EPPO Datasheet: *Xanthomonas citri pv. fuscans*

Last updated: 2023-02-08

This datasheet covers the two bacterial species and pathovars that are associated with the common bacterial blight of bean: *Xanthomonas phaseoli* pv. *phaseoli* (XANTPH) and *X. citri* pv. *fuscans* (XANTFF).

IDENTITY

Preferred name: *Xanthomonas citri pv. fuscans*
Authority: (Schaad et al.) Constantin et al.
Taxonomic position: Bacteria: Proteobacteria: Gammaproteobacteria: Lysobacterales: Lysobacteraceae
Other scientific names: *Pseudomonas phaseoli var. fuscans* (Burkholder) Stapp, *Xanthomonas fuscans subsp. fuscans* Schaad et al., *Xanthomonas phaseoli var. fuscans* (Burkholder) Starr & Burkholder
Common names: bacterial blight of bean, bacterial leaf pustule of bean, common blight of bean, fuscous blight of bean
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EPPO Categorization: A2 list
view more categorizations online...
EU Categorization: RNQP ((EU) 2019/2072 Annex IV)
EPPO Code: XANTFF



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Notes on taxonomy and nomenclature

The causal agent of common bacterial blight of bean was first identified in 1897 as *Bacillus phaseoli* (Smith, 1897). Variant strains isolated in 1924 by Burkholder produced a brown pigment on tyrosine-containing medium and were thus described as fuscous strains (Burkholder, 1930). Different revisions of the taxonomy led the fuscous and non-fuscous strains to be grouped under the names *Xanthomonas phaseoli* (Corey & Starr, 1957), then *X. campestris* pv. *phaseoli* (Dye *et al.*, 1980), and then *X. axonopodis* pv. *phaseoli* (Vauterin *et al.*, 1995). Molecular divergence between fuscous and non-fuscous strains led to the proposition of a taxonomic distinction between the two, leading to the fuscous strains being renamed as *X. fuscans* subsp. *fuscans*, while the non-fuscous strains conserved the name *X. axonopodis* pv. *phaseoli* (Schaad *et al.*, 2005). Later on, heterogeneity within *X. axonopodis* pv. *phaseoli* was revealed based on AFLP analysis, leading to the description of three non-fuscous genetic lineages: GL1, GL2, and GL3 (Alavi *et al.*, 2008). Lineages GL2 and GL3 were, however, genetically distant from GL1 and grouped with the fuscous strains from *X. fuscans* subsp. *fuscans*. The four lineages of bacterial pathogens responsible for common bacterial blight of bean are currently distributed across two species within the *Xanthomonas* genus. Lineage GL1 corresponds to *X. phaseoli* pv. *phaseoli*, while the other three lineages (GL2, GL3, and fuscans) group under the name *X. citri* pv. *fuscans* (Constantin *et al.*, 2016).

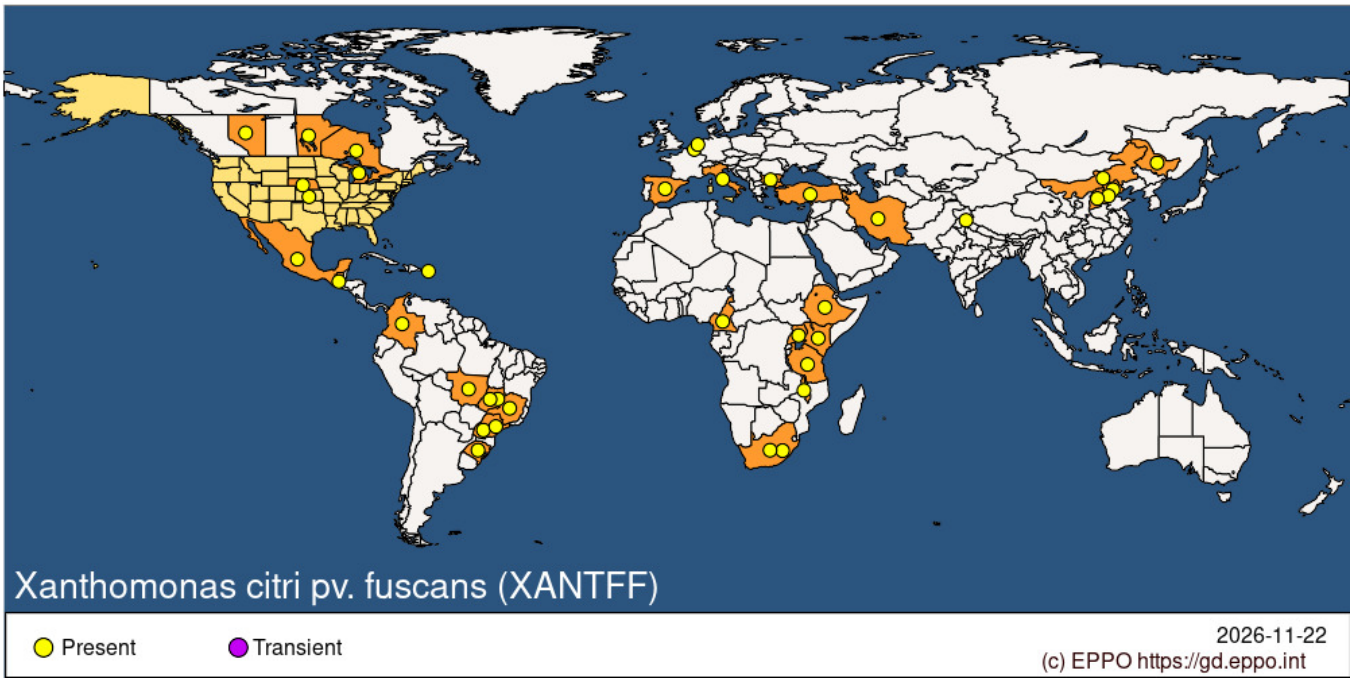
HOSTS

Besides common bean (*Phaseolus vulgaris*), which is the main host of *X. phaseoli* pv. *phaseoli* and *X. citri* pv. *fuscans*, natural infections have been reported on diverse other legume species (see host list, Bradbury, 1986; Gilbertson *et al.*, 1992). Additional hosts were reported after artificial inoculation. *X. phaseoli* pv. *phaseoli* was also reported from asymptomatic *Digitaria scalarum* and *Senna hirsuta* (Opio *et al.*, 1996).

Host list: *Phaseolus vulgaris*

GEOGRAPHICAL DISTRIBUTION

Common bacterial blight of bean is widely distributed over 100 countries across the five inhabited continents. The disease was reported in most regions where common bean is cultivated except in dry tropical areas. Although one can hypothesize that the bacteria originated from the same centre of diversity as their wild host *P. vulgaris* (from Mexico to Northern Argentina), the native range of the disease is unknown. This is due in part to the lack of geographic structuring of the strains, which may result from continuous movements of bacteria between regions through the global seed market (Mahuku *et al.*, 2006). Because most commonly used detection methods do not differentiate between *X. phaseoli* pv. *phaseoli* and *X. citri* pv. *fuscans* (Grimault *et al.*, 2014), it is still difficult to assess if the disease presence in a country is due to one species, the other or both.



EPPO Region: Belgium, Bulgaria, Italy (mainland), Netherlands, Spain (mainland), Türkiye
Africa: Cameroon, Ethiopia, Kenya, Lesotho, Malawi, South Africa, Tanzania, United Republic of, Uganda
Asia: China (Beijing, Hebei, Heilongjiang, Neimenggu, Shanxi), India (Himachal Pradesh), Iran, Islamic Republic of
North America: Canada (Alberta, Manitoba, Ontario), Mexico, United States of America (Michigan, Nebraska)
Central America and Caribbean: Guatemala, Puerto Rico
South America: Brazil (Distrito Federal, Goias, Mato Grosso, Minas Gerais, Parana, Rio Grande do Sul, Sao Paulo), Colombia

BIOLOGY

The main source of inoculum is infected seed. Bacteria can reside on both sides of the seed coat and on the surface of the embryo (Darrasse *et al.*, 2018; Zaumeyer, 1930), allowing overwintering and up to 30 years survival (Saettler, 1989). One seed in a lot of 10 000 to 30 000 is sufficient to cause a disease outbreak (Darrasse *et al.*, 2007; Opio *et al.*, 1993; Sutton & Wallen, 1970; Zaumeyer & Thomas, 1957). In tropical and subtropical areas, survival on weeds and crop residues represents an important source for bacterial dissemination (Fininsa & Tefera, 2001; Fininsa & Yuen, 2002; Santana, 1991). Epiphytic life is possible on various alternative hosts and weeds (Angeles-Ramos *et al.*, 1991; Cafati & Saettler, 1980; Gent *et al.*, 2005; Karavina *et al.*, 2011).

Primary infection usually starts with an epiphytic phase facilitated by aggregation in biofilms where bacterial populations grow and stabilize (Jacques *et al.*, 2005; Weller & Saettler, 1980). After growing on the leaf surface, bacteria enter the host tissues through openings such as stomata, hydathodes, or wounds (Rudolph, 1993; Zaumeyer, 1930). Within the host tissues, bacteria multiply exponentially and may express their pathogenicity when inoculated at ~10^6 bacterial cells per cm^2 or above (Weller & Saettler, 1980). Bacterial progression leads to colonization of the vascular tissues, which can lead to plant wilting in the most severe cases (Vidaver, 1993). In temperate areas, colonization of the plant is often asymptomatic but can still lead to efficient vertical transmission of the bacteria (Darrasse *et al.*, 2007; Weller & Saettler, 1980). Disease spread, incidence and severity are favored under warm

temperatures (28-32°C) and above 80% relative humidity (Weller & Saettler, 1980).

DETECTION AND IDENTIFICATION

Symptoms

All aerial parts of bean plants (seedling, leaf, stem, pod, and seed) can present symptoms caused by *X. phaseoli* pv. *phaseoli* or *X. citri* pv. *fuscans* (Gilbertson *et al.*, 1992; Zaumeyer, 1930). Symptoms on pods and/or leaves are very similar to those caused by *Pseudomonas savastanoi* pv. *phaseolicola*, the causative agent of halo blight, and it is seldom possible from visual examination to be certain which of these pathogens is present.

On leaves, symptoms appear as water-soaked spots usually starting from hydathodes and evolving into dry and brown necrotic lesions surrounded by a narrow yellow halo (Chupp & Sherf, 1960). These spots may merge, resulting in a burnt appearance, with possible defoliation and death of the plant. In case of systemic infection, a reddish-brown discoloration of the veins with water-soaking of adjoining interveinal areas may be observed. Infected stems present reddish longitudinal streaks.

On pods, symptoms appear as water-soaked spots, later evolving into dark red-brown lesions, slightly depressed circular spots, and possible bacterial ooze. Shrinking and death of pods may occur in the case of severe infection.

On seeds, symptoms appear as butter yellow spots that turn brown and are localized according to the infection pathway: on the hilum area in case of vascular transmission, at the micropyle in case of floral infection, and on the entire surface of the seed coat in case of infection by contact (Darrasse *et al.*, 2010; Maude, 1997). In severe cases, the seed may be shriveled, affecting germination rate and vigour (Darrasse *et al.*, 2018).

When grown from infected seed, seedlings are usually asymptomatic if infected with relatively low population numbers (Darrasse *et al.*, 2007). In more severe infections, seedlings can present water-soaked symptoms on the stem, cotyledons and/or primary leaves (Gilbertson *et al.*, 1992; Zaumeyer & Thomas, 1957). Angular, water-soaked areas frequently occur on the opposite sides of the primary leaves, indicating that the initial infection occurred while they were still folded together (Zaumeyer, 1930). Lesions on the stems of young seedlings begin as small water-soaked spots that gradually enlarge and sometimes become sunken. Plants often exhibit a characteristic wilting during the heat of the day, with recovery of turgidity at night (Zaumeyer, 1930). In some cases, seedlings may present injured or entirely destroyed growing tips (Gilbertson *et al.*, 1992; Zaumeyer & Thomas, 1957). If these plants do not die, buds may arise in the axils of the cotyledons and produce dwarfed plants with few pods.

Morphology

X. phaseoli pv. *phaseoli* and *X. citri* pv. *fuscans* are motile, aerobic, Gram-negative rod-shaped bacteria of 0.4-0.9 x 0.6-2.6 µm, with a single polar flagellum. Agar colonies are convex, yellow and wet-shining. A brown, diffusible pigment is produced by strains from the *fuscans* lineage when grown on tyrosine-containing media.

Detection and inspection methods

Detection on seeds include isolation of the bacterium followed by *in planta* pathogenicity assays and/or molecular detection by PCR, as validated by the International Seed Testing Association (Audy *et al.*, 1994; Grimault *et al.*, 2014). Commercial ELISA kits are also available, as well as LAMP-PCR primers (de Paiva *et al.*, 2020).

PATHWAYS FOR MOVEMENT

Sources of primary inoculum are infected seeds, infected weeds or volunteers. The bacteria further spread naturally over relatively short distances within or between fields. The only means of long-distance dispersal is by human transport of infected bean seed (Zaumeyer & Thomas, 1957). Secondary spread in the field mainly occurs by direct contact between infected plants, wind-blown rain or splashing. Dissemination of bacteria can also be caused by transportation via farm workers, or agricultural equipment (Belete & Bastas, 2017; Saettler, 1991). The role of bean-feeding insects as vectors is still understudied, but has been reported for a long time (Sackett, 1905; Zaumeyer &

Thomas, 1957). Potential insect vectors include *Chalcodermus ebeninus*, *Empoasca* sp., *Nezara viridula*, *Cerotoma ruficornis*, and *Diaprepes abbreviata* (Kaiser & Vakili, 1978).

PEST SIGNIFICANCE

Economic impact

Common bacterial blight is a major disease impacting common bean production (Broughton *et al.*, 2003). Yield losses up to 45% were reported in susceptible genotypes (Saettler, 1989; Wallen & Jackson, 1975; Yoshii *et al.*, 1975). Common bacterial blight agents directly reduce the area of photosynthetic tissues impacting yield of pods and seeds. Symptomatic edible fresh pods and edible seeds become unsaleable. Additional economic losses are due to the time and costs involved in controlling the disease. Common bacterial blight is a major threat for seed quality, as the bacterium is seed-transmitted. Infected seed lots, even in the absence of symptoms, cannot be sold in many countries, in particular where the disease does not occur-or has a limited distribution.

Control

A cornerstone of common bacterial blight management is pathogen detection on seed lots and regulation (see "Phytosanitary measures"). The most efficient management strategy is to use pathogen-free seeds, by producing seeds in specified areas either free of the bacteria (de Boisgrollier, 1993) or whose climatic conditions are non-conducive for the disease (Gilbertson *et al.*, 1992) and checking the absence of the pathogens. Cultural practices are essential to control the disease. If watering is necessary, furrow irrigation should be used rather than overhead irrigation, which mimics rainfall and promotes secondary spread of the bacteria (Akhavan *et al.*, 2013). Burial of residues is an effective way to reduce the survival of the bacteria (Chávez & Granada, 1988; Wimalajeewa & Nancarrow, 1980). Regular cleaning of harvesting equipment and seed containers is a means to limit primary infection, as bacteria can survive in dust or dirt on contaminated equipment (Belete & Bastas, 2017). Likewise, it is recommended to eliminate weeds, infected beans, and other potential hosts (Gilbertson *et al.*, 1990; Saettler *et al.*, 1986). Epidemics can effectively be reduced through employing long crop-rotations of 3 years or more to limit the risk of contamination by pathogens surviving on alternate hosts and volunteers (Schwartz *et al.*, 2005). Resistant bean varieties have been developed for the American market (Osorno *et al.*, 2016, 2020; Urrea *et al.*, 2019; Viteri *et al.*, 2014). However, breeding resistance to common bacterial blight is complex as it has variable heritability and level of expression depending on the environment, the genetic background, or the epidemic pressure (Miklas *et al.*, 2006; Singh & Schwartz, 2010). In addition, different genetic systems appear to control resistance in pods and leaves, and resistance may not be effective against the high diversity of pathogenic strains responsible for common bacterial blight (Aggour *et al.*, 1989; Duncan *et al.*, 2011).

Phytosanitary risk

Common bean is widely cropped throughout the EPPO region, and the major threat posed by common bacterial blight concerns seed quality, impacting both the seed industry and edible seed production. Depending on weather conditions and the level of seed inoculum management, the risk in terms of yield and quality losses is moderate to high. EFSA (2014) considered that the climatic conditions are widely favourable for the disease development in Europe, except in the northern EU countries and that the impact of the disease is limited in the EU due to the existing regulations against these pathogens.

PHYTOSANITARY MEASURES

As contaminated seeds are the main dispersal pathway for *X. phaseoli* pv. *phaseoli* and *X. citri* pv. *fuscans*, phytosanitary (quarantine) measures can be implemented to reduce the risk of long-distance dissemination of these pathogens. It can be recommended that consignments of bean seeds should have been produced from pest-free areas, or from pest-free places of production. Seed material imported from areas where the disease is known to occur should be certified for disease freedom via field inspections and laboratory testing.

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Datasheet history

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CABI/EPPO (1992/1997) *Quarantine Pests for Europe (1st and 2nd edition).* CABI, Wallingford (GB).

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