varied in sclerotia of different species of Claviceps. On pearl millet C. fusiformis and C. microcephala, mentioned as different species, contained 0.32 and 0.625% alkaloids, respectively. It is reported that the alkaloids of C. purpurea from rye were not the same as those of C. fusiformis and C. microcephala from pearl millet. Tryptophane was stated to be a precursor of the ergot alkaloids, methionine to be the origin of the N-methyl group of alkaloids, and lysergic acid to be a precursor to lysergic acid amide and methyl carbinolamide. Lysergic acid diethylamine (LSD) is a hallucinogenic agent, ergonovine is a stimulant for the sympathetic nervous system, and ergotamine is a depressant of the sympathetic nervous system. Agroclavine causes inhibition of mammary hypertrophy and blocks pregnancy.

48. Kannaiyan, J., and Arjunan, G. 1975. Ergot disease of bajra and its control. Farmers and Parliament 10:19-20.

Ergot (Claviceps microcephala) was stated as

the most important disease of pearl millet in Rajasthan, Gujarat, Maharashtra, Delhi, Karnataka, Andhra Pradesh, Tamil Nadu, Punjab, Haryana and Uttar Pradesh. Symptoms and favorable environmental conditions for disease development are described. The other hosts for the pathogen included maize, Phragmites communis, Aira caespitosa, Molinia caerulea, Nardus stricta, Poa annua, Pennisetum purpureum, P. squamulatum, P. massicum, P. rupelli, P. hoenackeri, P. alepecurus, Echinochloa crusgalli, Cenchrus ciliaris, and C. setigerus. Various control measures suggested include spray with protective chemicals like Benlate, Duter, Dithane M-45, and Pytolan during flowering, uprooting grass hosts growing near the crop, brine water treatment to separate sclerotia from grain, crop rotation to reduce soil inoculum, repeated deep plowing, rouging of infected plants, using insecticides, early sowing of crop, and growing tolerant varieties.

49. Kannaiyan, J., Vidhyasekaran, P., Govindaswamy, C.V., and Kandaswamy, T.K. 1972. Screening of pearl millet varieties for the ergot disease resistance. Madras Agricultural Journal 59:561-562.

Twenty varieties of pearl millet evaluated for resistance to Claviceps microcephala by spraying the earheads with conidial suspension showed more than 20% mean ergot severity, and none was found resistant. Pennisetum purpureum was found resistant to ergot but F₁ plants of P. typhoides x P. purpureum was highly susceptible.

50. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1971. Mammalian toxicity of ergot of bajra. Current Science 20: 557-558.

Guinea pigs fed on sclerotia of Claviceps microcephala mixed with the pearl millet grains showed a decrease in body weight and change in color of the liver and lungs. Ergometrine, ergotamine, and ergokryptine were detected in extracts of sclerotia by paper chromatography. The amount of alkaloids in honeydew increased with time after inoculation, and the maximum alkaloid content was found in mature sclerotia.

51. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1973. Amino acid contents of bajra in relation to ergot disease resistance. Indian Phytopathology 26:358-359.

Panicles of a pearl millet hybrid, HB 1, contained significantly more asparagine, aspartic acid, and proline up to 8 days after emergence, when these were susceptible, and higher amount of tryptophane, in 12 and 20 day-old spikelets, when these were not susceptible to ergot. Glutamic acid, threonine, methionine, and leucine showed no relationship with ergot resistance/susceptibility. In vitro asparagine and proline increased the growth of C. microcephala; aspartic acid did not support growth, and threonine and tryptophane inhibited growth.

52. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1973. Effect of fertilizer application on the incidence of ergot disease of bajra (Pennisetum typhoides B. & S.) Indian Phytopathology 26:355-357.

Under greenhouse conditions, studies on the effect of different combinations of nitrogen (ammonium sulfate), phosphorus (superphosphate), and potassium (potassium sulfate) revealed severe incidence of Claviceps microcephala on artificially inoculated pearl millet plants supplied with N, and without P and K. A heavy application of K without P counteracted the adverse effect of heavy N. Individually, the application of N and P increased but K significantly reduced the ergot incidence. Application of N and K decreased the total ergot alkaloids, where as P caused an appreciable increase.

53. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1974. Possible role of organic acids in the ergot disease of bajra (Pennisetum typhoides L.). Current Science 43(16):521-522.

Biochemical analysis of bajra spikelets of different ages revealed the presence of seven organic acids. In 12 to 20-day-old spikelets, when the concentrations of the organic acids reached the maximum, the spikelets showed resistance to

C. avicena microcephala. Five organic acids (tartaric, succinic, malic, citric, and oxalic acids) did not support growth of the pathogen in vitro on Kirchoff's medium. The authors concluded that the accumulation of excess organic acids can be inhibitory to the development of the ergot pathogen. Organic acid content of bajra earheads analyzed at different stages of disease development revealed the presence of succinic acid only in diseased earheads.

54. Kannaiyan, J., Vidhyasakaran, P., and Kandaswamy, T.K. 1974. Toxin production by Claviceps microcephala. Indian Journal of Experimental Botany 12 (5):473.

The culture filtrate of C. microcephala inhibited plumule and radicle elongation in bajra seedlings by 61.3 and 70%, respectively, compared to the control (uninoculated medium). Four sugars, maltose, lactose, fructose, and sucrose, were equally effective in inducing the production of phytotoxins by the ergot pathogen, as were eight amino acids, aspartic tryptophane, leucine, methionine, threonine, proline, glutamic, and asparagine.

55. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1975. Production of pectolytic and cellulolytic enzymes by bajra ergot fungus. Indian Phytopathology 28:123-125.

The possible role of pectolytic and cellulolytic enzymes in pathogenesis of Claviceps microcephala was suggested. The fungus produced pectic methyl esterase (PME), endopolygalacturonase (endo PG) and exopolygalacturonase (PGTE) in vitro. Addition of pectin to the culture medium induced enzyme production. In infected tissues, the enzymes protopectinase (PP), PME, endo PG, pectin trans-eliminase (PTE), and PGTE were more active in the initial stage of infection than at later stages. These enzymes were absent in healthy tissues. The cellulolytic enzymes, C1 and Cx, were detected in vitro and only C1 in the infected tissue. Further studies on the specific role of these enzymes in disease development and disease control, by selection of some inhibitors against

them, are suggested.

56. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1975. Proteolytic enzyme activity of Claviceps microcephala causing ergot disease of bajra. Indian Phytopathology 28:111.

An assessment was made of proteolytic enzymes in culture filtrate of Claviceps microcephala by substituting asparagine with casien, egg albumin, peptone, or gelatin as N sources in Kirchoff's basal medium. Maximum enzyme activity occurred when either casien or egg albumin was used. Prolonged incubation also increased enzyme production in peptone and gelatin media. Possible reduction in protein N in ergot infected bajra earheads was due to proteolytic enzymes of the pathogen.

57. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1977. The role of phenolics in relation to bajra ergot disease. Indian Phytopathology 30:549-50.

The quantitative analysis of healthy bajra earheads at different stages of development revealed that the total phenolics increased gradually up to 12 days after panicle emergence and then decreased slightly. The phenol content in ergot-infected panicles increased in the later stages of ergot (Claviceps microcephala) development. Presence of phenolics in panicles might impart resistance to ergot.

58. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1977. Studies on increased respiration of bajra earheads infected with Claviceps microcephala. Indian Phytopathology 30(3):420-421.

With the help of a Warburg constant volume type respirometer it was found that there was a constant increase in respiration in the ergot-infected earheads of pearl millet. Increased cytochrome oxidase activity was found in the infected compared with healthy earheads.

59. Kannaiyan, J., Vidhyasekaran, P., and Kandaswamy, T.K. 1977. The starch content of pearl millet earheads infected with Claviceps microcephala.

Estimation by colorimeter method revealed that C. fusiformis infection adversely affected starch synthesis in pearl millet earheads. There was more than 88% decrease in starch synthesis in ergot-infected earheads over the healthy at the honeydew and sclerotial stages. It was probably due to inhibition of the phosphorylase enzyme by the pathogen.

60. Karangale, L.T. 1975. Host range, varietal resistance and chemical control of ergot (Claviceps microcephala (Waller.) Tull. of hakra. Post Graduated Institute Research Journal, Akola, India 2:23-24.
61. Kazi, S.K., and Venkataraman, S. 1982. Influence of weather conditions on the incidence of ergot of hakra in Gujarat: a preliminary study. Mausam 33(2):255-258.

Heavy precipitation with afternoon relative humidity about 80%, followed by clear weather, favors ergot (Claviceps fusiformis) infection in pearl millet.

62. King, S.B. 1970. Millet disease in Nigeria. Samaru Agricultural Newsletter 12:88-89.

The common occurrence of ergot (Claviceps microcephala) on pearl millet was reported in Nigeria. Nonavailability of resistant sources indicated the potential of this disease to reduce the grain yield. Germination of soilborne sclerotia to produce airborne spores, infection through the flower, and role of insects in the secondary spread of ergot were mentioned. Disease development by spraying aqueous conidial suspension, limitation in rating scale, and exploitation of host plant resistance for the control of the disease were highlighted. The other diseases discussed include downy mildew (Sc. erospora graminicola), smut (To. vossorium penicil ariae), rust (Puccinia penniseti), and leaf spots caused by Gloeocercospora, Pyricularia, and Cercospora.

63. King, S.B. 1970. Sorghum and millet pathology. African Soils 25:473-476.

Ergot (Claviceps fusiformis) was not a problem of economic importance in Nigeria, with less prevalence in low rainfall areas. The available genotypes of pearl millet were susceptible to ergot and to smut (Tolyposporium penicillariae). Other major disease problems of sorghum and pearl millet in Zaria (Nigeria), along with the sources of resistance, were discussed.

64. Krishnamachari, K.S.V.R., and Bhat, R.V. 1976. Poisoning of ergoty bajra (pearl millet) in man. Indian Journal of Medical Research 64(11):1624-1628.

In 20 villages surveyed in Jaipur and Sikar districts of Rajasthan, 78 persons belonging to 14 households suffered from poisoning due to consumption of ergoty (Claviceps microcephala) pearl millet grains. The characteristic symptoms were nausea, repeated vomiting, and giddiness, followed by drowsiness and prolonged sleepiness within 1 to 2 h after ingestion of sclerotia. The toxicity due to pearl millet ergot was distinguishable from European ergotism (rye ergot) based on differences in chemical nature of alkaloids, biological effects in animals and clinical symptoms in man. Pearl millet grain contaminated with sclerotia that contain less than 675 ug alkaloids per 100 g of grain may be considered a non-toxic quantum. Assuming that an adult weighing 50 kg consumes about 200 g pearl millet in a single meal the safe limit of the clavine group of alkaloids in these sclerotia was worked out to be around 28 ug/kg body weight.

65. Kulkarni, U.K. 1962. A simple method of germinating sclerotia of Claviceps microcephala (Wall.) Tul. Current Science 31:390-391.

The method involved keeping mature sclerotia of C. microcephala collected from pearl millet in soil under shade for 25 to 30 days, washing with dilute solution of potassium permanganate followed by water, and then placing them in a horizontal position partially buried in an upper layer of red

soil. The lower layer of sand was kept moist by keeping the base of the pots in water continuously. It took 10-15 days for sclerotia to rupture and another week for stroma to emerge.

66. Kulkarni, U.K. 1963. Initiation of the dikaryon in C. avicenis microcephala (Waller.) Tul. Mycopathology et Mycopathologia Applicata 21(1): 19-22.

Antheridia of C. microcephala were functional, and production of single nuclear fusion of the Clausen type was observed. This nuclear behavior was similar to that found in C. purpurea.

67. Kulkarni, U.K. 1971. Studies of the bajra ergot fungus. Page 74 in Abstracts of the Second International Symposium on Plant Pathology, 27 January to 3 February 1971, Indian Agricultural Research Institute, New Delhi, India.

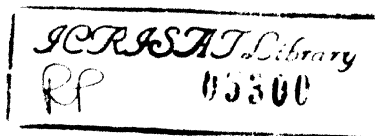
Ergot (Claviceps fusiformis) was reported on pearl millet hybrid HB 1. During the kharif 1967 the incidence varied from 5 to 100% in different states of India.

68. Kulkarni, C.S., and Moniz, L. 1974. Occurrence of hyperparasite Cerebella on ergot of bajra (Pennisetum typhoides (Burm F.) Stapf and C.E. Hubb.). Current Science 43:803.

Cerebella andropogonis Ces., reported for the first time from Gujarat (India) as a hyperparasite on Claviceps microcephala in the infected spikelets of pearl millet, inhibited formation of the ergot sclerotia. The sporodochia of C. andropogonis were convoluted, compact, and dark; conidiophores were often branched, pale brown, smooth, and 3-6 μ m long. Conidia were terminal, multicellular, globular, muriform with cross septa, smooth-walled with basal cells brown to dark brown, 7.4-25.6 x 7.4-22 μ m (av. 16.6 x 13.4 μ m). The fungus was common in Kaira district of Gujarat.

69. Kumar, A. 1977. Check ergot in bajra. Intensive Agriculture 15(5):16.

To control ergot (Claviceps fusiformis) in pearl millet several measures were suggested,



including spraying with Ziram (500-600 g/ha) 2 or 3 times starting just before ear emergence, early sowing, seed sanitation by removing sclerotia from seed with brine water treatment, and deep plowing.

70. Kumar, A. 1979. Production of phytotoxins in ergot of pearl millet. Seed Science and Technology 8:347-350.

The sclerotial filtrate of Claviceps fusiformis inhibited root and coleoptile elongation of pearl millet seedlings. Elongation was inhibited more in roots than in plumules. Increased concentrations of the filtrate caused a gradual increase in inhibition of root length. Seed germination was reduced from 70% in the control to 30% at 12.5% concentration of the filtrate. This was probably due to allelochemic interactions between sclerotial toxins and germinating seeds.

71. Kumar, A., and Arya, H.C. 1978. Estimation and identification of alkaloids produced by Claviceps fusiformis Loveless on some varieties of pearl millet. Current Science 47(17): 633-35.

Following thin layer chromatography technique and taking elymoclavine as standard samples the total alkaloid content was estimated between 0.182 and 0.362% (average 0.263%) in honeydew and between 0.160 and 0.548% in sclerotia of C. fusiformis collected from 20 varieties of pearl millet. The alkaloid content was maximum (0.548%) in variety B-463 and minimum (0.160%) in RC-216. Six alkaloids were detected: setoclavine, agroclavine, penniclavine, elymoclavine, chanoclavine, and one unidentified.

72. Kumar, A., and Arya, H.C. 1978. In vitro production of honeydew in Claviceps fusiformis Lov. Current Science 47: 781-782.

Claviceps fusiformis, isolated from pearl millet ergot sclerotia, produced honeydew-like secretion in pink to dark brown colonies after 20 days of incubation on calcium nitrate agar medium. Conidia from these cultures induced ergot symptoms upon artificially inoculating pearl millet hybrid HB 4. The authors considered that production of honeydew

was not a consequence of parasitism only.

73. Kumar, A., and Arya, H. C. 1981. Nature of resistance and susceptibility in vitro in ergot of pearl millet caused by Claviceps fusiformis Lov. *Phytopathologia Mediterranea* 20:43-45.

The calli of five varieties of Pennisetum americanum raised in modified Murashige and Skoog's medium were transferred to 0.6% water agar and inoculated with C. fusiformis. In highly susceptible genotypes (HB 4 (new), HB 5, PIB 228) almost all conidia that came in contact with callus tissue germinated and penetrated the cells to form inter- and intracellular mycelium, whereas in moderately and highly resistant genotypes (J 88, B 389) only a small number of conidia germinated.

74. Kumar, A., and Arya, H.C. 1981. Biology and control of ergot on pearl millet. Page 14 in Abstracts of the Third International Symposium on Plant Pathology, 14-18 December 1981. Indian Agricultural Research Institute, New Delhi, India.

Nearly 100% infection occurred on pearl millet earheads sprayed with Claviceps fusiformis conidia at a concentration of "10⁷/sq mm". Inoculation 5 days prior and 5 days after anthesis resulted in high levels of infection. Susceptibility declined after 12 days and was lost after 18 days of anthesis initiation. Hinosan and Cuman-L (500 ppm) partly controlled the growth of the fungus in vitro.

75. Kumar, A., and Arya, H.C. 1983. On taxonomy of the fungus causing ergot disease of pearl millet in India. Pages 278-279 in *Advancing Frontiers in Plant Sciences*, 26-30 November 1983, Jodhpur, Rajasthan, India (Arya, H.C., Sankhla, N., Tewari, M.N., Shekhawat, N.S., and Purohit, S.P. eds.) Scientific Publishers, Jodhpur, India.

Morphological studies and the presence of the clavine group of alkaloids in sclerotia of pearl millet in India confirmed the identity of the pathogen as Claviceps fusiformis.

76. Kumararaj, S., and Bhide, V.P. 1961. Varietal resistance in bajri (Pennisetum typhoides (Burm.) S. and H.) to ergot (Claviceps microcephala (Wallr.) Tul.). Current Science 31:76.

All the 144 cultures of pearl millet screened for resistance to C. microcephala under artificial inoculation conditions showed susceptibility. However 17 cultures -- PT 838-6, P 350, PT 833-4, Co 2, Australian 21-65, 168-A, RSK, S 530, Co 3, Bajra 2-L, Savargaon M-S, A-G-B, A K 297 x Amreli AGB, 179-G-4, Tharparkar, A 1-3, and Palanpur -- showed less than 5 sclerotia per ear compared to 10% or more in the others.

77. Loveless, A. R. 1967. Claviceps fusiformis sp. nov., the causal agent of an agalactica of sows. Transactions of the British Mycological Society 50: 15-18.

A new species of Claviceps having fusiform conidia on Pennisetum typhoides was distinguished from C. purpurea and was named as C. fusiformis. A morphological account was given from the samples collected from Rhodesia (now Zimbabwe) where it was known to cause a serious disease, i.e., agalactica of sows (pigs). The description of the type species is given below:

Claviceps fusiformis sp. nov.

Sclerotia obpyriform, 3.5 - 6 mm long, 2-3 mm wide, usually with irregular longitudinal furrows; tapering to a point and sometimes curved, blackish brown, the basal portion of the sclerotium globose or ovoid, greyish brown. Stromata 1-3, usually 1. Stipes mostly 4-5 mm long, sometimes up to 10 mm long, 0.3-0.5 mm wide, pale purple becoming creamy with age. Capitula, globose, 1-1.5 mm diam., greyish purple, slightly papillate at maturity. Perithecia ovate to pyriform, 130-175 um long and 60 x 95 um wide (mean of 20 median sections 155.6 x 77.6 um). Asci cylindrical, 95 - 135 um long by 3-5 um wide (mean of 20 asci 115 x 4 um). Ascospores slightly shorter than asci. Conidia hyaline, fusiform or broadly falcate, 9.5 - 22.5 um long by 3 - 5 um wide (mean of 100 spores 15.8 x 3.6 um). Collected 64.4 km south of Umtali, Rhodesia, 10

78. Hantle, P.G. 1968. Inhibition of lactation in mice following feeding with ergot sclerotia (*Claviceps fusiformis* Loveless) from the bulrush millet (*Pennisetum typhoides* Stapf and Hubbard) and an alkaloid component. Proceedings of the Royal Society B. 170:423-434.

The sclerotia of *C. fusiformis* when fed at 2 and 3% to female mice during the later part of pregnancy, caused failure to raise their litters. Feeding the mother alkaloid extracted from sclerotia induced pup mortality in a similar manner. The active principle, agroclavine, fed at 5 to 7 "mg%", inhibited the normal mammary hypertrophy. The discontinuance of alkaloid diet on the day before parturition resulted in rapid recovery from the effect of agroclavine. Agroclavine also suppressed well-established lactation. Exchanging litters of treated and control mice at birth showed survival and normal development of 77% of the pups of mothers which had received the agroclavine diet whereas all the control pups which were transferred to the mothers receiving agroclavine died.

79. Hantle, P.G. 1969. Interruption of early pregnancy in mice by oral administration of agroclavine and sclerotia of *Claviceps fusiformis* Loveless. Journal of Reproduction and Fertility 18:81-88.

The oral administration of agroclavine (slightly less than 300 ug/day) caused termination of pregnancy sometime during the first 6 days in female mice. A dose of 200 ug agroclavine given daily in food on the 3rd and 4th day of pregnancy (approximately 165 ug actual dose ingested) affected pregnancy in some replicates but daily intake of 250 ug agroclavine terminated the pregnancy without any sign of toxicity. Eight weeks of agroclavine treatment did not reduce subsequent fertility in mice, showing no significant effect on the process of conception. The alkaloid treatment during the first 2 days of pregnancy or after implantation as well as injection of 250 ug agroclavine did not interrupt pregnancy whereas a diet containing ergotamine, ergosine, and lysergic

acid hydroxyethylamide interrupted it.

80. Mathur, J.M.S., and Gupta, J.P. 1981. Production of pectolytic enzymes by Claviceps microcephala and their role in host cell wall degradation. Pages 25-26 in Abstracts of the Third International Symposium on Plant Pathology, 14-18 December 1981. Indian Agricultural Research Institute, New Delhi, India.

In ergot susceptible pearl millet varieties, it was shown that significant activities of the enzymes polygalactouronase (Exo-PG and Endo-PG), pectic methylgalactouronases (Exo-PMG and Endo-PMG), pectic acid transeliminase (PATE), and pectic transeliminase (PTE), at early stages of infection, indicated the production of diffusible extracellular pectolytic enzymes by Claviceps microcephala as a prerequisite for infection.

81. Modi, B.S., Bansal, V., and Bhora, C.P. 1979. Trials on inclined draper belt for ergot-bajra separation. Journal of Agricultural Engineering 16(3):95-102.

A draper belt separator was developed (details described) and tested for its efficiency to separate ergot sclerotia (Claviceps fusiformis) from infested pearl millet grains. Overall performance of the separator was satisfactory at 22° inclination of the belt. Proper adjustments were found essential for efficient functioning of the separator.

82. Murty, B.R. 1974. Mutation breeding for resistance to downy mildew and ergot in Pennisetum and to Aascochyta in chickpea. Pages 89-100 in Induced Mutation for Disease Resistance in Crop Plants, 4-8 June 1973. Novi Sas Yugoslavia Vienna, Austria. International Atomic Energy Association.

The creation of an artificial epiphytotic condition for development of ergot on pearl millet was successful. However, mutational rectification of pearl millet seed material with 60 Co was not successful in inducing resistance to Claviceps microcephala.

83. ~~Mand, Hira~~, J.M. 1983. Screening of ergot in pearl millet of five inbreds and one hybrid for a breeding programme. Zimbabwe Agricultural Journal 80:239-240.

In India, screening of five pearl millet inbred lines and a hybrid for resistance to Claviceps fusiformis following standard inoculation technique revealed mean ergot severity ranged between 0 and 10% in the inbred lines, and 95% in BJ-104 (susceptible check). The ergot severity was 0% in ICMPE 13-6 <1% in ICMPE 134-41, ICMPE 140-7-8, and ICMPE 134-6-31, and 10.9% on ICMPE 140-7-12. Ergot infection also reduced the total grain weight.

84. Mand, Hira; Kumar, Kamlesh; and Singh, Siyaram. 1981. Plant protection in dry land agriculture. Pesticides 15:3-7.

Surveys in some villages of Haryana (India) revealed ergot (Claviceps fusiformis) as the most widely spread disease of pearl millet causing considerable loss. Ergot was reported important by 100% of the farmers, downy mildew by 56%, and smut by 35%. There was unawareness among the farmers of the recommended integrated control measures. It was suggested that community adoption of practices, like collecting and destroying the diseased earheads and using disease-free seeds, can help control the disease.

85. Natarajan, U.S., Guruswamy, Raja, Selvaraj, S., and Parambaramani, C. 1974. Grain loss due to ergot disease in bajra hybrids. Indian Phytopathology 27:254-256.

Ergot (C. fusiformis) incidence and grain loss in 17 hybrids and 1 composite variety of pearl millet were recorded at Coimbatore, India. With an average ergot incidence of 62.4% a grain loss of 58.4% was estimated. All the cultivars were susceptible with no significant differences for either ergot incidence or grain loss. There was a strong, positive correlation between ergot incidence and grain loss.

86. Nene, Y.L., and Singh, S.D. 1976. Downy mildew and ergot of pearl millet. Pest Articles and News

The seriousness of ergot and downy mildew diseases on pearl millet was highlighted, since these diseases were the main reason for low yields of hybrids with high yield potentials. A review of the available literature on these diseases was presented to ascertain an up-to-date status of knowledge. Ergot (Claviceps fusiformis), a major disease in India and widely distributed in Africa, was reported to cause grain loss and, on consumption, poisoning in humans and cattle. The following priority areas of research were suggested: taxonomy of the pathogen, survival from one season to another, infectivity of ascospores, the role of conidia as infective propagules, host range, establishment of a standard procedure for evaluating resistance, development of a disease rating scale, identification of resistant sources, and biological control. Results of research on chemical control, removal of sclerotia by floatation in salt water, and the role of meteorological factors during anthesis in development of the disease were discussed. The need for research on downy mildew (Sclerospora graminicola) including seed-borne transmission, secondary spread through sporangia, physiological specialization, environmental races, and identification of stable resistance was also suggested.

87. Nicholas, I. 1975. Removal of ergot from grain or seed lots of bajra by gravity separators. Seed and Farms 1(9):4.

The specific gravity of ergot (Claviceps fusiformis) sclerotia was found to be lower than pearl millet grain. An effective mechanical method for removal of sclerotia from grain or seed lots by gravity separators was described. By this procedure sclerotia could be separated from more than 80% of the sample.

88. Pahil, V.S., and Thakur, D.P. 1985. Relative efficacy of different fungitoxicants against Claviceps fusiformis (Loveless) in vitro and in vivo. Haryana Agricultural University Journal of Research 15(3):279-283.

Out of 15 chemicals tested in vitro, Ridomil 25 WP, Benlate, Apron SD-35, Curzate, Delan, Cuman-L, and Difolatan, each at 500 ppm concentration, completely inhibited spore germination of all four isolates of Claviceps fusiformis collected from pearl millet at Hisar, Delhi, Patancheru (ICRISAT), and Jamnagar, and one isolated from Panicum antidotale at Hisar. On a hybrid, BJ 104, Ridomil 25 WP (200 ppm) most effectively reduced ergot incidence in the field when applied as protective sprays. Difolatan followed by Apron SD-35 and Cuman-L also checked the disease satisfactorily when applied as protective treatments.

89. Pathak, V.N., and Sharma, R.K. 1976. Reaction of bajra inbreds and hybrids to ergot at Jobner. Indian Journal of Mycology and Plant Pathology 5:199.

During the 1973 rainy season, all the 177 well established inbred lines and 72 F₁ hybrids of pearl millet showed susceptibility to ergot (Claviceps fusiformis) under natural conditions at Jobner, Rajasthan, India.

90. Pathak, V.N., Yadav, K.R., Langkild, M.E., and Mathur, S.B. 1984. Removal of ergot sclerotia from seeds of pearl millet. Seed Research 12(2):70-81.

Investigations comparing separation of ergot (Claviceps fusiformis) sclerotia from seeds of Pennisetum americanum were done for a Screen Air Separator (SAS), Specific Gravity Table (SGT), and SGT combined with a Brand Grader. Best removal was obtained by using the SGT. The SGT method was described as convenient, rapid, simple, economical, and more appropriate. Higher sclerotial contents in seed samples adversely affected the efficiency of the procedure. Although efficient sclerotial removal was also achieved by dipping the seed samples in 20% salt solution, and carefully and repeatedly skimming-off the floating and submerged sclerotia with the help of a tea strainer, this method proved slow and expensive, and it reduced seed germination and seedling growth.

91. Patil, B.P. 1980. Ergot of pearl millet. Pestology

A review of the findings on ergot of pearl millet caused by *Claviceps microcarpha* is presented. These include historical background, geographical distribution, extent of losses, ergot poisoning due to water-soluble alkaloids, survival and spread of the disease from one region to another by means of sclerotia, secondary spread by means of honeydew conidia, mode of infection through stigmas, meteorological factors congenial for disease development (high humidity, cloudy weather and temperature between 18-30 C), and collateral hosts. The control were also measures reviewed including agronomic practices, use of fungicides, growing resistant varieties, biological means, and use of sclerotia-free seeds. An integrated system of control was considered appropriate. The future priority areas for research included: development of an ergot forecasting system, devising suitable cultural practices, evaluation of systemic fungicides and development of suitable spray schedules, estimation of losses, studies on pathogenic variability, identification of resistance in wild grasses and their possible utilization in resistance breeding, investigation on mutation breeding for resistance, and development of biological and integrated control systems.

92. Pawar, C.P., Harrer, P.N., Gaikwad, A.C., Gaikwad, D.G., and Harinaryana, G. 1983. Genetics of disease resistance in pearl millet. Page 729 in Abstracts (pt2) of the 15th International Congress of Genetics, 12-21 December, 1983, New Delhi, India. Oxford and IBH Publishing Company, New Delhi.

The inheritance of ergot, downy mildew, and rust was studied using 9 x 9 diallel and generation mean analysis. General combining ability was highly significant for ergot only. Additive gene action was prevalent for disease resistance. However, generation mean analysis revealed non-allelic interactions of a duplicate, dominant, epistatic, or recessive nature.

93. Prakash, H.S., and Shetty, H.S. 1981. Influence of storage temperature on sclerotial germination of

C. avicens fusiformis. Current Science 50:870-871.

Germination was 3.5% in freshly collected sclerotia of C. fusiformis. A marked increase was observed in germination of sclerotia stored for 8 weeks at 23 to 37° C without chilling treatment. Storage temperature of 10 to 15° C and chilling treatment did not increase sclerotial germinability. Maximum germination (81.33%) was recorded in sclerotia stored at 37° C. The number of days required for germination were also reduced from 60 for freshly collected sclerotia to 16-38 for sclerotia incubated at 23 ± 1° C with 12 h photoperiod. The number of clavae produced per sclerotium ranged from 1 to 8.

94. Prakash, H.S., Shetty, H.S., and Safeeulla, K.M. 1980. Histology of carpel infection by Claviceps fusiformis in pearl millet. Proceedings of the Indian National Science Academy B 46:708-712.

The germ tubes of conidia of C. fusiformis penetrated directly through stigma, style and ovary wall of the pearl millet flower. The infection hyphae traversed through intercellular spaces and then proceeded down the stigma, style and ovary. The style withered after infection and three days after inoculation the hyphae reached the base of the ovary wall and invaded the interior cells of the ovary. On the 5th day after inoculation the cells of the ovary became pulpy and dead. The size of ovaries increased and honeydew was produced from the 6th to the 10th day after inoculation. The fungal hyphae replaced the ovary, but did not enter receptacle cells just below the ovary. Fifteen days after inoculation, the white, smooth layer around ovaries turned brownish, and ultimately became sclerotia.

95. Prakash, H.S., Shetty, H.S., and Safeeulla, K.M. 1983. Germination and nuclear behavior of sexual and asexual propagules of Claviceps fusiformis. Transactions of the British Mycological Society 81:65-69.

Filiform, aseptate, uninucleate ascospores of C. fusiformis, measuring 119 x 2 um, germinated and produced primary and secondary conidia. The

nucleus from the ascospore migrated into the primary conidium without undergoing division. Similarly, nuclear migration from primary to secondary conidia took place without division. In some ascospores the nucleus divided into two, resulting in the formation of two primary conidia. The asexually produced conidia in honeydew germinated within 24 h, producing secondary and tertiary conidia within 48 h. A few tertiary conidia also produced quaternary conidia. The last formed conidia were the only infective conidia, and the empty (non nucleate) primary or secondary conidia did not cause infection. The process of successive germination of conidia was considered favorable for prolonging the infectivity of conidia.

96. Prakash, H.S., Shetty, H.S., Subramanya, S., and Safeulla, K.M. 1981. Standardisation of sclerotial germination technique and infectivity of ascospores of *C. avicena fusiformis* Lov. Indian Journal of Agricultural Sciences 51(12):900-904.

Sclerotia of *C. fusiformis* germinated more quickly (within 20 days) and in higher percentage (18%) when placed in moist sand in petri plates than those placed in pots with moistened soil covered by polythene sheets at $23 \pm 1^{\circ}$ C with 12 hr photoperiod. At 28° C sclerotial germination percentage was lower and germination time longer. Sclerotia placed on moist blotters at $23 \pm 1^{\circ}$ C failed to germinate. Sclerotial germination was not influenced by light. Germination of freshly collected sclerotia was very low (4%) and required at least 65 days whereas germination of 9-month old sclerotia was higher (18%) and required only 20 days under similar conditions of moisture and temperature.

Up to eight stroma per sclerotium were produced. Ascospores (103.2×2.6 μ m) germinated with or without producing conidia at the terminal ends of the germ tube. Conidia resembled secondary conidia in shape and size. The ascosporial inoculum infected 44.25% of the inoculated florets.

97. Rachie, K.O., and Majmudar, J.V. 1980. Pearl Millet. The Pennsylvania State University Press. Univeristy Park and London.

A detailed account of ergot (C. avicenis fusiformis) on pearl millet is given, including its widespread occurrence on high yielding hybrids in India and on local land races in Africa, historical background and work on meteorological factors favorable for disease development, losses, biology and morphology of the pathogen, infection, and development of symptoms. Collateral hosts were found to be generally species of Pennisetum. Limited research on identification of resistance revealed lack of resistance in the germplasm screened. Cultural practices, like removal of infected heads, use of sclerotia-free seed, crop rotation, and chemical control through sprays of Ziram or mixtures of copper oxychloride and Zineb are discussed. Biological control and ergot-induced toxicity are also reviewed.

98. Ramakrishnan, T.S. 1952. Observations of ergots of Pennisetum and other grasses. Proceedings of the Indian Academy of Sciences B. 36(3):97-101.

Six species of Pennisetum, P. typhoides, P. purpureum, P. rupelli, P. hohenackeri, P. alopecuroides, and P. polystachyon, and two species of Cenchrus, C. ciliaris and C. setigerus, were found susceptible to the ergot pathogen (apparently Claviceps microcephala) collected from a hybrid, P. typhoides x P. purpureum. After inoculation, conidia germinated on stigmas in 48 h, completely invaded the ovary in 96 h, and honeydew in 5 days. Sclerotia did not germinate even after stratification and exposure to low temperature. Conidia germinated readily for up to 7 months after collection and could infect earheads for another 6 months.

99. Ramakrishnan, T.S. 1971. Diseases of Millets. Indian Council of Agricultural Research, New Delhi.

Ergot of pearl millet, caused by C. avicenis microcephala, was considered a disease of lesser importance than other diseases of the crop, although the potential threat was recognised. Distribution, pathogen morphology, alkaloid content, disease epidemiology, collateral hosts, and control measures, primarily through cultural

practices, are described.

100. Ramaswamy, C. 1968. Meteorological factors associated with the ergot epidemic of bajra (Pennisetum typhoides) in India during the Kharif season, 1967- a preliminary study. Current Science 37(12):331-335.

The meteorological factors associated with a kharif season when ergot (Claviceps fusiformis) did not appear (1966) and one when it did appear in epidemic form (1967) on pearl millet in Delhi were described to show their relationship to ergot. In 1966, the monsoon began on 25 July but there was low relative humidity (RH), little cloud cover and longer duration of sunshine during most of the period covering the last 10 days of August and first 2 weeks of September. In contrast, in 1967 there were daily showers between 2 and 8 July, and morning RH was 85-95% from 1-10 September. The evening RH was 75 to 90% from 1-5 September and 60 to 70% from 6-10 September. The total cloud cover was 6 to 8 Octa i.e. 75 to 100% sky coverage, both in the morning and the evening from 1 - 8 September which was much more than the sky coverage in the corresponding period in 1966. The total daily sunshine was only 1 to 5 h, and between 1 and 6 September there were rain showers daily. The surface wind-speed did not play a role; however, the higher disease incidence during 1967 was attributed to better rainfall distribution during the vegetative growth and flowering periods. Cloudy weather, less sunshine, and high RH were probably responsible for the higher disease incidence in 1967. The need of detailed meteorological studies was emphasised.

101. Reddy, K.D., Govindaswamy, C.V., and Vidhyasekaran, P. 1969. Studies on ergot disease of cumbu (Pennisetum typhoides). Madras Agricultural Journal 56(5):367-377.

Conidia from honeydew of Claviceps microcephala were more virulent on cumbu (P. typhoides) than conidia from culture medium. High relative humidity favored ergot development. Entry of the pathogen into the ovary was largely through the wall. Experiments on disease transmission

through seed or soil yielded negative results. Pennisetum squamulatum and P. massaicum were susceptible to the pathogen. Prophylactic application of fungicides was superior to post-inoculation sprays in minimizing the disease.

102. Saxena, M.B.L., Gupta, G.K., and Subba Rao, G.V. 1978. Influence of environment and genotypes on the development of ergot in pearl millet. Indian Journal of Agricultural Sciences 48(8):495-497.

Ergot (Claviceps fusiformis) incidence varied from 79.1 (on NHB 3) to 98.6% (on PHB 12) on nine F₁ hybrids of pearl millet, including HB 3, during the 1976 rainy season at Jodhpur. There were significant differences for ergot incidence and severity among the hybrids. The infection index ranged from 22.2% for NHB 3 to 29.7% for 5054 A x K 2457. There were daily showers during the protogyny to early anthesis period which appeared to play a significant role in the onset and spread of the disease. Percentage infection was positively correlated with relative humidity and rainfall, but the correlation with relative humidity was stronger. Sunshine and atmospheric temperature had direct effect on infection percentage, and were negatively correlated with it.

103. Selvaraj, J.C. 1980. Ergot disease of pearl millet in Nigeria. FAO Plant Protection Bulletin 28(4):129-132.

Ergot (Claviceps microcephala) on pearl millet in four ecological zones i.e. Sahel Savanna, Sudan Savanna, Northern Guinea Savanna and Southern Guinea Savanna was assessed in Nigeria during 1975, 1976, 1977, and 1978, using a 0-5 scale. The disease was mild in the Sahel and Sudan savanna zones in 1975 and 1976 and moderate in other zones. In 1977 and 1978 the disease was most severe in the northern and southern Guinea savanna zones, when it caused an estimated 2 to 3% crop loss. 'Gero millet' which matured during the rainy season was attacked more severely than 'Maiwa' millet which flowered later. The study demonstrated the potential threat of ergot and other diseases, like downy mildew and smut, to

pearl millet in Nigeria.

104. Sharma, M.P., Singh, R.S., and Tripathi, R.K. 1983. Estimation of loss caused by ergot disease of pearl millet (Pennisetum americanum (L.) Leeke). Madras Agricultural Journal 70(10):687-689.

There was a negative correlation between number of sclerotia and number of grains on ergot-infected pearl millet earheads. Ergot intensity below 50% did not affect grain size, but a significant reduction in size was observed when disease intensity exceeded 50%.

105. Sharma, O.P., and Chauhan, R.K.S. 1982. Phenomenon of infection of ergot disease of pearl millet. Current Science 51(20):994-995.

Studies on the occurrence of ergot (Claviceps fusiformis) on pearl millet hybrid HB 3, in sclerotia-infested isolation plots during 1978 and 1980 at Gwalior, India, indicated that the conidia trapped on slides were those that were produced on the surface of sclerotia. The role of these conidia as source of primary inoculum and those from honeydew as secondary inoculum was emphasized.

106. Sharma, O.P., and Chauhan, R.K.S. 1984. Identity of pathogen causing ergot of pearl millet. Indian Phytopathology 37:539-540.

Morphological studies of the ergot samples collected from pearl millet from Saiya, Rohta, Sevla (Uttar Pradesh), Bhind, Morena, Gwalior (Madhya Pradesh), Dholpur, Bargaon (Rajasthan), and Coimbatore (Tamil Nadu), India, confirmed that the causal organism was Claviceps fusiformis Loveless.

107. Sharma, R.K., Verma, O.P., Mazmudar, V.L., and Agnihotri, V.P. 1981. Conidial germination in Claviceps fusiformis, the incitant of pearl millet ergot in relation to physical and chemical factors. Page 25 in Abstracts of the Third International Symposium on Plant Pathology, 14-18 December, 1981. Indian Agricultural Research

Institute, New Delhi, India.

In vitro germination of Claviceps fusiformis conidia occurred at 15 to 45° C (optimum 25° C), 5 to 100% RH (optimum 100%), and 5.0 to 9.0 pH (optimum 7.5). Cuman-L (200 ppm) caused maximum inhibition in germination followed by Cela W 524 (200 ppm). Biodoxy (200 ppm), Thromycin, Phenergan, Brufen and Maleic hydrazide (50 ppm each) also caused significant reduction in the germination.

108. Sharma, R.K., Verma, O.P., and Pathak, V.M. 1982. Management of pearl millet ergot with fungicides and insecticides. Indian Journal of Mycology and Plant Pathology 12(1):109 (Abstract).

Aureofungin (50 ppm), Cuman-L (1500 ppm) and Ridomil (500 ppm) were effective in reducing ergot severity in pearl millet. Among insecticides, Dimecron, Ekalux, Chlorodane, Malathion, Diazinon and Parathion were also effective.

109. Sharma, R.K., Verma, O.P., and Pathak, V.M. 1984. Some physical and nutritional factors for the growth and sporulation of Claviceps fusiformis Lov. Cryptogamic Mycologia 5:269-275.

A linear relationship between sugar concentration and the growth of C. fusiformis, in vitro was reported. The highest sporulation occurred at a sugar concentration of 100 g/L at 25° C and pH 7.5. Asparagine was a better source of nitrogen than nitrate, and sulfates of barium, ferrous, copper, and bismuth completely stopped growth.

110. Sharma, Y.P., Singh, R.S., Tripathi, R.K. 1983. Role of insects in secondary spread of ergot disease of pearl millet (Pennisetum americanum). Indian Phytopathology 36(1):131-133.

The role of insects in transmission of Claviceps fusiformis in pearl millet was confirmed. Transmission efficiency of moths, honey bees, house flies, red cotton bug, plant bug, and flower beetle were 63, 57, 52, 45, 47 and 22%, respectively, in field tests and it varied

from 30 to 87% in greenhouse tests. In the greenhouse, among the small insects, the maximum transmission was by black ants (70%) followed by Coccinella septempunctata (60%), Chiomanes sexmaculata (60%), beetles (50%), and lygaeids (40%).

111. Sharma, Y.P., Singh, R.S., and Tripathi, R.K. 1984. Management of pearl millet ergot by integrating cultural practices and chemical control. Indian Journal of Mycology and Plant Pathology 14:69-79.

Application of nitrogen, phosphorus, and potash at the rate of 100, 50 and 40 kg ha, respectively, gave higher yield and also kept the ergot (Claviceps fusiformis) at a low level in pearl millet. Of 15 fungicides tested as pre- and post-inoculation sprays the systemic fungicides Bavistin, Benlate, and Brestanol, each at 0.055 a.i., gave 50 to 60% control. Ziram, Duter, Dithane - 45, and Difoltan gave 37.6 to 43.4% control when sprayed 24 h before inoculation. Systemic fungicides were superior to non-systemic ones. Sevin and Thiodan insecticides at 0.05% concentration also checked the disease. Better control resulted by using a combination of Bavistin, Benlate, or Brestanol with Sevin or Thiodan; three sprays of any combination at weekly intervals were effective.

112. Shinde, P.A., and Bhide, V.P. 1958. Ergot of bajri (Pennisetum typhoides) in Bombay state. Current Science 27(12):499-500.

The causal organism of ergot of bajri was described as Claviceps microcephala; it grew well on Kirchoff's original and modified media, Sabourands' medium, and modified Czapek's medium. The conidia of the pathogen germinated readily at 25-27° C with thermal death point around 55° C.

113. Shone, D.K., Phillip, J.R., and Christie, G.J. 1959. Agalactica of sows caused by feeding the ergot of bulrush millet, Pennisetum typhoides. Veterinary Research 71(7):129-132.

Feeding munga (Pennisetum typhoides) grain

contaminated with ergot sclerotia was described as a cause of agalactica of sows in Rhodesia (Zimbabwe).

114. Siddiqui, M.R., and Khan, I.D. 1972. Correlation between infection potential and mode of conidial germination of pearl millet ergot fungus. Proceedings of the Indian Science Congress 59(3):564-565. (Abstract)

Conidia of Claviceps fusiformis germinated in various ways and their percentage germination varied depending upon the source and time of production. Under favorable conditions, disease level in susceptible genotypes was directly proportional to the "infection potential" of the pathogen which was related to germination of conidia and prevalent relative humidity.

115. Siddiqui, M.R., and Khan, I.D. 1973. Renaming Claviceps microcephala ergot fungus on Pennisetum typhoides in India as Claviceps fusiformis. Transactions of Mycological Society of Japan 14(2):195-198.

Morphological studies of the ergot pathogen from material collected from naturally and artificially inoculated pearl millet varieties revealed conidia as hyaline and fusiform or broadly falcate, measuring 16.5 x 34.8 um. It was proposed that the causal organism of ergot on Pennisetum typhoides in India should be known as C. fusiformis Lov. instead of C. microcephala (Wallr.) Tul.

116. Siddiqui, M.R., and Khan, I.D. 1973. Dynamics of inoculum and environment in relation to ergot incidence on Pennisetum typhoides (Burm.) Stapf and Hubbard. Transactions of Mycological Society of Japan 14:280-288.

Data collected during 1968, 1969, and 1970 revealed that ergot (Claviceps fusiformis) disease of pearl millet was season bound at the Regional Research Station, Coimbatore. However, it was possible to maintain it throughout the year by

with fresh inoculum of the pathogen and providing relative humidity (RH) of more than 95% at the time of inoculation. In the field, a daily mean temperature between 18 and 30° C, mean RH above 90% and light showers during flowering were favorable for infection by the ergot pathogen. There was a gradual decline in germination percentage of conidia collected from previously inoculated plants which was probably responsible, along with the growth stage of the host, for a reduction in percentage of infection after December. Significance of the results in evaluation of varieties during various times of the year was discussed.

117. Singh, G., Bhatnagar, G.C., Choudhary, G.G., and Jat, R.G. 1978. Studies on ergot disease of bajra in Rajasthan. Indian Journal of Mycology and Plant Pathology 8:495. (Abstract)

The occurrence of ergot (Claviceps fusiformis) on pearl millet, its consequences in crop production, its spread through sclerotium-contaminated seeds, the non-availability of host plant resistance, effectiveness of Sulfex and Dithane M-45 sprays and late sowing for its control were mentioned.

118. Singh, G., Vyas, K.L., and Bhat, B.M. 1983. Occurrence of pearl millet ergot on Cenchrus ciliaris Pers. in Rajasthan. Indian Journal of Agricultural Sciences 53(6): 481-483.

The fungus from ergoty panicles of Cenchrus ciliaris at Durgapura, Rajasthan, was identified as Claviceps fusiformis. Pathogenicity tests revealed its ability to infect pearl millet (P. typhoides) as well as C. ciliaris. C. ciliaris possibly acted as a collateral host for increasing the period of multiplication of the inoculum under favorable conditions before flowering of pearl millet. This probably resulted in sudden, large-scale infection in late sown pearl millet varieties in Rajasthan.

119. Singh, H.N., and Husain, Akhtar. 1977. Saprophytic production of ergot alkaloids by bajra ergot (Claviceps fusiformis Loveless. Indian Journal of

C. avicena fusiformis isolated from Pennisetum typhoides provided only clavine type alkaloids like chanoclavine, elymoclavine, festuclavine and agroclavine in stationary liquid culture (on NL 406 medium), while it produced chanoclavine, elymoclavine, and agroclavine in submerged shake culture filtrate. Both ergotamine and ergometrine, which are therapeutically important alkaloids, were not produced in high quantity.

120. Singh, Rameshwar, and Singh, S.N. 1969. A note on effect of different dates of sowing HB 1 on grain yield and incidence of ergot (Claviceps microcephala (Wallr.) Tul.) Madras Agricultural Journal 56:140

A pearl millet hybrid, HB 1, sown on 15 and 30 July at Dholi, Bihar was not only free from ergot (Claviceps microcephala) but gave higher yields than that sown on 15 and 30 August which developed heavy ergot.

121. Singh, S.B., Bais, B.S., and Singh, D.R. 1972. Effect of different carbon and nitrogen sources on the growth and sporulation of Claviceps microcephala (Wallr.) Tul. Mycopathologia et Mycologia Applicata 46:373-378.

Cane sugar and glucose followed by sucrose and maltose were the best sources of carbon for in vitro growth and sporulation of C. fusiformis isolated from diseased pearl millet earheads. Peptone, glycine, L-asparagin, DL-valine, urea, magnesium nitrate and L-proline supported good growth and fair sporulation of the fungus.

122. Sivaprakasam, K. 1971. A note on the control of ergot disease of pearl millet (Pennisetum typhoides S. & H.). The Andhra Agricultural Journal 18(5):212-214.

In field and pot trials, Aureofungin, Duter, and Cuman sprayed onto pearl millet panicles significantly reduced ergot (Claviceps fusiformis) by 49.65, 32.87 and 28.80%, respectively. The differences in grain yields among treatments were,

however, not statistically significant.

123. Sivaprakasam, K., Chinnadurai, G., and Krishnamurthy, C.S. 1971. Alkaloid production by C. avicena microcephala on some varieties of pearl millet. Madras Agricultural Journal 58(6): 431-432.

The total content of alkaloids in honeydew and sclerotia of C. microcephala estimated in four artificially inoculated pearl millet hybrids was greatest in sclerotia (0.92%) on HB 1 and in honeydew (0.48%) on HB 3. The reduced alkaloid content in sclerotia of HB 3 was probably due to decreased supply of precursors for biosynthesis of alkaloids.

124. Sivaprakasam, K., Kandaswamy, D., Ramamully, T.G., Selvaraj, S., and Krishnamurthy, C.S. 1971. Effect of nitrogen on the incidence of ergot disease of pearl millet caused by Claviceps microcephala. Madras Agricultural Journal 58(11):811-813.

Studies on the influence of different levels of nitrogen on the incidence of ergot (Claviceps microcephala) on six pearl millet hybrids in field trials showed that the application of N increased the susceptibility of pearl millet lines to ergot, although there were no significant differences among the different levels of N. All six varieties tested were susceptible to the disease.

125. Sivaprakasam, K., Pillayarsamy, K., Jagganathan, R., and Annavaradham, L. 1976. The effect of micro-climatic weather elements on the incidence of cumbu ergot. Madras Agricultural Journal 63(3):194-196.

There was no ergot (Claviceps microcephala) incidence on a pearl millet hybrid NHB 3 during 1970, 1972 and 1974 when rainfalls were 11.1, 21.2 and 0.7 mm, respectively during the crop season. In contrast, there was 68.6 and 39.13% ergot during 1971 and 1973 when the rainfalls were 237.9 and 131.6 mm, respectively. It was concluded that the rainfall and humidity had a positive relationship to ergot incidence and sunshine had a negative relationship.

126. Sivaprakasam, K., Pillayarsamy, K., and Ramu, S. 1975. Role of nitrogen on the incidence of ergot disease of pearl millet. (Pennisetum typhoides (Burm.) Stapf and Hubb.). Madras Agricultural Journal 62(9):574-576.

The increase in level of nitrogen increased the ergot (Claviceps microcephala) incidence in field trials on three pearl millet cultivars. The incidence was highest with applications of 160 and 200 kg/ha.

127. Stoll, A., Brack, A., Kobel, H., Hoffman, A., and Brumer, R. 1954. Die Alkaloide eines Mutterkornpilzes von Pennisetum typhoideum Rich. und deren Bildung in saprophytischer Kultur; Mitteilung über Mutterkornalkaloide (Alkaloids of ergot on Pennisetum typhoideum and their formation in saprophytic culture). Helv. Chim. Acta. 37(6): 1815-1825.

128. Sulaiman, M., Ludage, G.M., and Dawkhar, G.S. 1966. Effect of some fungicides and antibiotics on sclerotial development and germination of ergot on Pennisetum typhoides. Hindustan Antibiotics Bulletin 9(2):94-96.

Germination of Claviceps microcephala sclerotia was completely inhibited when soaked for 1 h in 5 ppm Aureofungin, 1000 ppm Dithane M-22, or 500 ppm oxytetracycline hydrochloride, and for 2 h in 2500 ppm Captan. In a field experiment, Aureofungin at 5 ppm, when sprayed on ergot-inoculated earheads, inhibited sclerotial development up to 75%.

129. Sundaram, N.V. 1967. Ergot disease of bajra, its symptoms and control. Indian Farming 17(9):56-58.

In a general account it was reported that the ergot disease of bajra was of widespread occurrence in states of Madras, (Tamil Nadu), Mysore, (Karnataka), Andhra Pradesh, Uttar Pradesh, Delhi, Punjab, Rajasthan and Haryana. It included the description of honeydew and sclerotial symptoms. In humans, ergot poisoning appeared as stomach pain, giddiness, and vomiting

followed by diarrhea. For precautions to avoid poisoning, cutting and destroying the infected earheads, and removing sclerotia by immersing the contaminated grain in 2% salt solution and skimming off sclerotia, were suggested. The ways suggested to minimize the intensity of infection were, adjustment of date of sowing on a regional basis, use of certified sclerotia-free seeds, crop rotation with jowar, maize, and moong, spraying Ziram at 0.1 to 0.15% or copper oxychloride + Zineb (1:2) two to three times at 5 to 7 day-intervals during flowering, removal of infected earheads, and deep plowing soon after harvest to bury the sclerotia deep in the soil.

130. Sundaram, N.V. 1975. Ergot of bajra. Pages 155-160 in Advances in Mycology and Plant Pathology. Published by the Professor R.N. Tandon's birthday celebration committee. New Delhi, India.

The review of work done on ergot (Claviceps microcephala) of pearl millet described the distribution of the disease in Africa and India, honeydew and sclerotial symptoms, morphological and physiological studies on the pathogen, and alkaloid content in sclerotia and its adverse effects, like inhibition of mammary hypertrophy and pregnancy in mice and agalactia in sows. Also described were infection by the pathogen through stigmas as well as through the tender ovary wall, establishment of the pathogen in the ovary, high susceptibility of male-sterile lines, varietal reaction, host range, environmental factors responsible for disease development, and chemical and cultural control measures.

131. Sundaram, N.V., Bhowmick, T.P., and Khan, I.D. 1969. A new host for Claviceps microcephala (Wallr.) Tul. Indian Journal of Agricultural Sciences 39:703-705.

Of 45 grasses examined, a hybrid of Pennisetum orientale x P. purpureum was recorded as a new host for Claviceps microcephala. The pathogen from this grass freely infected P. typhoides and vice versa.

132. Sundaram, N.V., Bhowmick, T.P., and Khan, I.D.

1970. Water soluble alkaloid content of ergot (Claviceps microcephala (Wallr.) Tul.) sclerotia of pearl millet [(Pennisetum typhoides (Burm. F.) Stapf. and C.E. Hubb.)]. Indian Journal of Agricultural Sciences 40:569-572.

Fully developed sclerotia of C. microcephala collected from pearl millet contained 0.156 % water-soluble alkaloids. This was determined with the help of a Klett Somerson colorimeter using ergometrine solution as a standard.

133. Sundaram, N.V., Palmer, L.T., Nagarajan, K., and Prescott, J.M. 1972. Disease survey of sorghum and millet in India. Plant Disease Reporter 56: 740-743.

Surveys on millet and sorghum diseases in Delhi and Haryana, India, during the 1970 kharif revealed that ergot (Claviceps microcephala) incidence on pearl millet was generally 30%, but in some fields it was up to 100%. An average of 0.279% sclerotial contamination in grain was estimated. Early sowing generally escaped ergot. Downy mildew incidence in pearl millet ranged from 60 to 100%. Blast, rust, smut, and species of Fusarium, Penicillium, and Cladosporium were also recorded. Prevalence of diseases on sorghum is also described.

134. Thakur, D.P. 1983. Relevance of artificial inoculation techniques in analysing pearl millet gene pool for genetically durable disease resistance. Pages 55-56 in Abstracts of the National Seminar on Breeding Crop Plants for Resistance to Pests and Diseases. May 25-27, 1983. Tamil Nadu Agricultural University, Coimbatore, India.

Of 51 Pennisetum americanum hybrids and populations tested, only 1 hybrid and 3 populations recorded less than 5% ergot-infected florets/ear following artificial inoculation.

135. Thakur, D.P. 1983. Epidemiology and control of ergot disease of pearl millet. Seed Science and Technology 11:797-806.

A review on epidemiology and control of ergot (Claviceps fusiformis) in pearl millet was presented. It included perpetuation of the pathogen through sclerotia, relationship of host susceptibility to floral biology, and pollination-based escape resistance. The incidence of the disease was reported to increase with applications of higher levels of nitrogen and phosphorus to the crop whereas it decreased with increased application of potash. High relative humidity (85 - 95%), cloudy weather and rainfall in morning and evening during the first 10 days of flowering were highly conducive for the development of ergot. Various control measures suggested include: crop rotation, early sowing, application of potash, intercropping with moong bean, removal of plants or flowers of collateral hosts, seed treatment with Thiram, and sprays of Cuman-L and/or Difoltan. Lack of complete resistance and the availability of some tolerant lines developed by gene pyramiding were mentioned.

136. Thakur, D.P. 1984. Ergot disease of pearl millet. Review of Tropical Plant Pathology 1:297-328

A review of work done on ergot (C. avicens fusiformis) of pearl millet is presented. Various aspects discussed include geographical distribution, extent of grain loss, toxin production and food poisoning, disease symptoms, taxonomy, pathogen reproduction and variability, floral biology and the infection process, host range, host nutrition in relation to ergot incidence, disease cycle, predisposing factors, disease management through cultural practices, use of chemicals, resistant cultivars, and biological agents.

137. Thakur, D.P., and Kanwar, Z.S. 1978. Ability of naturally incident Claviceps microcephala from Panicum antidotale to produce ergot symptoms in Pennisetum typhoides. Indian Journal of Agricultural Sciences 48(9): 540-542.

Claviceps microcephala was observed on Panicum antidotale in April and May 1976 and 1977 at Hisar. The conidia of the fungus isolated on PDA from P. antidotale and Pennisetum typhoides were

identical and cross-inoculation tests were successful. However, when inoculum from the same host was used for inoculation, symptoms were produced much earlier than when inoculum came from the other host. The pattern of natural ergot occurrence suggested that infected panicles of P. anti-otale possibly act as the source of conidial inoculum on the main crop of pearl millet.

138. Thakur, D.P., Mathur, S.B., and Kandhari, J. 1982. Evaluation of improved cultivars of pearl millet for major diseases in Haryana and Delhi. Seed and Farms 8(4):25-26.

Local pearl millet cultivars like Mainpuri and Tolaja contracted less ergot incidence than NHB 3 or BJ 104, in Haryana and around Delhi.

139. Thakur, D.P., and Mehta, N. 1983. Ergot disease of bajra and its control. Haryana Farming 12(8):4-5.

Symptoms, spread, and control measures of ergot (Claviceps fusiformis) of pearl millet were described.

140. Thakur, D.P., and Mehta, N. 1985. Pathological analysis of national and regional pearl millet genepool for major diseases under multiple disease sick-plot. Haryana Agricultural University Journal of Research 15(1):64-70.

Of 51 pearl millet cultivars screened for ergot (Claviceps fusiformis) resistance, MBH 133, PSB 15, MP 81, and WC-C75 had less than 5% severity during 1980 and 1982.

141. Thakur, R.P. 1987. Diseases of pearl millet and their management. Pages 147-158 in Plant Protection in Field Crops (Rao, Veerbhadra, M. and Sithanatham, S. eds.). Plant Protection Association of India, Rajendranagar, Hyderabad, India.

The details of progress of research work on pearl millet diseases are given. Ergot (Claviceps fusiformis) deteriorates quality of grain by contamination with neurotoxic, alkaloid-containing sclerotia. Ergot is more serious on F₁ hybrids

than on varieties, and at ICRISAT Center grain loss up to 65% in hybrids and 55% in varieties have been recorded. Morphology of the pathogen, disease cycle, sclerotia in soil and/or with planting seed as primary inoculum and, secondary spread by conidia, are presented. Also described are critical factors for ergot development, including the short susceptible period of the host, pollen interference, and favorable weather conditions during flowering. Biocultural control through pollen management, biological control, and control through host plant resistance are described. Use of host plant resistance, including growing varieties with multiple disease resistance, are suggested as superior method to achieve economical control. Economic importance, biology, epidemiology, disease cycle, management practices, chemical control, and control through host plant resistance were also described for downy mildew, smut, and rust.

142. Thakur, R.P., and Chahal, S.S. 1987. Problems and strategies in the control of ergot and smut of pearl millet. Pages 173-182 in Proceedings of the International Pearl Millet Workshop, 7-11 April, 1986, ICRISAT Center, India, Patancheru, A.P. 502 324, India: ICRISAT.

Ergot (Claviceps fusiformis) is more severe on F₁ hybrids than on open pollinated varieties because of the pollen protection phenomenon being more effective in varieties than in hybrids. The most effective and economical control is possible through host plant resistance. Effective field based screening techniques were devised to identify resistance. Resistance to ergot, which could not be detected in germplasm accessions, was developed by gene pyramiding through pedigree selection. Stability of resistance was determined through international, multilocation testing and lines with stable and combined resistance to ergot, smut and downy mildew became available to breed resistant hybrids and varieties. Further research was suggested to better understand biology, epidemiology, and resistance, such as variation in the pathogen populations and existence of different pathotypes or races, mechanisms and genetics of resistance,

identification of new sources of resistance, and multilocal testing of resistant lines, particularly in Africa.

143. Thakur, R.P., and Rao, V.P. 1983. Resistance to ergot in pearl millet in India, Page 210 in Abstracts, Fourth International Congress of Plant Pathology, 17-24 August, 1983, Melbourne, Australia. Rowprint Service (Vic.) Pty. Ltd. North Melbourne, Victoria, Australia.

With the development of an effective field screening technique for ergot (Claviceps fusiformis) resistance in pearl millet, many resistant lines were detected in the progenies of crosses involving ergot low-susceptible lines following pedigree selection. These lines also showed high levels of resistance to downy mildew and smut across eight locations in India.

144. Thakur, R.P., Rao, V.P., and Williams, R.J. 1981. Screening pearl millet for resistance to ergot. Pages 24-25 in Abstracts of the Third International Symposium on Plant Pathology, 14-18 December, 1981. Indian Agricultural Research Institute, New Delhi.

More than seven thousand germplasm accessions and breeding lines of pearl millet were screened for resistance to Claviceps fusiformis at ICRISAT Center. All breeding lines were highly susceptible, but variation for ergot reaction was detected in a few germplasm accessions. Lines with higher levels of resistance were developed by intermating low susceptible lines and identifying resistance following pedigree selection. Several of these lines showed high levels of location non-specific resistance, while others showed location specific resistance.

145. Thakur, R.P., Rao, V.P., and Williams, R.J. 1984. The morphology and disease cycle of ergot caused by Claviceps fusiformis in pearl millet. Phytopathology 74:201-205.

Sclerotia of C. fusiformis collected from pearl millet were elongate to round, 3.6-6.1 x 1.3-1.8 mm, light to dark brown, and hard to

brittle with a thick outer rind. Incubation time required for germination was inconsistent, usually varying from 4 to 56 weeks. Sclerotia germinated in moist sand in the laboratory, in potted soil in a screenhouse, and in the field. On germination each sclerotium produced 1 to 16 fleshy, purplish, 6-26 mm long stipes with each stipe bearing a globular capitulum. Pyriform perithecia were ostiolate. The long asci with apical pores contained ascospores which measured 127.7 x 0.5 um. Macroconidia from honeydew were hyaline, unicellular, and fusiform, measuring 15.9 x 3.9 um. Microconidia were globular, unicellular, and hyaline, measuring 5.9 x 2.5 um. At 25° C it took 16 h for conidia to germinate producing 1 to 3 germ tubes. The times of sclerotial germination and pearl millet flowering coincided, possibly providing opportunities for disease initiation. The inflorescences exposed to germinating sclerotia developed 5 to 6% ergot within 6-7 days. Honeydew production occurred 4-6 days after infection; sclerotia became visible after 8-10 days, and sclerotia matured within 20-25 days after inoculation. The process of sclerotial formation was more rapid than that of grain in pearl millet. Use of sclerotia - free seed and adoption of cultural practices that reduce sclerotial germination were discussed.

146. Thakur, R.P., Rao, V.P., Williams R.J., Chahal, S.S., Mathur, S.B., Pawar, N.B., Mefade, S.D., Shetty, H.S., Singh, Govind, and Bangar, S.G. 1985. Identification of stable resistance to ergot in pearl millet. Plant Disease 69: 982-985.

Of more than 2500 germplasm accessions of pearl millet from African and Asian countries and about 7300 advanced breeding lines of ICRISAT Center screened for ergot, only a few from India, Nigeria and Uganda were identified as being less susceptible (<10% severity) to Claviceps fusiformis. These lines were intermated and following pedigree selection for several generations under high disease pressure, ergot-resistant lines were developed. The stability of resistance was tested through a cooperative international, multilocational testing program - the International Pearl Millet Ergot Nursery

(IPMEN). The locations were in India and several countries in West Africa. Seven inbred lines (ICMPE numbers) had mean ergot severities ranging from <1 to 7% in 30 tests over 3 years, and six sib-populations (ICMPES numbers) had from <1 to 3% in 20 tests over 2 years, with 30-65% ergot in the susceptible check. Thirteen such ergot-resistant lines were also resistant to downy mildew and smut in four and three tests, respectively. These lines can be utilized to develop cultivars with multiple disease resistance.

147. Thakur, R.P., Talukdar, B.S., and Rao, V.P. 1983. Genetics of ergot resistance in pearl millet. Page 737 in Abstracts (pt2) of the 15th International Congress of Genetics, 12-21 December 1983, New Delhi, India. Oxford and IBH Publishing Company, New Delhi.

The studies on behavior of the progenies from four crosses involving ergot susceptible (ICP 220 and J 104) and ergot resistant (ICMPE 13-6-9 and ICMPE 134-6-9) pearl millet parents against Claviceps fusiformis revealed that the inheritance of ergot resistance was quantitative and controlled by several genes. The estimated number of genes involved was 10 in one cross and 5 in another. The heritability estimates were 0.55 and 0.31, and genetic advances 39.2% and 19.4 % in the two crosses, ICMPE 134-6-9 x J 104 and ICP 220 x ICMPE 13-6-9, respectively. A possibility of effective transfer of ergot resistance in pearl millet lines was indicated.

148. Thakur, R.P., and Williams, R.J. 1978. The effect of pollination on ergot susceptibility in pearl millet. Page 122 in Abstracts Third International Congress of Plant Pathology, 16-23 August 1978, Munchen, West Germany. Paul Parley, Berlin, West Germany.

In pearl millet male-sterile lines, pollination prior to or at the same time as inoculation reduced ergot infection to a very low level and pollination up to 16 hr after inoculation gave significantly less infection than the inoculated, non-pollinated check, probably due

to relatively rapid withering of stigmas following pollination. The results indicated the possibility for ergot control through pollen management.

149. Thakur, R.P., and Williams, R.J. 1980. Pollination effects on pearl millet ergot. *Phytopathology* 70:80-84.

Emergence of stigmas in pearl millet inflorescences starting from near the tip and progressing further towards the tip and the base was recorded. The ergot (*Cl. vicens fusiformis*) incidence was maximum in bagged inflorescences that developed in a pollen-free environment and were inoculated at the fresh stigma stage. Pollination of three male-sterile lines, 5141 A, 111 A and 5054 A, before or simultaneously with inoculation reduced ergot to less than 3% and pollination 16 hr after inoculation reduced ergot to 7-21% , compared with 60-80% in the inoculated, nonpollinated checks. The germination of pearl millet pollen took much less time than germination of *C. fusiformis* conidia. It was concluded that infection occurs mainly through the stigmas and protection against infection was probably due to induction of rapid withering of stigmas after pollination. The higher susceptibility of F₁ hybrids compared with traditional cultivars was explained in light of the results of these findings. The importance of these observations on the ergot resistance screening procedure, the development of cultural practices, like selection of lines with reduced protogyny or overlapping of stigma emergence and anthesis, and the use of pollen donors were discussed.

150. Thakur, R.P., Williams, R.J., and Rao, V.P. 1982. Development of resistance to ergot in pearl millet. *Phytopathology* 72:406-408.

Repeated screening of more than 4000 pearl millet germplasm accessions against ergot (*C. vicens fusiformis*) using an improved screening technique at ICRISAT Center revealed 20 accessions which were consistently less susceptible than others. These lines were intermated and the progenies from selected lines were screened and selected at each generation from

F₂ to F₆. The level of ergot resistance steadily increased from F₂ to F₆ generation, and about 70% of the lines showed less than 10% ergot severity and 27 lines had less than 1% mean ergot severity compared with 76-95% severity in the susceptible check. The possibility of further increasing the level of ergot resistance and its utilization in association with resistance to downy mildew and smut, to develop single-cross, disease-resistant hybrids, was discussed.

151. Thakur, R.P., Williams, R.J., and Rao, V.P. 1983. Control of ergot in pearl millet through pollen management. *Annals of Applied Biology* 103:31-36.

Analysis of the data from field trials with four highly susceptible F₁ hybrids sown with and without a pollen donor line, and spray inoculated with *Claviceps fusiformis* conidial suspension, revealed a significant reduction in ergot incidence and severity on these hybrids when grown along with the pollen donor line. Significant increase in the grain yield and 1000 grain weight in hybrids grown along with the pollen donor line was probably because of reduced ergot infection due to rapid pollination by readily available pollen from the pollen donor line. The possibility of reducing ergot incidence and severity in pearl millet through pollen management in farmers' fields was discussed.

152. Thind, K.S., and Madan, Mira. 1973. Effect of various trace elements on the growth and sporulation of *Claviceps microcephala* and *Microxyphiella hibiscifolia*. *Proceedings of the Indian Academy of Sciences B* 78:222-233.

Of 15 trace elements tested, *C. microcephala* required Fe, Zn, Mn, Cu and Mo for its in vitro growth and sporulation at 0.2, 0.1, 1.0, 0.01 and 1.0 ppm concentrations, respectively. Trace element requirement of *M. hibiscifolia* was also given.

153. Thind, K.S., and Madan, Mira. 1973. Effect of various carbon and nitrogen sources on the growth and sporulation of *Claviceps microcephala*. *Proceedings of the Indian Academy of Sciences*

Of 41 carbon sources tested, the in vitro growth of C. microcephala isolated from Pennisetum typhoides was excellent with dextrose, sucrose, pectin, and methyl alcohol; good with mannose, fructose, and maltose; and fair with galactose, coconut oil, and isopropyl alcohol. Of 33 nitrogen sources, the growth of the pathogen was excellent with casein hydrolysate, yeast extract, asparagine, peptone, proline, glutamic acid, and aspartic acid, and it was good with ammonium nitrate and ammonium oxalate.

154. Tripathi, R.K., Kolte, S.J., and Nene, Y.L. 1981. Mycoparasites of Claviceps fusiformis, the causal fungus of ergot of pearl millet. Indian Journal of Mycology and Plant Pathology 11(1):114-115.

Sclerotia of C. fusiformis on pearl millet were reduced considerably in size and germinability (50% inhibition) when they were parasitised by Fusarium sambucinum and Dactylium fusarioides. The cultural filtrate of F. sambucinum showed inhibitory effect on germination of conidia of C. fusiformis. The inhibitory activity of the cultural filtrate was reduced by heating it at 90° C for 10 min. The possibility of biological control of ergot was suggested.

155. Tulpule, P.G., and Bhat, R. V. 1978. Food toxins and their implication in human health. Indian Journal of Medical Research 68 (Supplement):99-108.

The consumption of pearl millet grain contaminated with more than 1.5% (w/w) ergot (C. aviceps fusiformis) sclerotia containing the clavine group of alkaloids, caused enterocergotism with symptoms like nausea, vomiting, giddiness, and somnolence in man. These symptoms were different from those caused by ergot of rye and wheat that contain ergotoxin (ergotamine and ergometrine groups of alkaloids). Safe limits of pearl millet ergotism in man could not be determined using monkeys as experimental animals, because of variable effects of the toxins on man and monkey.

156. Venkatesan, V. 1980. Separation of ergot from infected bajra (Pennisetum typhoides L.). Bulletin of Grain Technology 18(3):213-215.

With the aid of a compartmental separator, a fairly good separation of sclerotia of the ergot pathogen (Claviceps purpurea) (syn. C. fusiformis on pearl millet) was achieved from grain samples having 0.45 to 3.20% contamination by ergot sclerotia. After separation, the samples contained 0.005% sclerotia (by weight), which was considered very low.

157. Verma, O.P., and Pathak, V.N. 1984. Role of insects in secondary spread of pearl millet ergot. Phytophylactica 16(3):257-258.

A survey of ergot-affected fields of pearl millet (Pennisetum americanum) during 1980, 1981, and 1982 revealed 80 species of insects contaminated with conidia of Claviceps fusiformis. Apis indica and Tabanus rubidus carried the heaviest conidial load. In laboratory studies Musa domestica and Apis indica were the most efficient vectors of ergot giving more than 92% transmission, whereas percentage transmission of the disease was 68.0 for Dysdercus cingulatus, 58.3 for Monomorium salomonis, 75.0 for Syrphus contractor, 76.7 for T. rubidus, 30.8 for Vespa orientalis, and 19.0 for V. tropica.

158. Verma, O.P., and Pathak, V.N. 1985. Comparison of inoculation methods for pearl millet ergot. Indian Phytopathology 38:353-355.

In tests for artificial development of ergot (Claviceps fusiformis) on pearl millet, dipping the earheads at full protogyny stage in inoculum suspension for 20 seconds was found superior to spraying inoculum suspension to run-off on individual earheads.

159. Verma, O.P., and Pathak, V.N. 1985. Detached earhead culture of pearl millet ergot. Current Science 54(20):1074-1075.

A technique to develop ergot (Claviceps

fusiformis) on detached inflorescences of Pennisetum americanum was described. Maximum infection (66.66%) with lowest incubation period (5.73 days) was obtained with inoculated earheads kept under high RH (94-96%) at night alternating with low RH (63-74%) during the day.

160. Virk, D.S., Chahal, S.S., Singh, M.B., and Srivastava, M. 1981. Effect of sowing date on the occurrence of ergot and some morphological characters in pearl millet. Crop Improvement 89(1):65-67.

The parabolic response of ergot (Claviceps fusiformis) on four pearl millet cultivars signified escape from this disease in early and late sown crops in the Punjab. Early sowing was suggested to avoid an adverse affect on yield. The variety x sowing date interaction was not significant for ergot, plant height, or ear length.

161. Virk, D.S., Chahal, S.S., Singh, M.B., and Srivastava, M. 1982. Inheritance of resistance to ergot of pearl millet. MILWAJ Newsletter 1:3. (Abstract)

Evaluation of all possible one way F₁ crosses among eight pearl millet inbred lines against Claviceps fusiformis revealed significance of additive genetic variance for resistance. The significant differences for specific combining ability were mainly attributed to unidirectional, nonadditive effects, indicating ineffectiveness of simple breeding procedures for incorporating polygenically controlled resistance to ergot.

162. Williams, R.J. 1984. Disease resistance in pearl millet. Pages 245-296 in Review of Tropical Plant Pathology I (Raychaudhuri, S.P., and Verma, J.P. eds.). Today and Tomorrows's Printers and Publishers, New Delhi.

The review included several aspects of pearl millet ergot (Claviceps fusiformis) research including the development of a reliable field screening technique, development of resistant sources, determination of stability of resistance

through multilocal testing, and utilization of resistance to breed hybrids and varieties. The importance of studies on variability in the pathogen and nature of genetic resistance in the host were emphasized. The review also dealt with downy mildew, smut, and leaf diseases of pearl millet.

163. Williams, R.J., and Andrews, D.J. 1983. Breeding for disease and pest resistance in pearl millet. FAO Plant Protection Bulletin 31(4):136-158.

The research work reviewed on ergot (*C. avicana fusiformis*) of pearl millet include screening technique, testing stability of resistance, and utilization of resistance in breeding ergot-resistant cultivars.

164. Willingale, J., and Mantle, P.G. 1987. Stigmatic constriction in pearl millet following infection by *Claviceps fusiformis*. Physiological and Molecular Plant Pathology 30:247-257.

Macro- and microconidia of *C. fusiformis* germinated only on the parts of stigmas emerging from pearl millet florets. Macroconidia often produced several germ tubes, forming a mycelial network. The germ tube penetrated the stigma and followed the same passage in stylodia as the millet pollen tube. Thirty-six hours after inoculation, a constricted region located in the fused stylodia appeared concurrently with hyphal invasion of the upper ovary wall. This response was similar to the stigmatic constriction formed in response to pollination. Stigmas then withered and abscised due to the weight of the aerial stylodia. It effectively isolated the ovary from pollen. Colonization of the ovary by the pathogen proceeded predominantly through the abaxial wall towards the vascular traces supplying the ovary. The hyphae remained intercellular throughout the invasion and colonization and honeydew exudation from florets marked the establishment of the sphacelium 4-5 days after inoculation. Hyphae did not penetrate the vascular strands below the ovary of the flower.

165. Willingale, J., Mantle, P.G., and Thakur, R.P.

1986. Postpollination stigmatic constriction, the basis of ergot resistance in selected lines of pearl millet. *Phytopathology* 76(5):536-539.

Five ergot-resistant pearl millet lines, developed at ICRISAT Center and showing varying degrees of protogyny, and two highly susceptible hybrids were inoculated artificially with the conidia of the pathogen (*Claviceps uniformis*). In the resistant lines, protogyny of individual inflorescences lasted for ≤ 48 h. The development of a localised stigmatic constriction which occurred 6 h after pollination helped ovaries escape the disease. In contrast, stigmas of highly susceptible hybrids remained receptive for up to 6 days, when an ageing constriction occurred, located similarly at the pollination-induced abscission site. Development of an ageing constriction in stigmas of susceptible lines, prior to self-anthesis, also resulted in self-incompatibility. An extended period between emergence of stigmas and anthers allowed establishment of the pathogen within the unfertilized ovary. Where protogyny lasted for >48 h, resistance could be conferred only by cross-pollination prior to gynoecial ageing. Possibly ergot resistance is based on a pollination escape phenomenon linked to normal events occurring during the flowering process. At ICRISAT Center ergot-resistant lines have been developed by selection of individuals in which stigmas emerge only a few hours before self pollen is shed, which results in rapid, self-induced stigmatic constriction.

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