



13. Annex: Regulated Pests

Pantoea stewartii* subsp. *stewartii

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1. Technical description of the pest

1.1 Disease name

English: Bacterial corn wilt, Stewart's disease of corn, Stewart's disease of corn

English: bacterial leaf blight of maize, bacterial wilt of maize, jackfruit bronzing, Stewart's disease, Stewart's wilt of maize.

1.1.1 Etiological agent

Preferred name: *Pantoea stewartii* subsp. *stewartii* (Smith) Mergaert, Verdonck & Kersters

Synonymy: *Aplanobacter stewartii*
Bacterium stewartii
Erwinia stewartii
Pseudomonas stewartii
Xanthomonas stewartii

Taxonomic categorization:

Class: Gammaproteobacteria
Order: Enterobacterales
Family: Enterobacteriaceae
Genus and species: *Pantoea stewartii*
Subspecies: *stewartii*

1.2 Host / Affected species

All types of maize (*Zea mays*) are hosts of *Pantoea stewartii* subsp. *stewartii* (CABI, 2020), especially sweet corn, but also susceptible varieties of dent corn and cultivars for popcorn and industry. Hybrid corn may be resistant to the first stage of the disease (wilt), but susceptible to the second stage (leaf blight) (ANPROS, 2020).

It occurs in *Poaceae* forages such as *Tripsacum dactyloides*, *Zea americana* (CABI and ANPROS 2020) and also several *Poaceae* weeds have been shown to act as asymptomatic hosts of the bacterium.

(ANPROS, 2020), including *Coix lacryma-jobi*, *Dactylis glomerata*, *Euchlaena perennis* and *Schlerachne punctata* (CABI, 2020).

1.3 Disease cycle

1.3.1 Transmission and survival

Pantoea stewartii subsp. *stewartii* survives on living host plants, insect vectors and seeds. There is no evidence that the bacterium is able to overwinter in soil or crop residues (CABI, 2020).

The route of long-distance dispersal is by seed (CIMMYT, 2004; Paliwal, 2001; CABI, 2020; ANPROS, 2020). However, in terms of international trade in seeds for planting, the probability of introducing (entry and establishment) of *Pantoea stewartii* to a new area, as a result of transmission by seed, is much lower than previously reported in the literature between the 1940s and 1990s (Pataky, 2003).

Maize plants are infected as a result of feeding wounds caused by insect vectors, which serve as entry points for the pathogen (De León, 1984, CIMMYT, 2004 and Paliwal, 2001).

The maize coleopteran, *Chaetocnema pulicaria*, is its main vector (De León, 1984; CIMMYT, 2004; CABI, 2020; ANPROS, 2020) and is very important in the production of maize (De León, 1984; CIMMYT, 2004; CABI, 2020; ANPROS, 2020).

important in the dissemination of *Pantoea stewartii* subsp. *stewartii* since after acquiring the bacterium it can carry and transmit it throughout its life (ANPROS, 2020).

The pathogen overwinters in the alimentary tract of *C. pulicaria*, overwinters in the soil in corn stover and animal manure, then emerges from hibernation and feeds on young corn plants, allowing the bacterium to be transmitted from one crop cycle to the next (CABI, 2020, CIMMYT, 2004).

It should be noted that the coleopteran *Chaetocnema pulicaria* is absent in Argentina (SINAVIMO, 2020).

Other possible vectors are *Diabrotica undecimpunctata howardi* (larva and adult), *Chaetocnema denticulata*, larva of *Delia platura*, *Agriotes mancus*, *Phyllophaga* sp. and larva of *Diabrotica longicornis barberis* (ANPROS, 2020).

As the survival of the pathogen within the other known insect vectors from one crop cycle to the next has not been tested, it is believed that the

The importance of its dissemination lies mainly within the crop cycle, intra- and inter-plant.

According to SENASA Peru, *Chaetocnema denticulata*, *Chaetocnema pulicaria* and *Agriotes mancus* are absent in Peru, while *Diabrotica undecimpunctata howardi* and *Delia platura* are present. According to information provided by the Argentine NPPO, the Plant Laboratory has tested samples for *Pantoea stewartii* pv. *stewartii* for more than 10 years, without finding positive results in any of the cases tested.

Early studies on the location of *Pantoea stewartii* subsp. *stewartii* in seed tissues indicated that the pathogen was present in the endosperm, but not in the seed coat. The pathogen was recovered from seeds up to 5 months after harvest, and in one study survived longer in corn stored at low temperatures, but disappeared after 200-250 days of storage at 8-15 °C (CABI, 2020).

The relationship between severity of plant infection and seed infection has been investigated. Seed transmission of *Pantoea stewartii* subsp. *stewartii* is closely associated with the severity of the parent plant infection, which is related to the susceptibility or resistance of the parent plant. Based on recent assessments of plant-to-seed and seed-to-seedling transmission rates, the likelihood of *P. stewartii* subsp. *stewartii* transmission in seed is extremely remote when seed is produced on resistant or moderately resistant parent plants, as resistance restricts the movement of *P. stewartii* subsp. *stewartii* in the vascular system of plants and prevents the plant from becoming systemically infected (CABI, 2020; Pataky, 2003).

According to Pataky (2003), plant-to-seed transmission is less than 0.3% for moderately resistant plants and less than 0.03% for resistant plants. When susceptible plants are infected systemically through natural methods, plant-to-seed transmission is approximately 10% or less. Therefore, it is likely that few seed lots have 35% or more infected kernels that have resulted in the highest rates of seed-to-seedling transmission. Seed-to-seedling transmission is likely to be very low (e.g., less than 0.06%) for seeds with less than 10% infected kernels, if *Pantoea stewartii* is transmitted in these seeds.

In the same vein, CABI (2020) refers to maximum seed infection estimates that were, for hybrids classified as resistant of 0.024%, for hybrids with moderate Stewart's wilt reactions of 0.19%, and for hybrids with susceptible reactions of 11.6% (adapted from CABI, 2020).

Petaky (2003) based on recent work, argues that it is evident that seed transmission of *E. stewartii* occurs at much lower rates than those recorded in the first half of the century. In modern maize hybrids and inbreds with improved levels of host resistance, transmission of *Pantoea stewartii* by seed is very low, if it occurs at all.

1.3.2 Incidence

According to Pataky & Ikin (2003), the bacterium has no environmental limitations for its development; conditions favorable for corn growth are also favorable for the bacterium. However, what can affect the incidence of the disease is the activity of the vector.

According to information provided by the Argentine National Pest Surveillance and Monitoring System (SINAVIMO), the main vector of the disease, *C. pulicaria*, is not present in Argentina.

1.3.3 Symptoms associated with the different organs and phenological stages

The severity of infection and the relative degree of resistance or susceptibility of a plant are associated with intraplant movement of *Pantoea stewartii* subsp. *stewartii*. In plants with highly susceptible reactions, infection is systemic and the bacterium can be isolated from tissues throughout the plant, including the seed. In plants with resistant reactions, the bacterium is generally restricted to tissues close to the site of infection, i.e., the wounds through which insects feed (CABI, 2020) and around these entry points the first symptoms develop (CIMMYT, 2004, De León, 1984 and Paliwal, 2001).

The disease has 2 stages or phases, which are differentiated by the moment in which the infection occurs.

1.3.3.1 Stage 1: Seedling stage

Infection occurs during the early stages of plant development (De León, 1984 and Paliwal, 2001). When susceptible cultivars are infected as seedlings, Stewart's disease of maize is distinguished by the presence of observable symptoms such as elongated, watery lesions with irregular or wavy margins along the leaves (CABI, 2020, CIMMYT, 2004, De León, 1984 and Paliwal, 2001). They then continue to develop along the veins (Figure 1) (De León, 1984) and acquire a light yellow color with irregular margins (Figure 2) (CIMMYT, 2004).

Systemic infection occurs on susceptible and moderately susceptible cultivars and distinctive foliar symptoms are evident on new leaves emerging from the plant whorl (CABI, 2020). In severe attacks, lesions eventually coalesce causing total leaf necrosis, infected seedlings show abnormal growth, wilt and often die shortly after flowering (De Leon, 1984 and Paliwal, 2001).

Systemic infections in seedling and vegetative stages cause stunting of plant growth and development, pollination will be delayed and these plants rarely reach reproductive stage (ANPROS and CABI, 2020). Severely infected plants do not produce ears (CIMMYT, 2004), show abnormally weak growth and die at flowering or immediately after (Paliwal, 2001).

If plants survive the infection, they will be so delayed that pollination will not occur or the seed will not mature at the indicated time (ANPROS, 2020) and those that do form seed will produce small, sterile ears or with poor grain formation (CIMMYT, 2004). In turn, the panicle will be whitish and weak in appearance (ANPROS, 2020) or may produce a premature, bleached and dead panicle (CABI, 2020).

Also, when infection is systemic, decay may form on the stems near the soil line, and in cases of severe infection, bacterial exudate may ooze through the stomata of the inner husks. The surface of the enveloped kernels may then become covered with bacterial slime (CABI, 2020).

Infection late in the growing season can cause severe leaf necrosis but not wilting (Figure 5) (CIMMYT, 2004).

The use of healthy seed and resistant varieties can control this disease (Paliwal, 2001). The impact of *Pantoea stewartii* subsp. *stewartii* on maize production has decreased due to the use of resistant hybrid lines, however, susceptible hybrids are still grown due to their desirable agronomic qualities (CABI, 2020).

In resistant cultivars, symptoms are generally limited to 2-3 cm around feeding wounds and systemic infection rarely occurs. If seedling infection occurs within a week of emergence, main stems may die, resulting in profuse tillering (CABI, 2020).

1.3.3.2 Stage 2: Panicle condition

After the initial infection, the infection may reach the stem. The leaf blight phase of Stewart's wilt occurs after panicle formation and infections that were not detected in the first phase will be clearly observed during the R2-R3 reproductive stages (ANPROS and CABI, 2020).

The bacterium multiplies and moves in the xylem of plants (CABI, 2020), clogging the vessels and causing the appearance of symptoms on leaves (Figure 3) (Mezzalama, 2016), and causing stunting, wilting and death of the plant (Figure 4) (De León, 1984, Paliwal, 2001 and CIMMYT, 2004).

Leaf symptoms are similar to those of the seedling wilt stage (CABI, 2020) and are characterized by leaf blight initially and after panicle. Pale green to yellow, irregular, short to long, irregular stripes appear and develop along the veins. Symptomatic tissue dies and turns straw-colored to brown (linear wilt) (ANPROS and CABI 2020).

Foliar symptoms originate from feeding wounds of the vectors. Like the foliar symptoms of the seedling wilt stage, necrotic tissues may extend the entire length of the leaves or symptoms may be limited to a few centimeters depending on the resistance or susceptibility of the cultivar (CABI, 2020).

While plants do not die at this stage, premature leaf death due to Stewart's wilt predisposes the weakened plant to stem rot and reduced yields (ANPROS and CABI, 2020).

1.3.4 Behavior and distribution in the lots

The behavior of *Pantoea stewartii* subsp. *stewartii* and its distribution in plots is closely related to its mode of dispersal. When the bacterium enters with the seed, a pattern of isolated patches with symptomatic plants is evident in the first instance. In the presence of a vector, the bacterium will spread rapidly through the lot, and the manifestation of symptoms on the plants will become uniform.

1.3.5 Similarities with other pathogens

Necrotic foliar symptoms of seedling wilt and Stewart's wilt stages may resemble the multiple and coalescing lesions of northern corn leaf blight (NCLB), caused by *Exserohilum turcicum* (t. *Setosphaeria turcica*). A simple microscopic examination of leaf tissue for bacterial exudate can easily differentiate Stewart's wilt and NCLB lesions (CABI, 2020).

Seedlings wilted by *Pantoea stewartii* subsp. *stewartii* may also resemble plants suffering from fungal seedling wilt, insect damage, drought stress, or nutritional deficiency. Bacterial exudate from symptomatic leaf tissue is also a simple diagnostic method to differentiate the seedling wilt stage from drought or other types of seedling stress (CABI, 2020).

2. Bibliography

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3. Annex: Figures



Figure 1: Watery spot continues to develop along the veins (De León, 1984).



Figure 2: Lesions with a watery appearance, with an irregular margin along the veins; they often turn yellow and extend towards the stem (CIMMYT, 2004).



Figure 3: Systemic infection in plants in the field (Mezzalama, 2016).



Figure 4: Damage can spread systematically in the stem and cause total wilting of the plant (De León, 1984).



Figure 5: Infection at the end of the crop cycle causes severe leaf necrosis but not wilting (CIMMYT, 2004).

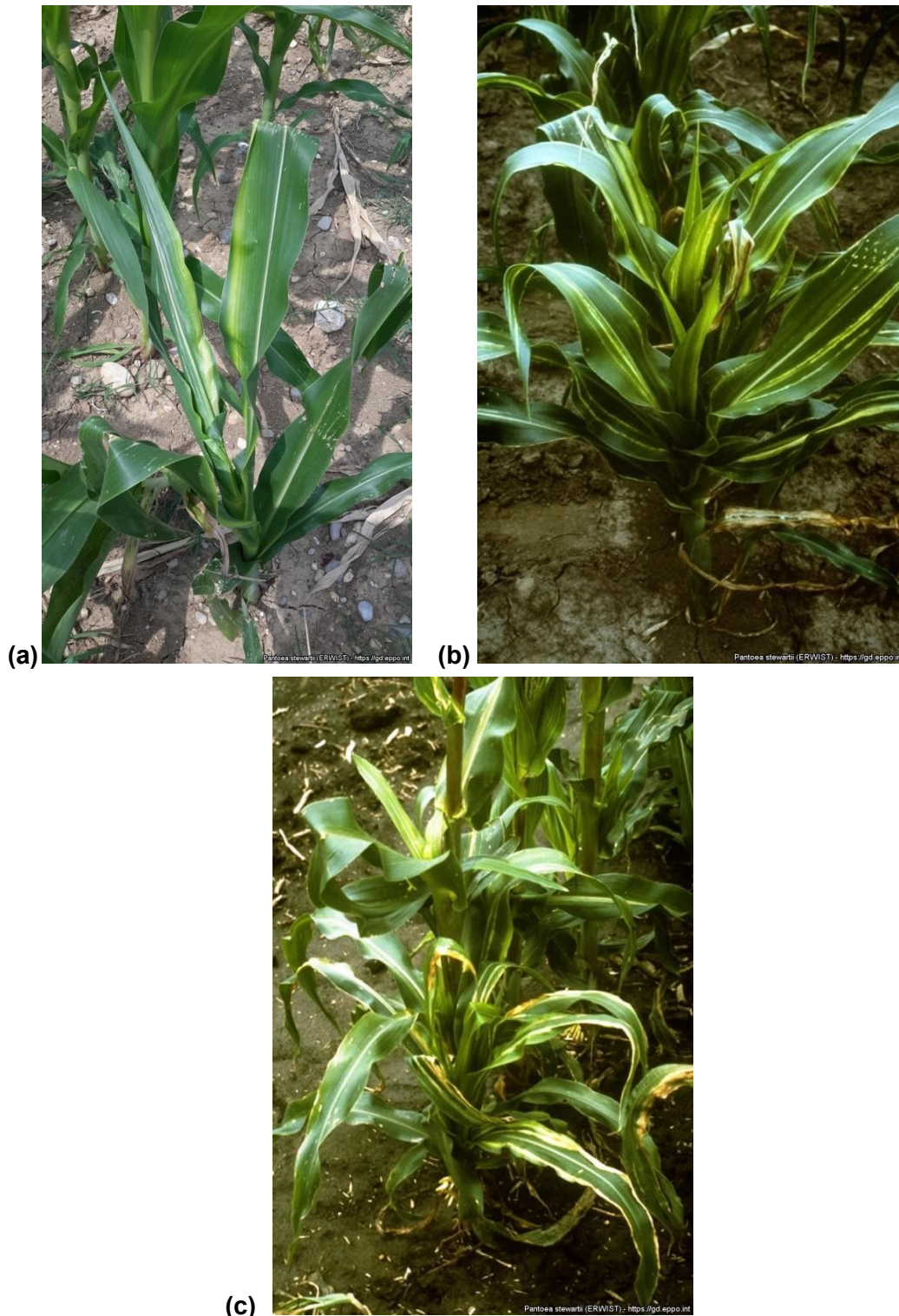


Figure 6: Maize plants infected with *Pantoea stewartii* subsp. *stewartii* showing shortening of internodes, in early crop stage with watery spots (a), chlorotic. (b) and in more advanced stages with necrotic spots (c) (EPPO, 2020).



Figure 7: Maize plant exhibiting symptoms of *Pantoea stewartii* subsp. *stewartii* on leaf (EPPO, 2020).