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Ergot: A New Disease Threat to Sorghum in the Americas and Australia

Sorghum, *Sorghum bicolor* (L.) Moench, is the world's fifth most important cereal crop, cultivated on about 45 million hectares for food, feed, beverage, and fodder. The most significant technological change since the 1960s has been the development and use of F_1 hybrid seed (14), which has lead to a dramatic improvement in the crop's productivity. Sorghum cultivation in intensive, commercialized systems where yields average 3 to 5 t ha^{-1} relies almost totally on hybrid seed. In contrast, yields vary widely and average less than 1 t ha^{-1} in low-input production systems.

Ergot is a serious disease that affects the production of F_1 hybrid seed. Ergot is particularly severe in male-sterile lines (A-lines) when either nonsynchronous flowering of A-line and restorer lines (R-lines) or adverse environmental conditions result in a lack of viable pollen and delayed seed set. In India, losses of 10 to 80% have been reported in hybrid seed production fields. Similarly, ergot epiphytotics in Zimbabwe result in regular annual losses of 12 to 25% and occasionally in total losses. Hybrid seed losses in seed production fields can be offset by the application of fungicides to panicles, but this is an uneconomic control measure in most other sorghum production situations, and possible fungicide residues in grain complicate its use. Under normal conditions, the disease is of little consequence in well-adapted male-fertile cultivars, but widespread damage to male-fertile cultivars subjected to unfavorable weather conditions in farmers' fields has been documented (4).

Sorghum ergot affects ovaries. Instead of normal pollination, fertilization, and production of seed, ovaries are colonized by fungal hyphae that develop into spore-bearing fungal masses (sphacelia). Less obvious but still substantial losses in seed quality occur when honeydew oozing from infected florets contaminates surrounding grains, which are then colonized by fungal saprophytes. Such seed may have decreased germination and seedling emergence and may be predisposed to other diseases (33). Infected late tillers or side branches may continue to produce honeydew when the main crop is mature, making combine harvesting difficult. Since the presence of dried honeydew, ergot sclerotia, or sphacelia on seeds increases the risk of disease transmission, contaminated seed may require further sanitation. Dried honeydew on seeds also interferes with the application of chemical seed dressing.

Ergot, therefore, reduces seed yields both directly and indirectly and increases seed production costs through such alterations in seed production practices as chemical applications, reduction in number of seed-producing A-lines to accommodate more R-lines, modifications in harvesting, threshing, and seed handling, and sanitation. However, none of these practices increases seed quality to that normally achieved in the absence of ergot. Because ergot is a disease of quarantine importance, international seed trade has been complicated by the recent sudden changes in the global distribution of the disease.

Expanding Geographical Distribution and Its Implications

In Asia, sorghum ergot, caused by *Claviceps sorghi*, was first observed in India in 1915 (38). In Africa, the disease was recognized first in Kenya in 1924. It is now widely distributed in eastern, western, and southern Africa (11,12) (Fig. 1). In Africa, with the first description of the teleomorph in 1991, the pathogen was recognized as a distinct species, *Claviceps africana*, Frederickson, Mantle, & de Milliano (18).

In 1988, *C. africana* was identified as the cause of ergot in Thailand (8,18). The identity of the pathogen reported in Taiwan in 1991 (10) is uncertain; in Japan, one of the two ergot species is *C. africana* (29). The second pathogen represents yet another distinct species, known currently as *Claviceps* species (T. Tsukiboshi, National Grassland Research Institute, Nishinasuno, Japan, *personal communication*).

The sudden, widespread appearance of ergot in both Brazil and Australia, and eventually throughout the Americas in recent years, demonstrates the potential impact of *C. africana* on the sorghum industry worldwide. In early 1995, sorghum ergot was noticed for the first time outside Asia and Africa when a widespread epidemic of the disease occurred in Brazil (42). Since then, ergot diffusion in South, Central, and North America and the Carib-

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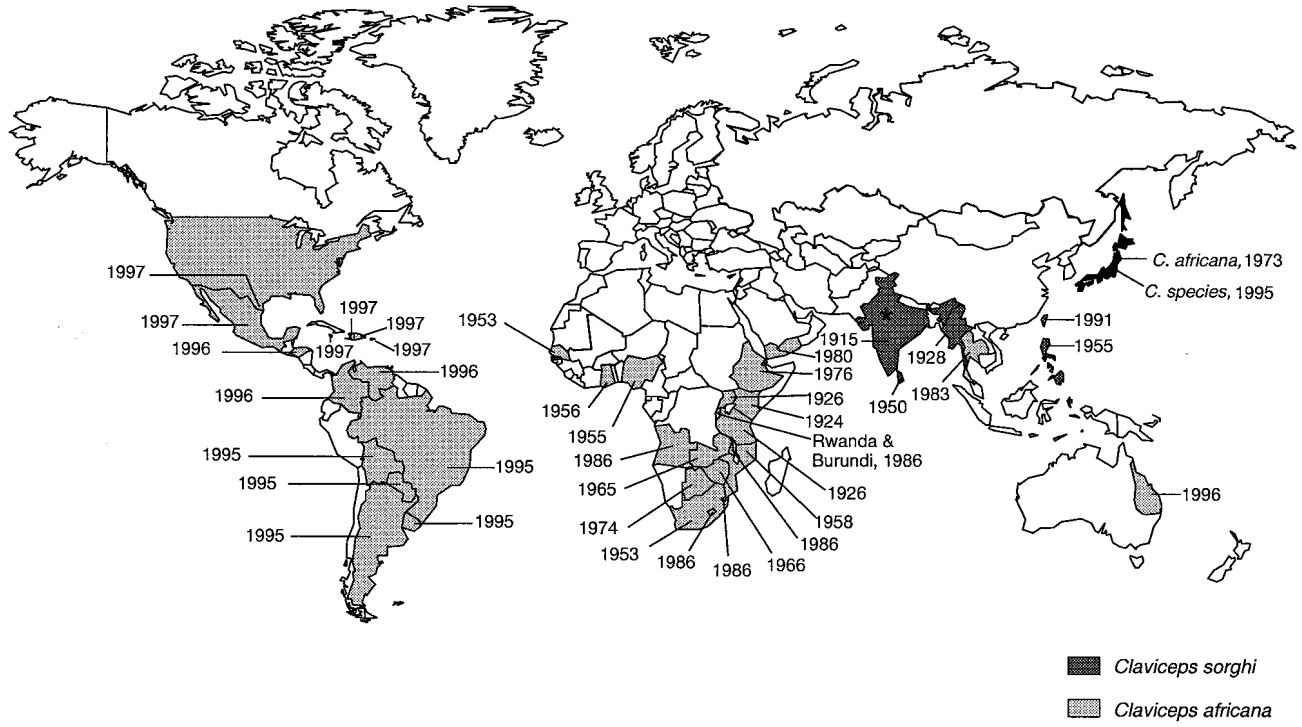

Fig. 1. Distribution of sorghum ergot and its pathogens worldwide with the year of first report of the pathogens in each country. The asterisk (*) indicates the country (India) where the disease was first reported.

bean has been monitored, given the high volume and frequency of seed exchange in the region for both research and trade. By mid-1996, the disease was recorded in Argentina, Bolivia, Paraguay, and Uruguay; by the end of 1996 in Colombia, Venezuela, and Honduras; and during the first quarter of 1997 in Puerto Rico, Haiti, the Dominican Republic, Jamaica, and Mexico. In late March 1997, ergot was observed on ratooned tillers in a sorghum field just north of the Rio Grande near Progresso, Texas. By October 1997, the disease had spread throughout Texas and was recorded in Georgia, Kansas, and Nebraska. The greatest threat from ergot is in commercial hybrid seed production areas, where the disease will not only make hybrid production difficult, but may also result in restricted distribution of the resultant seed. The commercial sorghum production areas of the northern Great Plains states of Kansas, Nebraska, and South Dakota might also be vulnerable because sorghum could be predisposed to ergot as it blooms and matures in increasingly cooler temperatures.

In Australia, *C. africana* was seen for the first time at Gatton in southern Queensland on 26 April 1996 (44). Immediately, a domestic quarantine was put in place, which was revoked when the disease was confirmed across the state in a matter of weeks. The Australian situation exemplifies that there is no zero-risk for disease introductions despite a strict and efficient quarantine. The disease had a significant impact on sorghum seed parent nurseries and has the potential to cause significant losses of irreplaceable breeding material. It was estimated that fungicidal control alone would cost approximately A$700 ha^{-1} (M. Ryley, R. Henzell, B. Boucher, D. Weier, and J. Obst, *unpublished data*). Costs related to seed processing would add another A$250 t^{-1} of seed. Adequate and reliable supply of good-quality seed will probably be jeopardized. Ergot will cost the seed industry A$4 million annually, and an additional A$20 will be required to produce each 25 kg of seed. Most farmers have lost confidence in the crop due to the lack of adequate information about actual losses and the perceived threat to the feed industry and export markets. The disease will also restrict such cropping practices as flexibility in sowing dates and ratooning (M. Ryley, R. Henzell, B. Boucher, D. Weier, and J. Obst, *unpublished data*).

Symptoms

Ergot only attacks unfertilized ovaries. A few or all ovaries within a panicle are individually infected. There are two obvious signs of the disease in the field. The earliest is honeydew oozing from infected florets. Honeydew is a thin or viscous, sweet, sticky fluid containing the sphacelial conidia. Newly formed honeydew droplets are colorless and transparent but become progressively opaque (Fig. 2). With time, honeydew may become uniformly yellow-brown to pink or superficially white. It may remain as intact droplets or may be so plentiful that it drips onto uninfected florets, seeds, leaves, and the ground below the panicle. When the relative humidity (RH) is high, honeydew droplets develop an opaque white, scum-like covering as secondary conidia are produced. Panicles thus infected are obvious, even from a distance, particularly if honeydew has spread over the panicle (Fig. 3). Infected panicles may also be highlighted by the growth of *Cerebella* sp., a

Fig 2. Light brown honeydew exuding from sorghum flowers infected by *Sphacelia sorghi*.

saprophytic fungus that forms a black growth covering the infected florets (Fig. 4).

The second sign of ergot is the presence of fungal sphacelia or sclerotia between the glumes of infected florets. In *C. africana* infections, a white to gray, compact cushion of mycelium (sphacelium) is noticeable on close scrutiny, usually a day before honeydew is produced (Fig. 5). The hard-textured sclerotia that develop later from the sphacelia rarely protrude for more than a few millimeters beyond the glumes (Fig. 6A). In contrast, cylindrical sclerotia of *C. sorghi* may protrude as much as 1.5 cm beyond the glumes (Fig. 6B).

Causal Organisms

Anamorph. The anamorph of the three *Claviceps* species that infect sorghum in different areas is *Sphacelia sorghi*. The pathogens produce three types of single-celled, hyaline spores: oblong to oval macroconidia, spherical microconidia, and pear-shaped secondary conidia.

Secondary conidiogenesis is unique because of the rapidity and the manner in which it occurs in the honeydew (5,17). Formation of secondary conidia results from iterative germination (i.e., germination of one spore to directly produce another spore) of macroconidia on the surface of thin honeydew when RH is high (Fig. 7). Macroconidia inside the viscous honeydew do not germinate because of the high osmotic potential generated by the sugars in the honeydew matrix or because of the inhibitory nature of the sugars themselves (30). However, water absorbed from rain or dew by hygroscopic honeydew lowers its osmotic potential or dilutes the inhibitory sugars and renders it thin. As a result, under humid conditions, macroconidia on the honeydew surface germinate iteratively by extending germ tubes outside the honeydew surface. These germ tubes function as sterigmata, ending in apical secondary conidia that are easily detached and disseminated by wind. Macroconidia in the outer layer of the honeydew also germinate to produce thick, branched or unbranched germ tubes that enmesh to form a firm hyphal mat that provides a stable surface on the otherwise fluid honeydew.

Teleomorphs. The stromatal initials of *C. africana* appear as pale, globose proliferation of the sclerotium (18). Fully extended stipes measure 8 to 15 × 0.3 to 0.6 mm and are colored purple adjacent to the capitulum. Capitula (0.5 to 1.3 mm) are subglobose and intensely purple (Fig. 8A). Perithecia measure 86 to 135 × 123 to 226 µm, with mature asci in situ 140 × 3 to 4 µm containing eight ascospores each up to 45 × 0.8 to 1.2 µm. The two to three stromata of *C. sorghi* (18,24,45) consist of stipes of a burnished bronze/deep terra-cotta color. Capitula, 0.7 mm in diameter, are buff colored, but with darker, papillate perithecial ostioles; the stipe insertion point may be surrounded by a white frill (Fig. 8B). Perithecia are 132.8 to 232.4 × 66.4 to 124.5 µm. The cylindrical asci (56 to 112 × 2.4 to 3.2 µm) have tapering ends and hyaline apical caps; the eight filiform ascospores measure 40 to 85 × 0.4 to 0.8 µm. The stromata of the Japanese *Claviceps* species consist of a buff to terra-cotta stipe and terra-cotta colored capitulum. The long ascospores measure 92.5 to 205 µm (T. Tsukiboshi, *personal communication*).

Germination of sclerotia. Most attempts to germinate *C. africana* sclerotia have proved futile across a wide range of conditions (18,28), but after 1 year in dry storage at 20 to 25°C, 10% of the sclerotia

Fig. 3. White coating of secondary conidia over a honeydew-smeared sorghum panicle infected by *Sphacelia sorghi*.

Fig. 4. Black, convoluted mass of the saprophyte *Cerebella* species associated with sphacelia of *Sphacelia sorghi* in a sorghum panicle.

Fig. 5. An infected sorghum ovary (left) converted into a fungal mass (sphacelium) of *Sphacelia sorghi* 6 days after infection. A healthy ovary is on the right.

from Matopos, Zimbabwe, germinated after 4 weeks at a depth of 3 cm in moist soil. However, only a few differentiating stipes and capitula reached maturity (18). In India, *C. sorghi* sclerotia germinated consistently on the soil surface at 27°C, with 55% of stromata differentiating fully (45). Germination occurred at 5 to 50°C, the optimum range being 20 to 30°C. Sclerotia of the Japanese *Claviceps* species germinate readily on moist sand under diffused light at 25°C and also in the field (T. Tsukiboshi, personal communication).

Diagnostic differences among the pathogens. The three species of sorghum-infecting *Claviceps* pathogens are distinguished mainly by differences in morphology of ascostromata and sclerotia, and by alkaloid production. From traditional mycological principles, the color of stipe and capitulum and the perithecial dimensions are the most critical determinants. The sclerotia of *C. africana* are spherical and largely confined within the host glumes, whereas *C. sorghi* sclerotia are thin, elongate, and protruding; and sclerotia of the Japanese *Claviceps* species are conical, elongate, and purple-black. The alkaloid dihydroergosine is synthesized in *C. africana* sclerotia (31), and the Japanese *Claviceps* species may contain small amounts of the alkaloid paliclavine, but *C. sorghi* sclerotia do not synthesize alkaloids. Under natural conditions, secondary conidiation is a common feature of *C. africana* and is occasionally observed in *C. sorghi* but has never been seen naturally on honeydew of the Japanese species. There is a need to re-examine the taxonomy and variation of the *Claviceps* species that infect sorghum based on a large number of isolates from different geographic areas in order to classify genetic relationships among them.

Toxicity to Animals

The question of the toxicity of alkaloids in sclerotia inevitably arises by analogy to the potent ergotamine alkaloids produced by *Claviceps purpurea* and agroclavine produced by *C. fusiformis* (26,46,54). The potential toxicity of an alkaloid similar to paliclavine, found in small amounts in the sclerotia of *Claviceps* spp., has not been investigated (T. Tsukiboshi, *personal communication*). The alkaloid content of *C. africana* sclerotia varies between 0.02 and 0.98% wt/wt, the unique dihydroergosine accounting for 88%. Diets of up to 50% sclerotia of *C. africana* (15 mg of alkaloid per day) tested on mice were harmless and did not induce clinical effects (27). In conjunction with the lack of reports of toxicity to livestock in Africa and Asia, the impact of sorghum ergot on livestock was expected to be low. However, in mid-1997 in central Queensland, several piggeries reported severe feed refusal and loss of litters due to failure of milk production by sows when fed diets based on grain

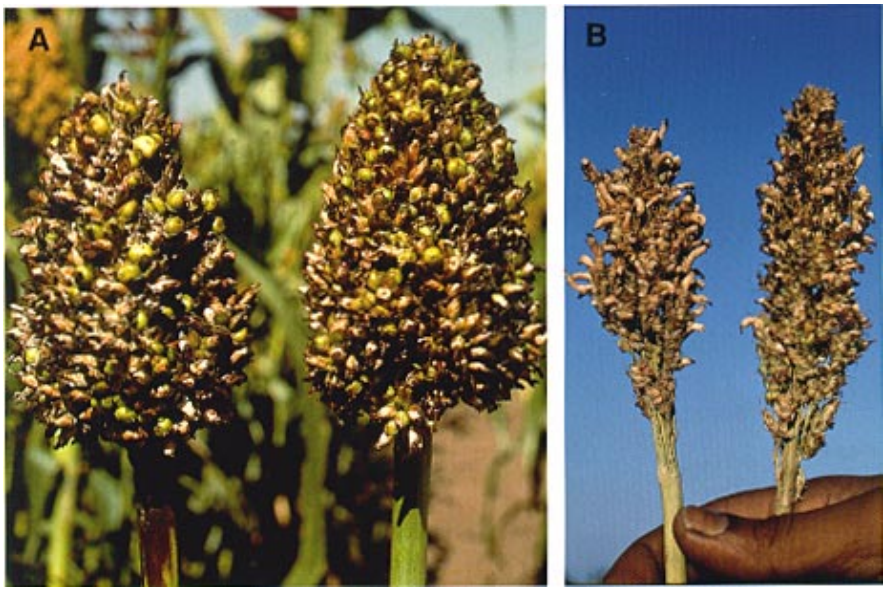

Fig. 6. Ergot sclerotia in sorghum panicles. (A) *Claviceps africana*, and (B) *Claviceps sorghi*.

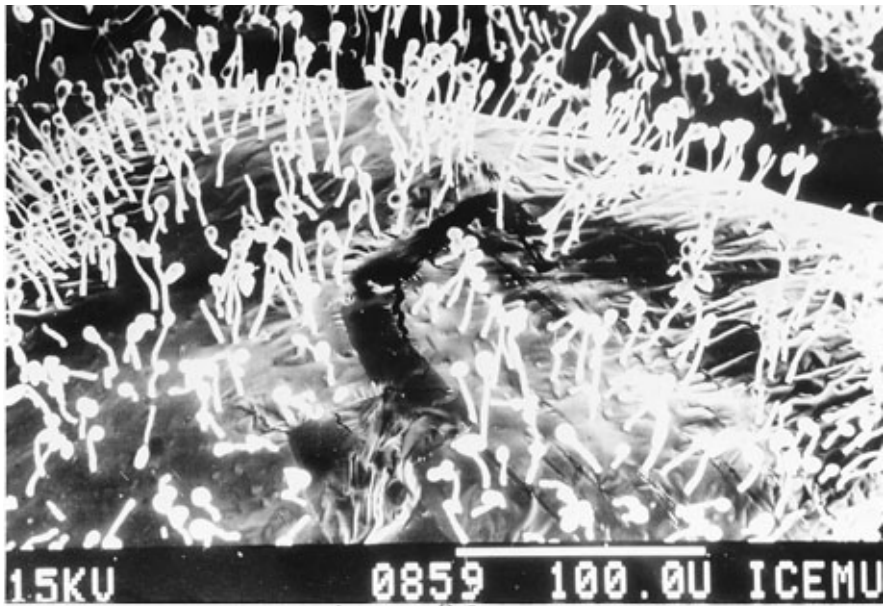

Fig. 7. Scanning electron micrograph showing secondary conidia of *Sphacelia sorghi* borne outside the honeydew at the apex of sterigmata that originated from macroconidia (not shown) embedded in the honeydew.

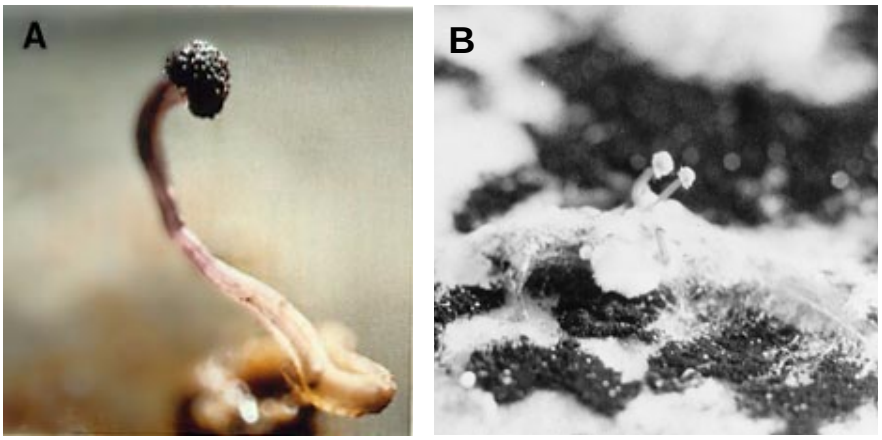

Fig. 8. Sexually germinated ergot sclerotia from sorghum panicles. (A) *Claviceps africana*, and (B) *Claviceps sorghi*.

containing 1 to 20% sclerotia (B. J. Blaney, *personal communication*). Crops from which the contaminated grain were harvested flowered when minimum temperatures were less than 13°C, which severely reduced pollination. Experimentally, pigs (20-kg body weight) fed on diets containing 1 to 5% sclerotia for 28 days showed a major reduction in blood prolactin concentrations, confirming the potential for agalactia in sows. Pigs fed with 5% sclerotia exhibited a reduction in feed intake and weight gain but no other ill effects (B. J. Blaney, Queensland Department of Primary Industries, Australia, *personal communication*). At the same time as the pig cases, several dairies reported drops in milk production by cows supplemented with grain containing up to 20% sclerotia, but the extent of this problem is less clear. Toxic effects of alkaloids were noticed in chickens fed with diets containing 2.5 and 5.0% sclerotia in feed. The chickens exhibited respiratory difficulties and diarrhea, and died. However, with diets containing 1.25% sclerotia, no significant clinical effects were seen (B. J. Blaney, *personal communication*). Feeding experiments need to be repeated and verified using sclerotia of isolates from Africa, Australia, and the Americas.

Path of Infection

All ergot diseases are organ-specific. The infection pathways of *C. africana* and *C. sorghi* on sorghum are similar, but the timing of infection events differs (3,16). The stigma is the principal site of infection, although conidia can germinate and infect through the style and ovary wall. Conidia germinate on the stigma after 16 to 24 h, producing one to four germ tubes, which penetrate the stigmatic papillae, grow intercellularly through the stigmatic hairs, and progress as infectious hyphae through the transmitting cells of the style to the ovary top (both pathogens, 3 days). The inner ovary wall tissues adjacent to the ovule are invaded next (4 days, *C. sorghi*), and infection progresses to the vascular bundles within the rachilla (5 days, *C. sorghi*). Only upon contact with the vascular bundles does the fungus commence acropetal colonization of the outer ovary wall tissues and ovule (4 days, *C. africana*; 6 days, *C. sorghi*), with hyphae simultaneously emerging onto the exterior surface. The entire ovary, apart from the apical region, is replaced by a sphacelium (6 to 8 days, *C. africana*; 8 to 10 days, *C. sorghi*). Initially, sphacelia secrete a clear liquid (7 to 8 days, *C. africana*; 9 to 11 days, *C. sorghi*), which becomes more opaque upon production and release of macroconidia originating from the short conidiophores at the surface of sphacelia. In time, sphacelia harden as they are converted into the more resilient sclerotial tissues. During the invasion, the fungi retain contact with the vascular bundles in the rachilla without destroying them, and thus continued nutrient transport supports the sphacelium and honeydew production.

Pollination, Fertilization, and Infection

Flowering behavior is one of many factors affecting disease escape, since ergot pathogens only infect and colonize unfertilized gynoecia. Effective pollination and fertilization enable pistils to escape or resist infection (22,39,45). There is a strong correlation (r = 0.87) between the percentage of nonpollinated florets and ergot infection. Rapid anthesis lowers the chance of infection (4), and any factor prolonging the period from floret opening to fertilization will promote ergot infection (22). When pollen and conidia are placed concurrently on the stigma, pollen germinates within 30 min, and fertilization occurs within another 2 to 12 h. In contrast, conidia require 8 to 24 h for germination and several days to reach the base of the ovary. Pollen tubes, therefore, reach the embryo sac faster than the colonizing hyphae (7). However, the times for these events vary among florets and genotypes and are strongly influenced by the environment, self incompatibility (Fig. 9), and other floral characteristics. For example, anthers dehisce early in the morning, but dehiscence, and therefore pollination and fertilization rates, may be delayed for several hours by damp weather. Protogyny and cleistogamy are floral characteristics that affect ergot severity (20). Protogynous genotypes are highly susceptible, since stigmas emerge from glumes and remain exposed for at least 72 h before anthesis, while cleistogamous genotypes are pollinated before the glumes open and thus escape infection. Such other host characters as stigma receptivity, stigma size, and the amount of pollen produced may influence the rate of pollination and concomitant exposure of the pistil to the pathogen.

In pollinated and fertilized pistils, the fungus grows slowly or fails to grow, thereby reducing the ergot severity. In a few cases, the infection hyphae and pollen

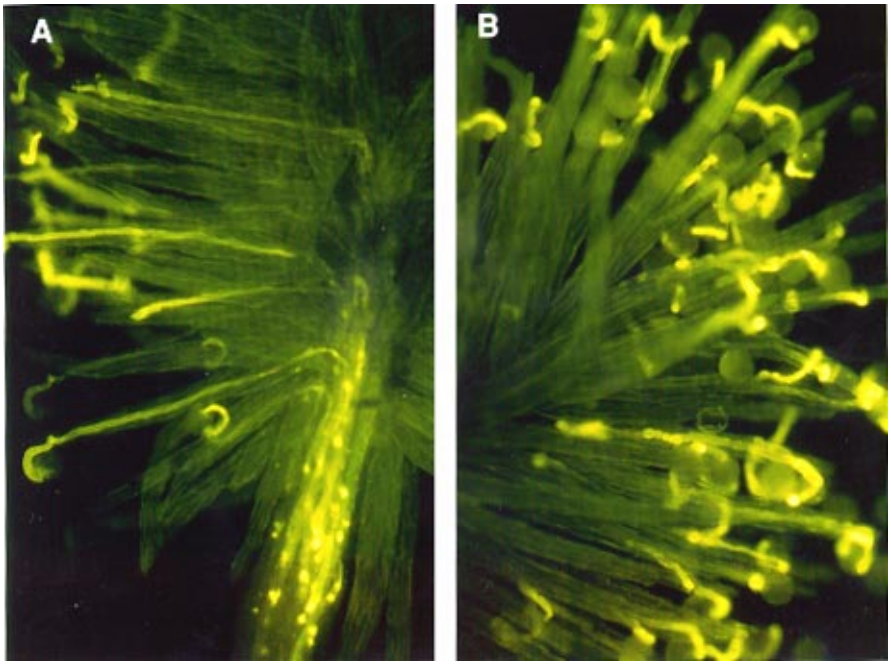

Fig. 9. Sorghum pollen germination on stigma 0.5 h after pollen deposition. (A) Efficient pollen germination and rapid pollen tube growth in a compatible reaction that leads to ergot escape. (B) Inefficient pollen germination and aborted pollen tube in an incompatible reaction that leads to ergot susceptibility.

Fig. 10. A fungal mass (sphacelium) of *Sphacelia sorghi* growing at the basal end of a developing sorghum ovary. This shows that *Sphacelia sorghi* can colonize developing grain.

tube grow together up to the ovule, and as a result, both sphacelia and grain develop together in some spikelets (Fig. 10). Most spikelets inoculated with conidia 1 to 4 days after anthesis are fertilized before infection can occur, but about 8% of the pollinated ovaries are not fertilized and thus remain susceptible. A concomitant level of 4 to 10% ergot infection occurs (Table 1). Therefore, fertilization, rather than pollination efficiency, is effective in ergot control.

Low-Temperature Predisposition and Disease

Night temperatures of 13°C or less during meiosis can induce male sterility in sorghum (9). The late archesporial cell–pollen mother cell development period to the leptotene stage of meiosis are the phases most sensitive to temperature. This critical stage occurs 2 to 3 weeks before anthesis (9).

Low temperature has a strong predisposing effect on ergot susceptibility through its indirect influence on pollen activity. For example, of the 70 lines that failed to develop the disease at Potchefstroom in the warmer North West Province of South Africa, all except three were susceptible when screened at Bethlehem, a cooler locality in the Free State. This was attributed to the cooler conditions affecting the flowering pattern and the pollination efficacy of sorghum genotypes (32). Average minimum temperatures of less than 12°C 23 to 27 days before flowering reduce pollen viability, reduce seed set, and predispose genotypes to infection (37). This critical temperature varies with genotypes that display a linear or quantitative sterility response to preflowering cold stress. Lines whose pollen remain viable at low temperature are most resistant to infection (35).

Epidemiology

Environment and disease. Cool (19 ± 1°C), wet, cloudy weather during floret opening and from the onset of anthesis to fertilization favors the rapid development of ergot (2,36). Ergot severity decreases with increasing temperature and is negligible at temperatures higher than 28 to 30°C (36). Near 100% RH for 24 h during anthesis is optimal for infection (23). When humidity is high, wetness or rainfall is not essential for ergot development. Cloudiness during anthesis aids disease development, probably due to delayed anther dehiscence and pollen deposition and activity (40). Moderate temperatures (14 to 28°C) combined with high RH (>90%) are conducive to conidial production and therefore pathogen spread, but not to the differentiation of sphacelia into sclerotia (5). In contrast, at 28 to 35°C and RH less than 90%, sphacelia develop into sclerotia, but honeydew and spore production are suppressed.

The environment affects ergot development indirectly by influencing the growth of saprophytes on developing sclerotia. Sclerotia develop poorly in the rainy season due to the growth of *Cerebella* sp. and other mold fungi that suppress their development; whereas dry weather after infection allows the formation of less-contaminated, mature sclerotia (5,23).

Dispersal of conidia. Infected spikelets exude sweet, sticky drops of fluid honeydew containing macroconidia and microconidia. Although insect transmission of the conidia of *Claviceps purpurea* is known (25), insects may not play a significant role in spreading sorghum ergot. Insects such as thrips, *Orius* species, beetles, midge flies, head bugs, and Hymenopterous insects can carry conidia attached to their bodies, but when such insects were released in cages containing susceptible panicles, they failed to transmit the pathogen (6). Spread of *C. sorghi* by water splash has been confirmed in field trials (6).

Wind dissemination of secondary conidia is the most important mode of dispersal for local and long-distance spread of *C. africana* (19) and may explain the rapid, long-distance disease spread in Australia and the Americas. Concentration of secondary conidia in the air shows a diurnal pattern, with the greatest occurrence at nightfall, coincident with the sharp rise in RH and fall in temperature. Dispersal of secondary conidia from even a very small initial infection focus results in a widespread epiphytotic (19). Therefore, even if the first infection focus is discovered, eradicating the disease is impossible because the secondary conidia of *C. africana* are windborne by then. Secondary conidia are also produced on honeydew that drips and falls onto wet soil. Such soilborne secondary conidia can infect plants in the field (6) and may also act as primary inoculum for disease initiation in the field.

Dispersal of honeydew containing conidia occurs during farming and postharvest operations. Honeydew may adhere to farm personnel and equipment, leading to the spread of the pathogen from field to field. Honeydew in contaminated seed lots is easily transferred to equipment and then to healthy seed lots during seed handling and processing. However, the epidemiological significance of seed contaminated with honeydew is unclear.

Disease spread. Experiences with ergot disease in Brazil and Australia illustrate its propensity for rapid, uncontrollable spread. In Brazil, *C. africana* was found over an area of 800,000 km^2 less than 1 month after the initial disease sighting in 1995 (4). Although several hypotheses concerning the means of introduction into these countries were put forward (4), disease dissemination remains poorly understood. An understanding of the mode of introduction of the pathogen may suggest methods to prevent its spread to new areas.

Ergot on sorghum was observed for the first time near Gatton, southern Queensland, on 26 April 1996, on A-lines in breeders' nurseries (Fig. 11). Within a week, the disease was confirmed over an area of approximately 16,000 km^2, and within a month it had expanded to 70,000 km^2 around the locality of the first sighting. On 15 May, ergot was sighted near Mareeba on the Atherton Tableland, in northern Queensland, approximately 1,400 km north of Gatton. Within another month, it was reported at several localities within 80 km of Mareeba. By 19 July, there were recordings from several places on or near the central Queensland coast between the northern and the southern outbreaks. Two

Table 1. Pollination, fertilization, and infection percentages of sorghum spikelets inoculated with *Claviceps sorghi* 1 to 4 days before and after anthesis, and at anthesis

	Spikelets (%)		
Inoculation	**Pollinated[a]**	**Fertilized[a]**	**Infected**
Days before anthesis			
4	91	14	95 ± 2.2
3	100	11	92 ± 2.2
2	96	11	87 ± 2.4
1	75	54	52 ± 2.5
At anthesis	100	77	32 ± 3.7
Days after anthesis			
1	100	83	10 ± 2.6
2	100	97	5 ± 2.8
3	100	91	6 ± 3.2
4	100	98	4 ± 1.9
Noninoculated control	…[b]	…	0
Correlation			
% pollinated spikelets	…	0.292	–0.332
% fertilized spikelets			–0.991**[c]

[a] Based on observation of pollen tube and germ tube growths (under a fluorescence microscope) in softened and squashed tissues of nearly 400 pistils per inoculation treatment.
[b] … = data not taken.
[c] ** = significantly different from 0, $P \leq 0.1$.

weeks later, ergot was found near Lakeland Downs, approximately 140 km north of Mareeba. In February 1997, it was found in the Macquarie Valley in New South Wales, approximately 500 km southwest of the previously known southern limit of sorghum ergot in Australia (not shown in Fig. 11). Secondary conidia have been implicated in spreading *C. africana* over moderate distances in Africa (17) and over long distances in Brazil (4), and may also have been responsible for the widespread dissemination of the pathogen in Australia.

In Australia, there is no conclusive evidence that the pathogen spread over a vast area from a single focus (Gatton) or two foci (Gatton and Mareeba). It is possible that *C. africana* had been in Queensland for a number of years at very low levels, but was undetectable due to a 5-year drought, and had spread slowly from a single source. The weather conditions in southern Queensland before and immediately after the first confirmed recording of *C. africana* were ideal for expression of symptoms and for the spread of the pathogen and may have been responsible for some subsequent recordings around the site of the first outbreak. Later recordings in northern areas were associated with low maximum (<25°C) and minimum (<15°C) daily temperatures 7 to 10 days before the appearance of ergot symptoms.

Host range. Records of collateral hosts of *C. africana* and *C. sorghi* are numerous but inconsistent and include experimentally unverified examples, making the literature confusing and misleading. Cross-inoculation studies have confirmed that other *Sorghum* species and pearl millet were hosts of the sorghum ergot pathogens. In Australia and the Americas, the continual flowering of *Sorghum halepense* is probably sufficient to perpetuate *C. africana* in the absence of cultivated *S. bicolor* or any other collateral host.

Caution is needed, because numerous *Claviceps* species naturally parasitize grasses. It is erroneous to conclude that a grass species at the periphery of a sorghum field is a collateral host just because it bears honeydew unless pathogen characterization and cross-inoculation studies have been made. In this way, *Cynodon dactylon*, *Urochloa brachyura*, *Brachiaria bryzantha*, *Digitaria tenata*, *Chloris guyana*, *Sporobolus pyrimidalis*, *Hyparrhenia* sp., and *Andropogon* sp. were eliminated as collateral hosts of *C. africana* in South Africa, Zambia, and Zimbabwe (15). Comparisons of ergot DNA from grasses, and between the three species attacking sorghum, would clarify host range and pathogen variability.

Disease cycle. During the growing season, infected sorghum flowers exude millions of primary conidia in the sphacelial honeydew that are spread locally by rain-splash (6) and secondary conidia that are wind-disseminated over large distances (17,19), leading to secondary infections (Fig. 12). Several cycles of secondary infection take place if flowering occurs for an extended period. By the end of the growing season, and following dry weather, well-differentiated sclerotia are produced. Although the sclerotium is the key survival propagule for cereal ergots via production of the ascosporogenous stage, its role in the disease cycle of sorghum ergot was established only for *C. sorghi* in India (45). Based on field observations, it was hypothesized that in India, ascospores and conidia of *C. sorghi* could infect collateral hosts that normally flower before sorghum, and that conidia in the honeydew of collateral hosts provide fresh inoculum to initiate primary infection in sorghum (45). Germination of *C. africana* sclerotia to produce the perfect stage was recently achieved (18). The present evidence suggests that the perfect stage of *C. africana* does not play a major role in perpetuating the pathogen compared with collateral hosts, conidia associated with sclerotia, and infected panicles bearing honeydew, sphacelia, or sclerotia. Mature sclerotia and sphacelia also readily become mixed with seeds, and honeydew acts as an external seed contaminant. The next season, conidia from any of these sources may provide the primary inoculum to directly infect sorghum or infect after passage through a collateral host to complete the disease cycle. Sorghum was successfully infected with honeydew stored dry for 9 to 12 months (18,19). Macroconidia and microconidia remain viable for 7 months in dried honeydew on infected panicles that fall to the ground (23). Although the initial infections from this inoculum are few (usually up to 10 sphacelia), the normal infectivity of honeydew is restored following one passage through the host (18,19).

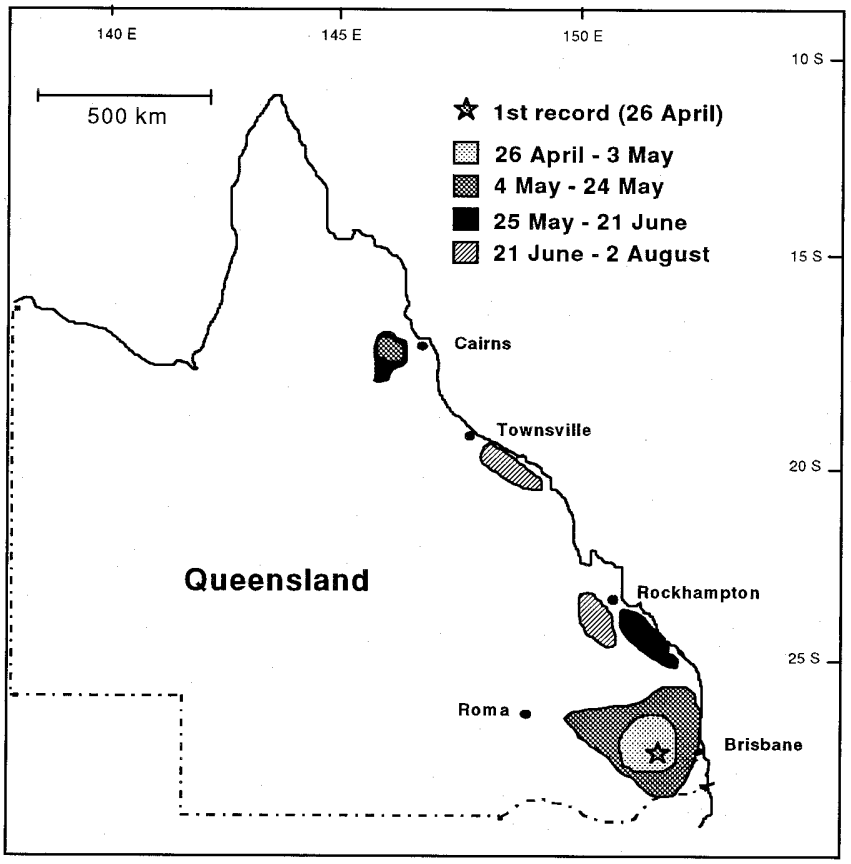

Fig. 11. Progressively increasing areas where ergot was seen on sorghum in Queensland, Australia, during April to August 1996.

Disease Control Options

Plant quarantine and policy. The recent experiences in the United States and Australia have shown that quarantine had limited impact in preventing disease introduction to these continents. Although the means by which *C. africana* arrived in Brazil and Australia is based on speculation, the windborne spread of secondary conidia is by far the most significant means of disease dissemination over such contiguous land masses as South America. Even if seedborne inoculum sources were eliminated, the disease would have spread between countries via the airborne inoculum. There is also potential for introduction through the high frequency of sorghum seed exchange among countries of South and Central America, and to some extent, the United States. The very nature of the hybrid sorghum industry, with seed production in one region and its distribution for sowing at multiple distant loca-

tions, increases the chance of disease spread. The high vulnerability of the male-sterile parent, and possibly the enhanced survival of the pathogen in cool, dry hybrid seed storage conditions, further increase disease risk.

Sclerotia and sphacelia in contaminated seed should not be ignored, and their undetermined role has prompted a few seed companies in Brazil to remove them from harvested seed. Until more sensitive techniques allow the rapid detection of *C. africana* propagules at low levels, close visual inspection of seed from ergot-affected countries will be necessary. With the presence of *C. africana* in all the major sorghum-growing countries, the pathogen has become internationally endemic, bypassing the benefits of quarantine. A total ban on seed imports from infected countries reporting ergot is not insurance against spread of *C. africana* across national boundaries because of the airborne dispersal of secondary conidia.

Cultural methods. Adjustments in sowing date and location to avoid ergot are common practices in many countries. In Zimbabwe, sowing in November may result in disease escape if flowering coincides with a midseason dry spell in January or February. For similar reasons, sowing in early July instead of August in India resulted in fewer *C. sorghi* infections (2). In India, most of the F_1 hybrid seed is multiplied in Andhra Pradesh and Karnataka states under irrigation in the dry season, when conditions do not favor ergot. In Zimbabwe, seed production plots in the hot Muzarabani area usually escape severe ergot infection. However, *C. africana* can infect A-lines virtually wherever they are grown in southern Africa, even in the more arid areas of northern Botswana, provided rain occurs near flowering.

Hybrid producers in Zimbabwe are aware of the potential benefits of good sanitation, e.g., removing infected panicles at harvest, incorporating crop residues into the soil, and sowing fields to sorghum every third year to reduce the inoculum level. Despite these practices, a serious epiphytotic usually occurs every 5 years. This is probably due to the ability of the pathogen to spread rapidly from even a small source of infection (19).

Although the ascosporic stages of *C. sorghi* and *C. africana* have not been con-

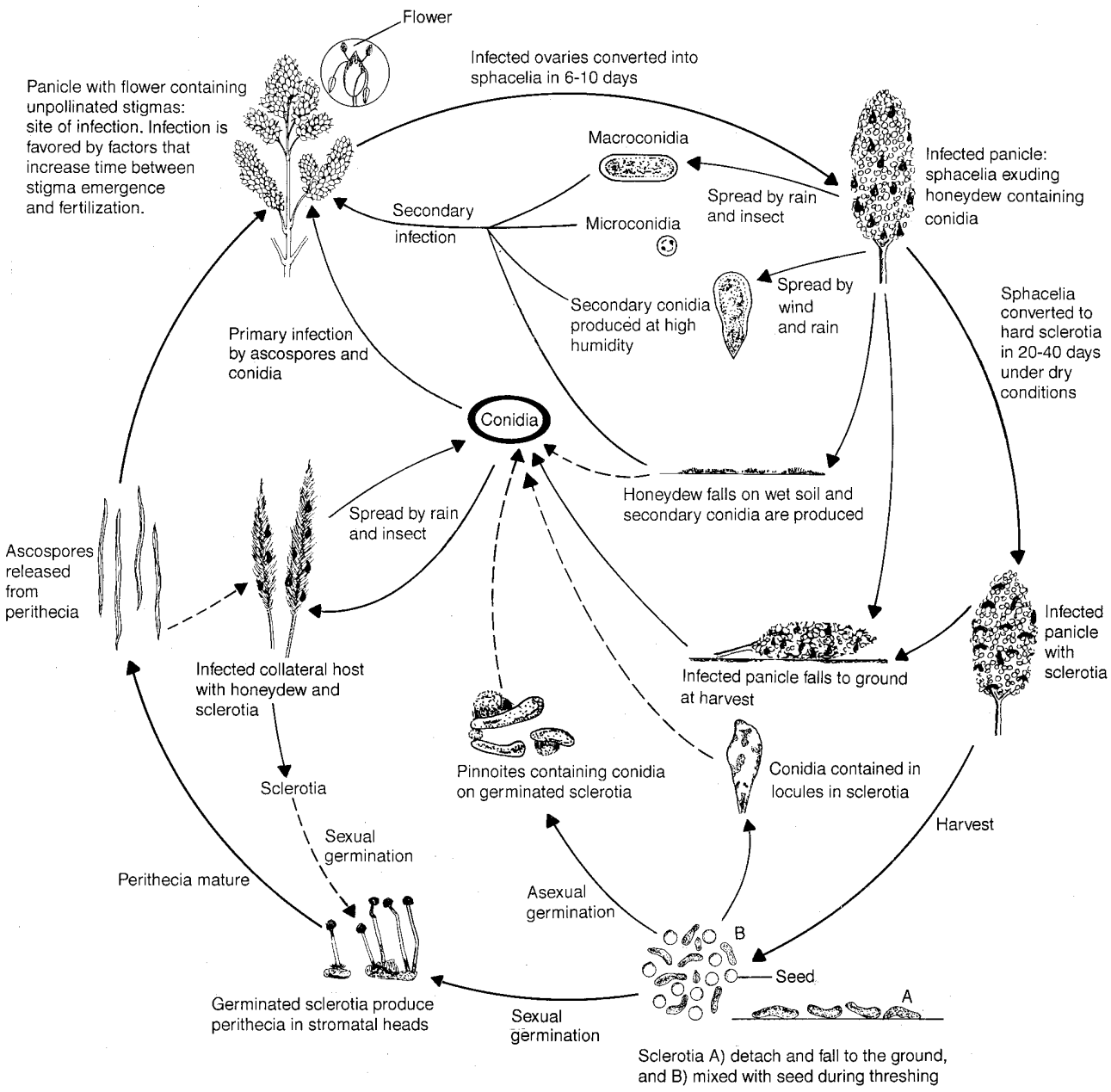

Fig. 12. Verified and unverified (distinguishable by dotted lines) aspects of disease cycle of sorghum ergot.

firmed in nature, sowing sclerotia-free seed will reduce the possibility of introducing the disease into new areas. Large numbers of sclerotia can accumulate in seed lots. It is advisable to remove contaminating sphacelia and sclerotia from seed batches. In India, the tolerance limit for certified seed has been set at 0.04% sclerotia by number (52), apparently without valid scientific evidence. Seed lots containing whole or broken sclerotia are considered contaminated, but those with honeydew lumps on the seeds are not. In Australia, seed from Queensland for hybrid production and breeders' nurseries will be accepted by authorities in Western Australia only if the seed was subjected to flotation in 10% salt solution, which removes 98 to 99% of *C. africana* sclerotia. In India, sclerotia are floated off from seed in 5% salt solution (3). If the seed is subsequently dried rapidly, loss of viability is minimized (33).

Chemical control. Sorghum ergot can be managed successfully in seed production fields and breeders' nurseries by spraying fungicides before anthesis until flowering is complete (1,13,34). However, use of fungicides for ergot control was not economical in South Africa, since the yield gains were not sufficient to offset the cost (34). In Zimbabwe, benomyl at 0.2% a.i. reduced ergot significantly in A-lines if they were sprayed once at heading, and this control schedule was economically feasible. In Brazil, propiconazole (Tilt) and tebuconazole (Folicur) applied in three to four sprays at the rate of 250 g a.i. ha^{-1} for each application at 5- to 7-day intervals have been used successfully by seed companies. Fungicides have little effect after the onset of the disease and are more effective as preventive measures. Control is good if inoculum pressure is low, and in the absence of rains.

Fungicides normally used for ergot control do not have a male gameticidal effect; thus seed set should not be affected. A key issue for fungicidal control is thorough spray coverage of the panicle. Due to the lack of systemic movement of fungicides from plant tissues to the stigma, it is essential that spray droplets wet the stigma (Fig. 13). Inadequate spray equipment and labor to cover large areas are major problems associated with intensive spray regimes. Although fungicides are not economical for small-scale farmers, their use is invaluable for the production of hybrid seed, even though control may not be absolute.

Seed treatment with thiram and captan was recommended, since these fungicides kill conidia on seed encrusted with honeydew (J Dahlberg, Mayaguez, Puerto Rico, *personal communication*). These fungicides can also reduce sclerotial germination (45).

Seed processing. In Brazil and Australia, removing sclerotia from seed during harvesting, threshing, and seed cleaning was successful without needing additional equipment (43). A gravity table, aided by an air stream, separates sclerotia of lower specific gravity than seeds. Seed stickiness due to contamination by honeydew results in aggregation of seed, making mechanized sowing difficult. Honeydew contamination was associated with reduced seedling vigor and predisposition of seedlings to infection by soilborne pathogens (33). Washing seed with large volumes of water reduces honeydew concentrations, increasing germination and seedling development. Seed washing and drying systems have been installed by South African seed companies.

Pollen-based management. Poor synchronization of flowering in A- and R-lines (i.e., nicking) and lack of viable pollen during stigma emergence are the main reasons for the increased susceptibility of A-lines in seed production plots. Efficient pollination reduces the length of the susceptible period between stigma emergence and fertilization in A-lines. Control of ergot in pearl millet was achieved by efficient pollen management systems in the field (51). In Zimbabwe, sorghum farmers routinely stagger the sowing of the pollen-donating R-lines to increase the period of pollen availability. One row of R-line is sown simultaneously with the A-lines, followed by the second row after another 7 to 10 days. Some farmers have increased the number of R-line rows from the usual two to four and reduced the number of A-line rows from six to four; this practice enhanced seed set and reduced rate of disease increase (21). The overriding effect of environment on pollen activity limits pollen management as a control method, particularly in ergot-favorable conditions.

Host plant resistance. There is no consistent evidence of the existence of physiological resistance to *Claviceps* species in any crop (53). Resistance to sorghum ergot is related to the ability of plants to pollinate and fertilize rapidly before infection can occur. In essence, this is acquired resistance that is strongly linked to a postfertilization mechanism(s). Therefore, most research in sorghum has dealt with identifying plants with traits that support acquired resistance.

Artificial inoculation methods that can generate ergot epidemics reliably have been developed and have been used to identify sources of resistance (39,47). Ergot incidence in sorghum is extremely sensitive to pre- and postflowering climatic conditions (32,36). Since genotype by environment interaction is significant for disease expression, it is necessary to qualify resistance in specific lines with respect to the limits of environmental conditions in which it is operable (20,32).

Resistance is feasible in male-fertile varieties, R-lines, and F_1 hybrids. McLaren (*unpublished*) found that under controlled inoculation conditions, flowering and the concomitant ergot-susceptible period in the sorghum line SA 858 lasted for 5 days, as opposed to 8 days in CK60B (Fig. 14). This means that the risk period was 37% shorter in SA 858 than in CK60B. Selection for a shorter ergot-susceptible period can contribute to integrated ergot risk reduction. Traits that contribute to a shorter ergot-susceptible period are protandry, a fast rate of flower opening, production of abundant functional pollen, efficient self-pollination, rapid fertilization, and a short period of stigma receptivity. Thus, many minor floral characteristics could, if selected for, contribute to plants that acquire resistance rapidly. These traits, to be meaningful, must be manifested in resis-

Fig. 13. Fungicide spraying in an F_1 hybrid seed production field of sorghum in Brazil. Note the arrangement of three spray nozzles to provide adequate spray coverage to the panicle.

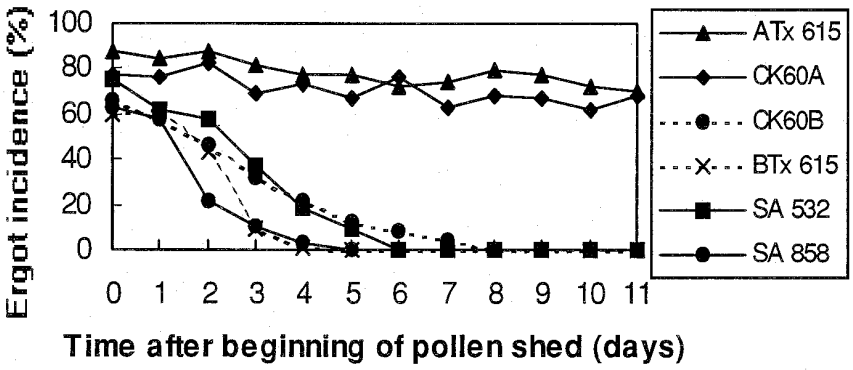

Fig. 14. Ergot incidence in male-sterile (ATx 615 and CK60A) and male-fertile (CK60B, BTx 615, SA 532, and SA 858) sorghum lines inoculated with conidia of *Sphacelia sorghi* at different times after pollen shed.

tant genotypes under such ergot-favorable conditions as low temperature. Selection for tolerance to cold-induced sterility can contribute to acquired resistance. The hybrid PAN8564, which is more cold-tolerant than other hybrids, is less susceptible to preflowering cold-induced sterility, and therefore more efficient at self-pollination, seed set, and ergot resistance (37). Ergot incidence and seed set were inversely correlated ($r = –0.92$).

Resistance to *Claviceps* species is needed in A-lines but is unavailable in sorghum, since most resistance mechanisms in sorghum relate to pollination (20). However, ergot-resistant male-sterile lines of pearl millet were developed (49), suggesting that the development of ergot-resistant A-lines in sorghum might be possible. Ergot-resistant maintainers, restorers, and hybrids of pearl millet are available (49). Use of biotechnology to incorporate antifungal proteins in stigmas offers exciting possibilities of inhibiting the pathogen at the site of infection.

Information on the genetics of ergot resistance is lacking in graminaceous crops, except pearl millet. Resistance in pearl millet is controlled by polygenic recessive genes (50), implying that to breed ergot-resistant hybrids, resistance should be incorporated into both male-sterile and pollen parents (41). A breeding strategy involving recurrent selection to concentrate the minor genes controlling polygenic resistance for intrapopulation improvement has been successful in pearl millet. Similar studies have yet to be conducted on sorghum. Good progress has been made in breeding for ergot resistance in pearl millet, and there is much to learn from the well-documented extensive research on pearl millet that was conducted during the 1980s and 1990s (48,49).

Integrated Ergot Management

Ergot management in hybrid seed production and in grain production requires different emphases. Pollen-based management, the selection of sowing locations and times to avoid high-risk situations, chemical control, and removal of sclerotia in seed-processing plants are of overriding importance in the production and processing of hybrid seeds. Host plant resistance is the most economically feasible control method in commercial grain production and should be available soon, given the current research efforts. In both production situations, methods that reduce inoculum in the field will assist in reducing damage. Further research is essential to test combinations of different ergot control methods, to develop strategies to suit the socioeconomic and technological needs of farmers at each location, and to reduce dependency on fungicides. Development and use of a disease forecasting system based on weather data (temperature, humidity, rainfall, solar radiation, etc.) and host factors (pollen production and viability, stigma receptivity, nicking, etc.) could help to better schedule and optimize fungicidal spray application.

Research Needs and Looking Ahead

Information is available on the infection process, the roles of pollination and fertilization in disease development, and techniques to screen for resistance. Research on pathogen biology, toxicology, disease epidemiology, chemical control, and host plant resistance needs to be undertaken to develop integrated ergot management practices (4). Quarantine, trade, the economics of sorghum production, international seed exchange, and other policy considerations require immediate attention. Experience gained from the recent outbreaks of Karnal bunt of wheat in the United States should be considered when formulating future control strategies.

The unfolding ergot story in Australia and the Americas and the responses of the sorghum research community have shown the interdependence of sorghum research worldwide. For example, a highly effective fungicidal control schedule developed in Brazil could be immediately employed in the United States or elsewhere to reduce losses. Continued communication of results will help exchange scientific information about ergot. The use of the World Wide Web (e.g., http://www.ars-grin.gov/ars/SoAtlantic/Mayaguez/sorghumnews.html) to globally exchange information about ergot has been successful.

Ergot is here to stay. However, it is not an unmanageable disease, given the experiences in India and South Africa.

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Literature Cited

1. Anahosur, K. H. 1979. Chemical control of ergot of sorghum. Indian Phytopathol. 32:487-489.
2. Anahosur, K. H., and Patil, H. S. 1982. Effect of date of sowing on the incidence of ergot of sorghum. Indian Phytopathol. 35:507-509.
3. Bandyopadhyay, R. 1992. Sorghum ergot. Pages 235-244 in: Sorghum and Millets Diseases: A Second World Review. W. A. J. de Milliano, R. A. Frederiksen, and G. D. Bengston, eds. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, India.
4. Bandyopadhyay, R., Frederickson, D. E., McLaren, N. W., and Odvody, G. N. 1996. Ergot — A global threat to sorghum. Int. Sorghum Millets Newsl. 37:1-32.
5. Bandyopadhyay, R., Mughogho, L. K., Manohar, S. K., and Satyanarayana, M. V. 1990. Stroma development, honeydew formation, and conidial production in *Claviceps sorghi*. Phytopathology 80:812-818.
6. Bandyopadhyay, R., Mughogho, L. K., and Sharma, H. C. 1991. Source of primary in-

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oculum and spread of ergot. Pages 24-25 in: Cereals Program Annual Report 1990. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, India.
7. Bandyopadhyay, R., Mughogho, L. K., and Tegegne, G. 1992. Role of pollination and inoculation on ergot development in sorghum. (Abstr.) Phytopathology 82:241.
8. Boon-long, T. 1992. Sorghum diseases in Thailand. Pages 41-43 in: Sorghum and Millets Diseases: A Second World Review. W. A. J. de Milliano, R. A. Frederiksen, and G. D. Bengston, eds. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, India.
9. Brooking, I. R. 1976. Male sterility in *Sorghum bicolor* (L.) Moench induced by low night temperature. I. Timing of the stage of sensitivity. Aust. J. Plant Physiol. 3:589-596.
10. Chen, G. M., Cheng, Q. H., and Yeh, C. C. 1991. Ergot disease of sorghum in Taiwan. Plant Prot. Bull. (Taiwan) 33:223-226.
11. Commonwealth Agricultural Bureau International. 1987. Commonwealth Mycological Institute Distribution Maps of Plant Diseases. Map No. 582, edn. 1, issued 1 Oct. 1987. CABI, Kew, Surrey, Eng.
12. de Milliano, W. A. J., Tavares Nogueira, M. F. R., Pomela, L. M., Msiska, F. S., Kunene, S., Matalaote, B., Mbwaga, A. M., Kaula, G. M., and Mtisi, E. 1991. New records of ergot of sorghum caused by *Sphacelia sorghi* in southern Africa. Plant Dis. 75:215.
13. Ferreira, A. S., Pinto, N. F. J. A., and Casela, C. R. 1996. Avaliacaode fungicidas para o controle de ergot ou doenca acucarada (*Claviceps africana*) em sorgho. Pesqui. Andamento. 14 April 1996.
14. Food and Agriculture Organization of the United Nations, and International Crops Research Institute for the Semi-Arid Tropics. 1996. The world sorghum and millet economies: Facts, trends and outlook. FAO, Viale delle Termie di Caracalla, 00100 Rome, Italy.
15. Frederickson, D. E. 1990. Ergot disease of sorghum. Ph.D. thesis. University of London, London.
16. Frederickson, D. E., and Mantle, P. G. 1988. The path of infection of sorghum by *Claviceps sorghi*. Physiol. Mol. Plant Pathol. 33:221-234.
17. Frederickson, D. E., Mantle, P. G., and de Milliano, W. A. J. 1989. Secondary conidiation of *Sphacelia sorghi* on sorghum, a novel factor in the epidemiology of ergot disease. Mycol. Res. 93:497-502.
18. Frederickson, D. E., Mantle, P. G., and de Milliano, W. A. J. 1991. *Claviceps africana* sp. nov., the distinctive ergot pathogen of sorghum in Africa. Mycol. Res. 95:1101-1107.
19. Frederickson, D. E., Mantle, P. G., and de Milliano, W. A. J. 1993. Windborne spread of ergot disease (*Claviceps africana*) in sorghum A-lines in Zimbabwe. Plant Pathol. 42:368-377.
20. Frederickson, D. E., Mantle, P. G., and de Milliano, W. A. J. 1994. Susceptibility to ergot in Zimbabwe of sorghums that remained uninfected in their native climates in Ethiopia and Rwanda. Plant Pathol. 43:27-32.
21. Frederickson, D. E., and Obilana, A. B. 1993. Manipulating the hybrid production system to control ergot (*Claviceps africana*). Page 1 in: ICRISAT Southern and Eastern Africa Regional Program Annual Report 1993. Southern African Development Community/International Crops Research Institute for the Semi-Arid Tropics, Sorghum and Millet Improvement Prog., Bulawayo, Zimbabwe.
22. Futrell, M. C., and Webster, O. J. 1965. Ergot infection and sterility in grain sorghum. Plant Dis. Rep. 49:680-683.
23. Futrell, M. C., and Webster, O. J. 1966. Host range and epidemiology of the sorghum ergot organism. Plant Dis. Rep. 50:828-831.
24. Kulkarni, B. G. P., Seshadri, V. S., and Hegde, R. K. 1976. The perfect stage of *Sphacelia sorghi* McRae. Mysore J. Agric. Sci. 10:286-289.
25. Langdon, R. F. N., and Champs, B. R. 1954. Insect vectors of *Claviceps purpurea* in Queensland. J. Aust. Inst. Agric. Sci. 20:24.
26. Loveless, A. R. 1967. *Claviceps fusiformis* sp. nov., the causal agent of an agalactia of sows. Trans. Br. Mycol. Soc. 50:15-18.
27. Mantle, P. G. 1968. Inhibition of lactation in mice following feeding with ergot sclerotia (*Claviceps fusiformis* Loveless) from the bulrush millet (*Pennisetum typhoides* Staph and Hubbard) and an alkaloid component. Proc. Royal Soc. London Ser. B. 170:423.
28. Mantle, P. G. 1968. Studies on *Sphacelia sorghi* McRae, an ergot of *Sorghum vulgare* Pers. Ann. Appl. Biol. 62:443-449.
29. Mantle, P. G., and Hassan, H. A. G. 1994. Widening geographical distribution of *Claviceps africana*, an important ovary pathogen of grain sorghum. Int. Sorghum Millets Newsl. 35:97-98.
30. Mantle, P. G., Hassan, H. A. G., and McLaren, N. W. Putative control of ergot disease epidemics in hybrid sorghum production through the inhibition of secondary sporulation by *Claviceps africana*. Proc. Global Conf. Ergot Sorghum, 1-8 June 1997, Sete Lagoas, Brazil. EMBRAPA/INTSORMIL/ICRISAT. In press.
31. Mantle, P. G., and Waight, E. S. 1968. Dihydroergosine: A new naturally occurring alkaloid from the sclerotia of *Sphacelia sorghi* (McRae). Nature 218:581-582.
32. McLaren, N. W. 1992. Quantifying resistance of sorghum genotypes to the sugary disease pathogen (*Claviceps africana*). Plant Dis. 76:986-988.
33. McLaren, N. W. 1993. Effect of sugary disease exudates on germination, seedling development and predisposition to seedling diseases of sorghum (*Sorghum bicolor*). S. Afr. J. Plant Soil 10:12-16.
34. McLaren, N. W. 1994. Efficacy of systemic fungicides and timing of preventative sprays in the control of sugary disease of grain sorghum (*Sorghum bicolor*). S. Afr. J. Plant Soil 11:30-33.
35. McLaren, N. W. 1997. Changes in pollen viability and concomitant increase in the incidence of sorghum ergot with flowering date and implications in selection for escape resistance. J. Phytopathol. 145:261-265.
36. McLaren, N. W., and Wehner, F. C. 1990. Relationship between climatic variables during early flowering of sorghum and the incidence of sugary disease caused by *Sphacelia sorghi*. J. Phytopathol. 130:82-88.
37. McLaren, N. W., and Wehner, F. C. 1992. Preflowering low temperature predisposition of sorghum to sugary disease (*Claviceps africana*). J. Phytopathol. 135:328-334.
38. McRae, W. 1917. Notes on some south Indian fungi. Madras Agric. Yearb. 1917:108-111.
39. Musabyimana, T., Sehene, C., and Bandyopadhyay, R. 1995. Ergot resistance in sorghum in relation to flowering, inoculation technique and disease development. Plant Pathol. 44:109-115.
40. Quinby, J. R. 1958. Grain sorghum production in Texas. Texas Agric. Exp. Stn. Bull. 912. Texas A&M University, College Station.
41. Rai, K. N., and Thakur, R. P. 1995. Ergot reaction of pearl millet hybrids affected by fertility restoration and genetic resistance of parental lines. Euphytica 83:225-231.
42. Reis, E. M., Mantle, P. G., and Hassan, H. A.-G. 1996. First report in the Americas of sorghum ergot disease, caused by a pathogen diagnosed as *Claviceps africana*. Plant Dis. 80:463.
43. Ribas, P. M. Strategy for the control of ergot in sorghum hybrid seed production in Brazil. Proc. Global Conf. Ergot Sorghum, 1-8 June 1997, Sete Lagoas, Brazil. EMBRAPA/INTSORMIL/ICRISAT. In press.
44. Ryley, M. J, Alcorn, J. L., Kochman, J. K., Kong, G. A., and Thompson, S. M. 1996. Ergot on *Sorghum* spp. in Australia. Aust. Plant Pathol. 25:214.
45. Sangitrao, C. S. 1982. Studies on ergot disease of sorghum in Vidarbha. Ph.D. thesis. Punjabrao Krishi Vidyapeeth, Akola, India.
46. Shone, D. K., Phillip, J. R., and Christie, G. J. 1959. Agalactia of sows caused by feeding the ergot of the bulrush millet *Pennisetum typhoides*. Vet. Rec. 71:129-132.
47. Tegegne, G., Bandyopadhyay, R., Mulatu, T., and Kebede, Y. 1994. Screening for ergot resistance in sorghum. Plant Dis. 78:873-876.
48. Thakur, R. P., and King, S. B. 1988. Ergot disease of pearl millet. International Crops Research Institute for the Semi-Arid Tropics, Inf. Bull. 24, Patancheru, India.
49. Thakur, R. P., Rai, K. N., King, S. B., and Rao, V. P. 1993. Identification and utilization of ergot resistance in pearl millet. International Crops Research Institute for the Semi-Arid Tropics, Res. Bull. 17, Patancheru, India.
50. Thakur, R. P., Rao, V. P., and King, S. B. 1989. Ergot susceptibility in relation to cytoplasmic male sterility in pearl millet. Plant Dis. 73:676-678.
51. Thakur, R. P., Williams, R. J., and Rao, V. P. 1983. Control of ergot in pearl millet through pollen management. Ann. Appl. Biol. 103:31-36.
52. Tunawar, N. S., and Singh, S. V. 1988. Sorghum. Pages 44-50 in: Indian Minimum Seed Certification Standards. Central Seed Certification Board, Department of Agriculture and Cooperation, Ministry of Agriculture, Government of India, New Delhi.
53. 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 76:536-539.
54. Youngken, H. W. 1947. Ergot — A blessing and a scourge. Econ. Bot. 1:372-380.