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HOST RANGE AND EPIDEMIOLOGY OF THE SORGHUM ERGOT ORGANISM

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Summary

The source of primary inoculum of ergot on sorghum in Nigeria is probably guinea grass growing wild. Ergot conidia, collected from guinea grass and used to inoculate sorghum and millet, produced the disease on both crops. The sorghum ergot was cross-inoculated on and infected maize (corn). Near 100% relative humidity was necessary during the first 12 to 36 hours (24 hours was the optimum) of the incubation period for infection. The incubation period ranged from 7 to 9 days.

The secondary spread of the disease was by flies, bees, and other insects, rapidly during the wet season but not at all in the dry season. Sorghum plants were easily infected during the dry season, if high humidity was maintained under plastic bags for the first 24 hours of the incubation period. Sclerotia produced during the wet season were poor because of fungal parasites. Excellent sclerotia were produced during the dry season. Sorghum was inoculated and infected for 9 months, from January to October. In August, maize (corn) was inoculated and infected with ergot conidia from sorghum. Ergot conidia, collected on guinea grass, infected sorghum and millet when inoculated in May but not in February and March. The Indian Selection 1112 produced 9.1 grams of ergot sclerotia per head, and Combine Kafir-60 produced 2.8 grams per head.

INTRODUCTION

Ergot of sorghum caused by \textit{Sphaecella-sorghi} McRae was epiphytotic in the breeding nurseries at Samaru in 1963, 1964, and 1965. The source of inoculum for these epiphytotics, and the conditions favoring infection, have not been determined. High incidence of the disease occurred almost spontaneously in August and September in each of these years. The disease has been more noticeable with increased use of cytoplasmic sterility in the breeding program. Burton and Lefebvre (2) have shown that nonfertilized or sterile florets of \textit{Bahia} grass, \textit{Panicum notatum}, were susceptible to ergot. Futrell and Webster (4) reported the same with sorghum. Both studies showed that after pollination florets are no longer susceptible to ergot. Tarr (8) suggested that wild grasses may be a source of primary inoculum of ergot of sorghum. We initiated this study to determine which species of plants were susceptible to the ergot organism, and to define more precisely the environmental conditions favoring infection and spread of the disease.

MATERIALS AND METHODS

The purposes of our field experiments were as follows:

1. To determine if maize (corn), \textit{Zea mays} 'Mexico-5', and millet, \textit{Pennisetum typhoides}, were susceptible to ergot conidia collected on sorghum, \textit{Sorghum vulgare} 'Combine Kafir-60', A line; and to determine if Combine Kafir-60, A line, and millet were susceptible to ergot conidia collected on common guinea grass, \textit{Panicum maximum}.

2. To determine the length of the period of high humidity that best favored infection of sorghum with the ergot organism, and to study the spread of the disease during 9 consecutive months of the year.

3. To determine the weight of ergot sclerotia produced on a sorghum panicle.

Inoculations on sorghum and millet were made artificially by immersing the unpollinated panicles in honeydew suspended in water. Inoculations on maize (corn) were made by injecting a water suspension of conidia into ear shoots with a hypodermic needle. High humidity was maintained by bagging the inoculated inflorescences with a plastic bag.
Honeydew conidial inoculum for these studies was collected in the field in October 1964, dried at room temperature, and kept under refrigeration at -10°F.

RESULTS

The results of cross-inoculation studies are given in Table 1. The highest percentage of infection was from sorghum to sorghum, followed by sorghum to maize, then by guinea grass to sorghum. The poorest infection was from guinea grass to millet. Millet inoculated with conidia collected on sorghum did not show visible honeydew, but the florets were always killed and took on a dusty appearance 1 week after inoculation. Not all of the inoculations on maize were successful; only those inoculated 7 days after the silks emerged were infected. Florets inoculated 2, 3, 4, and 14 days after the silks emerged did not become infected, and inoculation of fertilized florets did not result in infection.

Successful inoculations from sorghum to sorghum were made monthly from January through September. The cross-inoculation from sorghum to maize was made in August 1965. The successful cross-inoculations from guinea grass to sorghum and millet were made in May 1966. Failures to infect sorghum and millet with conidia from guinea grass occurred after the February and March inoculations. Cause of failure is not known. The inoculation period on all hosts studied ranged from 7 to 9 days. High relative humidity (near 100%) was necessary to establish infection. Most infection was obtained when florets were held at high relative humidity for 24 hours after inoculation. High humidity periods of 12 and 36 hours also favored infection, but disease developed on flowers held at high humidity for 9 days (216 hours) (Table 2).

Table 1. Percentage of florets infected with the ergot organism in cross-inoculation tests.

<table>
<thead>
<tr>
<th>Inoculation</th>
<th>Number of florets inoculated</th>
<th>% Florets infected</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. vulgare to S. vulgare</td>
<td>2000</td>
<td>55</td>
</tr>
<tr>
<td>S. vulgare to Z. mays</td>
<td>2500</td>
<td>25</td>
</tr>
<tr>
<td>S. vulgare to P. typhoides</td>
<td>2000</td>
<td>0</td>
</tr>
<tr>
<td>P. maximum to S. vulgare</td>
<td>500</td>
<td>6</td>
</tr>
<tr>
<td>P. maximum to P. typhoides</td>
<td>500</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 2. Effect of varying periods of high relative humidity (near 100%) on ergot infection of Combine Kafir-60 A line inoculated in the dry season. Data for 1965 and 1966.

<table>
<thead>
<tr>
<th>Number of hours</th>
<th>Number of florets inoculated</th>
<th>% Florets that became infected</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1000</td>
<td>0</td>
</tr>
<tr>
<td>12</td>
<td>1000</td>
<td>25</td>
</tr>
<tr>
<td>24</td>
<td>1000</td>
<td>55</td>
</tr>
<tr>
<td>36</td>
<td>1000</td>
<td>25</td>
</tr>
<tr>
<td>216</td>
<td>1000</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 3. Quantity of ergot sclerotia produced on three sorghum lines grown at Samaru, Nigeria, in 1964.

<table>
<thead>
<tr>
<th>Line</th>
<th>Number of sclerotia per panicle</th>
<th>Weight of sclerotia per panicle (in grams)</th>
<th>Average weight of sclerotium (in milligrams)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indian Selection 1112</td>
<td>816</td>
<td>0.1</td>
<td>11.1</td>
</tr>
<tr>
<td>Combine Kafir-60 A Line</td>
<td>331</td>
<td>2.8</td>
<td>8.5</td>
</tr>
<tr>
<td>Indian Selection 1052</td>
<td>354</td>
<td>2.7</td>
<td>7.5</td>
</tr>
</tbody>
</table>
After infection was established, the disease spread by flies, bees and other insects from plant to plant during the wet season (May to September). Sclerotial development was very poor during the wet season owing to the heavy growth of fungal parasites in the honeydew. The following parasites were found in the honeydew, and on the surface of the sclerotia: *Fusarium moniliforme* Sheld., *F. roseum* Lk. ex Fr. f. sp. *cerealis* (Cke.) Synd. & Hans., *Corebella* sp. and *Cladosporium* sp.

There was no secondary spread of the ergot disease during the dry season of very low relative humidity (October to May). That low humidity was the limiting factor during the dry season was shown by the fact that good infection was produced in the dry season when panicles were humidified under plastic bags. The best sclerotia, with clean honeydew, and free from fungal parasites, were produced from artificial inoculations made in February and March. Four or 5 weeks after inoculation were required for sclerotia to develop to maturity. Sorghum florets that became infected at the end of the wet season and developed in the dry season produced sclerotia free from fungal parasites.

The total and average weights of sclerotia produced on panicles of three varieties of sorghum are given in Table 3. Large quantities of ergot sclerotia were produced on two Indian Selections, I.S. 1112 and I.S. 1052, both of which have two fertile florets per sessile spikelet. The range in weight of ergot sclerotia produced on unpollinated florets of Combine Kafir-60, A line, was from 1.6 to 4.2 grams per panicle with an average of 2.8 grams.

Honeydew inoculum collected in October 1964, dried at room temperature and stored at -10°F, was still viable in May 1966. Honeydew on panicles stored in the laboratory at room temperature, left in the field during the dry season of 1964-65, became nonviable after 7 months.

**DISCUSSION**

This cross-inoculation study shows that ergot conidia collected on sorghum will infect *Z. mays*, and ergot collected on *P. maximum* will attack sorghum and millet. The perfect stage of the sorghum ergot organism has never been assigned to a species of *Claviceps*, although it is known to occur (5). Therefore, the following species of ergot must all be related. *C. gigantea* Fuentes, et al. attacks *Z. mays* (3), *C. maximensis* Theis attacks *P. maximum* (9), *C. purpurea* (Fr.) Tul. attacks *P. maximum* (10), *C. paspali* F. L. Stevens & J. G. Hall attacks *P. maximum* (7).

Sprague (7) summarized the literature on *C. paspali*, and showed that there are five or six physiologic races of this fungus. This cross-inoculation study would indicate that the sorghum ergot organism may be a physiologic race of *C. purpurea*. The Index of Plant Diseases in the United States (10) lists *P. maximum* as a host of *C. purpurea*; this is probably the oldest Latin binomial to be applied to the ergot fungus.

A knowledge of the life cycle of a plant pathogen is essential for efficient control of the disease that it causes. The life cycle of the sorghum ergot organism in Nigeria is not known. Although Singh (6) found the perfect stage of the fungus in India, the perfect stage has never been found in Africa. Tarr (8) suggested that wild grasses were a source of inoculum for sorghum; this is important in determining the spread of the disease in Nigeria. The disease was observed on *P. maximum* and other plants in southern Nigeria during February and March. The disease could have spread north on grasses as the rains moved northward. In the sorghum-growing area of northern Nigeria *P. maximum* and other wild grasses come into flower soon after the start of the rains, much earlier than sorghum does. The ergot organism builds up on the grass flowers, and thus provides inoculum for sorghum. This could account for the fact that the disease becomes epiphytotic on sorghum in August and September. Ergot honeydew can live through the dry season for 7 months on sorghum refuse; with the advent of rains the ergot organism could spread from honeydew to early flowering wild grasses, and then be transmitted to sorghum in late summer. Whenever a plant pathogen can propagate itself asexually, it will do so. The generation time is much shorter for the production of conidia than for the production of ascospores. A much greater quantity of inoculum is produced in an asexual phase of the life cycle than in the sexual phase; therefore in a locality where the ergot organism can propagate itself asexually, there is a tendency for the organism not to produce the sexual stage. *C. purpurea* requires a dormancy period, followed by cold temperatures, to germinate. Such is not the case in Africa, which lacks cold temperatures.

In spite of the results alluded to, cross-inoculation of sorghum ergot on millet may have been successful. In studies on grasses, Sprague (7) cited instances in which the ergot organism killed the florets before visible honeydew and sclerotia were produced in sufficient quantities to
be seen. He indicated that grass florets exhibiting these symptoms take on a dusty appearance. In our study a similar dusty appearance was observed on millet florets inoculated with sorghum ergot on three different dates; and all millet florets inoculated with sorghum ergot conidia were dead 1 week after inoculation. If the sorghum race of ergot was highly virulent on millet florets, the quick invasion of the floral parts might produce necrosis and death of the florets before the honeydew could be produced. The concentration of inoculum may have been too heavy. A further cross-inoculation study is needed with millet.

Failure of the sorghum ergot organism to infect fertilized florets of *Z. mays* further supports the theory of Burton and Lefebvre (2) -- substantiated by Futrell and Webster (4) -- that nonfertilized florets are susceptible to the ergot disease, but that fertilized florets are not susceptible. Ergot does not cause sterility. These facts should be taken into consideration in a program of screening for ergot resistance in any host plant.

The toxicity of the sorghum ergot sclerotia has never been established. Recent work in Australia (1) with *C. paspali* has shown that 1.68 ounces of ergot sclerotia will cause ergotism in a cow. If sorghum sclerotia are as toxic as those of *C. paspali* studied in Australia, fewer than six panicles of I.S. 1112 -- which in our study infected ergot -- would produce ergotism in a cow. If calculated on a weight basis, fewer than 1.68 ounces would be required to cause ergotism in a human being. Shands (5) demonstrated that barley affected with scab caused by *Fusarium* sp. was toxic to pigs. *Fusarium* spp. were found in abundance on ergot sclerotia, and on sorghum grains produced at Samaru in the wet seasons of 1964 and 1965. Are the toxic effects of ergot and scab additive? Inasmuch as sorghum is more and more widely used for animal and human food in many parts of the world, this question deserves more study.

**Literature Cited**


CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND AHMADU BELLO UNIVERSITY, INSTITUTE FOR AGRICULTURAL RESEARCH, SAMARU-ZARIA, NIGERIA