Sugary disease or Asian ergot of sorghum - *Claviceps sorghi*

Claviceps sorghi is a pathogen of *Sorghum bicolor* found only in India and Southeast Asia. The more widespread *C. africana* is predominant even in India; *C. sorghi* appears to be marginalized in its restricted range. A significant difference in invasiveness is the relative production of inoculum that spreads plant-to-plant. *Claviceps sorghi* produces few to no secondary conidia from macroconidia on infected florets, while *C. africana* produces large numbers of these airborne propagules. The macroconidia of *C. sorghi* may themselves be transported in honeydew by wind, rainsplash, insects or direct contact between plants, but these are more limited means. Fungal sclerotia and/or Sphacelia state may be carried among harvested seed, but the seed lots can be cleaned or treated with fungicides (Bandyopadhyay et al., 1996). Alternative hosts are pearl millet (Pennisetum glaucum), wild and weedy relatives of *Sorghum bicolor*, and wild grasses

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Sphacelia cream-white to grey; elongate, straight to curved, 3-14 x 1-2.5 mm, producing two types of conidia: macroconidia oblong with polar vacuoles and slight central constriction, 8-19 x 4-6 µm; microconidia spherical, 2.5 µm diam.

Sclerotia develop within glumes, with distal tissue forming the thin, red core of the protruding sphacelium. Germination sclerotium produces two or three stromata, stalks bronze to reddish-brown, 6-8 x 0.5 mm, capitulum buff 0.7 mm diameter, perithecial ostioles dark, papillate . Stalk insertion surrounded by a white frill.

Ascomata perithecial 130-250 x 60-125 µm diam. Asci cylindrical56-114 x 2.5-3 µm ends tapering, apical caps hyaline. Ascospores eight, 40-97 × 0.4-0.8 µm (Frederickson et al., 1991).

In culture on a defined medium, *C. sorghi* isolates from young sphacelia white, cottony to velvety, even colonies with diffuse margins, no puckering or sulcation. Macro- and microconidia produced in pale brown honeydew-like droplets at the centre of the colony (Muthusubramanian et al., 2006)

Host range: Primarily known on *Sorghum bicolor* (grain sorghum) but also infects *Heteropogon triticeus, Pennisetum glaucum, Pennisetum spp., Sorghum arundinaceum, S. halepense, S. versicolor, and S. virgatum.*

Geographic distribution: Central and southern India and parts of Thailand and Vietnam. The distribution is difficult to determine because of the confusion with *C. africana*. A recent survey of sorghum ergot in India found that *C. sorghi* occurs in Andhra Pradesh and Maharashtra states, but that *C. africana* is predominant in the sorghum-growing regions of India (Muthusubramanian et al. 2006).

Remarks: Two additional species of *Claviceps*, *C. africana* and *C. sorghicola* infect sorghum but only *C. africana* has an overlapping distribution with *C. sorghi* in India. *Claviceps sorghicola* is found only in central Japan. Records of *C. sorghi* in India go back to 1917 (McRae, 1917). Both *C. sorghi* and *C. sorghicola* form elongate sphacelia and sclerotia, but those of *C. sorghicola* are conical rather than straight-sided. *Claviceps africana* forms subglobose sphacelia and sclerotia. Additionally, honeydew of *C. sorghi* honeydew appears before the sphacelia are visible, so infected florets show the honeydew ooze before the fungus is visible. The converse is true for *C. africana* because the bulky sphacelium causes the glumes to bulge wide open before honeydew is produced. Neither conidia of *C. sorghi* nor those of *C. sorghicola* produce secondary conidia in nature, so their honeydew never appears superficially white. In contrast, secondary conidiation on the honeydew surface is a striking feature of *C. africana* infections. At the optimum temperature of 25 C, *C. sorghi* isolates produced few secondary conidia from honeydew on plants in growth chambers compared to the quantities produced by Indian *C. africana* isolates (Muthusubramanian et al., 2006).Pazoutova et al. (2000) used nucleotide sequence differences of ITS1 and part of 5.8S rDNA to distinguish between the three species. Different genetic tests were used by Tooley et al. (2006) to characterize and distinguish Indian isolates of sorghum ergot, providing molecular data to support species identifications based on morphology (Muthusubramanian et al., 2006). For a full comparison of *C. sorghi* and *C. africana* see Frederickson et al. (1991) and Muthusubramanian et al. (2006). For details of *C. sorghicola*, see Tsukiboshi et al. (1999b). The sori of covered kernel smut (Sporisorium sorghi (= Sphacelotheca sorghi)) and long smut (Tolyposporium ehrenbergii) may be confused with sphacelia of *C. sorghi*. However, the sack-like sori of the smut fungi are comprised of a smooth, cream to grey outer covering or peridium enclosing the dry, powdery-black teliospores (Frederiksen, 1986; Hilu, 1986). Spores are released by rupture of the peridium at the tip. Sphacelia of ergots are devoid of an outer covering, being solid, spongy bodies with convolutions which are spore-bearing cavities. Spores are washed-out of the sphacelium in the oozing honeydew.

Notes on taxonomy and nomenclature

Ergot of sorghum with the common name sugary disease was known worldwide by the scientific name Sphacelia sorghi until the teleomorph (sexual stage) from India was described in 1976 as *Claviceps sorghi* (Kulkarni et al., 1976). In 1991, when the teleomorph of a pathogen in Zimbabwe was generated (Frederickson et al., 1991), it became clear that a different pathogen was responsible for ergot disease of sorghum in Africa and that species was named *C. africana*. Before this distinction became known, ergot in Africa was often referred to as *C. sorghi*. With the arrival of sorghum ergot in the Americas during 1995-97, confusion still prevailed because even some of these later records erroneously referred to *C. sorghi* instead of *C. africana*. Therefore, care is needed when interpreting reports of *C. sorghi* in the literature (see also Geographic Distribution).

HOST RANGE

Although reports of various hosts of *C. sorghi* are extensive, most require substantiation by the application of Koch's postulates in properly-controlled, cross-inoculation tests. Sundaram and Singh (1975) found sugary disease on Ischaemum pilosum and managed to infect the grass with honeydew from sorghum. They similarly inoculated and infected *Pennisetum ciliare* (as *Cenchrus ciliaris* and *P. setigerum* as *C. setigerus*. Of 54 species tested by Chen et al. (1995a), *C. sorghi* would only infect *Pennisetum glaucum* (pearl millet) and *Sorghum halepense* (Johnson grass). Muthusubramanian et al. (2005) inoculated a number of grasses with conidia and found that four other species of Sorghum,as well as *P. glaucum* became infected. Pazoutova et al. (2002) used molecular techniques to confirm the identity of the ergot fungus on Heteropogon triticeus in India as *C. sorghi*. Continued revision of grass host taxonomy must also be considered.

Affected Plant Stages: Flowering stage.

Affected Plant Parts: Fruits/pods, inflorescence, leaves and stems.

Individual florets of the inflorescence are affected by sphacelia, sclerotia and honeydew; the panicle, seeds, leaves and stalk can be coated by honeydew.

GEOGRAPHIC DISTRIBUTION

Claviceps sorghi was first found in India in 1915 (McRae, 1917) and Ramakrishnan recorded it in 1948, although the pathogen was then known through its anamorph as *Sphacelia sorghi*, until the description of the teleomorph in 1976 (Kulkarni et al., 1976). *C. sorghi* was assumed to be the causal agent of ergot disease of sorghum worldwide until 1991, when ergot in Zimbabwe was described as a new species, *C. africana* (Frederickson et al., 1991). Any reports of *C. sorghi* in Africa should therefore correctly refer to *C. africana*. In 1998, *C. africana* was confirmed present in India also (Bogo and Mantle, 1999; Pazoutova et al., 2000). In retrospect it has become clear that since the late 1980s all ergot samples from India have been of *C. africana* and, it was hypothesized that *C. africana* may have replaced, or marginalized, *C. sorghi* (RAFrederiksen, Texas A&M University, USA, personal communication, 1999). The earlier report of Kulkarni et al. (1976) describes both the elongate sclerotia typical of *C. sorghi* and small, subglobose sclerotia characteristic of *C. africana*, indicating that *C. africana* was already present in India in 1976. Bandyopadhyay (ICRISAT, India, personal communication, 1998) reported *C. africana* in Maharashtra, Karnataka, Andhra Pradesh, Gujarat, Tamil Nadu and Uttar Pradesh states in India. The continued presence of *C. sorghi* in India was confirmed by an isolation in Karnataka province (Pazoutova and Bogo, 2002) and a survey was y undertaken to determine the distribution and prevalence of both *C. sorghi* and *C. africana*. Muthusubramanian et al. (2006) reported the survey results that found *C. sorghi* in Andhra Pradesh and Maharashtra states, but their data showed that *C. africana* is, in fact, predominant in the sorghum-growing regions of India.From the literature it is impossible to verify the reports of *C. sorghi* in Taiwan. The optimum temperature of conidial germination suggests *C. sorghi* (Chen et al., 1995b). Conversely, reports of secondary conidiation (Lin, 1993) suggest *C. africana*, but this trait is a feature of all sorghum ergot pathogens in vitro (Pazoutova et al., 2004). The narrow host range also suggests *C. africana* (Chen et al., 1995a). Records from Myanmar (CMI, 1987; specimens from the herb. IMI, c/o CABI Bioscience, Egham, UK, accession numbers 1471 and 1472) may possibly represent C. sorghicola (Tsukiboshi et al,, 1999b), while those from the Philippines and Yemen (CMI, 1987) are inconclusive and require expert verification. *Claviceps africana* has been found in Yemen (CABI/EPPO, 2006). Tonapi et al. (2003a) reported that at least some ergot specimens collected in Myanmar, Thailand and Vietnam appeared to be *C. sorghi*.

BIOLOGY AND ECOLOGY

In a study of the infection process in sorghum ergot, Frederickson and Mantle (1988) showed that macroconidia germinate on exserted stigmas of unpollinated, male-sterile sorghum ovaries within 1-2 days of inoculation. The germ tube penetrates the stigmatic surface and hyphae grow though pollen transmission tracts in the style, entering the ovary wall within 3 days. Infection via the ovary wall can occur but is probably not a significant means of entry under natural conditions. Hyphae grow down the

inner ovary wall to the base, occupying the tissues adjacent to the rachilla, which provides the necessary nutrients to support proliferation of hyphae. All but the very apex of the ovary is colonized. Six days after inoculation, hyphae of the pathogen simultaneously erupt from the lower aspect of the outer ovary wall and invade the ovule through the chalazal region. The incubation period is 8-10 days, after which time honeydew, containing large numbers of conidia, is exuded from infected florets. The sphacelium remains very small and is contained within the floret until about 15 days after germination, when it emerges from between the glume tips (Frederickson and Mantle, 1988; Frederickson, 1990). Secondary conidiation (Frederickson et al., 1989) is not a natural feature of *C. sorghi* infections, although it occurs readily in vitro (Frederickson, 1990; Pazoutova et al., 2004). Infected florets eventually produce typical elongated sclerotia of *C. sorghi*.

Ovaries resist infection and colonization by the pathogen after fertilization. For ergot diseases in general, rapid anthesis lowers the chance of infection and any factor prolonging the period from flower opening to fertilization will promote infection (Futrell and Webster, 1965). Musabyimana et al. (1995) found that each day of delay in fertilization after inoculation resulted in 8.3% more ergot. However, ovaries could be infected up to 5 days after pollination (Puranik et al., 1973). Despite various studies that have related environmental factors such as humidity, temperature and rainfall to floral infection, no definitive information is available to clarify these associations (Bandyopadhyay et al., 1996). Spore germination occurs at 12-36°C; optimum 24-28°C (Frederickson, 1990; Chen et al., 1995b). Relative humidity >60% encourages honeydew formation (Frederickson, 1990). Chen et al. (1995a) reported that low temperatures and high rainfall were critical factors favouring infection. Dry conditions favour development of sclerotia (Sangitrao and Bade, 1979; Mohan and Jeyarajan, 1991). Sclerotia are not separate entities but form from within sphacelia. The proximal sclerotial tissues form an oval to cylindrical structure iwithin the glumes; distal sclerotial tissues project into the uppermost sphacelial tissue forming a cylindrical core. However, the apex of the structure is actually all sphacelial tissue (Frederickson et al., 1991). Either sclerotia or sphacelia may facilitate survival. Sangitrao (1982) experimentally germinated sclerotia to produce the teleomorph. Germination occurred at 5-50°C, optimum 20-30°C. At 27°C, 55% of stromata differentiated fully. Sclerotia were still viable after 10 years in storage at room temperature in the laboratory (Tonapi et al., 2003b) . Frederickson et al. (1991) germinated C. sorghi sclerotia from Akola, Maharashtra, India, by incubation in moist sand in the laboratory at 24°C. Stromata were produced within 5 weeks. Germination has not been observed in the field and the significance of the teleomorph is unclear (Frederickson and McLaren, 2000)

Sclerotia may also be important as vehicles of conidia in the attached sphacelia. Conidia of ergot survived for 9 months in infected heads on the soil surface (Lakshmanan et al., 1990). Honeydew on seed may be a source of infection but it can be eliminated by seed treatment with captan (Odvody, 1997). In the absence of secondary conidiation, it is not clear how macroconidia on seed or plant debris would infect stigmas.

Alternative hosts of *C. sorghi* have been reported (see Host Range), but most have not been investigated by cross-inoculation tests following Koch's postulates. Their role, if any, in the spread and survival of *C. sorghi* remains unclear. Insects are known to carry ergot conidia non-specifically on their bodies after feeding on honeydew (Prom et al., 2003; Prom, 2005). Their role in specific transmission of *C. sorghi* has not been investigated.

MEANS OF MOVEMENT AND DISPERSAL

Plant parts liable to carry the pest in trade/transport

- Flowers/Inflorescences/Cones/Calyx: Spores, Sclerotia, Hyphae; borne internally; borne externally; visible to naked eye.

- Leaves: Spores; borne externally; visible to naked eye.

- Stems (above Ground)/Shoots/Trunks/Branches: Spores; borne externally; visible to naked eye.

- True Seeds (inc. Grain): Spores, Sclerotia; borne externally; visible to naked eye.

Plant parts not known to carry the pest in trade/transport

- Bark

- Bulbs/Tubers/Corms/Rhizomes

- Fruits (inc. Pods)

- Growing Medium Accompanying Plants

- Seedlings/Micropropagated Plants

- Roots

- Wood

SEEDBORNE ASPECTS

Incidence

Extensive infection of sorghum seeds by *C. sorghi* occurs in hybrid seed production. A 1989 survey in Taiwan of the autumn crop of cv. Taichung No. 5 indicated that 64% of the female parent (cytoplasmic male sterile line) and 12% of the male parent (restoring line) were infected (Chen et al., 1991). Seed lots may be contaminated with sclerotia, or sphacelia, and seed may be contaminated with honeydew (Bandyopadhyay et al., 1998).

Effect On Seed Quality

Ergot contamination of sorghum seed lots has a negative effect on quality standards in seed certification programmes (Bandyopadhyay, 1992).

Pathogen Transmission

Ascospore inoculum from sclerotia of *C. sorghi* that are planted with seeds is assumed to be a primary inoculum source, but proof is lacking. Conidia that survive on other hosts or on panicle debris in the soil were considered to be a more likely source of primary inoculum (Bandyopadhyay, 1992). Conidia carried in honeydew on seeds will not provide inoculum at the appropriate time for infection of the crop even if seeds are untreated (Dahlberg et al., 1999).

Seed Treatment

Steeping seeds in 5% salt solution is considered a practical way of removing sclerotia of *C. sorghi* from seed lots but is probably not 100% effective (Bandyopadhyay, 1992). Sprays of carbendazim + tridemorph followed by carbendazim + TMTD (thiram) and thiophanate-methyl gave good control of *C. sorghi* infection and reduced contamination of seeds (Lakshmanan and Mohan, 1988). Fungicide sprays to the seed crop, however, have generally been considered impractical and uneconomic (Bandyopadhyay, 1992). However, captan-treatment of seed is both economic and effective against Claviceps africana conidia and presumably also against those of *C. sorghi* (Dahlberg et al., 1999).Seed processing equipment such as gravity tables can separate sclerotia from seeds (Bandyopadhyay et al., 1998). See also *C. africana*.

Seed Health Tests

Visual examination (Bandyopadhyay et al., 1998): Seed lots are examined for the presence of cylindrical, cream-white to grey cushions of fungal growth (sphacelia), or for thin, elongate sclerotia, mixed with the seeds. Examination at a magnification of at least x10 is recommended to avoid the problems of mis-identification frequently encountered when using the naked eye (Alderman et al., 1999).

IMPACT

Ergot disease is primarily an economic problem in F1 hybrid seed production. It 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 lack of viable pollen and delayed seed set (Bandyopadhyay et al., 1998). Losses of 10-80% have been reported in hybrid seed production fields in India. However, *C. sorghi* has limited economic impact because of its narrow distribution.The incidence and severity of ergot (*C. sorghi* and *C. africana*) on sorghum (*Sorghum bicolor*) was surveyed in the Indian states Andhra Pradesh, Gujarat, Tamil Nadu, Maharashtra, Karnataka, Rajasthan and Uttar Pradesh from 1999 to 2002. Crops were examined at vegetative to physiological maturity stages, and disease incidence (number of plants infected) and severity (percentage, based on the number of florets infected within panicles) recorded in 12 m² areas. Percentage incidence of ergot infection varied with location, with Rajasthan and Gujarat having only trace infections from 1999 to 2002 and Karnataka with 27-60% infection. Disease severity followed the same pattern (Navi et al., 2002).

SYMPTOMS

Individual ovaries between the glumes of some or all florets are colonized by the parasite. Infected florets become evident when sticky,orange-brown liquid droplets of spore-bearing honeydew are exuded. The soft, white, narrow and elongate growth of mycelium forming thesphacelium becomes evident soon after, appearing from between the glume tips; the glumes are barely distorted laterally. The sphacelium grows at the inner end and may achieve a length of 14 mm (Frederickson and Mantle, 1988). Honeydew droplets may coat panicles, seeds, leaves and the stalk.In the case of colonization of the honeydew and sphacelia by the hyperparasitic fungus *Cerebella andropogonis*, black, spherical, convoluted growths are seen at floret tips (Mohan and Jeyarajan, 1991; Bandyopadhyay et al., 1998). Upon dissection, a discoloured sphacelium of reduced size is found underneath. Other moulds may also grow on the

honeydew.

Symptoms by affected plant part

• Fruits/pods: honeydew or sooty mould.

• Inflorescence: honeydew or sooty mould.

• Leaves: honeydew or sooty mould.

• Stems: honeydew or sooty mould.

DETECTION AND INSPECTION

In the field, *Claviceps sorghi* infection is usually obvious from the dripping of honeydew from infected florets, honeydew deposition on the panicle and the protruding sphacelia. The pathogen is less easily detected in seed batches, even though sclerotia are of distinct shape and size (Tonapi et al., 2003b), which poses potential problems for seed exchange. If the fungus could be cultured from seed, the description provided by Muthusubramanian et al. (2006) would be of use. See Seed Health Tests.

CONTROL

Cultural Control and Sanitary Methods

In India, early sowing reduced the incidence of *C. sorghi*. Planting in the first 2 weeks of June gave the best results. Planting in July, on the other hand, was recommended to facilitate screening under natural disease pressure (Singh, 1964; Sangitrao et al., 1979; Anahosur and Patil, 1982). Field practices aimed at reducing the risk or severity of all ergot pathogens include the removal of infected panicles at harvest, crop rotations and deep ploughing of crop residues. However, these methods have limited impact (Frederickson and Leuschner, 1997).

Host-Plant Resistance

There is currently no source of resistance to any species of sorghum ergot for deployment in the field in A-lines, although many workers have devised screening methodologies for *C. sorghi* and report variable success in finding 'resistant' lines (Khadke et al., 1979; Chandrasekaran et al., 1983, 1985; Rajkule et al., 1983, 1985; Lakshmanan et al., 1988a, b; Lakshmanan and Mohan, 1989a, b; Palanisamy et al., 1989; Anahosur et al., 1990; Hiremath and Lakshman, 1990). In trying to evaluate resistance to *C. africana*, McLaren (1992) concluded that simple comparisons of severity data from tests of genotypes from different localities, following either natural infection or artificial inoculation, is meaningless, and this will also be the case for *C. sorghi* resistance. Susceptibility to ergot is extremely sensitive to environmental factors at flowering and a few weeks before (McLaren and Wehner, 1990, 1992; McLaren, 1997). Cool nights of <12°C at 2-3 weeks before anthesis result in pollen sterility and increased ergot severity. Therefore, tolerance of low, pre-flowering minimum temperatures is important for disease avoidance (McLaren, 1997). Similarly, the mean maximum temperature 1-4 days after pollen shed affects fertilization and thus affects ergot incidence. Interactions between genotype, location and flowering date must be compared by regression analyses because differences in flowering date of even a day or two affect the severity of ergot (McLaren, 1992; McLaren and Flett, 1998).

Careful screening and selection for floral characteristics that reduce disease may prove to be one useful strategy, and results from research on *C. africana* are also applicable to the development of resistance to *C. sorghi.* For example, in Puerto Rico, Dahlberg and Bandyopadhyay (USDA-ARS-TARS, Puerto Rico, personal communication, 1999) found a male-fertile accession with glumes which tightly clasp the ovary, apparently conferring tolerance to high inoculum loads of *C. africana*. The line also showed potential in a male sterile background. In the USA, many A-line sorghums have a protracted stigma receptivity period, enhancing ergot susceptibility (Odvody, 1997) and disease reduction may possibly be achieved by decreasing that ergot-susceptible period of the A-line stigma. Other advantageous modifications might include reducing the floret gaping period, selecting for more rapid post-fertilization changes in the A-line, breeding for cold-temperature tolerance in R-line pollen production and extending the pollen production period.

Chemical Control

Chemical control is barely cost effective, is only feasible for controlling disease on the A-lines, and is unnecessary on the hybrids themselves. In India, however, the use of fungicides has been extensively explored. In field tests, three earhead sprays of ziram and zineb were more effective than two (Gangadharan et al., 1976). Mancozeb, captafol, thiophanate-methyl and carbendazim all gave good yield increases (Anahosur, 1979; Lakshmanan et al., 1986; Lakshmanan and Mohan, 1988). Schedules involving ground applications of triazoles, such as propiconazole and tebuconazole, are effective against *C. africana* and, presumably, *C. sorghi* (Odvody, 1997). See also Seed Treatments.

Development of IPM Strategies

Ergot caused by *C.sorghi* is only a very limited problem in sorghum production; it is not a global problem. The survey of the distribution of *C. sorghi* and *C. africana* in India (Muthusubramanian et al, 2006; Tooley et al., 2006) indicated that serious losses from sorghum ergot in India are mostly due to the more widespread *C. africana*. Future combined control options such as host genetic resistance, disease prediction, manipulation of the pollination system and limited fungicide use during high risk periods are therefore likely to be useful benefits from research on the more globally important ergot pathogen, *C. africana*.

References

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