# *Sorghum Downy Mildew: A Global Perspective*

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*Resumen: La cenicilla, causada por Peronosclerospora sorghi se originó en la India y se diseminó a Asia, y luego a Africa, y más recientemente a las Américas. Debido a la rápida reproducción de las esporas asexuales, las epidemias son creadas en un ciclo de cultivo en campos con poca o ninguna historia de la enfermedad. Suelos con temperaturas menores de 20° C y con predominancia de suelos saturados o secos en las primeras dos semanas después de la siembra son considerados como que tienen poco o ningún potencial para el desarrollo de la cenicilla.*

*El control basado en la resistencia de plantas hospederas, prácticas culturales y el uso de fungicidas han reducido la enfermedad a ésas de menor importancia económica en Asia, la región del mediterráneo y norteamérica. Los patrones de éxito para controlar la cenicilla son probables en centroamérica. El antídoto o protector de semillas contra herbicidas, Concep II, aumentó la frecuencia de las plantas infectadas con cenicilla en un cultivar susceptible. Este problema puede ser resuelto tratando la semilla con metalaxyl.*

*La evaluación de la colección de USDA de dieciséis diferentes aislamientos de P. sorghi coleccionados alrededor del mündo (dos provenientes de Honduras) mostraron que cada uno de ellos fue diferente. Quince cultivares de sorgo han sido identificados como resistentes a todos los aislamientos de la colección. Muchas de las razas fisiológicas están presentes virtualmente en todas las poblaciones. Para hacerle frente o lidiar con esta variabilidad, la población debe ser evaluada anualmente probando las fuentes de resistencia más importantes para determinar cambios en la virulencia del patógeno predominante. Las evaluaciones deben ser hechas en áreas en las cuales existan inoculos de oosporas así como conídias. Actualmente patotipos de P. sorghi pueden ser solamente diferenciados usando cultivares.*

*Los programas de mejoramiento deben tener buenas fuentes de resistencia y tecnologías apropiadas para la evaluación y selección de cultivares. Genotipos con resistencia a inoculos de* conIdias *y de oosporas deberían de ser desarrollados seleccionando en viveros con niveles altos de oosporas en el suelo y en una área con tendencia a infección por conídias. La variabilidad en el patógeno requiere que diferentes fuentes de resistencia sean usadas. Resistencia de las plantas hospederas a la cenicilla puede ser*

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*aumentada evitando el monocultivo de sorgo. Las oosporas sobreviven relativamente pocos años, y pueden ser sustancialmente eliminadas del suelo en un período de tres años. En áreas de monocultivo o de períodos muy cortos de rotación de cultivos, la semilla podría ser tratada con fungicidas tal como metalaxyl. El metalaxyl es eficiente en dosis muy bajas pero podría reducir la habilidad del sorgo para establecer una buena población. El cultivo de sorgo intercalado con maíz podría también contribuir a la diseminación del patógeno. Mejoramiento genético de maíz para resistencia a cenicilla también podría reducir las pérdidas del cultivo y el porcentaje de diseminación del patógeno.*

## ABSTRACT

Sorghum downy mildew, caused by *Peronosclerospora sorghi* (Weston and Uppal) C. G. Shaw, is widely distributed and recognized as a real and potentially damaging pathogen of sorghum and maize (Frederiksen and Renfro, 1977). The pathogen has appeared in most sorghum growing regions of the world, probably throughout history. *Sorghum* spp. were introduced to *P. sorghi* in India and over the ensuing millennia spread across the globe; first in Asia, then Africa and more recently to the Americas. The pathogen spread at will prior to the nineteenth century simply because it was not recognized. Even as recently as the middle of this century we did not appreciate the role of oospores in the rapid distribution of the pathogen with both seed and grain (Frederiksen, 1980b). With advent of the disease and its actual or perceived distribution we must accept its presence and work with it as we would any other biological constraint. Because of rapid reproduction and cycling of asexual spores, epidemics are created within a single growing season in a field of plants with little or no previous history of the disease. This lightening fast, explosive nature of downy mildew along with its frightening destructiveness accounts for the respect we share for this remarkable disease.

In 1980, Frederiksen (1980a) reviewed the status of sorghum downy mildew in the US. Since 1980, the disease has essentially remained static in its distribution. Occasionally, new or unusual epidemics develop such as in Nebraska, Kansas, and Indiana in 1987 (personal communications, S. Jensen, L. Claflin, J.H. Warren, respectively). While not yet fully documented, it appears that in each example the weather permitted repeated cycles of asexual inoculum in susceptible cultivars. Schuh et al. (1987a, 1987b) have evaluated selected environmental parameters on the host-parasite interaction contributing to downy mildew epidemics in Texas. The specific soil moisture, texture and temperature along with inoculum define, in part, the probability of an epidemic in any one year. Schuh et al. (1987a) demonstrated that soils with a temperature below 20 °C and a predominance of saturated or dry soils, in the first 2 weeks after planting, could be considered to have low or no potential for sorghum downy mildew.

Problems with downy mildew are not limited to the U.S. Among the Southern African countries, the disease is becoming increasingly more important as growers shift from traditional to more extensive cultivation of sorghum hybrids. Fortunately, new germplasm being developed for the Southern African region will have high levels of resistance (Williams, 1984).

Downy mildew continues as a concern in South America, particularly Brazil, Argentina, and Venezuela, in part because of the importance of maize, an important collateral host, and because the disease is important at times in various sorghum weed hosts. For the most part however, sorghum downy mildew and related tropical downy mildews are much less important today than they were 2 decades earlier (Frederiksen and Renfro, 1977). Controls based on host resistance and fungicides have reduced the disease to that of minor economic importance particularly in Asia, the Mediterranean region, and North America. The patterns of success for controlling downy mildew are as probable for Central America as they were elsewhere.

## CURRENT CONCERNS REGARDING THE DOWNY MILDEW PATHOGENS

### Variability of the Pathogen.

The appearance of pathotype 5 in Honduras was disappointing but not completely unexpected. *Peronosclerospora sorghi* is a highly variable organism (Fernández and Meckenstock, 1987). Pawar (1986) evaluated the reaction of sixteen different isolates from around the world and found that each was different (Table 1). He even detected differences between two isolates from Honduras in the USDA Maryland

Table 1. Reactions of TAMU-USDA sorghum converted lines to Sixteen Isolates† of *Peronosclerospora sorghi*.

| Cultivar | SC No. | Group | Tx1 | Tx3 | Ar1 | Ar2 | Ar3 | Br1 | Br2 | Hd1 | Hd2 | Id1 | Id2 | Id3 | Id4 | Et1 | Et2 | Ng1 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| IS 12646C | 155 | Sub Dur Doc | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | S |
| IS 12558C | 38 | Durra | R | R | R | R | R | R | R | R | R | R | R | S | S | R | R | R |
| IS 6418C | 493 | Nandyal | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | * |
| IS 2816C | 120 | Zerazera | R | R | R | R | R | R | * | R | R | R | R | R | R | * | R | R |
| IS 12543C | 23 | Durra | R | R | R | R | R | R | R | R | R | R | R | S | S | R | - | R |
| IS 12553C | 33 | Durra | R | R | R | R | R | R | R | R | R | R | R | S | S | R | S | R |
| IS 12556C | 36 | Durra | R | R | R | R | R | R | R | R | R | R | S | S | S | R | R | R |
| IS 2816C | 120 | Sub Dur Doc | R | R | R | R | - | R | R | R | R | R | R | S | R | - | R | - |
| IS 12672C | 181 | Dur Sub | R | R | R | R | R | R | R | R | R | R | R | S | S | S | S | S |
| IS 2508C | 414-12 | Cau Kaf | R | R | R | R | R | R | R | R | R | R | S | S | - | - | R | - |
| IS 12623C(?) | 136 | Dur Doc | R | R | R | R | R | R | R | R | R | R | S | S | S | S | S | S |
| IS 12628C | 137 | Dur Doc | R | R | R | R | R | R | R | R | R | R | S | S | S | S | S | S |
| IS 12632C | 141 | Dur Doc | R | R | R | R | R | R | R | R | R | R | S | S | S | S | S | S |
| IS 2462C | 325 | Nigricans | R | R | R | R | R | R | R | R | R | R | S | S | S | S | S | S |
| IS 3553C | 806 | Cau Dur | R | R | R | R | R | R | R | R | R | R | S | S | S | S | S | S |
| IS 3598C | 502 | Dur Nig | R | R | R | R | R | R | R | R | R | S | S | S | S | S | S | S |
| IS 12610C | 110 | Zerazera | R | S | R | R | R | S | R | R | R | S | R | S | - | - | R | S |
| IS 3072C | 575 | Cau Dur | R | - | S | S | S | S | S | - | - | R | S | S | S | S | S | S |
| IS 12555C | 35 | Durra | S | S | S | S | S | S | S | S | S | S | S | S | S | S | S | S |
| IS 12622C | 131 | Dur Doc | S | S | S | S | S | S | S | S | S | S | S | S | S | S | S | S |

†Tx1 = Texas Pathotype 1, Tx3 = Texas Pathotype 3, Ar1-Ar3 = Isolates from Argentina, Br1-Br2 = Isolates from Brazil, Hd1-Hd2 = Isolates from Honduras, Id1-Id4 = Isolates from India, Et1-Et2 = Isolates from Ethiopia, Ng1 = Isolate from Nigeria.

*Reaction is unknown: No reaction is given (possible allelic heterogeneity). - datos no disponibles collection of *P. sorghi* at Frederick, MD. Honduran isolate Hd1 was similar to the Texas pathotype 1 and Hd2 resembled pathotype 3. Clearly, additional isolates would have permitted further characterizations of the population. In evaluation of other sorghums, Pawar found several entries to be resistant to all of the collections (Table 2). An important item to note is that *P. sorghi* is variable and that many physiologic races are present in virtually all populations. To cope with the variability, the population must be evaluated annually using the important sources of resistance as a screen. Evaluations must be made in areas challenged by both oosporic and conidial inoculum.

Currently, isolates of *P. sorghi* can only be differentiated using cultivars, although isozyme analysis and related procedures have a potential for characterizing among different species of *Peronosclerospora* (Micales et al., 1987).

## Breeding for Resistant to Sorghum Downy Mildew

For downy mildew resistance to be effective, the breeding program must have good parents as sources and appropriate screening technologies. Currently, we recommend a program of selecting for resistance in a field nursery with both high levels of oospores in the soil and in an area conducive to infection by conidia. Even from low levels of initial infection with oospores, conidia can spread rapidly to create an epidemic in a field of sorghum. In this manner, selection can be made for sorghums with both positive levels of resistance to conidial as well as oosporic inoculum. Currently the Craig techniques for inoculating sorghum relies on asexual inoculum (Craig, 1987; Craig and Frederiksen, 1980). The most recent adaptation utilizes freshly collected conidia atomized on seedlings at the 2 leaf stage of growth. Controlled inoculations have many advantages in a breeding program because off-season testing can be employed to reduce the number of test entries and evaluations can be made in the early breeding generations.

In Honduras, Wall (1986) found that sorghum downy mildew caused significant, field losses (Fig. 1). These losses were essentially linear and directly proportional to the incidence of disease. The incidence of 43 percent downy mildew resulted in a similar loss in yield bases on comparisons with an isogenic population in the same field.

sources of resistance be used and that it cannot be assured that a particular source will be "the" durable source.

Host resistance to downy mildew can be augmented by avoiding monocropping of sorghum. Oospores, survive for relatively few years and can be substantially depleted from the soil in a 3 year period, consequently, the growing of non sorghum hosts reduces both the quantity and the quality of overseasoning inoculum. In areas where monocropping or very short term rotations are practiced, seed can be treated with a fungicide such as metalaxyl. Metalaxyl is very effective at very low rates, but can reduce the ability of sorghum to establish a good stand. The cause of this phytotoxicity is not clearly understood, but seedlings develop more slowly and the effect is stronger on seed lots with lower germination. The growing of sorghum as an intercrop with maize may also contribute to the slow rate of spread. Genetic improvement of maize for resistance to sorghum downy mildew might also reduce the probable damage and rate of spread of the pathogen in maize/sorghum intercrop fields.

Recognition of sorghum downy mildew as a normal production constraint and application of prudent safeguards will tend to keep sorghum downy mildew in perspective over the next decade.

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