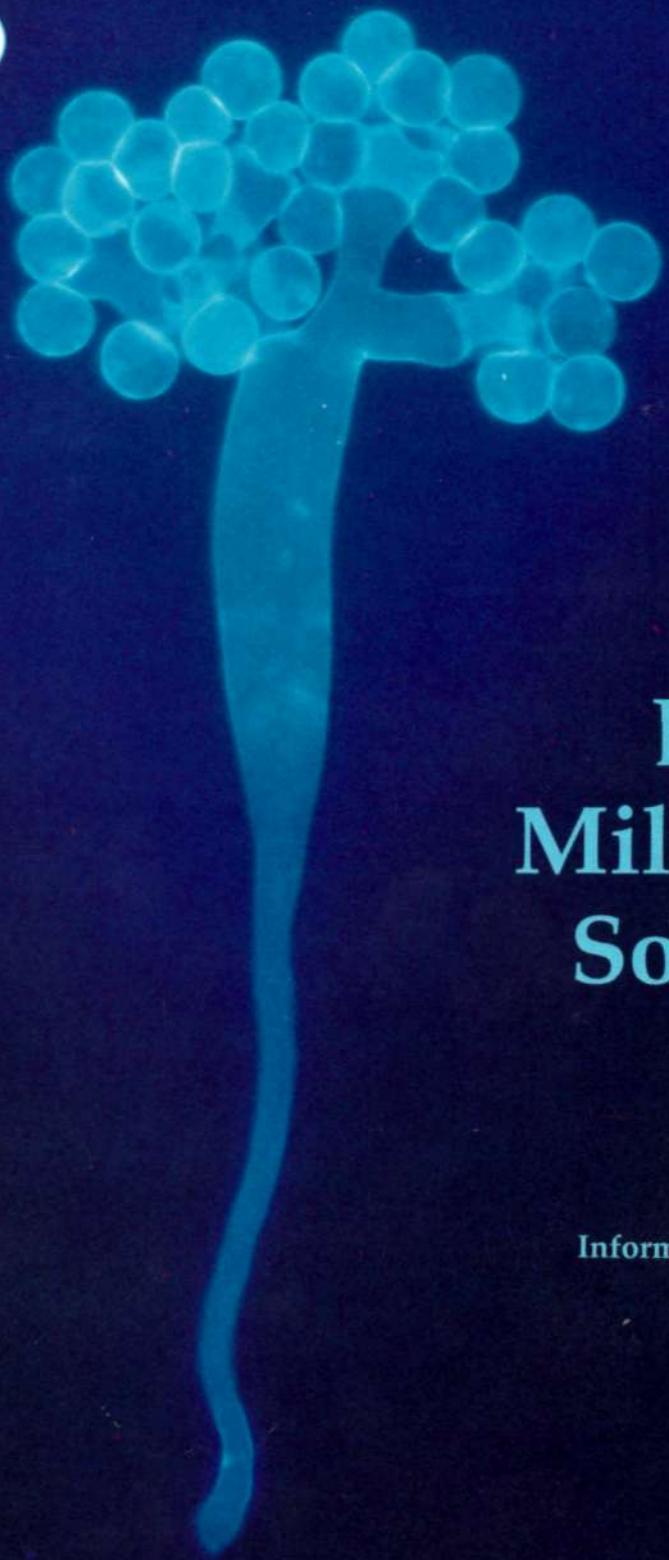

# Downy Mildew of Sorghum

Information Bulletin no. 51

International Crops Research Institute for the Semi-Arid Tropics

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**Abstract**

Downy mildew of sorghum (*Peronosclerospora sorghi*) is one of the most important diseases of sorghum and maize. It is distributed widely in Africa, Asia, and the Americas, where serious epidemics occur. Systemic infection causes complete or partial sterility of the panicle, resulting in yield loss roughly proportional to the disease incidence. The seriousness of the problem has resulted in significant investment to increase knowledge of the pathogen and to investigate various disease control measures. Several alternatives for the control of sorghum downy mildew are now available including cultural and chemical control, and the deployment of resistant varieties. The current state of knowledge of the pathogen's biology, epidemiology, variability, and control are described in this bulletin, together with practical aspects of disease management.

**Résumé**

Le mildiou du sorgho (*Peronosclerospora sorghi*) est l'une des plus importantes contraintes à la production du sorgho et du maïs. La maladie sévit en Afrique, en Asie et aux Amériques, parfois sous forme d'épidémies. L'infection systémique cause la stérilité complète ou partielle de la panicule conduisant à une perte de rendement presque égale à l'incidence de la maladie. Des chercheurs ont fait beaucoup d'efforts pour intensifier leur connaissance du pathogène et pour élaborer des méthodes de lutte contre le mildiou. Des stratégies de lutte – culturales, chimiques et variétés résistantes – sont disponibles. Cette publication fait le point de la recherche sur la biologie, l'épidémiologie, la variabilité et les méthodes de lutte y compris les aspects pratiques de lutte.

**Resumen**

El mildeo velloso del sorgo (*Peronosclerospora sorghi*) es una de las enfermedades más importantes en los cultivos de sorgo y maíz. Esta ampliamente distribuida en Africa, Asia y las Americas, donde ocurren serias epidemias. La infección sistémica causa completa o parcial esterilidad en la panicula, dando como resultado perdidas severas en la producción proporcionales a la incidencia de la enfermedad. La seriedad del problema ha dado como resultado significativas investigaciones para incrementar el conocimiento del patógeno y las diferentes medidas de control de la enfermedad. Diferentes alternativas de control para el Mildeo velloso del sorgo estan disponibles actualmente incluyendo controles culturales y químicos, asi como el desarrollo de variedades resistentes. El actual estado del conocimiento sobre la biología del patógeno, su epidemiología, variabilidad y control están descritas en esté boletin, asi como los aspectos prácticos del manejo de la enfermedad.

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**1997**

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## Introduction

Sorghum (*Sorghum bicolor* L. Moench.) is an important crop for both human consumption and animal fodder. Much of the crop's production area is in the semi-arid regions of the less-developed world, including Africa, Asia, and South America, although there is also substantial sorghum production in the USA. The current worldwide trends in sorghum production since 1961 suggest that sorghum will remain an important crop in these regions.

The sorghum downy mildew (SDM) fungus (*Peronosclerospora sorghi* [Weston and Uppal (Shaw)], infects both sorghum and maize (*Zea mays* L.), and is widespread in many tropical and sub-tropical regions of the world where sorghum and maize crops are grown. Butler (1907) was the first to report downy mildew of sorghum. However, the true taxonomic status of the pathogen as *P. sorghi* was satisfactorily established much later (Weston and Uppal 1932; Shaw 1976,1978, and 1980). It has caused severe epidemics in both sorghum and maize crops in many countries (Kenneth 1976, Williams 1984).

Effective disease control through well-informed disease management strategies can help contribute to sustainable food production in endemic areas, particularly those that support a burgeoning population. This bulletin covers many aspects of the biology, epidemiology, and control of SDM - but does not claim to be comprehensive. It is intended to act as an introduction to the disease and to provide information of interest and use to farmers, extension agents, students, and researchers. Although *P. sorghi* infects both sorghum and maize this bulletin concentrates on the disease of sorghum, and only consider infection of maize where it is deemed necessary, particularly if information is lacking on a particular aspect for sorghum. The reader requiring more detailed information is directed towards the source literature.

## Economic damage

Sorghum downy mildew is particularly destructive since systemic infection of the host results in a barren inflorescence (Frederiksen et al. 1973). There are many reports of the effect of SDM on yield. In a single season in the USA, a SDM epidemic in grain sorghum in the coastal counties of Texas caused an estimated loss of US$ 2.5 million (Frederiksen et al. 1969). Payak (1975) reported that in parts of India annual yield loss due to SDM was at least $1.0 \times 10^5$ metric tons. In Venezuela, crop losses were so severe in the early 1970s that a national emergency was declared (Frederiksen and Renfro 1977). In Israel both forage sorghum and maize have been severely infected with incidences up to 50% (Kenneth 1976), and in the USA the incidence can even be as high as 90% in some fields (Frederiksen et al. 1969). Models have been developed that show a linear relationship exists between the incidence of systemic SDM and yield loss at normal sowing densities (Frederiksen et al. 1973, Tuleen and Frederiksen 1981, Craig et al. 1989).

## Origins and geographic distribution

The SDM pathogen has been confirmed on all continents in tropical and sub-tropical zones of the world (Williams 1984). Countries from which it has been reported are listed in Table 1, and its geographic distribution is illustrated in Figure 1. *Peronosclerospora sorghi* is considered an 'Old World' pathogen, having originated in Africa or Asia (Shaw 1981, Williams 1984). It subsequently spread to the Americas in the late 1950s, where it was probably introduced (Toler et al. 1959, Frederiksen 1980a).

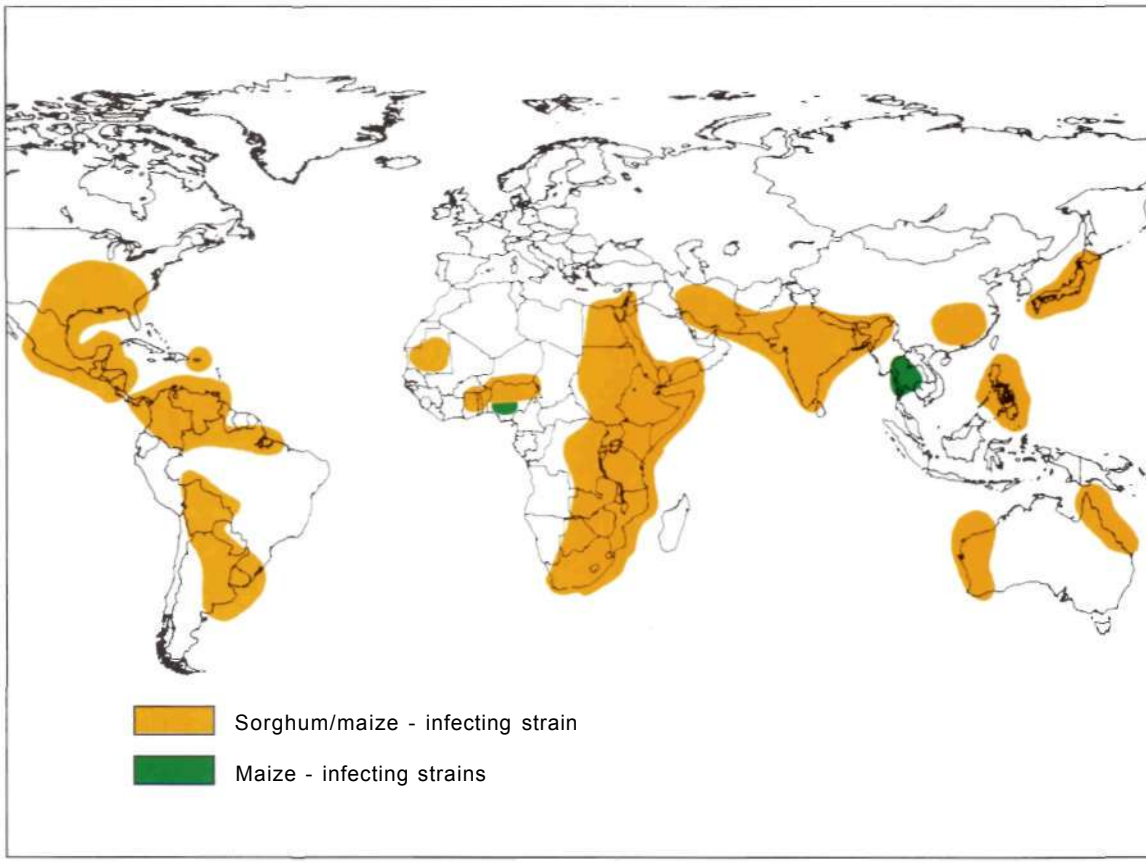


***Figure 1. Geographical distribution of* Peronosclerospora sorghi *showing regions where the sorghum/maize and maize infecting strains occur (the maize strain in Thailand has recently been designated* Peronosclerospora zeae *(Yao 1991).***

## Causal organism

The following description of *P. sorghi* is based on that of Weston and Uppal (1932). The fungus produces asexual conidia (Fig. 2) and sexually produced oospores (Fig. 3). Conidia are produced on the leaf surface on erect conidiophores that grow out through stomata. The conidiophore comprises a basal cell and a more or less complex, usually dichotomously branched, expanded apex (Fig. 4). The basal cell is knobbed or bulbous at the base, then of fairly uniform diameter (7-9 µm) for a length of approximately 100-150 urn. It is usually delimited from the main axis by a complete septum.

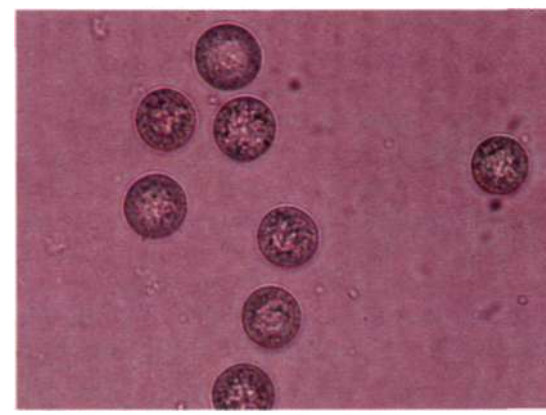

***Figure 2. The asexual phase of* Peronosclerospora sorghi. *Conidia are sub-orbicular, hyaline, and thin-walled (x350).***

**Table 1. Countries from which *Peronosclerospora sorghi* has been reported[1].**

| Region or continent | Country |
|---|---|
| Africa | Benin, Botswana, Burundi, Egypt, Ethiopia, Ghana, Kenya, Malawi, Mozambique, Mauritania (Frison and Sadio 1987), Nigeria, Rwanda, Somalia, South Africa, Sudan, Swaziland, Tanzania, Uganda, Zimbabwe, Zambia (de Milliano 1992) |
| Asia[2] | Bangladesh, China, India, Japan, Pakistan, Philippines |
| Australia | Australia (Queensland, Western Australia) |
| North America | Mexico, USA (Alabama, Arkansas, Georgia, Illinois, Indiana, Kansas, Kentucky, Louisiana, Minnesota, Montana, Nebraska, New Mexico, Oklahoma, Tennessee, Texas) |
| Central America and the West Indies | El Salvador, Guatemala, Honduras, Panama, Puerto Rico |
| South America | Argentina, Bolivia, Brazil, Colombia (Buritica et al. 1992), Uruguay, Venezuela |
| Middle East | Israel, Iran, Yemen |

1. Unless otherwise indicated, all reports were obtained from the Commonwealth Mycological Institute Distribution Maps of Plant Diseases, Map no. 179, Edition 5, Issued 1 April 1988.
2. Previously a maize-infecting strain of P. *sorghi* was thought to occur in Thailand (Bonman et al. 1983). This race is now considered a separate species, *P. zeae* (Yao 1991).

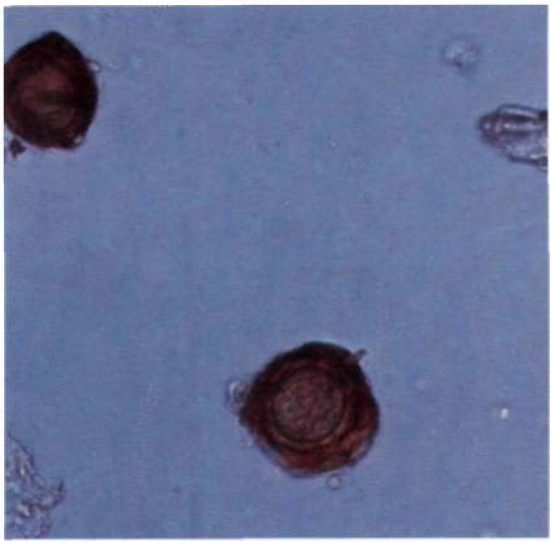

***Figure 3.The sexual phase of* Peronosclerospora sorghi. *Oospores are spherical and thick-walled (x 475).***

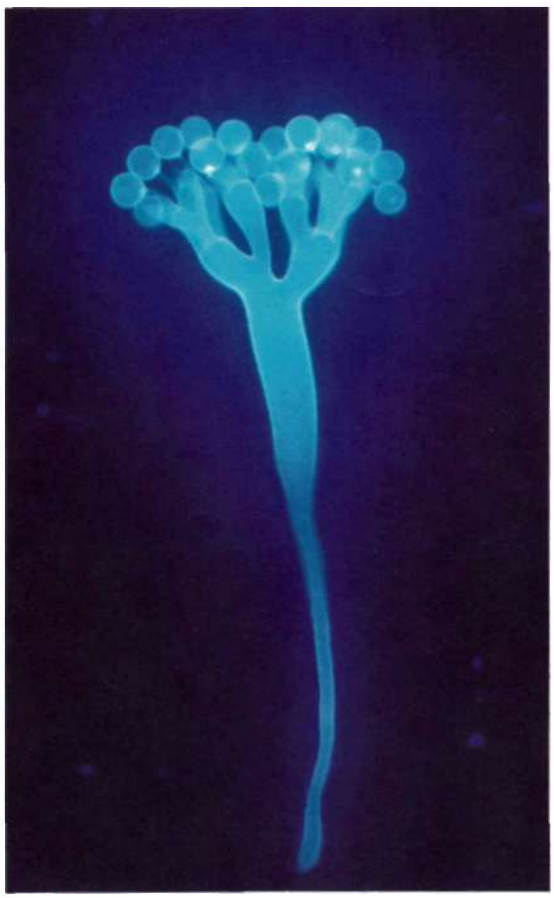

*Figure 4. A mature conidiophore of* Peronosclerospora sorghi *showing the basal cell, the main body of the conidiophore, and the conidia attached to the sterigmata (x375).*

The main axis has a diameter of 15-20 µm, and is usually less than, or equal to, the basal cell in length (80-150 µm from the septum of the basal cell to the beginning of the branch system). Branching of the conidiophore is by a succession of short, stout dichotomies usually involving primary, secondary, and tertiary branches terminating in tapering sterigmata approximately 13 µm long. The branches are arranged so the conidia borne on the sterigmata lie approximately in a hemispherical plane. The conidia are sub-orbicular (21-24.9 µm x 19.0-22.9 µm), hyaline, with a thin wall, and germinate directly by a hyphal germ-tube (Fig. 5).



***Figure 5. Conidia germinate by means of a single, unbranched hyphal germ-tube (x300).***

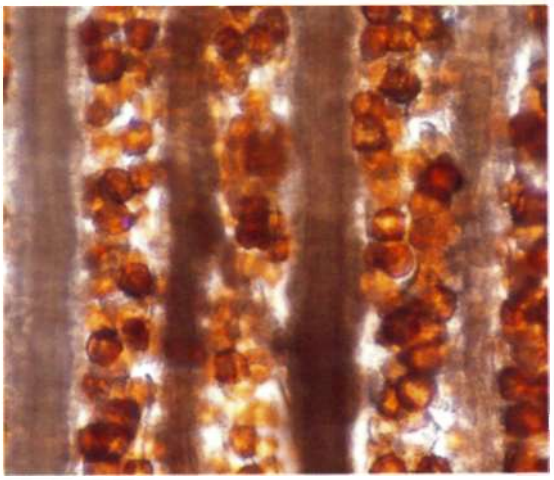

***Figure 6. Oospores of* Peronosclerospora sorghi *are typically produced in parallel bands between the fibro-vascular strands of the leaf (x 150).***

Oospores are produced within the leaf mesophyll between the fibro-vascular bundles (Fig. 6). They are spherical, the majority being 31.0-36.9 µm in diameter. The oospore wall is 1.1-2.7 µm thick and orange-yellow in color. The oospore contains finely granular material with masses of oil globules. The oospore germinates by means of an nonseptate, usually branched, hyaline germ-tube, averaging 4.4 µm in width.

*Peronosclerospora sorghi* is an obligate parasite. However, it has been successfully grown in dual culture with host tissue on a modified White's medium (Kaveriappa et al. 1980). Bhat and Gowda (1985) later described an improved method for obtaining contaminant-free dual cultures, but the inherent problems of maintaining these cultures has prevented them being widely used. Most culture maintenance therefore depends on inoculating seedlings of the host with conidia or oospores and using infected plants as a source of inoculum (Craig 1976).

## Disease symptoms

### Systemic infection

Systemic infection (Fig. 7) can manifest itself at any stage from about one week after seedling emergence. The symptoms first appear as chlorotic areas emanating from the base of the first leaves showing the infection. This often covers half the lamina (the 'half-leaf symptom, Fig. 8). Progressively greater proportions of the lamina of younger leaves show this symptom until the whole lamina becomes chlorotic (Fig. 9). In cool, humid weather the asexual reproductive structures of the fungus, i.e., the conidiophores and conidia, form during the night on the leaves, particularly on the abaxial surfaces. This gives a white down-like appearance to the infected leaves (Fig. 10). As the plant ages white, chlorotic streaks develop from the base of the younger leaves (Fig. 11), these turn pale to reddish-brown as the interveinal tissue dies (Fig. 12), and oospores develop. As the streaks turn brown they start to shred into long strips, the lamina disintegrates along the fibro-vascular strands of the leaf thus resulting in typical 'leaf-shredding' symptoms (Fig. 13).



***Figure 7. Sorghum plants showing typical symptoms of systemic sorghum downy mildew infection. The leaves are chlorotic with some pale streaking (arrowed) on the younger leaves; the leaves tend to be narrow and the plants have an upright habit.***



*Figure 8. The typical 'half-leaf symptom first manifested in a sorghum plant systemically infected with sorghum downy mildew. The lower part of the leaf is infected and chlorotic, while the upper portion remains green and uninfected.*



*Figure 9. The whole-leaf chlorosis that develops as the systemic infection progresses.*



*Figure 10. The downy appearance of leaves infected with sorghum downy mildew resulting from the asexual sporulation of* **Peronosclerospora sorghi.**



*Figure 11. The typical pale, chlorotic streaking of the younger leaves of a sorghum plant systemically infected with sorghum downy mildew. This symptom is indicative of the early stages of oospore production.*



*Figure 12. As the oospores mature the leaf streaking darkens and becomes brown in color.*



***Figure 13. 'Leaf-shredding' typically observed in older sorghum plants infected with sorghum downy mildew. As the oospores reach maturity the leaves start to shred along the fibro-vascular strands, releasing the oospores into air currents that probably allow them to be dispersed from the host.***

Plants that are systemically infected as seedlings remain stunted and often die. Sytemically infected plants are upright in habit, with narrow foliage, and are generally barren (Fig. 14), although some grain may be produced (Fig. 15). Occasionally a plant may recover and produce healthy, viable grain (symptom remission), but the basis for this phenomenon is unknown (Singh and de Milliano 1989b). The production of a normal grain-bearing panicle on a systemically infected plant has also been reported (Singh and de Milliano 1989a).

### Local lesions

The local lesion phase (Fig. 16) can occur on any leaf of an infected sorghum plant. Lesions develop as discrete chlorotic areas, variable in size, but generally elongate with parallel edges (1-4 mm x 5-15 mm). Asexual spores are produced mostly on the abaxial surface of leaves displaying these lesions.

## Epidemiology and biology

The life cycle of *P. sorghi* is shown in Figure 17. Conidia of P. *sorghi* are copiously produced, thin-walled, and ephemeral, they can cause the rapid build-up of an epidemic. Oospores are



***Figure 14. Generally plants systemically infected with sorghum downy mildew have barren panicles (arrowed), hence no yield is produced.***



***Figure 15. Systemically infected plants may have panicles that are partially fertile and produce some grain (arrowed). The amount of grain produced will depend on the proportion of the panicle colonized by the fungus.***

tough-walled and long-lived, and provide both a perennating stage for the pathogen and a mechanism for long-distance transport.

## Conidia production, dispersal, and infection

The infected plant must be subject to a preconditioning period of light for at least 4 h prior to producing conidia in the dark (Schmitt and Freytag 1974). The conidiophores subsequently form in the dark from within the stomata over a period of about 6 h (Lal 1981). During this time high relative humidity (RH) is essential. Shetty and Safeeulla (1981) showed that systemically infected sorghum leaves held in the dark at 20°C produced a maximum of 10,800 conidia $cnv^2$ at 100% RH, but only 3,600 conidia $cm^{-2}$ at 85% RH, and none at 80% RH. Leaf wetness is another important factor influencing conidia production. The optimum temperature for sporulation of *P. sorghi* on maize is 15-23°C (Bonde et al. 1985). Maximum spore germination occurs at 15°C, and the greatest germ-tube growth at 22°C, although germination is good at 10-19°C, and germ-tube growth is rapid at 14-22°C. A dew period of about 4 h and temperatures of 10-33°C support systemic infection (Bonde et al. 1978). Conidia are usually produced between midnight and 0500 h when the temperature is about 20°C and the RH exceeds 85% (Shenoi and Ramalingam 1979). Conidia are dispersed in air currents (Rajasab et al. 1979). On a leaf, the conidium germinates



***Figure 16. Typical local lesions on sorghum caused by infection with conidia of* Peronosclerospora sorghi. *The lesions can be seen as discreet tan areas on the leaf lamina.***

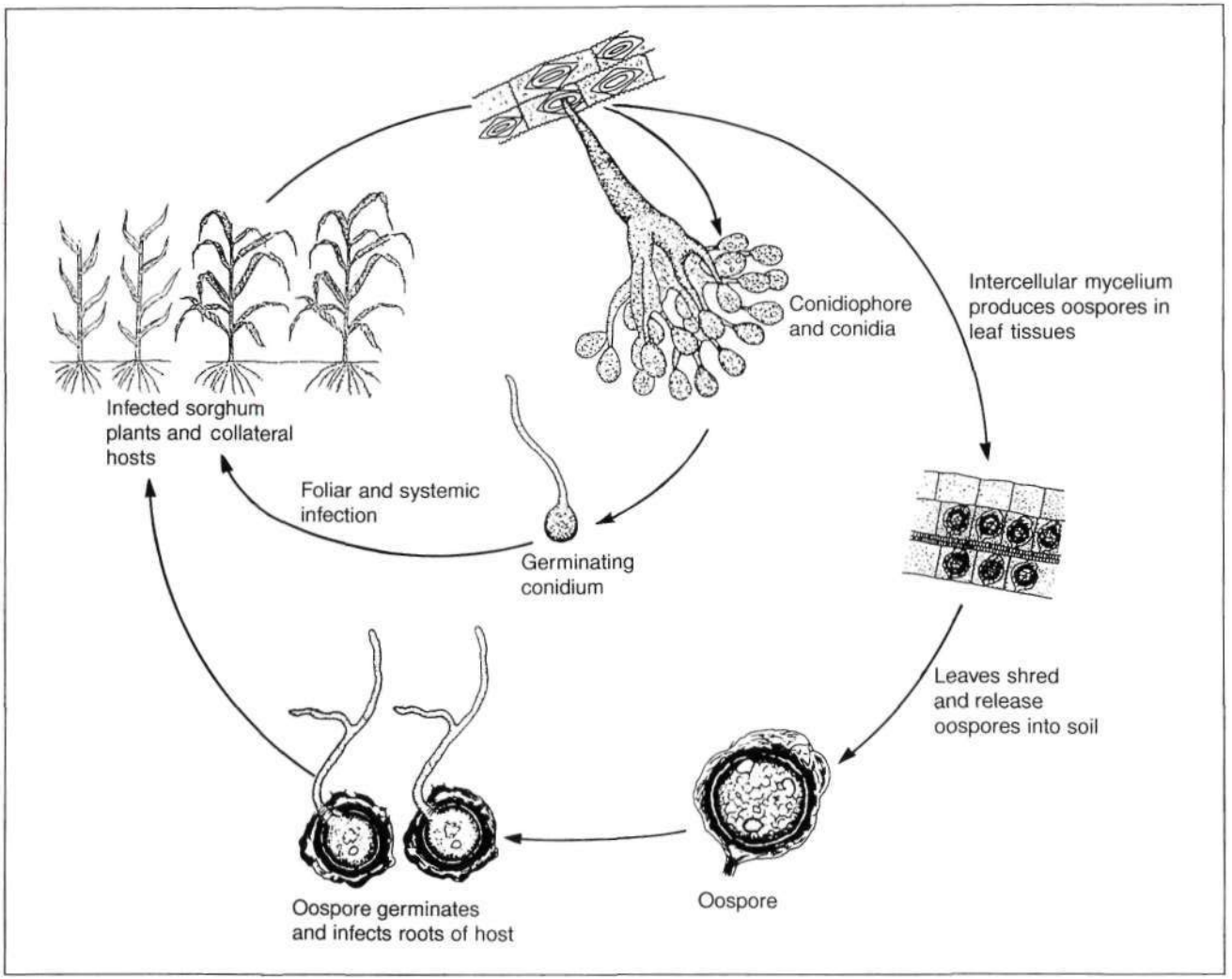


*Figure 17. The disease cycle of* **Peronosclerospora sorghi.** *Whereas sexually produced oospores will generally provide only one cycle of infection per season, the asexually produced conidia from an infected plant can infect fresh hosts within the same season thus allowing rapid build up of an epidemic of sorghum downy mildew.*

when mature, and the germ-tube grows at right angles to the long axis of the leaf until it encounters a stomata. An appressorium forms over the stomatal opening, and the penetrating structure enlarges to form an oval-shaped substomatal vesicle, that then gives rise to one or more infection hyphae (Jones 1971a). Sorghum plants are susceptible to systemic infection for only 2-3 weeks after emergence (Jones 1978). If a systemic infection develops, intercellular hyphae proceed to the apical meristem of the plant and invade the developing leaves and flowering parts. Symptoms can manifest themselves at any time up to flowering. Local lesion infections can occur at any stage, and take approximately 7 days to develop (Cohen and Sherman 1977).

At the point of host colonization, differences are observed between resistant and susceptible cultivars (Mauch-Mani et al. 1989). In resistant cultivars necrosis occurs at the penetration site. In susceptible cultivars systemic colonization proceeds with the progress of hyphal growth through the intercellular spaces of the mesophyll cells, and the development of haustoria with up to eight finger-like tubes.

## Oospore production, dispersal, and infection

Oospores of SDM develop subsequent to the fusion of oogonia and antheridia initials in the mesophyll of sorghum leaves (Safeeulla and Thirumalachar 1955). Oospores may be dispersed by man or on farm animals in soil adhering to their feet, or on farm implements (Williams 1984). They can survive passage through the bovine digestive tract, so dispersal in manure is implicated (Safeeulla 1976). Oospores can be seed-transmitted (Bain and Alford 1969), or may also be wind- (Bock et al. 1997) or water-dispersed (Rajasab et al. 1979). They can survive adverse conditions for several years (Safeeulla 1976). The highest incidence of infection is found in sandy-textured soils at temperatures of 24-29°C, and soil moisture contents of about 0.2 bar (Schuh et al. 1987). Host and nonhost roots can stimulate oospore germination (Pratt 1978). The germ-tube grows towards the meristematic region of the root where it forms an appressorium and infection peg (Safeeulla 1976).

## Collateral hosts

Collateral hosts, common in many areas where sorghum and maize crops are grown, can act as reservoirs of both conidial and oospore inocula. Several species of Graminae from the tribes Andropogonae, Maydae, and Panicae can be infected with *P. sorghi* (Table 2). Where collateral hosts are a source of inoculum for crops it may be feasible to consider roguing those plants in the vicinity of the cropping area.

## Seed transmission

Oospores can be dispersed on the surface of sorghum seed (Bain and Alford 1969). In sorghum and maize there is also evidence that oospores (Upadhyay 1987) and mycelium (Kaveriappa and Safeeulla 1978, Prabhu et al. 1983) of P. *sorghi* might be internally seedborne. Frederiksen (1980b) discussed ways in which oospore contamination and internal mycelial transmission of SDM could be avoided. Drying seed to below 20% moisture content, using healthy seed, producing seed in areas not prone to epidemics of SDM, breeding resistant hybrids, and observing strict quarantine measures are all practices that can be used to avoid seed infection. In laboratories where the technology is available, molecular probes and DNA hybridization can be used to test for seed transmission of graminaceous downy mildews (Yao et al. 1990).

## Seasonal disease development

To recommend control methods, an understanding of the factors that contribute to disease initiation and epidemic development is necessary. Oospores and conidia may vary in their importance in epidemic initiation and

**Table 2. Host range of *Peronosclerospora sorghi.***

| Host | Author |
|---|---|
| *Panicum trypheron* Shult. | McRae (1934) |
| *Pennisetum americanum* (L.) Leeke | Castellani (1939) |
| Para-sorghum sp. | Karunakar et al. (1994) |
| *Sorghastrum rigidifolium* Stapf. | Karunakar et al. (1994) |
| *Sorghum aethiopicum* (Hack.) Stapf. | Karunakar et al. (1994) |
| *Sorghum almum* Perodi. | Tarr (1962) |
| *S. arundinacium* (Willd.) Stapf. | Karunakar et al. (1994) |
| *S. bicolor* x *S. sudanense* (Piper) Stapf. | Futrell and Bain (1967) |
| *S. bicolor* (L.) Moench | Bonde and Freytag (1979). |
| *S. controversum* (Steud.) Snowden | Karunakar et al. 1994 |
| *S. drummondii* (Steud.) Millsp. & Chase | Karunakar et al. (1994) |
| *S. halepense* (L.) Pers. | Frederiksen et al. (1965) |
| *S. hewisonii* (Piper) Longley | Bonde and Freytag (1979) |
| *S. lanceolatum* Stapf. | Bonde and Freytag (1979) |
| *S. miliaceum* (Roxb.) Snowden | Karunakar et al. (1994) |
| *S. niloticum* (Stapf. ex Piper) Snowden | Bonde and Freytag (1979) |
| *S. plumosum* (R. Br.) Beauv. | Nagarajan et al. (1970) |
| *S. propinquum* (Kunth.) Hitch. | Bonde and Freytag (1979) |
| *S. pugionifolium* Snowden | Bonde and Freytag (1979) |
| *S. purpureo-serecium* (A. Rich.) Aschers. & Schwerf. | Karunakar et al. 1994 |
| *S. sudanense* (Piper) Stapf. | Nagarajan et al. (1970) |
| *S. verticilliflorum* (Steud.) Stapf. | Tarr (1962) |
| *S. controversum* (Steud.) Snowden | Bonde and Freytag (1979) |
| *S. usamberance* Snowden | Karunakar et al. (1994) |
| *S. versicolor* Anderss. | Bonde and Freytag (1979) |
| *S. virgatum* (Hack.) Stapf. | Nagarajan, et al. (1970) |
| Zea *mais* ssp. *mexicana* (L.) (Schrad.) litis. | Uppal and Desai (1932) |
| Zea mais (L.) | Bonde and Freytag (1979) |

build-up, depending on the particular region. In some areas initial infections may be through soilborne oospores. Provided conditions are favorable for conidial production, these early infections lead to an increased inoculum load of conidia later in the season, and this results in an increased incidence of SDM on late-sown crops. An increased risk of infection in late-sown crops has been illustrated in several regions, including parts of India (Ramalingam and Rajasab 1981) and Israel (Cohen and Sherman 1977). In other areas, including the USA, oospores are the major source of infection (Frederiksen et al. 1980a, Tuleen et al. 1980). Soil temperature, moisture and texture probably influence the incidence of systemic infections in these regions (Schuh et al. 1987). The presence of infected collateral hosts can also influence epidemic development.

## Variability within *P. sorghi*

*Peronosclerospora sorghi* can infect both sorghum and maize. However, strains that infect only maize, but not sorghum are recognized. The affiliations of these so-called maize strains' is unclear. Generally, as more information becomes available they are designated as separate species. A maize-infecting strain of the SDM pathogen in Rajasthan was eventually shown to be a different species and designated *P. heteropogonii* (Siradhana et al.. 1980). Similarly, recent studies suggest that a maize pathotype from Thailand should be given

specific rank as *P. zeae* (Micales et al. 1988, Yao 1991, Bonde et al. 1992). Another maize-infecting downy mildew currently considered a maize pathotype of the SDM pathogen occurs in Nigeria (Fajemisin 1980, Anaso et al. 1987).

Morphological variation between strains of *P. sorghi* is limited (Bock 1995). There is little adaptation to specific environments as suggested by the environmental requirements of a range of isolates from diverse locations (Bonde et al. 1985, Bock 1995).

The first indication of pathogenic variability on sorghum was observed in the USA in the late 1970s (Craig and Frederiksen 1980). A previously resistant hybrid became susceptible to SDM. Subsequently, three distinct pathotypes have been identified in the USA by the differential reaction of the varieties Tx 412, Tx 430, CS 3541, and QL 3 (Table 3, Craig 1983, Craig and Frederiksen 1983). Other pathotypes do occur, and have been identified in Brazil (Fernandes and Schaffert 1983), Honduras (Craig and Odvody 1992), and Zimbabwe (de Milliano and Veld 1990). Pawar et al. (1985) tested 75 sorghum varieties for their reaction to 16 isolates from different geographic regions. They found a differential reaction that identified each isolate as a different pathotype. Those from Africa (Nigeria and Ethiopia) and Asia had greater virulence than those from the Americas.

# Control

## Chemical control

Prior to the late 1970s several different fungicides had been tested in attempts to control the graminaceous downy mildews (Singh et al. 1970). None of these had proved effective. However, the discovery of the acyl-alanine fungicide metalaxyl [N-(2-methoxyacetyl)-N-(2,6-xylyl)-DL-alaninate] revolutionized the chemical control of SDM (Schwinn 1980). In India, seed treatments of 1 g ai $kg^{-1}$ of seed plus a foliar spray of 1 g ai $L^{-1}$ 40 days after emergence (DAE) or foliar sprays of 2 g ai $L^{-1}$ at 10, 20 or 40, and 50 DAE gave complete control of systemic SDM (Venugopal and Safeeulla 1978, Anahosur and Patil 1980). Seed treatment alone did not fully protect the plant or nodal tillers from systemic infection, and the spray regime was required to prevent a low incidence of late systemic infection and to control local lesions. At some locations concentrations of metalaxyl as low as 0.05 g ai $kg^{-1}$ seed can give complete control of systemic SDM, and in some cases concentrations greater than 1 g ai $kg^{-1}$ seed can cause seedling death (Odvody and Frederiksen 1984). There is a risk that SDM may develop resistance to metalaxyl, a view supported by the fact that other oomycetes have developed resistance to this fungicide

**Table 3. Identification of pathotypes i, II and III of *Peronosclerospora sorghi* in the USA by the differential reaction of four sorghum inbred lines'.**

| | Reaction to infection | | |
|---|---|---|---|
| Sorghum variety | Pathotype I[2] | Pathotype II | Pathotype III |
| Tx 412 | S | S | S |
| Tx 430 | R | R | S |
| CS 3541 | R | s | s |
| QL 3 | R | R | R |

1. Source: Craig and Frederiksen 1983
2. S = susceptible, and R = resistant to infection.

(Georgopoulos and Grigoriu 1981, Bruck et al. 1982).

Soil sterilization has also been shown to be effective in reducing infection through soil-borne oospores, but is probably not economic (Matocha et al. 1974).

## Cultural control

### Crop rotation

Roots of host and non-host plants trigger germination of oospores (Pratt 1978) and bait crops' (e.g., *Linum usitatissimum*) grown in infested soil can reduce the incidence of infection in susceptible sorghum crops sown in the same soil (Tuleen et al. 1980). This may be of value where oospores are the principal source of inoculum and soils are heavily infested.

### Deep tillage

Both the incidence of SDM and the oospore content in the upper strata of infested soil are reduced by deep tillage (Tuleen et al. 1980, Janke et al. 1983). However, it is an expensive operation, and probably not cost-effective as a means of control.

### Over-sowing and rouging diseased plants

Sowing at seed rates of up to 50% more than the recommended agronomic optimum can lead to an acceptable plant density of healthy plants remaining at harvest after stand losses due to 20-30% disease incidence (Frederiksen et al. 1973).

### Sowing date

In areas where the asexual inoculum rapidly increases as the season progresses it is expedient to sow early. In Israel early-sown crops avoided the disease (Cohen and Sherman 1977). However, Tuleen et al. (1980) found a lower incidence of the disease in later-sown crops in the USA, where oospores are the principal source of infection.

### Effect of host plant nutrition

There are conflicting reports about the effect of nutrition on the incidence of systemic SDM (Balasubramanian 1973, Gupta and Siradhana 1978). The effect of nutrition is probably associated with such other factors as plant age, inoculum type and pressure, and other variables.

### Biological control

A chytrid fungus (*Gaertennomyces* sp.) was found to be effective in reducing the incidence of systemic infection in soils heavily infested with oospores by as much as 58% (Kunene et al. 1990). However, field application of this organism has not been investigated and it is unlikely bio-control of SDM by this means will become a practical reality in the near future. Other potential bio-control agents have also been reported although they have not been studied in detail (de Diaz and Polanco 1984, Lakshmanan et al. 1990).

### Host-plant resistance

The cultivation of disease-resistant varieties is probably the most cost-effective and feasible long-term method for the control of SDM. Breeding disease-resistant varieties requires both the identification of a stable source of resistance, and its subsequent utilization in a breeding program. Breeding programs aim to incorporate resistance into a variety with high yield potential, good grain quality, and other agronomic traits that are acceptable in the relevant consumer market.

### Identification of resistance

By 1988 the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) had screened 13,101 sorghum accessions from 73 countries using a field-based screening system at Dharwad, India. Of these 46 accessions from geographically diverse sources were resistant to *P. sorghi* (Table 4). Using similar field-screen-

**Table 4. Origin and number of accessions from the world collection of sorghum germplasm screened in the field from 1981-88 and found to be resistant to sorghum downy mildew at Dharwad, Karnataka, India[1].**

| Origin | Number of countries | Number of lines screened | Number of lines resistant |
|---|---|---|---|
| Eastern Africa | 7 | 3345 | 18 |
| Western Africa | 14 | 3324 | 4 |
| Southern Africa | 9 | 930 | 2 |
| Middle East | 8 | 252 | 0 |
| Indian Subcontinent | 5 | 3403 | 12 |
| Southeast Asia and the Far East | 8 | 218 | 0 |
| North and Central America | 7 | 1476 | 4 |
| South America | 3 | 19 | 0 |
| Europe | 9 | 71 | 0 |
| Eastern Europe | - | 40 | 0 |
| Australia and Oceania | 2 | 23 | 6 |
| Total | 73 | 13101 | 46 |

1. Eastern Africa: Ethiopia, Kenya, Sudan, Somalia, Tanzania, Uganda, Zaire.
Western Africa: Benin, Burkina Faso, Cameroon, Congo, Central African Republic, Chad, Ghana, Gambia, Côte d'Ivoire, Mali, Nigeria, Niger, Sierra Leone, Senegal.
Southern Africa: Angola, Botswana, Lesotho, Malawi, Malagasy Republic, South Africa, Swaziland, Zambia, Zimbabwe.
North Africa and the Middle East: Egypt, Israel, Iran, Iraq, Lebanon, Syria, Saudi Arabia, Yemen.
Indian Subcontinent: Afghanistan, Bangladesh, India, Nepal, Pakistan.
Southeast Asia and Far East: China, Indonesia, Japan, Myanmar, Philippines, South Korea, Taiwan, Thailand.
North and Central America: Cuba, El Salvador, Guatemala, Mexico, Nicaragua, USA, West Indies.
South America: Argentina, Uruguay, Venezuela.
Europe: Belgium, Cyprus, France, Greece, Hungary, Italy, Portugal, Spain, Turkey.
Eastern Europe.
Australia and Oceania: Australia, Papua New Guinea.

ing techniques more than 3000 germplasm accessions and 2000 breeding lines were again evaluated by ICRISAT at Dharwad. Of these, 151 germplasm accessions and 334 breeding lines were resistant to SDM. At Matopos, Zimbabwe, ICRISAT screened 4500 sorghum entries, and found 1008 resistant. Over 5000 sorghum and maize entries were screened at Golden Valley, Zambia, where 928 sorghums were found resistant (S Pande, unpublished).

The International Sorghum Downy Mildew Nursery (ISDMN) was established in 1976 to screen sorghum germplasm to identify stable resistance and differences in pathogen virulence between locations (Williams et al. 1980). The results of multilocational testing of selected resistant sorghum accessions at ISDMN test locations are shown in Table 5. Although the results of the ISDMN did not initially indicate pathogen variability of *P. sorghi* on sorghum, the existence of pathotypes of *P. sorghi* on sorghum was subsequently reported (Craig and Frederiksen 1983, Pawar et al. 1985). Pathogen variability underscores the need to identify stable resistance to this pathogen.

## Screening techniques

### Field screening

Several field-screening techniques have been used to identify resistance to SDM in sorghum and maize (Anahosur and Hegde 1979, Cardwell et al. 1993). A method that challenges resistance to both conidia and oospores is optimal if environmental conditions permit. In places where oospores are the most important component of epidemics, soils heavily infested with oospores are used to screen breeding ma-

**Table 5. Sorghum downy mildew incidence in selected resistant accessions in the International Sorghum Downy Mildew Nursery during, 1976-86.**

| | | Highest disease incidence at locations (% plants infected) | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Entry | Origin | Manfredi[a] | Pergamino[a] | Jaboticabal[b] | Samaru[c] | Dharwad[d] | Mysore[d] | Texas[e] |
| IS 1317 | Tanzania | -[2] | 0(1)[3] | - | - | 3(5) | 0(3) | 0(1) |
| IS 2132 | USA | - | 0(1) | - | - | 0(5) | 3(3) | 0(1) |
| IS 2204 | India | - | 0(1) | - | - | 0(5) | 6(5) | 0(1) |
| IS 2473 | USA | - | 0(1) | - | - | 3(5) | 6(3) | 0(1) |
| IS 2482 | USA | - | 0(1) | - | - | 2(5) | 6(5) | 0(1) |
| IS 3443 | Sudan | 0(3) | 3(4) | 0(1) | 0(1) | 7(5) | 6(6) | - |
| IS 3546 | Sudan | - | 0(1) | - | - | 1(5) | 6(3) | 0(1) |
| IS 3547 | Sudan | 0(3) | 0(4) | 0(1) | 0(2) | 0(6) | 0(3) | - |
| IS 4696 | India | - | 0(1) | - | - | 0(5) | 0(2) | 0(1) |
| IS 5616 | India | - | 0(1) | - | - | 4(5) | 0(3) | 0(1) |
| IS 5628 | India | - | 0(1) | - | - | 5(5) | 0(2) | 0(1) |
| IS 5651 | India | - | 0(1) | - | - | 4(5) | 0(2) | 0(1) |
| IS 5665 | India | - | 0(1) | - | - | 0(5) | 0(2) | 0(1) |
| IS 5743 | India | - | 0(1) | - | - | 0(5) | 0(2) | 0(1) |
| IS 7528 | Nigeria | 0(3) | 0(4) | 0(2) | 0(2) | 5(5) | 14(3) | - |
| IS 8185 | Uganda | 0(3) | 0(4) | 0(2) | 0(2) | 3(6) | 15(5) | - |
| IS 8283 | Uganda | 6(2) | 0(2) | 0(1) | 0(1) | 2(5) | 3(5) | - |
| IS 8607 | Uganda | 3(3) | 0(4) | 0(2) | 0(2) | 5(6) | 13(6) | - |
| IS 14387 | Zimbabwe | - | 0(1) | - | - | 0(5) | 2(2) | 0(1) |
| IS 18757 | Australia | 0(4) | 0(5) | 0(2) | 0(2) | 0(11) | 0(11) | - |
| IS 22227 | Australia | 0(4) | 0(5) | 0(1) | 0(2) | 0(7) | 12(4) | - |
| IS 22228 | Australia | 0(4) | 0(5) | 0(1) | 0(1) | 4(7) | 11(4) | - |
| IS 22229 | Australia | 0(4) | 0(5) | 0(1) | 0(1) | 0(7) | 28(4) | - |
| IS 27042 | India | 0(2) | 0(3) | - | 0(1) | 0(5) | 7(5) | - |
| Susceptible control | | | | | | | | |
| DMS 652 | India | 100(4) | 36(5) | 9(2) | 100(2) | 100(5) | 100(5) | 45(1) |

1. Locations: a. Argentina, b. Brazil, c. Nigeria, d. India, and e. USA.
2. - not tested at that location.
3. Numbers in parentheses indicates number of years tested at each location.

terial. The oospore content of the soil is bulked up by incorporating infected crop residues from the previous season. However, even in some of these areas host reaction to conidial inoculum is checked by artificial inoculation in the greenhouse (Frederiksen 1980a). A large-scale field-screening technique utilizing primarily windborne conidia of *P. sorghi* was developed at ICRISAT (Pande and Singh 1992). The system was designed so that conidial showers were blown by wind from infector rows onto test materials (Fig. 18). The two critical aspects for the successful use of this technique are the establishment of disease infector rows, and favorable temperatures and humidities for abundant conidial production from the systemically infected sorghum plants in the infector rows. Ideally therefore, screening should be conducted at locations known to be favorable for the disease, as at Dharwad, Golden Valley, and Matopos. It is desirable to use the same field for several years to encourage an accumulation of oospore inoculum in the soil that will act as an additional source of infection.



***Figure 18. The spreader-row technique employed by breeders to ensure effective screening of sorghum germplasm against sorghum downy mildew. Note the older infector rows sown to the left and right of the four rows of test material. The infector rows were planted about three weeks prior to the test material.***

The field-screening technique has the following components:

1. Source and off-season maintenance of inoculum. Infected material for inoculation of the spreader rows may come from different sources. Infected volunteers or collateral hosts may be readily available in the vicinity (Pande and Singh 1992). Alternatively, infected plants can be maintained by the breeding program throughout the season. A simple and effective way of maintaining infected plants is to inoculate sorghum seedlings using the 'sandwich' technique. The procedure can be done in petri dishes lined with moist filter paper to maintain high RH. Pre-germinated seedlings (with 0.5-1.0 mm long plumules and radicles) are incubated by placing them on the adaxial surface of a piece of systemically infected leaf, and covering them with another section of systemically infected leaf (adaxial surface touching the seeds), thus 'sandwiching' the seedlings (Fig. 19). They are placed in petri dishes and incubated in darkness at 18-20°C for 12-16 h. After this period sporulation, germination, and infection will have occurred. If incubators and petri dishes are not available, but environmental conditions at night are favorable for SDM, this seedling inoculation technique can be modified (Pande and Singh 1992). Germinated seeds can be either spread on a polythene sheet (1-2 m x 0.75 m) or on seedbeds (2 m x 1 m). Systemically infected leaves are laid abaxial surface down over the seed-

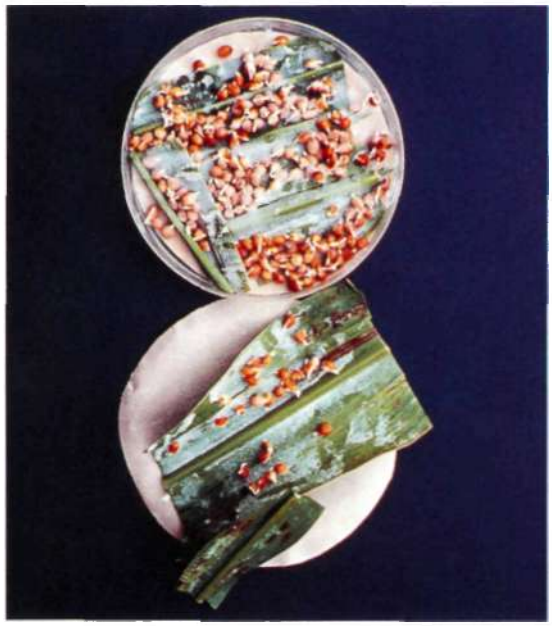

*Figure 19. Inoculation of pre-germinated sorghum seedlings using the 'sandwich' technique. The seedlings are placed between the abaxial sides of sorghum leaves systemically infected with sorghum downy mildew and incubated in conditions conducive to sporulation and infection.*

lings, and covered with moist newspaper or gunny bags and polythene sheets to maintain high RH.

2. **Establishing infector rows.** The most important components of the ICRISAT SDM screening technique are the infector rows. To establish these, seedlings of a susceptible variety (DMS 652, IS 643, Marupantse, Sugardrip, or a locally available susceptible variety or landrace) are inoculated using the sandwich technique. Immediately after inoculation the pre-germinated seedlings are sown in the spreader row area, as every 5th or 10th row, depending on prevailing wind conditions.

3. **Sowing test materials and indicator rows.** Once the systemically infected plants in infector rows are established and show heavy sporulation (20-25 days after sowing), the test material should be sown. Test lines are sown in the intervening rows between the spreaders. The indicator rows are sown to the same SDM-susceptible variety as the infector rows, alongside the test material at regular intervals (generally every 10-20 rows). Indicator material provides a measure of the disease pressure in the nursery.

   It is also useful to include genotypes with variable levels of resistance as controls in screening trials. Williams et al. (1981) used a sprinkler system to increase humidity and provide optimal conditions for asexual sporulation of the infector rows when screening for resistance to pearl millet downy mildew. This method can also be applied to SDM screening.

4. **Evaluating resistance.** To compare host reactions it is necessary to develop an effective (both accurate and precise) assessment method. Scoring systemic infection is straightforward. The incidence of systemically infected plants in the indicator rows and test material should be recorded on at least two occasions during the season (at seedling and flowering stages) to provide a realistic estimate of disease incidence (Williams 1984). Sorghum lines with no more than 5% systemically infected plants are regarded as resistant. Various severity scales have been developed to assess local lesions. A 1-5 scale has been used to score this type of infection (Singburaudom and Williams 1978, Frederiksen 1980a) and Shenoi and Ramalingam (1976) developed a 1-4 scale to assess the severity of local lesion infection on individual leaves.

**Greenhouse and laboratory screening**

Several greenhouse and laboratory screening techniques using both oospores and conidia have been standardized (Jones 1971b, Craig 1983, Naryana et al. 1996). Oospores can be incorporated into pot soil at a known rate, the test material then sown in the container, and the subsequent development of systemic infection assessed (Craig 1983). Alternatively,

conidia could be used as the source of inoculum to infect the plants. Conidia are produced on pieces of systemically infected sorghum leaves incubated in the dark for 6-7 h at 18-20°C and >90% RH (Fig. 20). Conidia are harvested from the leaves by gently brushing them into cold distilled water (to prevent conidial germination) using a paint brush (Fig. 21). The conidial suspension is adjusted to $1x10^5$ conidia $mL^{-1}$. The suspension is generally applied to seedlings at the coleoptile to one-leaf stage using a hand-held sprayer (Fig. 22), although seedlings may simply be dipped in the suspension (Naryana et al. 1996). Spraying seedlings is a useful way to mass-screen breeding materials in the off-season. Alternatively, if inoculum quantity is crucial to the study, then a syringe-inoculation method can be used to accurately apply a known number of conidia (Fig. 23). In this technique a drop of inoculum is placed precisely at the apex of the 12-24 h old coleoptile and allowed to run off to the base. Once the seedlings have been inoculated they are incubated at 18-20°C and RH >90%



***Figure 21. Preparing a conidial inoculum of* Peronosclerospora sorghi *in cold water by brushing sporulated infected leaves.***



***Figure 20. Sporulation of* Peronosclerospora sorghi *conidia on pieces of systemically infected leaves incubated in a moist chamber.***



***Figure 22. The seedling spray inoculation method. When the coleoptiles are 1-2 cm long the seedlings are spray inoculated with a conidial suspension of* Peronosclerospora sorghi *until they are evenly coated with droplets.***

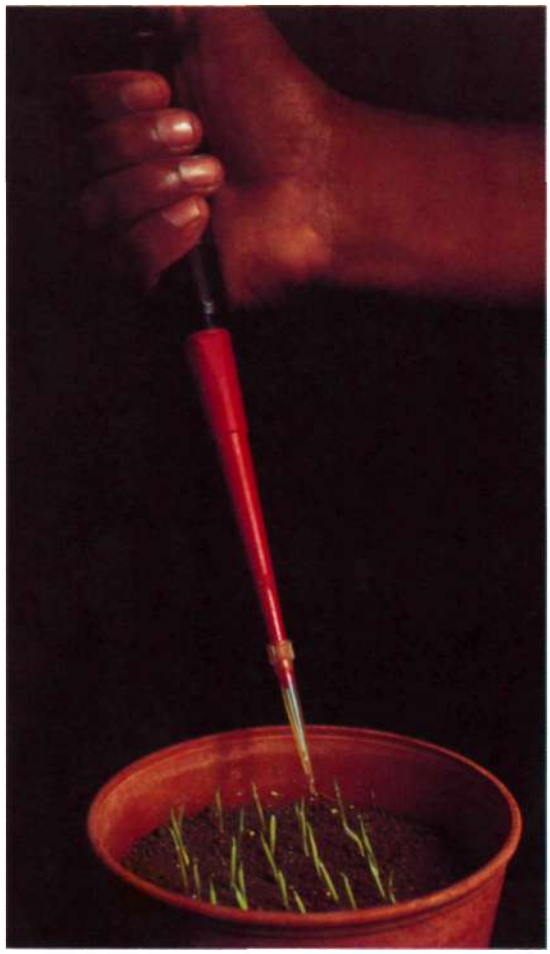

***Figure 23. The seedling droplet inoculation method. A pipette is used to administer a known volume of conidial suspension of* Peronosclerospora sorghi *to each coleoptile.***

for 12-16 h. They are subsequently transferred to greenhouse benches. Symptoms of systemic infections start to appear 8-12 days after inoculation, after 15 days they are obvious (Fig. 24).

A comparison of field and laboratory techniques undertaken at ICRISAT indicates controlled inoculation with conidia is the more discerning test for putative resistance to SDM (Table 6). Many sources of resistance have been reported using both field-screening and laboratory techniques (Frederiksen et al. 1973, Williams et al. 1982, Naryana et al. 1996).

## Inheritance of resistance

Knowledge of the inheritance of resistance, or at least a working knowledge of the ease of resistance transfer, is essential to a breeding program. Sorghum is a self-pollinated species which means genetic uniformity can be attained (Frederiksen et al. 1973). However, it can be induced to cross-pollinate. Studies of the inheritance of resistance in sorghum undertaken by various authors suggest that resistance is dominant to susceptibility (Rana et al. 1982, Sifuentes and Frederiksen 1988, Rosenow 1992, Thakur and Pande 1995) although some earlier workers found dominance of susceptibility (Puttarudrappa et al.



***Figure 24. Ten to fifteen days after inoculation of the seedlings the symptoms of systemic infection are clearly visible in susceptible lines and assessments can start.***

**Table 6. The incidence of sorghum downy mildew (SDM) on selected resistant varieties and a susceptible control using different methods of inoculation at ICRISAT Patancheru.**

| Sorghum accessions | Downy mildew incidence (% plants systemically infected) | | | | | |
|---|---|---|---|---|---|---|
| | Field exposure to conidia and oospores[1] | | Seedlings sprayed with conidial suspension | | Sprouted seeds dipped in conidial suspension | |
| | Mean | Max[2] | Mean | Max[3] | Mean | Max[3] |
| IS 1032 | 0 | 0 | 6 | 8 | 36 | 42 |
| IS 1317 | 1 | 3 | 4 | 5 | 4 | 8 |
| IS 1331 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 2132 | 0 | 0 | 5 | 8 | 26 | 29 |
| IS 2233 | 2 | 5 | 46 | 47 | 61 | 64 |
| IS 2234 | 3 | 4 | 67 | 82 | 71 | 73 |
| IS 2266 | 0 | 0 | 10 | 13 | 19 | 21 |
| IS 2474 | 0 | 0 | 0 | 0 | 5 | 10 |
| IS 3443 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 3547 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 3882 | 4 | 5 | 40 | 42 | 48 | 54 |
| IS 4298 | 3 | 5 | 39 | 44 | 36 | 43 |
| IS 5616 | 2 | 4 | <2 | 2 | 27 | 33 |
| IS 5628 | 2 | 5 | 6 | 8 | 10 | 13 |
| IS 5646 | 0 | 0 | 12 | 14 | 38 | 42 |
| IS 5665 | 1 | 2 | 5 | 6 | 63 | 67 |
| IS 5743 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 6094 | 1 | 2 | 18 | 23 | 14 | 18 |
| IS 6918 | 1 | 4 | 2 | 4 | 6 | 12 |
| IS 7179 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 7528 | 2 | 3 | 4 | 5 | 8 | 9 |
| IS 8185 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 8247 | 0 | 0 | 3 | 4 | 10 | 13 |
| IS 8274 | 2 | 4 | 5 | 9 | 8 | 8 |
| IS 8276 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 8283 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 8589 | 1 | 3 | 7 | 8 | 18 | 23 |
| IS 8607 | 1 | 2 | 2 | 3 | 1 | 2 |
| IS 8615 | <1 | 1 | 2 | 4 | 10 | 13 |
| IS 8864 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 8906 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 8954 | 0 | 0 | 0 | 0 | 2 | 4 |
| IS 14387 | 0 | 0 | 11 | 12 | 15 | 21 |
| IS 22228 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 22229 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 22230 | 0 | 0 | 0 | 0 | 0 | 0 |
| IS 27042 | 0 | 0 | 7 | 10 | 27 | 38 |
| Susceptible control DMS 652 | 87 | 88 | 100 | 100 | 100 | 100 |

1. Mean percentage incidence of SDM over 5-7 years.
2. Mean maximum incidence in 5-7 years.
3. Maximum percentage incidence from 4 replications.

1972, Miller 1966). Craig and Schertz (1985) illustrated that the resistance to SDM expressed by the inbred line SC 414-12 was conferred by a single dominant gene. This resulted in an incompatible host-pathogen interaction that inhibited pathogen development and sporulation on inoculated leaves. Gimenes-Fernandes et al. (1984) found that resistance was conferred by one or two dominant or partially dominant genes that were different. Sifuentes and Frederiksen (1988) investigated the inheritance of resistance to three pathotypes of *P. sorghi.* Their results indicated that the resistant variety QL 3 has two dominant genes conditioning resistance to each of the three pathotypes. Reddy et al. (1992) found resistance in QL 3 dominant to susceptibility: a two-loci model with independent segregation, and a combination of complementary and inhibitory inter-allelic interaction appeared to be the most appropriate in explaining the inheritance pattern they observed. Further investigations are needed to characterize the inheritance of resistance to SDM in more sorghum lines, and the mechanisms of this resistance, which remain poorly understood. Resistance, preferably of a durable nature needs to be incorporated into agronomically suitable varieties. Symptom remission has been observed in systemically infected plants. This might be another resistance mechanism that could be utilized (Singh and de Milliano 1989a and b).

### Resistance sources in breeding, and resistant varieties used by farmers

Sorghum downy mildew resistant lines are being successfully used at ICRISAT to breed SDM-resistant varieties and hybrids. Other breeding programs including the one at Texas A&M University in the USA also make use of SDM-resistant sources in their sorghum breeding programs (Frederiksen et al. 1973). In the USA varieties resistant to SDM have been commercially available since the early 1970s. At ICRISAT large numbers of crosses involving QL 3, IS 3443, and IS 8283 as resistant parents have been made. Several SDM-resistant varieties and populations have been developed at Dharwad (Anahosur 1992) and in various southern African countries (de Milliano 1992).

### Integrated control

Sorghum downy mildew lends itself to integrated control that involves the use of two or more methods of control to bring about a reduction in disease incidence (Odvody et al. 1983). A good knowledge of disease epidemiology and control options available to farmers in a given area are needed before an integrated package can be implemented (Fig. 25). Integrated control might involve chemical control (e.g., metalaxyl seed treatment), cultural control (e.g., deep plowing or use of crop rotation), and the use of host-plant resistance. The combination of control methods can be mutually beneficial. For example, Odvody and Frederiksen (1984) suggested the use of a resistant variety and seed treatment could extend the life of the host resistance and prevent development of fungicide resistance in the pathogen. The final methods chosen must depend on their effectiveness in a particular situation, and on the farmers' ability to use them.

## Technology Transfer and Adoption

Technology transfer is an extension activity in which products of scientific investigation in any area of crop improvement are applied to practical use in other, usually distant places. Technology transfer is an ICRISAT mandate. In this case the intended products are the SDM resistance screening techniques that have been transferred to other locations by the following methods:

1. Scientists from locations where the SDM resistance screening technology is required are trained in the methods at ICRISAT where the screening techniques were developed. The scientists then establish the technology at their own locations on their return.

2. The SDM resistance-screening techniques are demonstrated at the location where the scientists are based and the technology is required. Locally available resources are used and adapted as necessary.

The second method has proved the more successful in transferring SDM screening techniques. Individuals receiving training in a familiar environment appear to gain confidence by modifying, as needed, the locally available facilities to establish the technique. Techniques have been successfully established in Zimbabwe and Zambia (Pande and Singh 1992). The sandwich inoculation component of the technique has been used to establish infector rows for in Rwanda. Components of the technique have been adopted by several other national programs, such as the All India Coordinated Sorghum Improvement Program at the University of Agricultural Sciences, Dharwad, Karnataka (K H Anahosur, personal communication). The reasons for the wide adoption of this technique are its simplicity, flexibility, and effectiveness.



***Figure 25. Sorghum downy mildew infected crop in a farmer's field near Kigali, Rwanda. An integrated disease management package could reduce crop losses in such situations.***

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# About ICRISAT

The semi-arid tropics (SAT) encompasses parts of 48 developing countries including most of India, parts of southeast Asia, a swathe across sub-Saharan Africa, much of southern and eastern Africa, and parts of Latin America. Many of these countries are among the poorest in the world. Approximately one-sixth of the world's population lives in the SAT, which is typified by unpredictable weather, limited and erratic rainfall, and nutrient-poor soils.

ICRISAT's mandate crops are sorghum, pearl millet, finger millet, chickpea, pigeonpea, and groundnut; these six crops are vital to life for the ever-increasing populations of the semi-arid tropics. ICRISAT's mission is to conduct research which can lead to enhanced sustainable production of these crops and to improved management of the limited natural resources of the SAT. ICRISAT communicates information on technologies as they are developed through workshops, networks, training, library services, and publishing.

ICRISAT was established in 1972. It is one of 16 nonprofit, research and training centers funded through the Consultative Group on International Agricultural Research (CGIAR). The CGIAR is an informal association of approximately 50 public and private sector donors; it is co-sponsored by the Food and Agriculture Organization of the United Nations (FAO), the United Nations Development Programme (UNDP), the United Nations Environment Programme (UNEP), and the World Bank.



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