**EPPO Datasheet: *Xanthomonas phaseoli pv. phaseoli***

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*](https://gd.eppo.int/taxon/XANTFF/datasheet) (XANTFF).

**IDENTITY**

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| **Preferred name:** *Xanthomonas phaseoli pv. phaseoli***Authority:** (Smith) Constantin, Cleenwerck, Maes, Baeyen, Van Malderghem, De Vos, Cottyn**Taxonomic position:** Bacteria: Proteobacteria: Gammaproteobacteria: Lysobacterales: Lysobacteraceae**Other scientific names:** *Pseudomonas phaseoli* (Smith) Bergey et al., *Xanthomonas axonopodis pv. phaseoli* (Smith) Vauterin, Hoste, Kersters & Swings, *Xanthomonas campestris pv. phaseoli* (Smith) Dye, *Xanthomonas phaseoli* (Smith) Gabriel, Kingsley, Hunter & Gottwald**Common names in English:** bacterial blight of bean, bacterial leaf pustule of bean, common blight of bean, fuscous blight of bean[view more common names online...](https://gd.eppo.int/taxon/XANTPH/)**EPPO Categorization:** A2 list**EU Categorization:** RNQP (Annex IV)[view more categorizations online...](https://gd.eppo.int/taxon/XANTPH/categorization)**EPPO Code:** XANTPH | 4124.jpg[more photos...](https://gd.eppo.int/taxon/XANTPH/photos) |

**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*), whichis 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:** *Digitaria abyssinica*, *Helianthus annuus*, *Lablab purpureus*, *Mucuna deeringiana*, *Phaseolus aconitifolius*, *Phaseolus acutifolius*, *Phaseolus coccineus*, *Phaseolus lunatus*, *Phaseolus vulgaris*, *Phaseolus*, *Senna hirsuta*, *Solanum nigrum*, *Vigna angularis*, *Vigna mungo*, *Vigna radiata*, *Vigna unguiculata*

**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* (fromMexico 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 otheror both.

 **EPPO Region:** Austria, Belarus, Belgium, Bosnia and Herzegovina, Bulgaria, Cyprus, Czech Republic, France (mainland), Georgia, Germany, Greece (mainland), Hungary, Italy (mainland), Jordan, Lithuania, Moldova, Netherlands, Poland, Portugal (Madeira), Romania, Russia (Central Russia, Far East, Southern Russia), Serbia, Slovakia, Slovenia, Spain (mainland), Switzerland, Tunisia, Türkiye **Africa:** Angola, Burundi, Central African Republic, Congo, Democratic republic of the, Egypt, Eritrea, Eswatini, Ethiopia, Kenya, Lesotho, Madagascar, Malawi, Mauritius, Mozambique, Nigeria, Reunion, Rwanda, Somalia, South Africa, Sudan, Tanzania, Tunisia, Uganda, Zambia, Zimbabwe **Asia:** Bangladesh, Brunei Darussalam, Cambodia, China (Beijing, Heilongjiang, Henan, Hunan, Jiangsu, Jilin, Liaoning, Neimenggu, Shanxi, Xianggang (Hong Kong), Zhejiang), India (Delhi, Madhya Pradesh, Maharashtra, Punjab, Rajasthan, Uttar Pradesh), Indonesia, Iran, Japan, Jordan, Korea Dem. People's Republic, Korea, Republic, Lebanon, Malaysia (West), Myanmar, Nepal, Philippines, Sri Lanka, Taiwan, Thailand, United Arab Emirates, Vietnam, Yemen **North America:** Canada (Alberta, British Columbia, Manitoba, New Brunswick, Nova Scotia, Ontario, Prince Edward Island, Québec, Saskatchewan), Mexico, United States of America (Colorado, Connecticut, Florida, Georgia, Hawaii, Kentucky, Maine, Massachusetts, Michigan, Mississippi, Montana, Nebraska, New Hampshire, New York, North Carolina, North Dakota, South Dakota, Texas, Wisconsin, Wyoming) **Central America and Caribbean:** Barbados, Costa Rica, Cuba, Dominica, Dominican Republic, El Salvador, Guatemala, Honduras, Jamaica, Martinique, Nicaragua, Panama, Puerto Rico, St Vincent and the Grenadines, Trinidad and Tobago **South America:** Argentina, Brazil (Espirito Santo, Goias, Minas Gerais, Parana, Rio de Janeiro, Rio Grande do Sul, Santa Catarina, Sao Paulo), Chile, Colombia, Ecuador, Paraguay, Uruguay, Venezuela **Oceania:** American Samoa, Australia (New South Wales, Northern Territory, Queensland, Tasmania, Victoria, Western Australia), New Caledonia, New Zealand, Papua New Guinea, Samoa

 **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 ~106 bacterial cells per cm2 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.

**REFERENCES**

Aggour AR Coyne DP & Vidaver AK (1989) Comparison of leaf and pod disease reactions of beans (*Phaseolus vulgaris* L.) inoculated by different methods with strains of *Xanthomonas campestris*pv*. phaseoli* (Smith) dye. *Euphytica* **43**(1–2), 143–152. <https://doi.org/10.1007/BF00037907>

Akhavan A, Bahar M, Askarian H, Lak MR, Nazemi A & Zamani Z (2013) Bean common bacterial blight: Pathogen epiphytic life and effect of irrigation practices. *SpringerPlus* **2**(1), 1–9. <https://doi.org/10.1186/2193-1801-2-41>

Alavi SM, Sanjari S, Durand F, Brin C, Manceau C & Poussier S (2008) Assessment of the genetic diversity of *Xanthomonas axonopodis*pv*. phaseoli*and *Xanthomonas fuscans*subsp*. fuscans* as a basis to identify putative pathogenicity genes and a type III secretion system of the SPI-1 family by multiple suppression subtractive hybridizations. *Applied and Environmental Microbiology* **74**(10), 3295–3301. <https://doi.org/10.1128/AEM.02507-07>

Angeles-Ramos R, Vidaver AK & Flynn P (1991) Characterization of epiphytic*Xanthomonas campestris*pv*. phaseoli*and pectolytic Xanthomonads recovered from symptomless weeds in the Dominican Republic. *Phytopathology* **81**(6), 677–681.

Audy P, Laroche A, Saindon G, Huang HC & Gilbertson RL (1994) Detection of the bean common blight bacteria,*Xanthomonas campestris*pv*. phaseoli*and *X. c. phaseoli*var*. fuscans*, using the polymerase chain reaction. *Phytopathology* **84**(10), 1185–1192. <https://doi.org/10.1094/phyto-84-1185>

Belete T & Bastas KKK (2017) Common bacterial blight (*Xanthomonas axonopodis*pv*. phaseoli*) of beans with special focus on Ethiopian condition. *Journal of Plant Pathology & Microbiology***8**(3), 403. <https://doi.org/10.4172/2157-7471.1000403>

Bradbury JF (1986) *Guide to Plant Pathogenic Bacteria.* CAB international.

Burkholder WH (1930) The bacterial diseases of the bean. In *Cornell University Agricultural Experiment Station* (Vol. 127).

Cafati CR & Saettler AW (1980) Effect of host on multiplication and distribution of bean common blight bacteria. *Phytopathology* **70**(7), 675–679. <https://doi.org/10.1094/phyto-70-675>

Chávez CL & Granada GA (1988) Supervivencia de *Xanthomonas campestris*pv*. phaseoli,* agente causal de la bacteriosis del frijol, bajo condiciones del Valle del Cauca, Colombia. *Fitopatologia Colombiana* **12**, 9–14.

Chupp C & Sherf AF (1960) *Vegetable diseases and their control*. John Wiley & Sons. USA

Constantin EC, Cleenwerck I, Maes M, Baeyen S, Van Malderghem C, De Vos P & Cottyn B (2016) Genetic characterization of strains named as *Xanthomonas axonopodis*pv*. dieffenbachiae*leads to a taxonomic revision of the*X. axonopodis* species complex. *Plant Pathology* **65**(5), 792–806.

Corey RR & Starr MP (1957) Colony types of*Xanthomonas phaseoli*. *Journal of Bacteriology* **74**(2), 137–140. <https://doi.org/10.1128/jb.74.2.137-140.1957>

Darrasse A, Barret M, Cesbron S, Compant S & Jacques M (2018) Niches and routes of transmission of *Xanthomonas citri*pv*. fuscans* to bean seeds. *Plant and Soil***422***(*1–2), 115–128. <https://doi.org/10.1007/s11104-017-3329-3>

Darrasse A, Bureau C, Samson R, Morris CE & Jacques MA (2007) Contamination of bean seeds by *Xanthomonas axonopodis*pv*. phaseoli*associated with low bacterial densities in the phyllosphere under field and greenhouse conditions. *European Journal of Plant Pathology* **119**(2), 203–215. <https://doi.org/10.1007/s10658-007-9164-2>

Darrasse A, Darsonval A, Boureau T, Brisset MN, Durand K & Jacques MA (2010) Transmission of plant-pathogenic bacteria by nonhost seeds without induction of an associated defense reaction at emergence. *Applied and Environmental Microbiology* **76**(20), 6787–6796. <https://doi.org/10.1128/AEM.01098-10>

de Boisgrollier H (1993) Bilan des zones indemnes de bactérioses. *Bulletin Semences de La FNAMS* **124**, 41–44.

de Paiva BA, Wendland A, Teixeira NC & Ferreira MA (2020) Rapid detection of *Xanthomonas citri*pv*. fuscans* and *Xanthomonas phaseoli*pv*. phaseoli* in common bean by Loop-Mediated Isothermal Amplification. *Plant Disease* **104**(1), 198–203. <https://doi.org/10.1094/PDIS-02-19-0325-RE>

Duncan RW, Singh SP & Gilbertson RL (2011) Interaction of common bacterial blight bacteria with disease resistance quantitative trait Loci in common bean. *Phytopathology* **101**(4), 425–435. <https://doi.org/10.1094/PHYTO-03-10-0095>

Dye DW, Bradbury JF, Goto M, Hayward AC, Lelliott RA & Schroth MN (1980) International standards for naming pathovars of phytopathogenic bacteria and a list of pathovar names and pathotype strains. *Review of Plant Pathology* **59**(4), 153–159.

EFSA (2014) Scientific opinion on the pest categorisation of *Xanthomonas axonopodis*pv*. phaseoli* and *Xanthomonas fuscans*subsp*. fuscans*. *EFSA Journal* **12**(10), p.3856

Fininsa C & Tefera T (2001) Effect of primary inoculum sources of bean common bacterial blight on early epidemics, seed yield and quality aspects. *International Journal of Pest Management* **47**(3), 221–225. <https://doi.org/10.1080/09670870110044030>

Fininsa C & Yuen J (2002) Temporal progression of bean common bacterial blight (*Xanthomonas campestris*pv*. phaseoli*) in sole and intercropping systems. *European Journal of Plant Pathology* **108**(6), 485–495.

Gent DH, Lang JM & Schwartz HF (2005) Epiphytic survival of *Xanthomonas axonopodis*pv*. allii*and *X. axonopodis*pv*. phaseoli* on leguminous hosts and onion. *Plant Disease* **89**(6), 558–564. <https://doi.org/10.1094/PD-89-0558>

Gilbertson RL, Rand RE & Hagedorn DJ (1990) Survival of *Xanthomonas campestris*pv*. phaseol*i and pectolytic strains of *X. campestris*in bean debris. *Plant Disease* **74**(4), 322–327.

Gilbertson RL & Maxwell DP (1992) Common bacterial blight of bean. *Plant diseases of international importance. Volume II. Diseases of vegetables and oil seed crops,* 18–39.

Grimault V, Olivier V, Rolland M, Darrasse A & Jacques M-AA (2014) Detection of*Xanthomonas axonopodis*pv*. phaseoli*on*Phaseolus vulgaris*. In *International rules for seed testing, Annex to chapter 7: Seed health testing methods: 7-021*. International Seed Testing Association (ISTA), Bassersdorf, Switzerland.

Jacques M-A, Josi K, Darrasse A & Samson R (2005) X*anthomonas axonopodis*pv*. phaseoli* var. *fuscans* is aggregated in stable biofilm population sizes in the phyllosphere of field- grown beans. *Applied and Environmental Microbiology* **71**(4), 2008–2015. <https://doi.org/10.1128/AEM.71.4.2008-2015.2005>

Kaiser WJ & Vakili NG (1978) Insect transmission of pathogenic Xanthomonads to bean and cowpea in Puerto Rico. *Phytopathology* **68**, 1057–1063. <https://www.apsnet.org/publications/phytopathology/backissues/Documents/1978Articles/Phyto68n07_1057.PDF>

Karavina C, Mandumbu R, Parwada C & Tibugari H (2011) A review of the occurrence, biology and management of common bacterial blight. *Journal of Agricultural Technology* **7**(6), 1459–1474. <https://pdfs.semanticscholar.org/944a/5b1e16f4f8b08eb65a84ffc7049e3621febc.pdf>

Mahuku GS, Jara C, Henriquez MA, Castellanos G & Cuasquer J (2006) Genotypic characterization of the common bean bacterial blight pathogens,*Xanthomonas axonopodis*pv*. phaseoli* and *Xanthomonas axonopodis*pv*. phaseoli*var*. fuscans* by rep-PCR and PCR-RFLP of the ribosomal genes. *Journal of Phytopathology* **154**(1), 35–44.

Maude RB (1997) Seedborne diseases and their control: principles and practice. *CABI*, 280 pp.

Miklas PN, Kelly JD, Beebe SE & Blair MW (2006) Common bean breeding for resistance against biotic and abiotic stresses: From classical to MAS breeding. *Euphytica* **147**(1–2), 105–131. <https://doi.org/10.1007/s10681-006-4600-5>

Opio AF, Teri JM & Allen DJ (1993) Studies on seed transmission of *Xanthomonas campestris*pv*. phaseoli* in common beans in Uganda. *African Crop Science Journal* **1**(1), 59–67.

Opio AF, Allen DJ & Teri JM (1996) Pathogenic variation in *Xanthonomas campestris*pv*. phaseoli*, the causal agent of common bacterial blight in *Phaseolus* beans. *Plant Pathology***45**(6), 1126–1133.

Osorno JM, Grafton KF, Vander Wal AJ, Kloberdanz M, Schroder S, Vasquez JE, Ghising K & Pasche JS (2017) Improved tolerance to root rot and bacterial blights in kidney bean: Registration of ‘Talon’dark red kidney and ‘Rosie’light red kidney. *Journal of Plant Registrations* **11**(1),1-8.

Osorno JM, Grafton KF, Vander Wal AJ, Pasche JS, Posch J & Simons K (2020) *‘* ND Whitetail ’, a new white kidney bean with high seed yield and intermediate resistance to white mold and bacterial blights. *Journal of Plant Registration***14**, 102-109.

Rudolph K (1993) Infection of the plant by *Xanthomonas*. In *Xanthomonas* (pp. 193–264). Springer, Dordrecht (NL).

Sackett WG (1905) *Some bacterial diseases of plants prevalent in Michigan* (Vol. 230). Michigan State Agricultural College Experiment Station.

Saettler AW (1989) Common bacterial blight. *Bean Production Problems in the Tropics* **2**, 261–283.

Saettler AW (1991) Diseases caused by bacteria. In R. Hall (Ed.), *Compendium of Bean Diseases* (pp. 29–32). APS Press, St. Paul, MN, USA.

Saettler AW, Cafati CR & Weller DM (1986) Nonoverwintering of *Xanthomonas* bean blight bacteria in Michigan. *Plant Disease* **70**(4), 285. <https://doi.org/10.1094/PD-70-285>

Santana EA (1991) Longevity of *Xanthomonas campestris*pv*. phaseoli* in naturally infested dry bean (*Phaseolus vulgaris*) debris. *Plant Disease* **75**(9), 952. <https://doi.org/10.1094/PD-75-0952>

Schaad NW, Postnikova E, Lacy GH, Sechler A, Agarkova I, Stromberg PE, Stromberg VK & Vidaver AK (2005) Reclassification of *Xanthomonas campestris*pv*. citri* (ex Hasse 1915) Dye 1978 forms A, B/C/D, and E as *X. smithii*subsp*. citri* (ex Hasse) sp. nov. nom. rev. comb. nov.,*X. fuscans*subsp*. aurantifolii*(ex Gabriel 1989) sp. nov. nom. rev. comb. nov., and X. *Systematic and Applied Microbiology* **28**(6), 494–518. <https://doi.org/10.1016/j.syapm.2005.03.017>

Schwartz HF, Steadman JR, Hall R & Forster RL (2005) Compendium of bean diseases (Issue Ed. 2). *American Phytopathological Society* (APS Press), St. Paul, MN, USA.

Singh SP & Schwartz HF (2010) Breeding common bean for resistance to diseases: A review. *Crop Science* **50**(6), 2199–2223. <https://doi.org/10.2135/cropsci2009.03.0163>

Smith EF (1897) Description of *Bacillus phaseoli* n. sp. *Botanical Gazette* **24**, 192.

Sutton MD & Wallen VR (1970) Epidemiological and ecological relations of *Xanthomonas phaseoli* and *X. phaseoli*var*. fuscans* in southwestern Ontario, 1961-1968. *Canadian Journal of Botany* **48**, 1329–1334.

Urrea CA, Hurtado‐Gonzales OP, Pastor‐Corrales MA & Steadman JR (2019) Registration of great northern common bean cultivar ‘Panhandle Pride’ with enhanced disease resistance to bean rust and common bacterial blight. *Journal of Plant Registrations* **13**(3), 311-315.

Vauterin L, Hoste B, Kersters K & Swings J (1995) Reclassification of Xanthomonas. *International Journal of Systematic Bacteriology* **45**(3), 472–489. <https://doi.org/10.1099/00207713-45-3-472>

Vidaver AK (1993) *Xanthomonas campestris*pv*. phaseoli:* cause of common bacterial blight of bean. In JG Swings, EL Civerolo (Eds.), *Xanthomonas* (pp. 40–44). Chapman & Hall, London, UK.

Wallen VR & Jackson HR (1975) Model for yield loss determination of bacterial blight of field beans utilizing aerial infrared photography combined with field plot studies. *Phytopathology* **65**(9), 942-948.

Weller DM & Saettler AW (1980) Colonization and distribution of *Xanthomonas phaseol*i and *Xanthomonas phaseoli*var*. fuscans* in field-grown navy beans. *Phytopathology* **70**, 500–506.

Wimalajeewa DLS & Nancarrow RJ (1980) Survival in soil of bacteria causing common and halo blights of French bean in Victoria. *Australian Journal of Experimental Agriculture and Animal Husbandry* **20**, 102–104.

Yoshii K, Galvez GE & Alvarez G (1975) Estimation of yield losses in beans caused by common blight. *Fitopatologia Colombian*a **6**(2), 141-142.

Zaumeyer WJ (1930) The bacterial blight of beans caused by *Bacterium phaseoli*. *United States Department of Agriculture*.

Zaumeyer WJ & Thomas HR (1957) A monographic study of bean diseases and methods for their control. In *Technical Bulletins 169625, United States Department of Agriculture, Economic Research Service.*

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

EPPO (1978) EPPO Data Sheet on Quarantine Organisms no 60: *Xanthomonas phaseoli*subsp*. phaseoli. EPPO Bulletin* **13**(1), 62-66. <https://doi.org/10.1111/j.1365-2338.1978.tb02772.x>

