



COSAVE

13. Annex: Regulated Pests

Magnaporthiopsis maydis

July 2023

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1. Technical description of the plague

1.1 Disease name in Spanish:

Marchitez tardía del maíz

English: Maize late wilt disease (LWD)

1.1.1 Etiological agent

Name preferred: *Magnaporthiopsis maydis* (Samra, Sabet & Hing.) Klaubauf, Lebrun & Crous, 2014.

Synonymy: *Cephalosporium maydis* Samra Sabet & Hing
Harpophora maydis (Samra, Sabet & Hing.) W. Gams

Taxonomic categorization:

Class: Sordariomycetes
Order: Magnaporthales
Family: Magnaporthaceae
Genus: *Magnaporthiopsis*
Species: *Magnaporthiopsis maydis*

1.2 Hosts / Species affected

Zea mays is the main host (CAB International, 2021). Secondary hosts (Dor,

S., Degani, O., 2019; Degani, O., 2021): *Setaria viridis*

Gossypium hirsutum

Citrullus lanatus

Lupinus termis

1.3 Cycle of the disease

The cycle of *Magnaporthiopsis maydis* is well documented. The pathogen infects maize and, besides being able to survive in the soil, it can also have secondary hosts such as *Setaria viridis*, *Gossypium hirsutum*, *Citrullus lanatus* and *Lupinus termis*.

According to Sabet *et al.* (1970a) infection occurs during the first three weeks of growth.

According to Deganil, O., 2021, citing other papers, the pathogen easily infects corn shoots. In the same work, it is reported that the fungus penetrated the roots and was first identified in the xylem, 21 days after planting. On day 35 after planting, the pathogen reached the first internode of the stalk. At 49 days, *M. maydis* spread to the fourth internode. At that stage (50 days after planting), relatively low but identifiable amounts of fungal DNA were measured by PCR in various parts of the plant. When corn panicles first emerged (day 63), the fungus was found throughout the stalk, although there was a lower concentration of the fungus towards the upper parts of the plant. DNA levels of *M. maydis* peaked in the stalks at this age of the plant and the first disease symptoms appeared shortly thereafter. At the late stage of the disease near harvest (75-85 in sweet cultivars), the fungus can be traditionally isolated from the cobs or more sensitively identified using PCR (Figure 1).

1.3.1 Transmission and survival

Late wilt of corn is a vascular disease caused by *Magnaporthiopsis maydis*. This fungus survives in the soil and seeds and infects seedling roots, invading the xylem vessels and translocating from them to the stalk and even to the grain (Sabet *et al.*, 1970b), in some cases causing rotting (Khokhar *et al.*, 2014). The initial growth of the fungus in the roots causes swollen, short, brown, thick-walled cells (Sabet *et al.*, 1970b).

Tests by Sabet *et al.* (1970a) showed that *Magnaporthiopsis maydis* has a low competitive saprophytic ability in soil and is inhibited by the growth of other soil microorganisms.

Singh and Siradhana (1988) determined that the mycelium of this fungus, when inoculated in soil under natural conditions, survived up to one and a half months, while when inoculated in sterile soil, it survived up to 3 months.

In soil, the main survival structures of *Harpophora maydis*, the sclerotia, can remain viable for up to 10 months at high temperatures (Singh and Siradhana, 1987a). In plant debris sclerotia can maintain viability for 12 to 15 months (Singh and Siradhana, 1987b). Moreover, inoculum persistence is localized in the first 20 centimeters of soil (Sabet *et al.*, 1970b).

Magnaporthiopsis maydis was found to remain viable and virulent in seed for up to 10 months under laboratory conditions.

At the local level, dispersal can occur via conidia, or movement of plant parts with reproductive structures, such as pycnidia in plant debris.

Over long distances, dispersal occurs through seeds (Galarza Bazan, 2005). The mycelium of the fungus can be present in the endosperm and embryo of maize seeds (EPPO, 2020; CAB International, 2021).

1.3.2 Incidence

Disease incidence and development are highly influenced by abiotic factors such as irrigation. Thus, in plants subjected to water stress, disease severity intensifies (Abd El Rahim *et al.*, 1998).

Other factors that predispose to infection are soil temperature and pH. Thus, temperature values outside the range 20-32°C and pH 4-8, hinder infection (Degani and Goldblat, 2014).

Maximum disease development in the field occurred at a constant temperature of 24°C or in the range of 20 to 32°C. There was less disease development at a constant temperature of 36 °C (Singh and Siradhana, 1987a).

In the laboratory, the optimum temperature for fungal growth is 30°C, with a maximum of 36°C, with no growth at 8°C (Samra *et al.*, 1963).

1.3.3 Symptoms associated with different organs and phenological stages

This disease appears at advanced stages of crop development, showing wilt symptoms around the flowering period, extending to the end of the cycle (Samra *et al.*, 1963). As a good example of vascular wilt, the symptoms are quite nonspecific and are usually distributed in the form of stands in affected fields.

Initially, the lower leaves begin to dry from the edge to the midrib and these symptoms rapidly ascend to the upper leaves. As the infection progresses, reddish-brown streaking is also visible on the basal internodes of the stem, which begin to dry out, shrink and, consequently, break. In addition, the ears

The number of grains per row is lower and even, in severe infections, they do not develop (Samra *et al.*, 1963) (Figure 2).

The fungus infects corn seedlings during the first month of growth, and no infections have been detected on plants older than 50 days (Sabet *et al.*, 1970a).

Brown cortical lesions develop on the seeds in the internode between the scutellum and coleoptile; the seminal roots are frequently destroyed. Single or confluent oval-shaped, irregular or elongated lesions, 1-10 cm long, with a pale cream-brown center and darker indeterminate margins, are present on the stem (Figure 3). The wilted leaves become dry and turn grayish-green, showing symptoms resembling frost damage. The pycnidium of the fungus appears as an orange-yellow pustule on the upper surface of the leaves.

Ear infection begins at the base of the ear, moving upward from the stalk; the entire ear becomes grayish-brown, shrunken, decaying and then lightening. In early infections, discoloration is prominent on the bracts of the ear. Black pycnidia may spread to the bracts or the sides of the kernel (Figure 4).

In late infected ears, no symptoms are shown, but the grains tend to detach; a whitish mold is commonly found between the grains (Galarza Bazan, 2005).

The internal symptoms caused by this fungus consist of darkening and subsequent necrosis of the vascular tissue of the plant (Figure 5).

1.3.4 Behavior and distribution in the batches

The fungus was detected in 39 of 42 seed samples in Egypt (Mohamed *et al.*, 1967). In Hungary, Michail *et al.* (1999), detected this fungus in a higher percentage in white maize seeds (1-9%), than in yellow cultivars (1-3%).

The fungus was detected in different parts of the ear, such as the ear stalk, seeds and the husk of naturally infected maize cultivars. In none of the parts did the level of infection exceed 10%.

Magnaporthiopsis maydis, was detected in the embryo, endosperm and seed coat in 12 of the 13 samples analyzed, with the exception of cv. Amon (Michail *et al.*, 1999).

1.3.5 Similarities with other pathogens

Magnaporthiopsis maydis, lacks a known teleomorph, but is similar to the anamorph of the genus *Harpophora* of the species *Gaeumannomyces* on culture medium (Saleh *et al.*, 2003).

Magnaporthiopsis maydis, can be distinguished from other *Acremonium* species, due to its rapid growth on complex culture media, minimal growth on Czapek agar and with eventual dark pigmentation (Samra *et al.*, 1963).

Conidiophores can be quite long and conidia are generally larger than those of other *Acremonium* species (Samra *et al.*, 1963; Gams, 2000).

According to Degani, O.,(2021), some scientific articles confused *Magnaporthiopsis maydis* with *Acremonium maydis*, but *Acremonium maydis* is another fungus. This was probably a misidentification.

2. Bibliography

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3. Annex: Figures

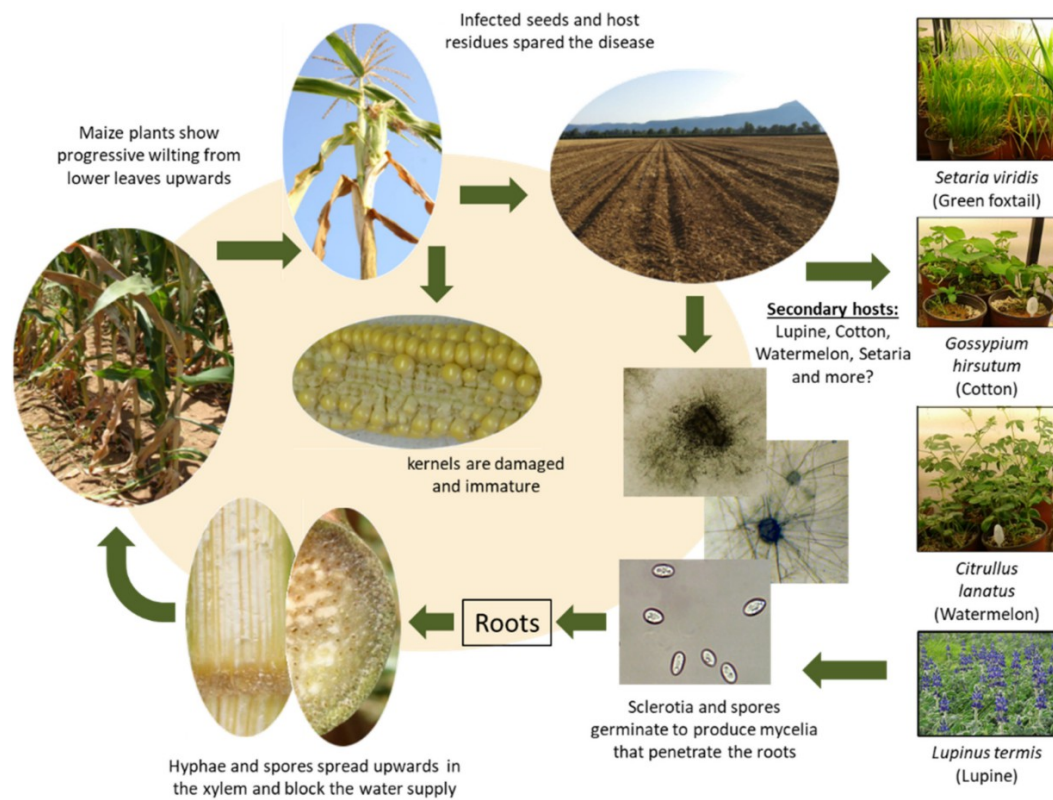


Figure 1: Disease cycle of *Magnaportheopsis maydis* (Replicated from DEGANI, Ofir, 2021).

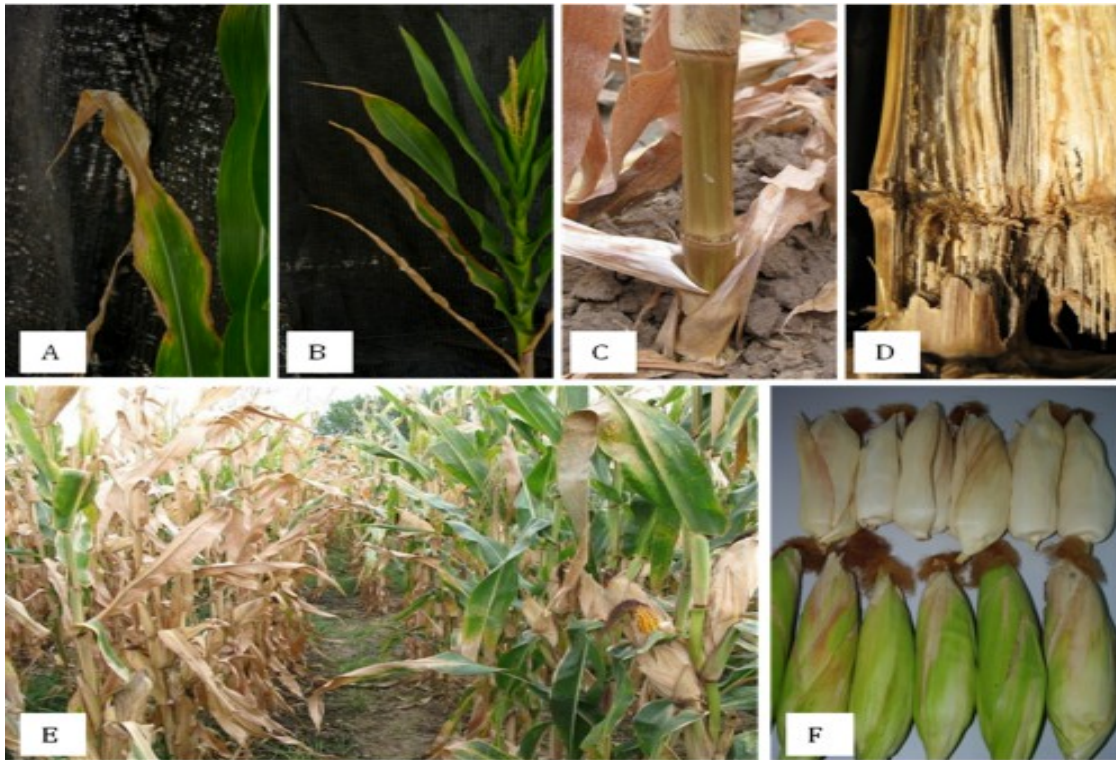


Figure 2: Symptoms of late wilt of maize. A) Advancement of wilt on the surface of a leaf; B) Disease progression from lower to upper leaves of the plant; C) Striation in basal internodes of the stalk; D) Drying and cupping in maize stalk; E) Plants of a variety susceptible to *Harpophora maydis* (left) and of a resistant one (right); F) Cobs of *Harpophora maydis* infected plants (top) and of healthy plants (bottom) (Ortiz Bustos, 2017).



Figure 3: Symptoms of late wilt of maize on stalk infected by *Harpophora maydis*. (Drori et al., 2013).



Figure 4: Symptoms of late corn wilt in ears infected by *Magnaporthiopsis maydis* (Plantix, 2022)

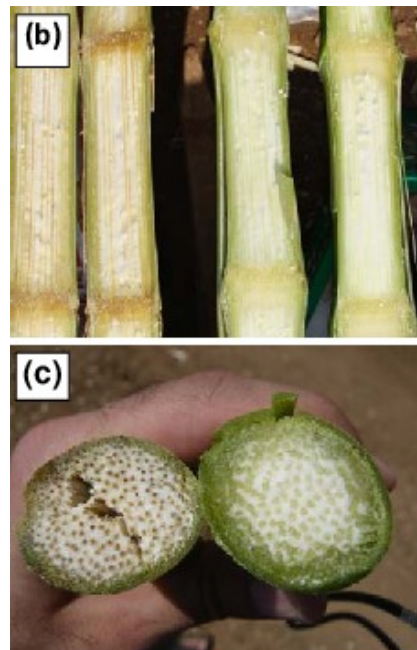


Figure 5: Internal symptoms of maize plant vascular tissue caused by *Harpophora maydis* (Degani and Cernica, 2014).

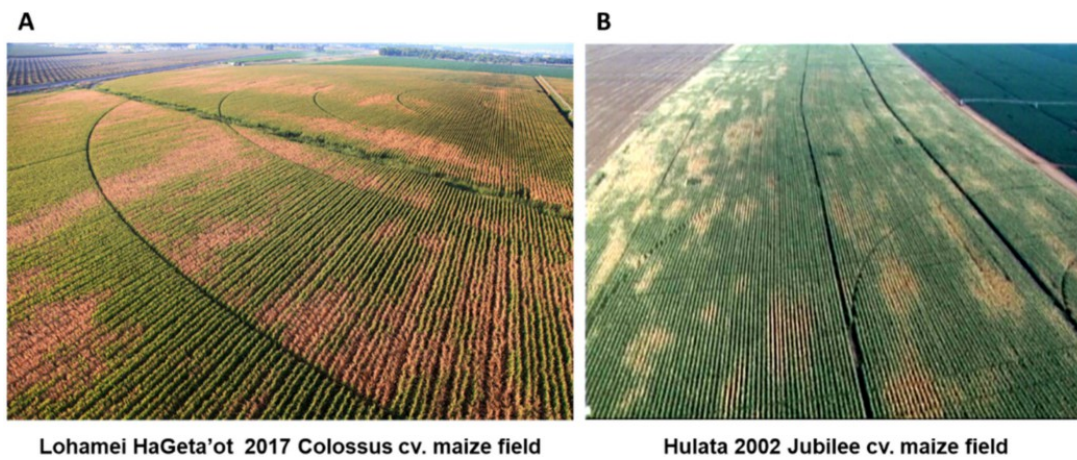


Figure 6: Aerial view of *Magnaporthiopsis maydis* symptoms in maize fields in Israel (Replicated photos from DEGANI article, Ofir, 2021).