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EC00-1879 Sorghum Ergot in the Northern Great Plains

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Sorghum is grown throughout Nebraska on approximately 0.6 million acres of land. It is grown as a forage crop as well as a grain crop. There is no significant commercial seed production in Nebraska. Grain sorghum is used domestically as livestock feed, in ethanol production, and to a limited extent as a food crop. Grain sorghum is also exported to several countries. All sorghum hybrids (grain and forage) are susceptible to ergot disease. Ergot is a disease that impacts sorghum production directly by infecting unfertilized flowers and preventing seed development. Ergot also impacts sorghum production indirectly. Affected fields with honeydew-covered panicles can be difficult to harvest due to gumming up combines. Additionally, honeydew-coated seed can cause problems with postharvest shipping and handling.

Pathogen

Identity

The pathogens that cause sorghum ergot are different from the pathogens that cause ergot in other cereals, including rye and wheat. Sorghum ergot is caused by three species of fungus. The geographic distribution of each species has been changing over the last few years with significant implications for the international sorghum industry. *Claviceps sorghi* was first described in India and until recently was believed to be the sole cause of sorghum ergot in that country. *Claviceps africana* is the most widespread species that causes sorghum ergot; it has been found in Africa, Australia, South America, Central America, North America, and the Caribbean basin. Recently this species was discovered in India. There is some speculation that *C. africana* is displacing *C. sorghi* in India. A new species, *C. sorghicola*, was described as the causal pathogen of sorghum ergot in Japan. The pathogen life cycles and symptoms caused by these three species of *Claviceps* are similar but not identical. The primary differences among the three species are:

1) the role of secondary conidia in pathogen dispersal and epidemic development,
2) the importance of sclerotia to survival,
3) the presence of a sexual reproductive stage in nature, and
4) the synthesis of potentially toxic alkaloids.

Claviceps africana is the only sorghum ergot pathogen which has been found in the United States.

Unlike other species of *Claviceps* (e.g., *C. purpurea*), sexual reproduction is not believed to be important to the survival or to epidemic development for *C. africana*. Although the sexual structures have been observed in laboratory studies, they have never been observed in nature. Genetic variation between U.S. populations of *C. africana* and populations from other countries has been reported. Variation among U.S. populations is being investigated. The potential for genetic variation leading to altered virulence or host range is uncertain.

Geographic Distribution

Sorghum ergot is an important disease worldwide. It has occurred in India and South Africa for several decades. Although ergot can limit yield and reduce grain quality when severe, the sorghum industries of these two countries remain healthy. Sorghum ergot has also occurred in southeast Asia and Japan to a lesser extent. On March 28, 1997, the disease was first observed in the United States in the Rio Grande Valley of south Texas. During the 1997 growing season, *C. africana* spread north from Corpus Christi, to Uvalde, and Hillsboro and then into the major sorghum seed production area between Lubbock and Amarillo. On August 28, 1997, ergot was observed in a field of sterile forage sorghum just north of Wichita, Kansas, approximately 185 miles south of Nebraska. It was observed in eight fields in Kansas within a 14-mile radius of the original site. Disease incidence (number of heads infected) in a few fields of forage sorghum was estimated to be approximately 60 percent, while in other fields it was much lower.

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Figure 1. The pathogen infects the plant only through unfertilized florets, resulting in the production of fungal structures called sphacelia (white structure shown above) in place of seed. A single fungal structure is called a sphacelium.

Its current geographic distribution in the United States extends from Texas to Nebraska and from New Mexico to Georgia. The disease is considered a production problem only in the hybrid seed production areas of Texas. The dynamics and mechanisms of dispersal for the ergot pathogen in North America have not yet been determined. This pathogen has been present in Africa for many years and the disease it causes has been well studied. Whether information obtained in Africa can be applied to the United States and in particular to the Northern Great Plains remains uncertain. Since its arrival in 1997, sorghum ergot was reported in Texas and in central Kansas each year to a limited extent. It was found in Nebraska in only one field (in Virginia, Nebraska) in 1997 and has not been observed since then.

Disease Development

The symptoms of sorghum ergot are very distinctive. The ergot pathogen infects through unfertilized flowers in a manner that mimics pollination. The conidium lands on the stigma, germinates, grows down the style, and penetrates the ovary. The pathogen colonizes the ovary and converts it into a mass of fungal tissue called a sphacelium *(Figure 1)* approximately six days after infection. Direct infection of the ovary has

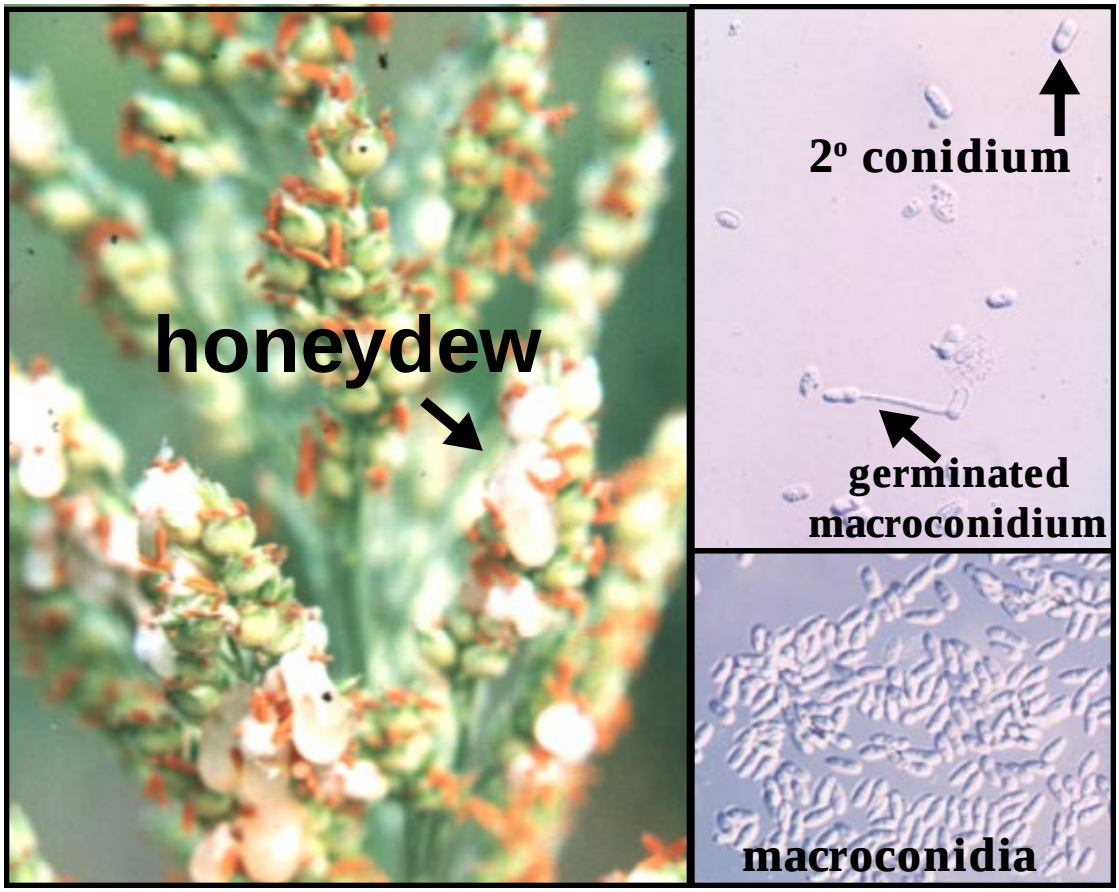

Figure 2. A very viscous exudate "ergot honeydew" with high sugar content is produced from the sphacelium 7-10 days after infection. This honeydew contains spores of the pathogen.

Figure 3. Exudate from the sphacelium, called honeydew, flows down the panicle and drips onto leaves and soil leaving the appearance of bird droppings when dried.

also been reported. The most diagnostic symptom of sorghum ergot is the production of exudate *(Figure 2a)* from the sphacelia that develop in place of seed. The exudate, called honeydew, contains a high concentration of sugar. After flowing down the panicle, it coats seed and drips onto leaves and soil leaving the appearance of bird droppings *(Figure 3a-b)*. Other fungi commonly colonize the honeydew giving the panicle a discolored appearance *(Figure 4a-c)*.

Claviceps africana produces at least two spore types, macroconidia and secondary conidia. The honeydew contains very high populations of macroconidia *(Figure 2b)*. As the honeydew flows down the panicle, the macroconidia can directly infect unfertilized florets. Because of the high sugar concentration, the honeydew is very attractive to insects. Insects that come into contact with the honeydew can serve as vectors of the pathogen by carrying honeydew containing

Figure 4. If the humidity remains high other fungi can colonize the exudate. Three fungi commonly found on ergot infected panicles are *Alternaria* (left), *Cladosporium* (center), and *Cerebella* (right). Although the presence of these other fungi may cause reduced grain quality, their full significance is undetermined.

macroconidia to other sorghum plants. The macroconidia also germinate by producing secondary conidia *(Figure 2c)*. If the relative humidity is high, secondary conidia are produced on the honeydew surface. The secondary conidia can be wind dispersed at least 900 feet within and between fields. It is believed the conidia are capable of long range dispersal (region to region) via wind and / or insects. The production and dispersal of secondary conidia are considered to be the primary reasons for the rapid spread of sorghum ergot through Australia within one season and throughout South America, Central America and into North America within two years (1995-1997). If deposited on a receptive stigma of an unfertilized sorghum floret, the secondary conidium can cause infection via the same mechanism as the macroconidium. The secondary conidium is also capable of iterative (repeated) germination. If it is deposited onto a surface other than an unfertilized floret, it can germinate by producing another conidium for aerial dispersal. Secondary conidia are capable of at least three germination cycles.

Impact of Sorghum Ergot

Direct Effect on Yield

The ergot pathogen infects unfertilized sorghum flowers preventing seed development. Consequently, ergot directly reduces yield by preventing seed production. The higher the disease incidence in the field (the number of plants with ergot) and the more severe the disease (the number of florets per panicle infected by the ergot pathogen) the greater the impact on final yield. This will in large part be determined by the weather conditions, planting date, hybrid maturity, and the flowering characteristics of the hybrid.

Indirect Effect on Yield

Ergot-colonized sorghum panicles frequently contain seeds coated with honeydew. The honeydew is extremely viscous and can interfere with harvesting operations by gumming-up the combine. Often the combine must be dismantled and cleaned prior to resuming harvest. In a severely infected field this process may have to be performed several times to complete harvest, significantly lengthening harvest. After periods of low relative humidity, the honeydew may be of low viscosity, allowing for a normal harvest. If however, the harvested seed is exposed to high relative humidity during transport or storage, the honeydew becomes viscous again and the seed may clump together and be difficult to handle. With alternate periods of high and low humidity, the seed may be cemented together.

Direct Effects on Grain Quality

Alkaloid production by *Claviceps* species is well known and a quality problem in some grains such as wheat and rye. Research indicates that in the United States the sorghum ergot pathogen, *Claviceps africana*, does not produce significant amounts of toxic alkaloids in the honeydew. No cases of toxicity or feed refusal have been reported in the United States. Consequently, there is no health risk to animals fed grain from ergot-infested fields. Research in Australia has indicated toxicity (feed refusal and pulmonary dysfunction) to swine and poultry fed sorghum with very high levels of ergot. The ergot levels that elicited the toxic effects were higher than what had been naturally encountered.

Indirect Effects on Grain Quality

Many species of fungi (filamentous and yeast-like) and bacteria (*Bacillus* spp. and *Pseudomonas* spp.) naturally colonize the surface or interior tissues of sorghum seed. Some of these organisms are seed-borne pathogens (e.g., *Bipolaris sorghi, Colletotrichum sublineolum*, *Curvularia* spp., *Fusarium* spp.) while others can affect grain quality by direct discoloration (e.g., *Alternaria* spp., *Cladosporium* spp., *Epicoccum* sp., *Fusarium* spp.) of the seed or by the production of toxic compounds, i.e. mycotoxins (e.g., *Alternaria* spp.). Ergot can increase the incidence and severity of these grain molds. The honeydew produced subsequent to infection and exuded onto the panicles contains not only the macroconidia of *C. africana* but also high concentrations of sucrose. This sucrose serves as a readily utilizable energy source for many species of bacteria and fungi (both filamentous and yeast). A common sight in ergot infested fields after periods of high relative humidity are the discolored panicles *(Figure 4)*. The most common secondary colonizers of ergot honeydew are filamentous fungi (*Alternaria* spp., *Cladosporium* spp., and *Cerebella* sp.), yeasts, and bacteria (*Pseudomonas* spp.). Whether the honeydew provides conditions that change the species composition of sorghum seed or alter the relative populations has not been determined. It is possible that the honeydew coating on seed surfaces can provide a protective habitat for not only the *C. africana* macroconidia but also the propagules of other sorghum pathogens and grain mold species.

Long-term Potential Impact for Nebraska

For a pathogen to become endemic (permanent inhabitant) in an area and expand into adjacent areas, many factors need to exist:

- a timely source of viable inoculum,
- effective dispersal mechanism(s),
- spatial and temporal availability of susceptible host tissue, and
- environmental conditions conducive to pathogen survival, dispersal, and disease development.

There are critical questions to answer before predicting the eventual impact to the Nebraska sorghum industry. Chief among them is whether this pathogen can survive harsh Nebraska winters. If the pathogen becomes endemic in Nebraska, then the population may reach levels capable of causing damage to the commercial grain sorghum crop in seasons where the weather conditions favor disease development. However, if this pathogen only survives the winter in Texas and disease in Nebraska is dependent upon inoculum dispersed from Texas, then in normal years the amount of disease in Nebraska will not likely be high enough to cause significant effects on either yield or grain quality. If the pathogen arrives in Nebraska late in the season, heads on the main stalk of commercial grain sorghum will have been fertilized. Since this pathogen only infects through unfertilized florets, the chance for widespread infection and significant damage to grain sorghum will be minimal. Newly developing tillers on grain sorghum plants that still produce florets could become infected if exposed to ergot spores. This could result in some fields in localized areas experiencing observable disease levels. Forage sorghum fields with high levels of male sterility might be at greater risk if tillering is prevalent.

Disease Management

It's likely that different components of the sorghum industry — seed production, grain production and forage — will use different strategies to manage the disease. Successful management will require cooperation among production areas and distribution channels for both seed and grain production. Managing ergot in each sorghum-growing area may involve the following management options:

Pollen Management

Weather-based decision tools are under development to help within-season management decisions. Night temperatures lower than 53°F to 60°F three to five weeks before flowering and at the time of pollination will increase the risk of ergot. This is primarily due to lowered fertilization of flowers as a result of the effects of low temperatures on pollen production and pollen viability.

Cropping Sequence

The sorghum ergot pathogen does not infect crop plants other than sorghum and certain sorghum-sudan hybrids grown as crops. The sorghum ergot pathogen does infect and cause disease in Johnsongrass, shattercane, and other uncultivated sorghum species and sudan grasses *(Figure 5)*. These alternate hosts can serve as a survival reservoir for the pathogen in the absence of a flowering sorghum crop and then serve as a source of inoculum (both macrocondia and secondary conidia) for sorghum at the time of flowering. Consequently, these weed species need to be managed within and adjacent to sorghum fields. Rotation to non-host crops may reduce the risk of ergot disease, but it will not be effective as the sole disease management practice.

Prior Ergot Epidemic

At present it is not known how far north in the Great Plains the ergot pathogen will overwinter. Until the survival capability of the pathogen is determined the risk from prior epidemics is unknown. If the source of ergot inoculum for epidemics in the Northern Great Plains is in the Southern Great Plains, the incidence and severity of ergot in Texas over the previous year may indicate the potential for ergot north of there.

Figure 5. Left, *Claviceps africana* infects and causes disease in Johnson grass (*Sorghum halapense* L.) and right, shattercane (*S. bicolor*) resulting in similar symptoms as to those in cultivated sorghum. The honeydew from these alternate hosts contains macroconidia capable of infecting cultivated sorghum.

Tillage

The effect of tillage on the pathogen population has not been determined in the Northern Great Plains. Honeydew that drips onto leaves, soil, or seed contains macroconidia of the pathogen that can germinate to produce secondary conidia for aerial dispersal *(Figure 6)*. The survival of macroconidia on seed, leaf debris, on the soil surface, or buried in soil has not been quantified under environmental conditions typical for Nebraska. Sphacelia and sclerotia left in the field as a result of the harvest operation may serve as a source of inoculum if they can survive the period between flowering sorghum crops. In Africa, tillage to bury plant tissues containing the pathogen was not effective in reducing ergot in the next sorghum crop. This was presumably due to the rapid dispersal of secondary conidia from small amounts of primary inoculum.

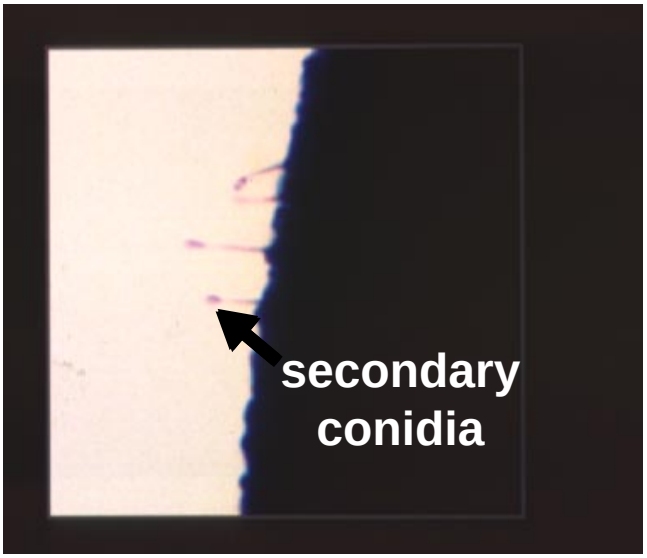

Figure 6. Macroconidia on honeydew-coated seed can germinate to produce secondary conidia available for aerial dispersal. The survival dynamics of macroconidia on seed surfaces are not known.

Hybrid Susceptibility

The ergot pathogen infects through unfertilized florets competing with pollen for infection sites. Anything that interferes with pollination (i.e., pollen production, pollen viability and vigor, or stigma receptivity) may lead to increased ergot infection. Hybrids vary greatly in flowering dynamics and sensitivity to stresses that impair pollination; e.g., sensitivity to low night temperature effects on pollen production and viability. The less synchronous the flowering dynamics across a field the longer the window of vulnerability to ergot for each hybrid. Hybrids with the greatest potential for full fertilization of the panicle and least sensitivity to pollination stresses should be selected.

Fungicide Application

Under high disease pressure in seed production fields, timely fungicide applications can greatly minimize impact on yield; however, these applications are costly and usually only practical for seed production.

Unresolved Issues for Sorghum Ergot

Among the unresolved issues for managing sorghum ergot are the following:

1) how far north the ergot pathogen will overwinter,
2) the mechanisms and dynamics of dispersal of the ergot pathogen,
3) the importance of planting date, hybrid maturity, and plant population density to the incidence and severity of ergot, and
4) the role of alternate hosts in the life cycle of the ergot pathogen in the Northern Great Plains.

Relevant Web Sites

Several Web sites can provide additional information about sorghum ergot including images of symptoms. Additional Web sites allow monitoring of the weather in any Nebraska county.

Sorghum Ergot Sites:

American Phytopathological Society, http://www.scisoc.org/feature/ergot/top.html
Texas A&M University publications, http://agpublications.tamu.edu
USDA sorghum information, http://www.ars.grin.gov/ars/SoAtlantic/Mayaguez/sorghumnews.html
Purdue University, Plant Pathology, http://www.btny.purdue.edu/Extension/Pathology/

Weather Sites:

High Plains Climate Center, http://hpccsun.unl.edu/
National Oceanic and Atmospheric Administration, http://www.crh.noaa.gov/