Downy Mildew of Sorghum

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Introduction

Sorghum (*Sorghum bicolor* L. Moench.) is an important crop for human consumption and animal fodder in many areas of the world where semi-arid conditions prevail, including Africa, Asia, and the Americas. In 1994 sorghum was grown on 4.37×10^7 ha worldwide, which compares with 3.77×10^7 ha for pearl millet, the other major cereal grown in the semi-arid tropics (FAO Agrostat-PC, 1991-1996). The sorghum plant has good drought tolerance and produces a yield where other crops such as maize may succumb due to lack of moisture. Sorghum is also an ancient crop, having been cultivated for at least the last 5000 years. It was probably originally domesticated in northeastern Africa (Mann et al. 1983).

Sorghum suffers from many diseases, several of which have an adverse effect on yield. Investment in studying these pathogens can lead to disease control through well-informed disease management strategies. This in turn contributes to sustainable food production in epidemic-prone areas, particularly those that support a burgeoning population. Sorghum downy mildew, or SDM, (*Peronosclerospora sorghi* (Weston and Uppal (C G Shaw)) infects both sorghum and maize (*Zea mays* L.) and has caused serious economic yield losses in these crops. A major investment was made during the 1960's-1980's in an attempt to control this disease.

Twelve years ago Williams (1984) provided an excellent and comprehensive review of the different graminaceous downy mildews, including *P. sorghi*. This
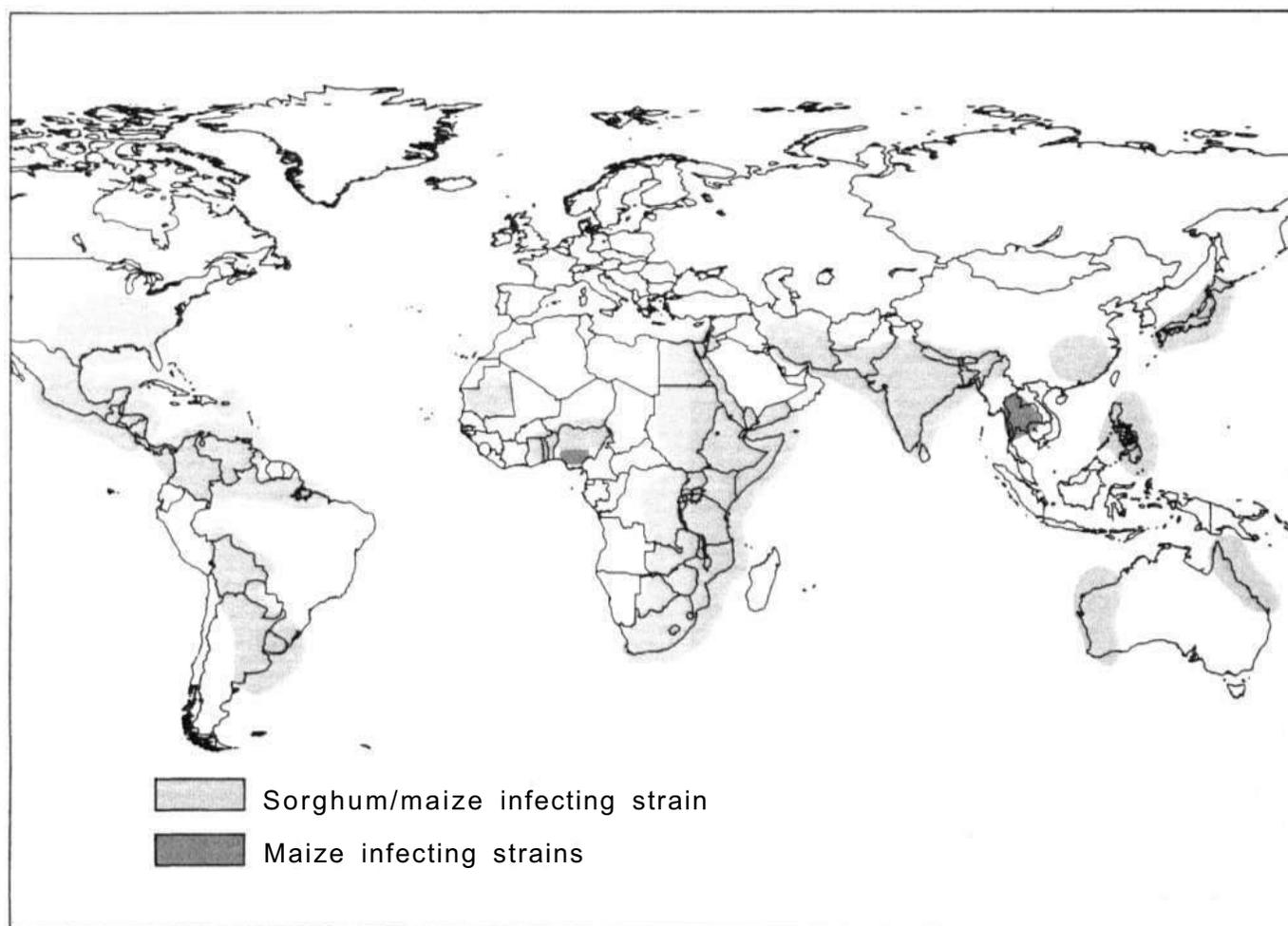

Figure I. Geographical distribution of *Peronosclerospora sorghi* showing regions where the sorghum/maize- and maize-infecting strains occur (the maize strain in Thailand has recently been designated specific rank as *P. zeae;* Yao 1991).

Legend:
- Sorghum/maize infecting strain
- Maize infecting strains

report presents our current state of knowledge of SDM with respect to sorghum, taking into account advances since the Williams review. Infection of maize or other hosts is only considered where deemed necessary, particularly if information is lacking on a particular aspect for sorghum.

Crop loss

Sorghum downy mildew is particularly destructive because systemic infection of the host generally results in a barren infloresence. The effect of infection on yield is best illustrated by reference to several reports in the literature. 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 10^5 t. In Venezuela, crop loss was so severe in the early 1970's that a national emergency was declared (Frederiksen and Renfro 1977). In Israel both forage sorghum and maize were severely infected with incidences of up to 50% (Kenneth 1976), and in the USA incidences of 90% have been reported (Frederiksen et al. 1969). The effect of systemic SDM is now more clearly understood, since models have been developed that show a linear relationship between incidence of systemic SDM and yield loss at normal sowing densities (Craig et al. 1989; Frederiksen et al. 1973; Tuleen and Frederiksen 1981).

Taxonomic history

A downy mildew infecting sorghum was first mentioned in India by Butler (1907), who considered it to be *Sclerospora graminicola.* Subsequently, Kulkarni (1913) observed the asexual phase germinated directly by means of a germ tube from conidia (rather than through zoospores from sporangia), and primarily on this basis he recommended designating it varietal rank - *S. graminicola* var. *Andropogonis-sorghi.* Further investigation of morphology and host range established the downy mildew infecting sorghum as *S. sorghi* (Weston and Uppal 1932). The differences observed in the mode of germination was deemed sufficient to eventually designate the new genus *Peronosclerospora* to house those graminaceous downy mildews, including *P. sorghi,* that produced conidia (Shaw 1976, 1978, and 1980). The genus *Sclerospora* was retained for species that germinated by means of zoospores from sporangia.

Origins and geographic distribution

The geographic distribution of SDM is illustrated in Figure 1. The pathogen has been reported to infect sorghum, maize, or wild hosts in at least 43 countries (Table 1). It is thought to be an 'Old World' disease, originating in Africa or Asia (Shaw 1981; Williams 1984) and subsequently spreading to theAmericas in the 1950's, where it was probably introduced (Frederiksen 1980a; Toler et al. 1959).

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). The conidia are produced on the leaf surface on erect conidiophores which grow out through stomata. The conidiophore comprises a basal cell and a more or less complex, usually dichotomously branched, expanded top (Figure 4). The basal cell is knobbed or bulbous at the bottom, then of fairly uniform diameter (7-9 µm) for a length of approximately 100-150 µm. It is usually delimited from the main axis by a complete septum. The main axis has a diameter of 15-20 µm, and is usually 80-150 µm long from the septum to the beginning of the branch system. The conidiophore branches by a succession of short, stout dichotomies involving primary, secondary, and tertiary branches. These terminate in tapering sterigmata approximately 13 µm long. The branches are arranged so the conidia borne on the sterigmata lie in a hemispherical plane. Conidia are suborbicular (15.0-28.9 µm x 15.0-26.9 µm, most frequently 21.0-24.9 µm x 19.0-22.9 µm), hyaline, with a thin wall and germinate directly by a hyphal germ tube.
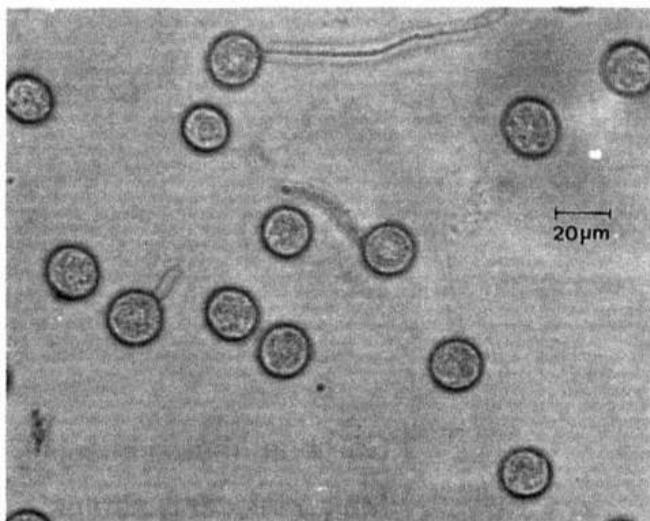

Figure 2. The asexual phase of *Peronosclerospora sorghi.* Conidia are sub-orbicular, hyaline and thin-walled. Some conidia are in the process of germinating by a single, unbranched hyphal germ tube.

Oospores are produced within the leaf mesophyll between the fibro-vascular bundles (Fig. 5). They are spherical, the majority being 31.0-36.9 μm in diameter, extremes range from 25.0-42.9 μm. The wall color is a light shade of Mars Yellow, and is 1.1-2.7 μm thick (extremes range from 0.3-4.3 μm). The oospore contains finely granular material with masses of oil globules. The oospore germinates by means of an unseptate, usually branched, hyaline germ tube, averaging 4.4 μm in width, extremes ranging from 2.5-8.3 μm.

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

Peronosclerospora sorghi is an obligate parasite. Recently, however, *P. sorghi* has been successfullygrown 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 and most culture maintenance depends on inoculating seedlings of the host with conidia or oospores and using infected plants as a source of inoculum.

Disease symptoms

These are well described (Frederiksen et al. 1973; Williams 1984). Two types of symptoms can develop as a

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	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 (Burtica 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 Disease, Map no. 179, Edition 5, Issued 1 Apr 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).
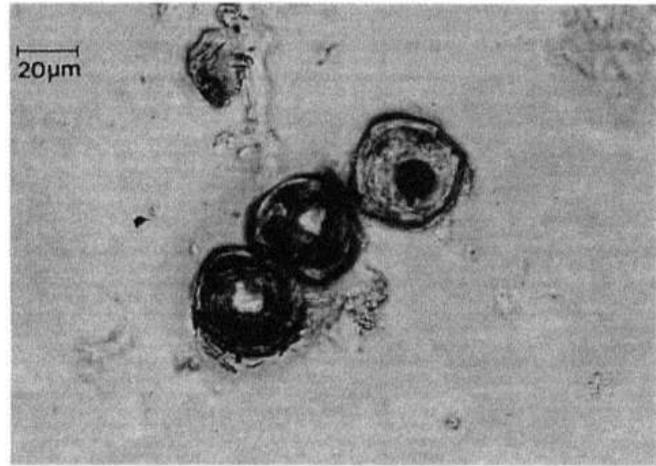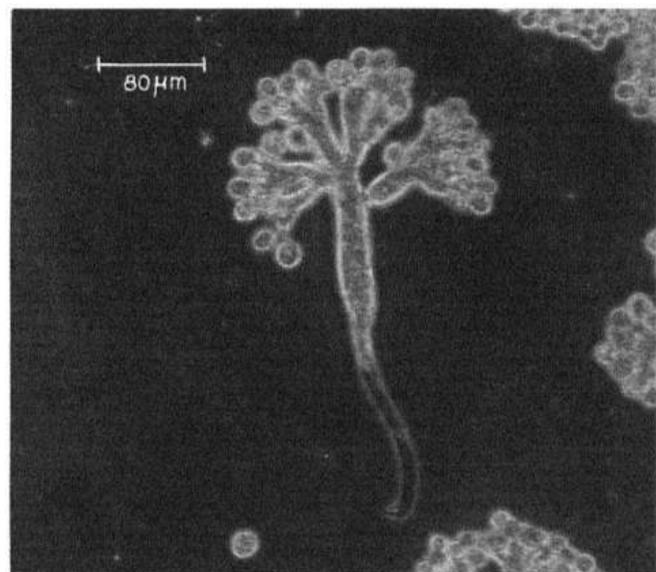

Figure 4. A mature conidiophore of *Peronosclerospora sorghi* showing the basal cell, the main body of the conidiophore and the conidia attached to the sterigmata.
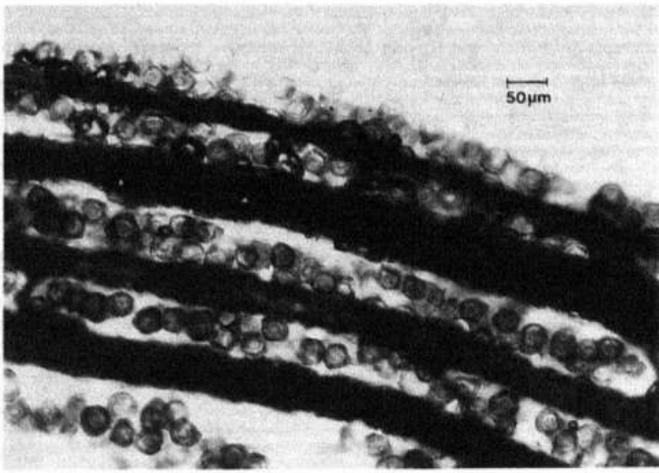
Figure 5. Oospores of *Peronosclerospora sorghi* are typically produced in parallel bands between the hbro-vaseular strands of the leaf.

Figure 6. A sorghum plant showing typical symptoms of systemic sorghum downy mildew infection. The leaves exhibit chlorosis with some pale streaking in the younger leaves; the leaves tend to be narrow, and the plant has an upright habit.

result of infection, either systemic symptoms (Fig. 6) resulting from an early infection and colonization of the growing point, or local lesions (Fig. 7) resulting from localized infection of the leaf lamina by conidia.

Systemic infection

Systemic infection resulting from infection by conidia or oospores can manifest itself at any stage from about one week after emergence. The symptoms are first seen as chlorotic areas emanating from the leaf base of the first leaves showing the infection. This chlorosis often covers half the lamina (called the 'half-leaf' symptom, Figure 8). Progressively greater proportions of the lamina of younger leaves show this symptom until the whole lamina is chlorotic. As the plant ages, white or pale yellow streaks develop from the base of the younger leaves (Fig. 9), which turn reddish brown as the inter-veinal tissue dies and the oospores develop. As the streaks turn brown they start to shred into long strips along the fibro vascular
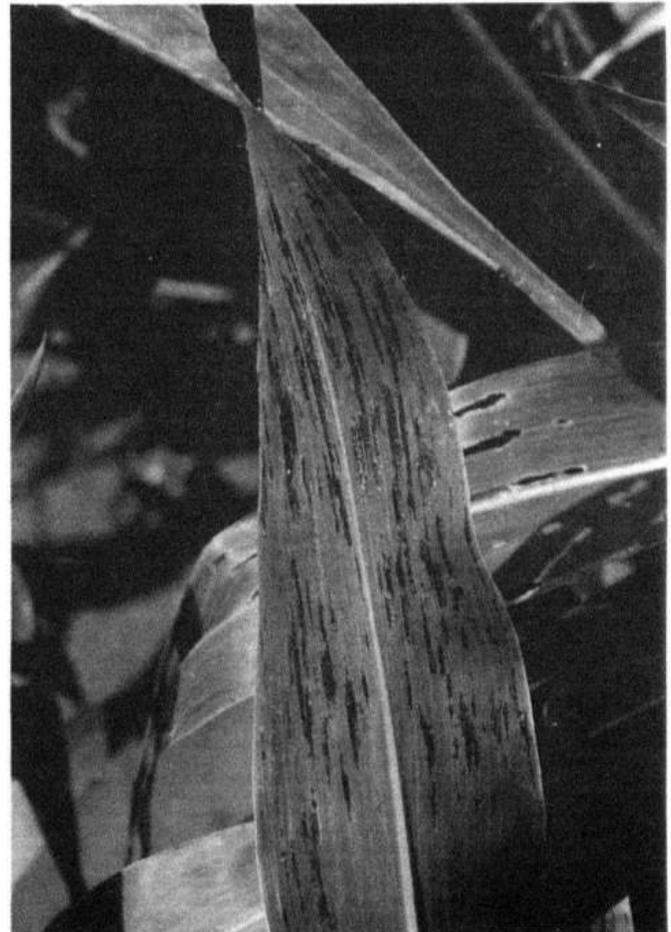

Figure 7. Typical symptoms of local lesions on sorghum caused by infection with conidia of *Peronosclerospora sorghi*. The local lesions can be seen as discrete chlorotic to purple-tan areas on the leaf lamina.
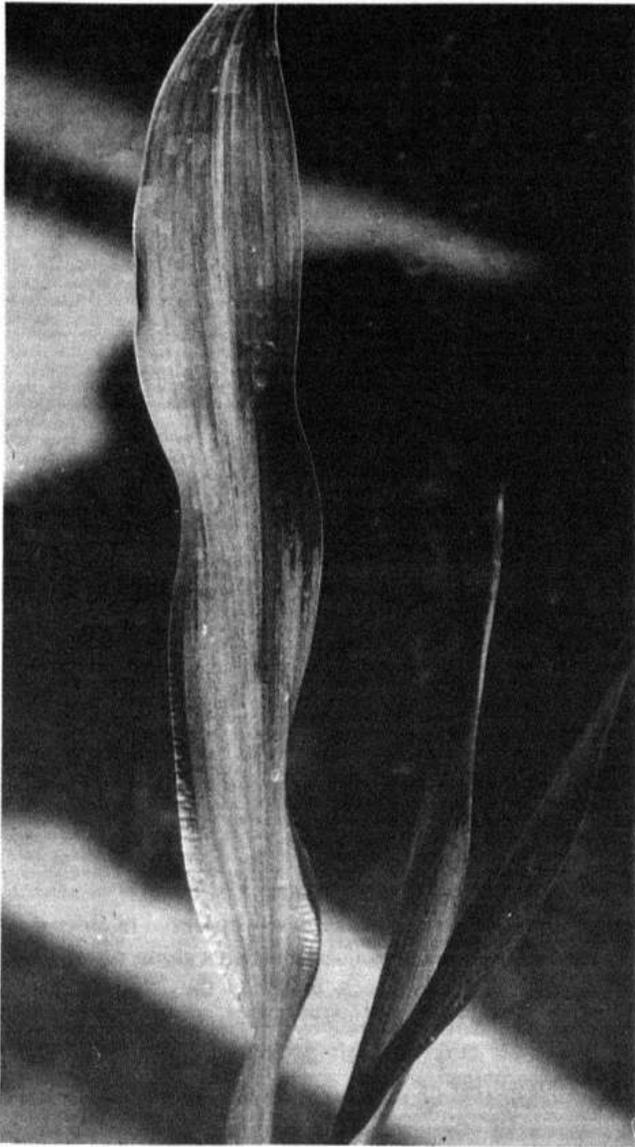
Figure 8. The typical 'half-leaf' symptom first manifested in a sorghum plant systemically infected with sorghum downy mildew. The lower part of the leaf is infected and chlorotic, while the upper portion remains green and non-infected.

strands of the leaf resulting in the symptoms of 'leaf-shredding' (Fig. 10). Plants that are systemically infected as seedlings can remain stunted and often die. Systemically infected plants are upright in habit, with narrowing of the foliage, and are generally barren, although some grain might be produced. Occasionally, a plant can recover and produce normal panicles with healthy, viable grain (symptom remission), but the basis for this phenomenon is unknown (Singh and de Milliano, 1989a and b).

Local lesions

Local lesions of SDM can occur on any leaf of a sorghum plant. Such lesions develop as discreet chlorotic to purple-tan areas, variable in size, but generally elongate with

parallel edges (1-4 mm x 5-15 mm). In cool, humid weather conidia are produced on the leaves of sytemically infected plants and on local lesions during the night, particularly on the abaxial surface. This gives infected parts of the plant a white down-like appearance (Fig. 11).

Epidemiology and biology

The life cycle of P. *sorghi* is shown in Figure 12. Conidia of P. *sorghi* are produced in large numbers, they are thin-walled, ephemeral, and can cause the rapid build up of an epidemic. Oospores are tough walled, long-lived, and provide a perennating stage for the pathogen, as well as a mechanism for long-distance transport.

Conidia production, dispersal, and infection

Peronosclerospora sorghi has exacting environmental requirements for asexual reproduction and infection

Figure 9. The downy appearance of leaves infected with sorghum downy mildew resulting from the asexual sporulation of *Peronosclerospora sorghi*.

(Bonde 1982), Prior to conidiation in the dark, the host must be subject to a minimum period of 4 h of high light intensity (Schmitt and Freytag 1974). In maize, conidiophores form from within stomata under suitable environmental conditions over a period of about 6 h (Lal 1981). High relative humidity (RH) is crucial. Shetty and Safeeulla (1981a) found that systemieally infected sorghum leaves held in the dark at 20°C produced a maximum of 10,800 conidia cm^{-2} at 100% RH, but only 3,600 conidia cm^{-2} at 85% RH. None were produced at 80% RH. The optimum temperature for sporulation of an American isolate on maize was between 15°C and 23°C (Bonde et al. 1985). The optimum temperature for germination was 15°C and for germ tube growth was 22°C. However, germination was good at 10-19°C and germ tube growth rapid at 14-22°C. A dew period temperature of 10-33°C is required for 4 h for infection (Bonde et al.

Figure 11. 'Leaf-shredding' typically observed in older sorghum plants systemieally 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; this probably allows them to be dispersed from the host.

Figure 10. The typical pale, chlorotic streaking in the younger leaves of a sorghum plant systemieally infected with sorghum downy mildew. This symptom is indicative of the early stages of oospore production.

1978). Conidial production in the field has a marked periodicity of release, and has a close relationship with temperature and moisture. Conidia are produced between midnight and 0500 h when temperatures are about 20°C and the RH > 85% (Shenoi and Ramalingham 1979). Conidia of SDM can be dispersed in air currents as far as 80 m (Rajasab et al. 1979). Germination occurs when conidia arc mature. The germ tubes grow at random over the leaf surface until encountering a stomata, when an appressorium forms over the stomatal opening (Jones 1971). The penetrating structure enlarges to form an oval shaped sub-stomatal vesicle, which gives rise to one or more infection hyphae. In susceptible cultivars, systemic colonization progresses by the development of haustoria that have up to eight finger-like tubes. Hyphal growth proceeds through the intercellular spaces of the mesophyll cells (Mauch-Mani et al. 1989). If a systemic infection develops, hyphae proceed to the apical meristem of
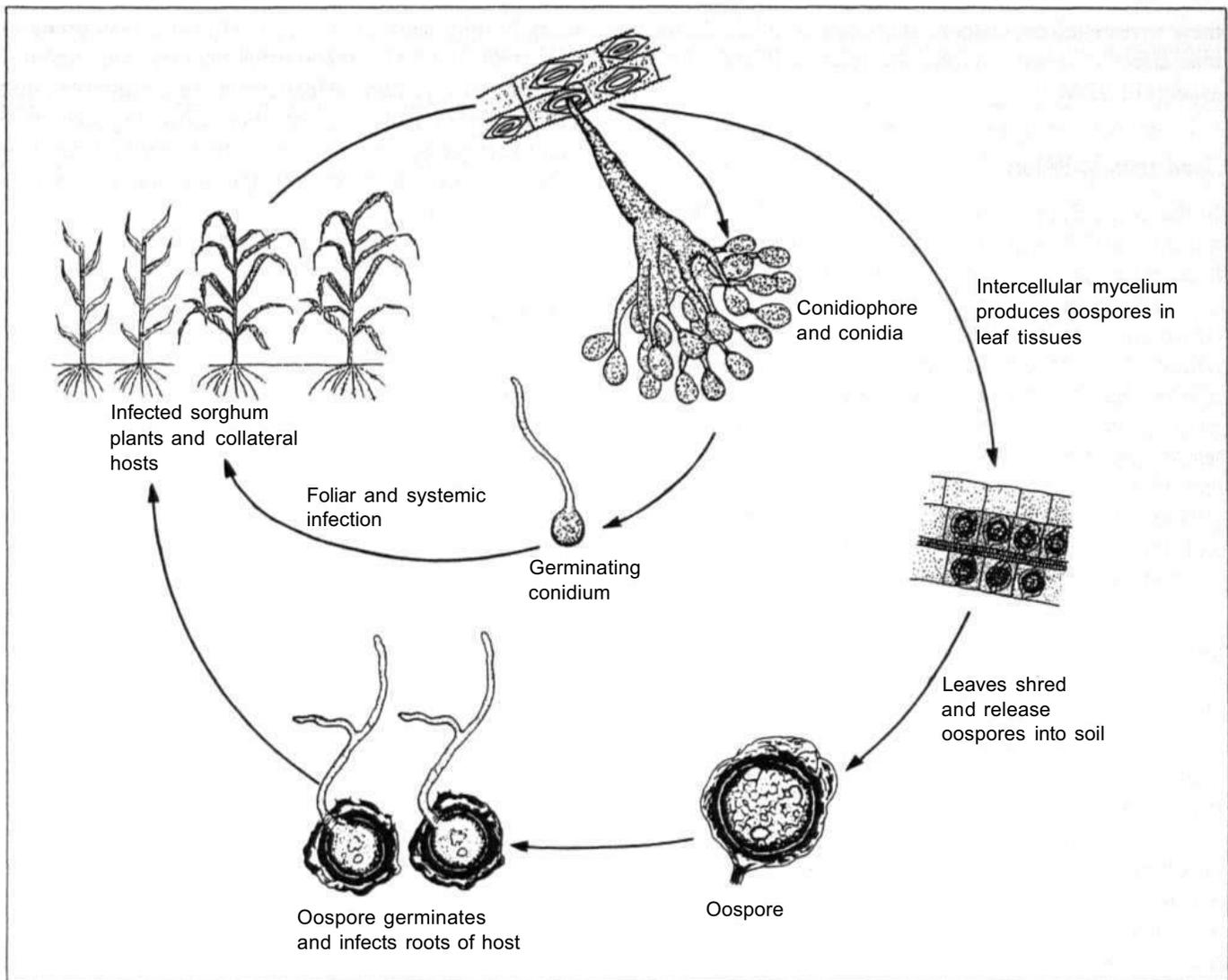
Figure 12. The disease cycle of *Peronosclerospora sorghi.* Whereas sexually produced oospores will generally provide only one cycle of infection per season, the asexually produced conidia from an infected plant can infect fresh hosts within the same season allowing rapid build up of an epidemic of sorghum downy mildew.

the plant and invade the developing leaves and flowering parts; the symptoms being manifest after at least 7 days. Plants are most vulnerable to systemic infection caused by conidia for approximately 20 days after emergence, after which time only local lesions are produced (Jones 1978; Shetty and Safeeulla 1981b). Local lesions develop approximately 7 days after infection (Cohen and Sherman 1977). In resistant cultivars necrosis occurs at the penetration site (Mauch-Mani et al. 1989).

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 can be dispersed by man or animals in soil adhering to feet or implements (Williams 1984). They can survive passage through the digestive tract of a cow and thus dispersal in manure is implicated (Safeeulla 1976). Seed transmission of oospores can also occur (Bain and Alford, 1969). They can also be dispersed by wind (Bock et al. 1995), and by water (Rajasab et al. 1979). Oospores can survive adverse conditions for several years (Safeeulla 1976). In soils the greatest incidence of infection was observed when the temperature was 25°C and the soil moisture potential 0.2 bar (Schuh et al. 1987). Soils with a high sand content support greater infection (Pratt and Janke 1978; Schuh et al. 1987). Host and nonhost roots can stimulate germination of oospores (Pratt 1978), the germ tube growing towards the meristematic region of the root where it forms an appressorium and infection peg (Safeeulla, 1976). Despite

these investigations, oospore germination and the factors that affect it remain among the least well understood aspects of SDM.

Seed transmission

In the USA, Bain and Alford (1969) illustrated external transmission of oospores with sorghum seed. Studies in India have indicated that *P. sorghi* could be transmitted internally in sorghum and maize seed either as mycelium (Kaveriappa and Safeeulla 1978) or as oospores (Up-adhyay 1987). Mycelium was reported in both reproductive structures and in the endosperm of sorghum seed from systemically infected plants and a direct correlation was observed between seed transmission and embryo infection (Prabhu et al. 1983; Upadhyay 1987). Frederiksen (1980b) discussed ways in which oospore contamination and internal mycelial transmission of SDM can be avoided. Seed drying 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 legislation are all practises that can be used to avoid seed infection. Checking seed samples using molecular probes and DNA hybridization can also be used to check for seed transmission, of *P. sorghi* (Yao et al. 1990).

Collateral hosts

Collateral hosts, common in many areas where sorghum and maize crops are grown, are known to act as reservoirs for infection (Malaguti 1977). They can act either as a source of conidia early in the season or as a source of oospores that can infest the soil. Several species of gram-inae from the tribes Andropogonac, Maydae, and Panicae are reported to be susceptible to *P. sorghi* and are potential collateral hosts (Table 2).

Table 2. Host range of *Peronosclerospora sorghi*

Host	Author
Panicwn 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 x *almum* Perodi.	Tarr(1962)
S. arundinacium (Willd.) Stap.	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)
5. *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. purpurea-serecium (A. Rich.) Aschers. & Schwcrf.	Karunakar et al., 1994
S. sudanense (Piper) Stapf.	Nagarajan et al. (1970)
S. verticilliflorum (Steud.) Stapf.	Tarr(1962)
S. controversion (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)

Table 3. Identification of pathotypes I, II, and III of *Peronosclerospora sorghi* in the USA by the differential reaction of four sorghum inbred lines[1].

Sorghum variety	Reaction to infection with pathotype I[2]	Reaction to infection with pathotype II	Reaction to infection with pathotype III
Tx412	S	S	S
Tx430	R	R	S
CS 3541	R	S	S
QL 3	R	R	R

1. Source; Craig and Frederiksen, 1983.

2. S = susceptible to infection, R - resistant to infection.

Seasonal disease development

An understanding of the factors that contribute to disease initiation and epidemic development is necessary if appropriate control methods are to be recommended. Studies of infection of sorghum and maize crops in several geographic regions indicate that oospores and conidia vary in importance as causal agents of infection. In parts of India where weather conditions are conducive to asexual spore production conidia are responsible for most of the infections, and early sowings can escape disease (Rajasab et al. 1980; Ramalingham and Rajasab, 1981). In other areas, including the USA, oospores are the major source of infection (Schuh et al. 1987). Soil temperature, moisture, and texture are likely to influence the incidence of systemic infection in these regions. In South America collateral hosts are thought to be an important source of disease each season (Malaguti, 1977). Although it has not been applied to downy mildew of sorghum, Drepper et al. (1993) illustrated the potential use of modeling epidemics to identify conditions conducive to epidemic development of the maize downy mildew in Thailand. This could provide a useful tool for understanding the conditions that support epidemics of SDM at different locations.

Variability within *Peronosclerospora sorghi*

Morphological variability between isolates of *P. sorghi* is limited (Bock 1995). Adaptation to specific environmental conditions is not apparent either. There appears to be little variability in environmental requirements between isolates from diverse locations (Bock 1995; Bonde et al. 1985).

Variability is thought to occur at the host range level. As a result, *P. sorghi* has been subdivided into 'sorghum/maize' and 'maize' infecting strains. However, as more information has become available on the affiliations of the maize strains, most have eventually been designated as separate species. For example, *P. heteropogonii* was designated specific rank after studies of a maize strain of *P. sorghi* showed it to be ,n a different species (Siradhana et al. 1980). Similarly, recent studies suggest that a maize strain of *P. sorghi* from Thailand should be given specific rank as *P. zeae* (Bonde et al. 1992; Yao 1991). Molecular and biochemical tools have proven useful for investigating the variability of these species (Bonde et al. 1984, Micales et al. 1988, Yao 1991). Further observations indicate that at least one other maize strain of *P. sorghi* occurs in Africa (Anaso ct al. 1987; Fajemisin 1980). Further work is needed to confirm its identity.

The first indication of pathogenic variability of *P. sorghi* on sorghum was observed in the USA in the late 1970's when a previously resistant and popular sorghum hybrid was observed to be infected with SDM (Craig and Frederiksen 1980). Subsequently three distinct pathotypes were identified on sorghum in the USA from the differential reaction of the inbred lines Tx412, Tx430, CS 3541, and QL 3 (Table 3, Craig and Frederiksen 1983). Other pathotypes have also been identified in Brazil (Fernandes and Schaffert 1983), Honduras (Craig and Odvody 1992), and Zimbabwe where there are reports of the resistant variety QL 3 being susceptible (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 that differential reactions identified each of the isolates 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 1970's several different fungicides had been used in an attempt to control the graminaceous downy mildews (Balasubramanian 1975; Singh et al. 1970). None of these had proved effective. However, in the late 1970's the discovery of the acyl-alanine fungicide

metalaxyl-([N-(2-methoxyacetyl)-N-(2,6-xylyl)-DL-al-aninate) revolutionized the chemical control of these pathogens (Schwinn 1980). In India, seed treatments of 1 g a i kg^{-1} of seed plus a foliar spray of 1 g a i L^{-1} 40 days after emergence (DAE) or foliar sprays of 2 g a i litre^{-1} at 10 plus 40, or 20 plus 50 DAE gave complete control of systemic SDM (Anahosur and Patil, 1980, Venugopal and Safeeulla, 1978). Seed treatment alone did not fully protect the plant or nodal tillers from systemic infection, and a spray regime was required to prevent a low incidence of late systemic infection developing and to control local lesions. However, in most cases it is unlikely that the additional spray treatments are economic. Other studies in the USA, where oospores constitute the bulk of systemic infection, indicated concentrations of metalaxyl as low as 0.05 g a i kg^{-1} seed gave complete control, and concentrations greater than 1 g a i kg^{-1} seed caused seedling death (Odvody and Frederiksen, 1984).

It remains a possibility that the graminaceous downy mildews may develop resistance to metalaxyl, a view that is supported by the fact that other oomycetes have developed resistance to this fungicide (Bruck et al. 1982; Georgopoulos and Grigoriu 1981). Judicious use of metalaxy as a seed treatment is recommended, perhaps in conjunction with other means of disease control. Apart from the use of metalaxyl, soil sterilization has also been shown to be effective in reducing infection through soilborne oospores, but may not be practical or economic (Matocha et al. 1974).

Cultural control

Crop rotation. Roots of host and non-host plants cause 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 will have greatest effect where oospores are the principal source of systemic infection and infestation of the soils is severe.

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

Over-sowing and roguing of diseased plants. By sowing up to 50% more than the recommended agronomic optimum, the stand loss due to moderate disease incidence leads to an acceptable plant density of healthy plants at harvest (Frederiksen et al. 1973). A disease incidence of 20-30% can be borne at this level of oversowing before yield is reduced. Roguing results in a reduced oospore population, and consequently the incidence of systemic infection in subsequent crops is re-

duced (Janke et al. 1983). It also reduces the source of within-season asexual spores. Roguing can also be usefully applied to weed hosts so as to reduce the sources of external inoculum (Malaguti 1977).

Sowing date. In Dharwad, India, late sowings of sorghum had an increased incidence of SDM (Balasubramanian 1974). Similarly in Israel, early sowings of sweet corn avoided the disease (Cohen and Sherman 1977). This is because conidia produced in large quantities from plants that were infected early provided an increased inoculum pressure, resulting in a higher disease incidence on late-sown crops. However, where conidia are not the major source of inoculum late sowings might not have a higher disease incidence. Tuleen et al. (1980) found a lower incidence of systemic SDM in late sowings in the USA, where oospores were the principal source of infection.

The effect of host-plant nutrition. There is no clear effect of nutrition on the incidence of systemic SDM. Balasubramanian (1973) found that phosphorus added to the soil increased the incidence of SDM on sorghum plants, but nitrogen levels had no effect. Gupta and Siradhana (1978) observed that the absence of phosphorous, and deficiency of nitrogen reduced incidence of systemic infection, but potassium deficiency caused greater incidence of SDM on maize grown in a nutrient solution,

Bonman and Pittipornchai (1984) found that in early-sown maize crops in Thailand the incidence of maize downy mildew was lowered by the application of nitrogen or nitrogen plus phosphorus, but late-sown crops had a high incidence regardless of treatment. It is likely that the effect of nutrition is associated with plant age, inoculum type and pressure, and other environmental variables.

Biological control

A chytrid fungus (*Gaertennomyces* sp.) was found to effectively parasitize oospores (Kunene et al. 1990). It can reduce the incidence of infection in treated soils by up to 58%. However, field application of this organism has not been developed and it is unlikely that bio-control of SDM will become a practical reality in the near future. Other parasites of oospores have also been observed although they have not been studied in detail (de Diaz and Polanco 1984, Lakshmanan et al. 1990a).

Host-plant resistance

There are many reports in the literature of screening sorghum lines for resistance to SDM. (Anahosur et al. 1984; de Milliano et al. 1990; Frederiksen et al. 1973; Henzell et al. 1982; Kumar et al. 1979; Lakshrnananet al. 1990b; Lu et al. 1990; Sarwar and Rao 1979; Shivana and

Anahosur 1988; Shivana and Anahosur 1990; Williams et al. 1982). In India, the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) has screened a great deal of germplasm. Up to 1988, a total of 13,101 accessions from 73 countries had been screened in the field for resistance to *P. sorghi*. Of these 46 accessions were resistant to *P. sorghi* (Y D Narayana, personal communication). These accessions were from geographically

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

Origin[1]	Number of coun tries	Number of lines screened	Number of lines resistant
Eastern Africa	7	3345	18
Western Africa	14	3324	4
Southern Africa	9	930	2
Northern Africa and the 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, Cote d'Ivoire, Mali, Nigeria, Niger, Sierra Loene, Senegal.
   Southern Africa: Angola, Botswana, Lesotho, Malawi, Malagasy Republic, South Africa, Swaziland, Zambia, Zimbabwe.
   Northern Africa and the Middle East: Egypt, Israel, Iran, Iraq, Lebanon, Syria, Saudi Arabia, Yemen.
   Indian subcontinent: Afganistan, Bangladesh, India, Nepal, Pakistan.
   Southeast Asia and the Far East: Myanmar, China, Indonesia, Japan, Philippines, South Korea, Taiwan, Thailand.
   North and Central America: Cuba, El Salvador, Gautamela, Mexico, Nicaragua, USA, West Indies.
   South America: Argentina, Uraguay, Venzuela.
   Europe: Belgium, Cyprus, France, Greece, Hungary, Italy, Portugal, Spain, Turkey.
   Eastern Europe.
   Australia and Oceania: Australia, Papau New Guniea.

Figure 13. The spreader row technique employed by breeders to ensure effective screening of sorghum germplasm against sorghum downy mildew. Note the older infeetor rows sown to the left and right of the four rows of test material. The infector rows were sown about 3 weeks prior to the test material.

diverse sources (Table 4). Many lines of germplasm have also been screened in the USA (Frederiksen et al. 1993).

In an attempt to screen sorghum cultivars and to identify stable resistance and differences in pathogen-virulence between locations the International SDM Nursery (ISDMN) was established in 1976 (Williams et al. 1980). Selected results of multilocational testing of resistant sorghum accessions at ISDMN test sites are shown in Table 5 (Dr Y D Narayana, personal communication). Although the results of the ISDMN did not initially indicate pathogen variability of *P. sorghi* on sorghum, the existence of pathotypes of *P. sorghi* was subsequently reported (Craig and Frederiksen 1983; Pawar et al. 1985). This indicates that durable, broad-based resistance to this pathogen should be sought.

Methods for screening for resistance. To identify resistance reliably and to investigate the inheritance and genetics of resistance it has been necessary to develop effective screening methods:

• Natural infection (Anahosur and Hegde 1979). This method is probably the least effective as there is no attempt to ensure the exposure of different test materials to the same amounts of inoculum.

• Spreader rows as a source of asexual inoculum (Fig. 13; Cardwell et al. 1993; Anahosur and Hegde 1979). The spreader rows are generally inoculated to ensure a uniform and high level of infection (Cardwell et al. 1993). The test material is sown approximately 3 weeks after the spreader rows. This allows the systemic infections in the spreader rows to develop and produce large quantities of asexual spores over the period when

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

| Entry | Origin | Highest disease incidence at locations (% plants infected) | | | | | | |
		Mana[1]	Per	Jab	Sam	Dha	Mys	Tex
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)	-
DMS 652 (Susceptible check)	India	100(4)	36(5)	9(2)	100(2)	100(5)	100(5)	45(1)

1. Locations: Manfredi and Pergamino (Argentina), Jabaticobal (Brazil), Samaru (Nigeria), Dharwad and Mysore (India), Texas (USA).
2. - not tested at that location.
3. Numbers in parentheses indicate number of years tested at each location.

the test materials are susceptible. Humidity can be increased by using sprinkler irrigation to provide ideal conditions for asexual spore production as Williams and Singh (1981) illustrated using pearl millet downy mildew. Anahosur and Hedge (1979) compared five different methods, and found that the infector row technique was the most effective at producing a high and uniform incidence of infection in susceptible test materials.

- Oospore infested plots as a source of sexual inoculum (Craig 1980). With this technique monocropping and plowing in of infected sorghum from test plots and spreader rows is used to increase the oospore content of the soil. Test material is then sown. The main source of infection is through the oospores in the soil.

- A combination of spreader rows and oospore-infested plots (de Milliano, personal communication). This technique is used by 1CRISAT to screen for resistance to SDM both at Dharwad, in India, and at Matopos, in Zimbabwe. As in the previous technique, infected sorghum is incorporated to increase the oospore content of the soil. Spreader rows are also sown to act as a source of conidial inoculum. The advantage of this system is that plants are subject to infection by both oospores and conidia, which have different sites of entry, and for which there may be evidence of differential resistance, at least in maize (Frederiksen et al. 1973).

- Artificially applied asexual inoculum (Schmitt and Freytag 1974; Craig 1976; Narayana et al. 1995). This

system is generally used in a controlled environment. Seedlings of test material are either whorl- or spray-inoculated with a suspension of mature conidia. The advantage of this system is that optimal conditions can be maintained and the amount of inoculum is regulated. It can also provide a rapid technique for screening large quantities of material in a short time. The ephemeral nature of conidia of *P. sorghi* means they must reach the host within a short time of maturation so as to ensure infection. Craig (1987) utilized the natural infection cycle of SDM to develop a system for producing and storing conidia, that could later be used to inoculate material. However, the most successful long-term storage technique was developed by Gale et al. (1975) and Long et al. (1978). Maize seedlings could be infected with conidia of various *Peronosclerospora* spp. after more than 2 years storage in 10% dimethyl sulphoxide held in liquid nitrogen.

- Tissue culture. Currently this method of screening for resistance to SDM does not have a practical application. Mauch Mani et al. (1989) found that callus cultures of resistant cultivars were not infected by conidia of P. *sorghi,* while those of susceptible cultivars were. However, Gowda and Bhat (1992) obtained a viable dual culture when they applied asexual inoculum to the callus of a SDM-resistant cultivar of sorghum, although a second resistant line remained uninfected by *P. sorghi* in culture. This system needs to be investigated in greater depth before it can be used as a tool in resistance breeding.

Assessment methods. For comparing host reactions it is necessary to develop an effective (both accurate and precise) assessment method. Scoring of systemic infection is straightforward. The incidence of systemically infected plants can be recorded on at least two occasions during the season; This should provide a realistic estimate of the incidence of systemic infection (Williams 1984). Assessment of local lesions requires that both incidence and severity data be recorded. In the past a 1-5 scale has been used to score this type of infection (Singburaudom and Williams 1978; Frederiksen 1980a). Shenoi and Ramalingham (1976) developed a 1-4 scale to assess the severity of local lesion infection.

Genetics and inheritance of resistance. 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 it is dominant to susceptibility (Rana et al. 1978; Sifuentes and Frederiksen 1988) although earlier-workers found dominance of susceptibility (Miller 1966;

Puttarudrappa et al. 1972). Quantitative inheritance has also been observed by some authors. Puttarudappa et al. (1972) suggested that two complementary genes controlled resistance. Bhat et al. (1982) concluded that a primary dominant gene with either one or two duplicate genes and three complementary genes contributed to resistance. Nider et al. (1974) reported polygenic control. Craig and Schertz (1985) illustrated that the resistance to SDM expressed by the inbred line SC414-12 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. Neither author detected cytoplasmic factors of inheritance. 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) also 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 other sorghum lines, and the mechanisms of resistance, which remain poorly understood. Resistance, preferably of a durable nature needs to continue to be incorporated into agronomically suitable varieties. Recently symptom remission has been observed in systemically infected plants. It might be that this is another resistance mechanism that could be utilized (Singh and de Milliano, 1989a and b).

Integrated control

Integrated control involves the use of two or more methods of control to bring about a reduction in the incidence of the disease (Odvody et al., 1983). The suitability of the methods used depends on the local conditions. Thus, a good knowledge is needed of the epidemiology of the disease and control options available to a farmer in a given area before an integrated package can be implemented. Integrated control can involve chemical control (for example, metalaxyl seed treatment), cultural control (for example, deep plowing or crop rotation) and the use of host-plant resistance. The combination of control methods can be mutually beneficial. For example, Odvody and Frederiksen (1984) suggest 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 the farmer's ability to use them.

In conclusion

A great deal of work has been done to increase our knowledge base of downy mildew of sorghum during the last 30 years. The availability of a number of different disease management strategies attests to the success of this investment. However, there are aspects of this pathogen that remain poorly understood. Further investigation may enhance our understanding and contribute to the continued control of SDM. The pathogen remains a threat to the production of sorghum (and maize) in Africa, Asia, and the Americas. The breakdown of resistance to SDM in the USA in the late 1970's suggests we should not be too complacent. However, host plant resistance probably offers the best solution to the control of SDM, as well as being the most environmentally sound method for the future management of this disease.

Acknowledgments

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