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ERGOT INFECTION AND STERILITY IN GRAIN SORGHUM

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Summary

The sterility often associated with the ergot disease is apparently not caused by the pathogen. Unfertilized cytoplasmic male-sterile florets were susceptible to ergot infection. The disease did not develop on the B line of Combine Kafir-60, but the cytoplasmic male-sterile A line was the most susceptible line in the test. Two lines from India with "twin" seeds became infected. Experimental lines with female sterility caused by chromosomal aberrations showed a low degree of susceptibility to the fungus. Cytoplasmic male-sterile plants of the A line of Combine Kafir-60 had 50% of the florets infected with ergot when they were not pollinated. Pollination of the florets at the time they become receptive reduced ergot infection to 8%. The data indicated that the ergot fungus infected flowers only when they were not pollinated. Ergot could become a serious problem in hybrid seed production with male-sterile lines in Nigeria. These experiments indicated that this disease can be controlled or markedly reduced in a cytoplasmic male-sterile line by having an abundance of pollen available from the pollen parent when sterile florets become receptive.

INTRODUCTION

Ergot of sorghum is caused by Sphacelia sorghi McRae, the imperfect stage of an undetermined species of the genus Claviceps. According to Tarr (11), this disease was observed in India in 1915, Uganda and Kenya in 1926, Burma in 1941 and Tanganyika in 1949. The disease was reported in Nigeria by Harrison (5) in 1955. A high incidence of the disease was found in certain strains of grain sorghum growing in experimental plots at Samaru in 1963 and 1964.

EXPERIMENTAL

Experiments were designed to provide additional evidence that male-sterile sorghum florets were more susceptible to ergot infection than those that were fertile. This implies that a floret is no longer susceptible to ergot after fertilization has occurred. The following observations were made:

1. The percentage of spikelets infected with ergot was determined in the A line of Combine Kafir-60 (cytoplasmic male-sterile) and in numerous fertile varieties.
2. The percentage of infected spikelets of the A line of Combine Kafir-60, hand-pollinated at the time of blooming, was compared with male-sterile spikelets on adjacent plants that were permitted to open-pollinate in an area where there was very little pollen available.
3. The percentage of spikelets infected with ergot in plants of A lines and B lines (fertile) of Combine Kafir-60, planted in alternate rows, was compared.
4. The development of the ergot disease in plants of the A and B line of Combine Kafir-60 was compared with ergot development in three populations with varying degrees of female sterility caused by chromosomal aberrations.

RESULTS

The prevalence of ergot was determined in a collection of 1053 sorghum strains from 15 different countries. Table 1 gives data for 11 lines or varieties. Ergot was found on Indian selections I.S. 1052, I.S. 1112 and the A line of Combine Kafir-60.

Panicles of the A line of Combine Kafir-60 were hand-pollinated at the time the florets became receptive to determine whether this would reduce the number of florets infected with ergot. The hand-pollinated panicles had 8.4% of the spikelets infected, and the open-pollinated panicles had 50.3% of the spikelets infected (Table 2).

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Table 1. The percentage seed set, ergot-infected spikelets and blank spikelets in panicles of 11 varieties or lines of grain sorghum from 5 different countries, grown at Samaru, Nigeria in 1964.

Variety or Line[a]	Country of origin	% Seed set	% Ergot-infected spikelets	% Blank spikelets
Barqui	Sudan	79.9	0	20.1
Deng	Sudan	79.3	0	20.7
Mathinang	Sudan	80.3	0	19.7
White Shalsali	Sudan	79.5	0	20.5
M.F.G. Sel. F 1479	Argentina	75.9	0	24.1
B.C. 59	Nigeria	93.2	0	6.8
White Kaoliang	U.S.A.	96.4	0	3.6
Combine Kafir-60				
B Line	U.S.A.	96.4	0	3.6
A Line	U.S.A.	30.5	50.3	19.2
I.S. 1112	India	4.3	58.3	37.4
I.S. 1052	India	2.7	74.8	22.5

[a]A minimum of 2000 spikelets were counted per variety or line.

Table 2. The percentage seed set, ergot-infected spikelets, and blank spikelets in panicles of hand-pollinated and open-pollinated heads of the A line of Combine Kafir-60.

Treatment[a]	% Seed set	% Ergot-infected spikelets	% Blank spikelets
Open-pollinated	30.5	50.3	19.2
Hand-pollinated	71.1	8.4	20.5

[a]2000 spikelets per treatment counted.

Table 3. Comparison of percentage of ergot-infected and non-infected plants of A and B lines of Combine Kafir-60 and female-sterile lines.

Line	% Ergot-infected plants	% Non-infected plants
A line, Combine Kafir-60	54.8	45.2
70% Female Sterile	1.6	98.4
30% Female Sterile	0	100.0
15% Female Sterile	0	100.0
B line, Combine Kafir-60	0	100.0

Table 4. The percentage seed set, ergot-infected spikelets, and blank spikelets in panicles of the A and B lines of Combine Kafir-60 and female-sterile material.

Line[a]	% Seed set	% Ergot-infected spikelets	% Blank spikelets
A line, Combine Kafir-60	30.5	50.3	19.2
70% Female Sterile	20.0	9.5	70.5
30% Female Sterile	70.0	0	30.0
15% Female Sterile	85.0	0	15.0
B line, Combine Kafir-60	96.4	0	3.6

[a]A minimum of 2000 spikelets per line were studied.

The percentage of ergot-infected plants in the A and B lines of Corbine Kafir-60 and the three female-sterile lines are given in Table 3. A comparison of the percentages of spikelets infected in these lines is presented in Table 4. No disease was found in the lines with 15% and 30% female sterility, but 9.5% of the spikelets were infected in the plants with 70% female sterility. These data indicate that the disease is strongly associated with cytoplasmic male sterility and only slightly associated with female sterility.

DISCUSSION

The data indicate that unfertilized cytoplasmic male-sterile florets are susceptible to ergot infection. The two Indian varieties which were infected with ergot normally have "twin" seeds which result when both florets of the sterile spikelets are fertile. The lower floret of the spikelets of most varieties is reduced to a trim empty lemma. The seed set on the Indian selections was low and the percentage of ergot infection was high. "Twin" ergot sclerotia should be expected in the spikelets but none could be identified.

The ergot fungus has been cited many times as the cause of sterility in host plants. The data here presented suggest that the ergot fungus is more likely to infect sterile florets of sorghum. Sterility is prevalent in other species of plants affected by the ergot disease. Claviceps paspali Stevens & Hall parasitizes the flowers of Paspalum species which have apomictic reproduction with a high percentage of sterile flowers. When crosses were made between two species of Paspalum, selected progenies had better seed production and less ergot infection (8). Rye, one of the most common hosts of the ergot fungus, is self sterile and mostly cross pollinated. When Khapli emmer wheat was crossed with common wheats, sterility resulted and ergot infection was common in a large number of the crosses (4). Theis (12) reported heavy ergot infection on guinea grass, Panicum species in Puerto Rico. Hutchinson and Bashaw (6) demonstrated that apomictic reproduction occurs in Panicum species, especially those with higher chromosome numbers. Data on all the above species of plants strongly suggest that sterile flowers are susceptible to ergot infection and fertile flowers resist infection.

Further evidence that ergot is not the cause of sterility, but that it infects unfertilized or sterile flowers, can be found in studies of time of infection with different crops. Ramakrishnan (9) stated that sorghum could become infected with ergot only between the time the flowers open and the time of fertilization. Fuentes, et al. (3) were unable to infect maize with Claviceps gigantea Fuentes, et al. after fertilization had occurred. Any factor that prolongs the period from the time when the flower opens, until fertilization, promotes ergot infection. Many environmental factors are known to cause the pollen to become non-functional; among these are heat, cold and irradiation. When flowers of wheat, oats or barley open during a period of cold weather (35° to 40°F) the pollen becomes non-functional and sterility will result. Ergot infection often occurs under these conditions. Hot water has been used experimentally to sterilize grass flowers. Floral sterility caused by cytoplasmic male sterility, apomixis, self-incompatibility, spikelets with twin ovules and female sterility all establish conditions favorable for development of ergot. In the present study, the ergot fungus grew most profusely on cytoplasmic male-sterile lines. This was followed by the twin ovary condition in the two Indian selections. Ergot infections were lightest on female-sterile lines of sorghums.

Ergot sclerotia will grow only in the flowers of host plants. The sclerotia have never been grown on artificial media. Fuentes, et al. (3) grew the imperfect stage of the maize ergot fungus on 1% glucose, 1% yeast extract, and 1.5% agar medium, but after several transfers the ergot organism lost the ability to produce imperfect spores; and many of the cultures ceased to grow. Dickson (2) presented photomicrographs, showing that the ergot sclerotia become an integral part of the grass flower by attaching the absorbing mycelium to the rachilla. The point of attachment of the fungus is the area of the flower where the flowering hormones are concentrated. If the flower is not pollinated at the time it becomes receptive, it continues to produce female hormones excessively; this is the condition that exists in the non-pollinated cytoplasmic male-sterile plants; the flowers are producing only female hormones because the male flower parts are non-functional. This unbalanced hormone condition establishes a suitable medium to catalyze and promote successive growth of ergot sclerotia. What would the chemical nature of a hormone responsible for ergot development be? Audus (1) cited some evidence that flowering hormones contain small amounts of Vitamin E and large quantities of sterols. Love and Love (7) applied the animal female hormone oesterone and oestradiol in lanolin paste to the leaf axils of the male plants of Melandrium dioicum which caused the suppression of the flowers and promoted the development of female flower parts. Treating the pistillate flowers with the male hormone, testosterone, suppressed their development and

promoted the development of staminate flowers.

Hormones have also played a role in the susceptibility of M. dioicum to the smut disease. According to Shull (10) when Ustilago violaceae (Pers.) Fckl. attacks male flowers of M. dioicum the chlamydospores are formed in the pollen sacs. But when plants producing female flowers are attacked by this smut fungus, the plants cease to produce female plant parts and are stimulated to produce male plant parts. The chlamydospores of the smut fungus then develop in the pollen sacs. The hormones produced in flowers of grass plants probably account for the obligate nature of the growth of the ergot sclerotia. The use of the steroid ring in the flowering hormones probably furnishes the basic material for the synthesis of alkaloid drugs by the ergot fungus in the grass flowers.

The medicinal value of ergot sclerotia has long been recognized. Dickson (2) stated that the sclerotia produced on rye contain varying amounts of ergosterol, ergotoxin, ergotamin, ergosletrine, ergoclarin and other alkaloid-like compounds used in drug manufacture. Many of these compounds are chemically similar to the hormones. The male-sterile A line of Combine Kafir-60 affords a good tool for studying the relationship between plant hormones and sclerotial development. The possibility exists that these compounds may be identified, and that the sclerotia may be grown in culture on artificial media.

Sclerotia can be produced in mass amounts on cytoplasmic male-sterile lines of sorghum for physiology studies on animals to determine what alkaloids are present. The effect of sorghum ergot sclerotia on animal metabolism has not been but should be determined since sclerotia can be present in threshed sorghum seed.

The ergot disease appears to be a potential threat to hybrid sorghum seed production in Nigeria. This study demonstrates that one solution to the problem would be to have an abundance of pollen present to fertilize the flowers of male-sterile plants as soon as they become receptive.

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