# Peronosclerospora philippinensis

# **Scientific Name**

Peronosclerospora philippinensis (W. Weston) C.G. Shaw

### Synonyms:

Peronosclerospora sacchari, Sclerospora indica, Sclerospora maydis, and Sclerospora philippinensis

# **Common Name**

Philippine downy mildew, Java downy mildew of corn, and sugarcane downy mildew

# Type of Pest

Fungal-like

# **Taxonomic Position**

**Phylum:** Oomycota, **Class:** Oomycetes, **Order:** Sclerosporales, **Family:** Sclerosporaceae

# **Reason for Inclusion in Manual**

CAPS Target: AHP Prioritized Pest List – 2009; Agricultural Bioterrorism Protection Act of 2002 (Select Agent)

# **Pest Description**

Philippine downy mildew, caused by Peronosclerospora philippinensis, was initially reported from India in 1913. The pathogen at this time, however, was misidentified as Sclerospora maydis and later as S. indica (Bonde, 1982). The disease in India has never caused large losses (Payak, 1975). In contrast, in the Philippines, where the disease has been present at least since 1916 (Weston, 1920), it is economically very damaging. Philippine downy mildew is confined to parts of Asia and has not been reported within the United States.

Peronosclerospora philippinensis is an obligate parasite that will not grow on artificial media. The mycelia are branched, slender (8  $\mu$ m in diameter), irregularly constricted, and inflated. Erect conidiophores (15-26 x 150-400  $\mu$ m) grow out of stomata and are dichotomously branched two to four times. Branches are



**Figure 1.** Chlorotic symptoms of Philippine downy mildew. Photo courtesy of CIMMYT .

robust. Sterigmata are ovoid to subulate, slightly curved, and 10  $\mu$ m long. The conidia (17-21 x 27-39  $\mu$ m) are elongate ovoid to round cylindrical, hyaline, and slightly rounded at the apex (Weston, 1920; Smith and Renfro, 1999). Haustoria are simple, vesiculiform to subdigitate, 8 x 2  $\mu$ m.

Oospores are rarely produced and are not produced in corn tissue (Smith and Renfro, 1999). When produced, oospores are spherical, smooth-walled, approximately 22  $\mu$ m in diameter; they germinate by a side germ tube (CABI, 2007).

#### **Biology and Ecology**

Disease spreads locally via wind and rain from an infected crop. Perennial grass hosts may also serve as reservoir hosts to carry over the pathogen during unfavorable periods, or provide primary inoculum (Bonde, 1982). The known infective agents are the mycelium in these plants or the airborne conidia produced by the mycelium. Although conidia are produced from 18-23°C (64-73°F) and germinate from 10-35°C (50-95°F), the highest rates of infection occur at temperatures greater than 16°C (61°F) (Bonde et al., 1992). Dalmacio and Raymundo (1972) indicated that conidial production, germination, and infection required night time temperatures of 21-26°C (70-79°F). Movement of infected plant tissue could introduce Philippine downy mildew to new locations. Although Philippine downy mildew is reported to produce an over-wintering spore form (oospore), its role in the lifecycle has not been established.

In the host, the mycelium produces the conidiophores, which bear the conidia. The conidiophores emerge through host stomata in the chlorotic areas on both leaf surfaces (more so on the lower surface of corn, because of the higher density of stomata), and on sheaths, tassel rachis, glumes, and husks (Dalmacio and Exconde, 1969). The pathogen spreads intercellularly through the mesophyll cells. The fungus grows mainly downward through the leaf sheath to the stem where it moves into and persists in the shoot apex. When mycelium invades the meristematic tissues, chlorotic streaks soon appear on the leaves, followed by the fungus sporulating in these areas when conditions are favorable, producing secondary inoculum (Dalmacio and Exconde, 1969). The fungus can spread throughout the plant, but it is confined to the chlorotic, (not green) areas.

Spore production requires high humidity, with at least a thin film of water for 4-5 hours in the darkness on the infected leaf surface (Exconde, 1970; Dalmacio and Raymundo, 1972; Bonde, 1982). Germinating conidia produce germ tubes, which invade stomata. A mycelium develops in the mesophyll. Conidia form sparsely the morning after the first symptom appears, and abundantly after systemic symptoms appear (Dalmacio and Exconde, 1969). Sporulation may begin at midnight and continue until surface moisture dries (Weston, 1920). Sporulation on individual plants can continue for more than 2 months, releasing enormous number of conidia (Bonde, 1982). Once exposed, however, to drying conditions (such as sun, wind, or low humidity for 1-2 hours) the conidia shrivel and no longer germinate (Weston, 1920). Viable conidia germinate in less than 1 hour under favorable conditions. Penetration of the host occurs about 2 hours after inoculation (Dalmacio and Exconde, 1969).

Seedborne transmission may occur in corn, but there are no external symptoms on the seed. Transmission does not occur when seed moisture content is less than 14% (Exconde, 1976). In the laboratory, corn seed from infected plants produce infected seedlings, especially when hard dough kernels are planted soon after harvest. Because infected plants mature slower that uninfected plants, ears from the former are left in the field while the latter are harvested. The infected ears may then become a new inoculum source. No infections result from inoculations of healthy seed with conidia. The infective agent is the mycelium within the infected seed, reportedly located in the pericarp (Advincula and Exconde, 1976) or the embryo (Bains and Jhooty, 1982).

The number of systemically infected (non-yielding) plants is positively correlated with night time relative humidity, spore production, day time relative humidity and rainfall and negatively correlated with night and day temperatures and duration of sunlight (Bonde, 1982; Exconde, 1976).

# Symptoms/Signs

On corn, the first symptoms typically appear as chlorotic stripes at the first leaves as early as 9 days after planting. All leaves on a plant may show characteristic symptoms of long chlorotic (yellow) streaks (Fig. 1). Weston (1920) reported the collapse of badly infected cells and the destruction of chloroplasts, resulting in the characteristic yellow color of diseased leaves.

A downy (grayish) covering primarily on the underside of the leaves is characteristic beginning at the two-leaf stage and is present until the appearance of tassels and silks. This covering is the site of spore production (conidia on conidiophores) and the source for secondary spread of the disease to other susceptible plants.

As the plant ages, leaves may narrow, become abnormally erect, and appear somewhat dried-out. As the corn plant matures, tassels become malformed and produce less pollen, ear formation is interrupted, and sterility of



**Figure 2**. Downy growth characteristic of downy mildews. Photo of downy mildew of tobacco. Photo courtesy of Tom Creswell, North Carolina State University.

seeds can result (Expert Panel, 2006). If infection occurs early, plants are stunted and may die (Smith and Renfro, 1999).

There are no external symptoms on seeds. The fungus becomes established in the pericarp layer in the form of mycelium. The fungus is also present in the embryo and endosperm. There are no reports on the effect of *P. philippinensis* on seed quality (CABI, 2007).

Sugarcane has some of these symptoms.

## **Pest Importance**

Downy mildew of sorghum, maize, and sugarcane are among the world's most destructive diseases (Bonde et al., 1992). Heaviest losses are incurred when the disease becomes systemic (spreads throughout the plant host) (Kenneth, 1981). *P. philippinensis* is considered the most virulent of the maize downy mildew pathogens (Payak, 1975). This obligate pathogen causes a serious disease to two major crops, corn and sugarcane. Yield losses of 40-60% and 25% have been observed in corn and sugarcane respectively (USDA, 1986). The national yield loss for corn in the Philippines in the 1974-1975 crop year was estimated at 8%, valued at US \$23,000,000 (Exconde, 1976). No grain is produced because the seedlings quickly die or the plant slowly matures but produces little grain (Weston, 1920). For a susceptible sugar cane clone, 25-85% disease incidence was observed with losses in tons of cane per hectare ranging from 9 to 38%, losses in picul of sugar per ton from 2 to 35%, and losses in picul of sugar per ton from 2 to 35%, and losses in picul of sugar per hectare ranging from 10-58% (Husmillo, 1982).

# **Known Hosts**

Major hosts Saccharum officinarum (sugarcane) and Zea mays (corn)

#### Secondary hosts

Avena spp. (oats) and Sorghum bicolor (sorghum)

#### Wild hosts

Andropogon spp. (blue stem), Botriochloa spp. (bluestem), Euchlaena luxurians (Florida teosinte), Eulalia spp. (eulalia), Miscanthus spp., Saccharum spontaneum (wild sugarcane; kans grass), Sorghum halepense (Johnson grass), Sorghum plumosum (plum sorghum), Tripsacum spp. (gamagrasses), Zea diploperennis (diploperennial teosinte), Zea mexicana (teosinte), Zea perennis (perennial teosinte).

### Known Vectors (or associated insects)

There is a synergistic relationship between downy mildew fungi and maize streak virus (MSV) on corn. Infection by MSV can mask symptoms of downy mildew infection. Reduction in height and biomass were significantly greater with pathogen combinations than with single pathogens (Damsteegt et al., 1993).

### **Known Distribution**

**Asia:** China, India, Indonesia, Japan, Nepal, Pakistan, Philippines, and Thailand. **Africa:** Democratic Republic of Congo, Mauritius, and South Africa.

## **Potential Distribution within the United States**

A recent risk analysis by USDA-APHIS-PPQ-CPHST indicates that portions of Illinois, Indiana, Kentucky, and Ohio have the greatest risk for *P. philippinensis* establishment based on host availability and climate within the continental United States. The remaining states have low to moderate levels of risk for establishment of *P. philippinensis*.

## Survey

**<u>CAPS-Approved Method:</u>** Use visual survey, sentinel plots, spore trapping, or a combination of methods. For visual survey collect symptomatic plants. Spore traps, similar to those used in soybean rust monitoring, can be used to detect spores. Unsprayed, susceptible plants (sentinel plots) that are scouted regularly can also be used for early detection.

#### Literature-Based Methods:

<u>Visual survey:</u> Surveys should occur in areas of the country that are at the greatest risk for establishment. Survey for Philippine downy mildew is conducted via visual survey of plants for symptoms of the disease (chlorotic stripes or streaks) and signs of the pathogen (downy growth on underside of leaves consisting of conidia and conidiophores). Survey for diseased plants by examining corn seedlings that have larger and larger yellow streaks on succeeding leaves. Grayish white down will be in the chlorotic areas although sun or low humidity may have dried the mildew to matted fragments (USDA, 1986).

<u>Trapping:</u> Spore trapping using Burkhard spore traps and sentinel plots (unsprayed, susceptible plants that are scouted regularly) are suggested for early detection and have been employed for resistance screening for other exotic downy mildews (Cardwell et al., 1997; Expert Panel, 2006).

# **Key Diagnostics**

**<u>CAPS-Approved Method:</u>** Confirmation of *P. philippinensis* is by morphological identification. Pathogen may be identified morphologically by conidiophore structure and dimension and spore (conidia) shape and size. Isozyme comparisons have been used to identify *Peronosclerospora* spp., including *P. philippinensis* 

Literature-Based Methods: Peronosclerospora spp. and other downy mildew genera (including Sclerospora and Sclerophthora) are primarily differentiated by pathogen morphology, including conidiophore structure and dimension and spore (conidia) shape and size. However, these characteristics can vary considerably under different culture conditions, at different developmental stages, and on different hosts. Identification of *Peronosclerospora* species often is difficult. They may be easily divided into three categories according to the shape of conidia: globose, ovoid to slightly elongate, and long or slipper-shaped, but within each group there are usually only minor morphological differences. Species are differentiated by only variations in the size and shape of their conidia and conidiophores, and sometimes differences in host ranges,

presence or lack of oospores, and differences in morphology (Bonde et al., 1992). Characters for downy mildew pathogens are listed in Appendix A.

Isozyme comparisons have been used to identify *Peronosclerospora spp.*, including *P. philippinensis* (Bonde et al., 1984; Micales et al., 1988). *P. philippinensis* and *P. sacchari* can not be separated based on isozyme analysis. These species are believed to be conspecific (Yao et al., 1991b).

Some DNA-based approaches have been reported for other *Peronosclerospora* spp., which indicates that a molecular method for identification may be available in the future. Yao et al. (1991a) created a DNA clone that could be used as a species-specific hybridization probe for the detection and identification of *P. sorghi*. Yao et al. (1991b) then developed a DNA probe from a *P. maydis* genomic library to detect *P. sacchari* in maize leaves and seeds. Ladhalakshmi et al. (2009) used a DNA sequence characterized amplified region (SCAR) marker for identification of isolates of *P. sorghi* and related species that include this pathogen and will be helpful for identification and future PCR primer development (Perumal et al., 2008).

### **Easily Confused Pests**

Downy mildews of corn are caused by up to ten different species of Oomycete fungi in the genera *Peronosclerospora, Scleropthora* and *Sclerospora. Peronosclerospora philippinensis* may be confused with other *Peronosclerospora* spp. occurring on corn and other downy mildew genera. Other indigenous downy mildews (*e.g., P. sorghii*) and physiological conditions (fertility, weather, etc.) can cause similar symptoms.

### References

Advincula, B.A. and Exconde, O.R. 1976. Seed transmission of *Sclerospora philippinensis* Weston in maize. Philpp. Agric. 59: 244-245.

**Bains, S.S. and Jhooty, J.S.** 1982. Distribution, spread, and perpetuation of *Peronosclerospora philippinensis* in Punjab. Indian Phytopathology 35(4): 566-570.

**Bonde, M.R.** 1982. Epidemiology of downy mildew diseases of maize, sorghum, and pearl millet. Trop. Pest. Manage. 28(1): 49-60.

Bonde M.R., Peterson, G.L., Dowler, W.M., and May, B. 1984. Isozyme analysis to differentiate species of *Peronosclerospora* causing downy mildews of maize. Phytopathology 74(11): 1278-1283.

**Bonde, M.R., Peterson, G.L., Kenneth, R.G., Vermeulen, H.D., Sumartini, and Bustaman, M.** 1992. Effect of temperature on conidial germination and systemic infection of maize by *Peronsclerospora* species. Phytopathology 82(1): 104-109.

**CABI.** 2007. Crop Protection Compendium Wallingford, UK: CAB International. <u>http://www.cabi.org/compendia/cpc/</u>.

Cardwell, K.F., Kling, J.G., and Bock, C. 1997. Methods for screening maize against downy mildew *Peronosclerospora sorghi.* Plant Breeding 116: 221-226.

**Dalmacio**, **S.C. and Exconde**, **O.R.** 1969. Penetration and infection of *Sclerospora philippinensis* Weston on corn. Philippine Agriculturist 53: 35-52.

**Dalmacio**, **S.C. and Raymundo**, **A.D.** 1972. Spore density of *Sclerospora philippinensis* in relation to field temperature, relative humidity, and downy mildew incidence. Philippine Phytopathology 8: 72-77.

**Damsteegt, V.D., Bonde, M.R., and Hewings, A.D.** 1993. Interactions between maize streak virus and downy mildew fungi in susceptible maize cultivars. Plant Disease 77: 390-392.

Exconde, O.R. 1970. Philippine corn downy mildew. Indian Phytopathology 23: 275-284.

**Exconde, O.R.** 1976. Philippine corn downy mildew: assessment of present knowledge and future research needs. Kasetsart Journal 10: 94-100.

**Expert Panel.** 2006. Recovery plan for Philippine downy mildew and brown stripe downy mildew of corm caused by *Peronosclerospora philippinensis* and *Sclerophthora rayssiae* var. *zeae*, respectively. <u>http://www.ars.usda.gov/SP2UserFiles/Place/0000000/opmp/Corn%20Downy%20Mildew %2009-18-06.pdf</u>.

**Husmillo**, **F.R.**1982. Assessment of yield loss due to downy mildew of sugarcane caused by *Peronosclerospora philippinensis* (Weston) C.G. Shaw. Sugarcane Pathol. Newsl. 28: 17-24.

**Kenneth, R.G.** 1981. Downy mildew of Graminaceous crops. *In* The Downy Mildews. Spencer, D.M. (ed.). pgs. 367-394.

Ladhalakshmi, D., Vijayasamundeeswari, A., Paranidharan, V., Samiyappan, R., and Velazhahan, R. 2009. Molecular identification of isolates of *Peronosclerospora sorghi* from maize using PCR-based SCAR marker. World J. Microbiol. Biotechnol. 25: 2129-2135.

Micales, J.A., Bonde, M.R., and Peterson, G.L. 1988. Isozyme analysis and aminopeptidase activities within the genus *Peronosclerospora*. Phytopathology 78: 1396-1402.

**Payak, M.M.** 1975. Epidemiology of maize downy mildews with special reference to those occurring in Asia. Tropical Agriculture Research 8: 81-91.

Perumal, R., Nimmakayala, P. Erattaimuthu, S.R., No, E. –G., Reddy, U.K., Prom, L.K., Odovdy, G.N., Luster, D.G., and Magill, C.W. 2008. Simple sequence repeat markers useful for sorghum downy mildew (*Peronsclerospora sorghi*) and related species. BMC Genetics 9: 77.

Smith, D.R. and Renfro, B.L. 1999. Pg. 26, 28. *In*: D. G. White (ed.) Compendium of Corn Diseases, 3<sup>rd</sup> ed. APS Press, St. Paul, MN. 78 pp.

**USDA.** 1986. Pests not known to occur in the United States or of limited distribution. No. 77: Philippine downy mildew.

Weston, W.H. 1920. Philippine downy mildew of maize. J. Agric. Res., 19: 97-122.

**Yao, C.L., Magill, C.W., and Frederickson, R.A.** 1991a. Use of an A-T-rich DNA clone for identification and detection of *Peronosclerospora sorghi*. Applied and Environmental Microbiology 57(7): 2027-2032.

Yao, C.L., Magill, C.W., Frederickson, R.A., Bonde, M.R., Wang, Y., and Wu, P. 1991b. Detection and identification of *Peronosclerospora sacchari* in maize by DNA hybridization Phytopathology 81: 901-905.