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Data Sheets on Quarantine Pests

Xanthomonas axonopodis pv. *citri*

IDENTITY

Name: *Xanthomonas axonopodis* pv. *citri* (Hasse) Vauterin *et al.* 1995

Synonyms: *Xanthomonas campestris* pv. *citri* (Hasse) Dye 1978
Pseudomonas citri Hasse
Xanthomonas citri (Hasse) Dowson
Xanthomonas citri f.sp. *aurantifoliae* Namekata & Oliveira
Xanthomonas citri (ex Hasse) nom. rev. Gabriel *et al.*
Xanthomonas campestris pv. *aurantifolii* Gabriel *et al.*

Taxonomic position: Bacteria: Gracilicutes

Common names: Citrus canker, bacterial canker of citrus, citrus bacterial canker (all strains); Asiatic canker, canker A, cancrosis A; South American canker, false canker, canker B, cancrosis B; Mexican lime cancrosis, canker C; citrus bacteriosis, canker D (English)
Chancre bactérien des agrumes (French)
Cancrosis de los cítricos (all strains); cancrosis asiática (A strain); cancrosis de la lima ácida, cancrosis del limonero gallego (C strain) (Spanish)

Notes on taxonomy and nomenclature: Reinstatement of *X. campestris* pv. *citri* to *X. citri* and reclassification of some pv. *citri* strains as *X. campestris* pv. *aurantifolii* have been proposed by Gabriel *et al.* (1989). This reinstatement of pv. *citri* to species level was based essentially on differences in restriction fragment length polymorphism (RFLP) and, therefore, the proposed reclassification was not considered to be valid at that time without additional complementary data (Vauterin *et al.*, 1990). Similarly, the proposed reclassification of parts of pv. *citri*, notably B, C and D strains (see Biology) as *X. campestris* pv. *aurantifolii* based primarily on qualitative RFLP differences may not have been justified at that time (Vauterin *et al.*, 1990; Young *et al.*, 1991). More recently, Vauterin *et al.* (1995), on the additional basis of data on DNA-DNA hybridization and the use of BIOLOG microplates, have proposed new species delineations within the genus *Xanthomonas*. The new name *X. axonopodis* pv. *citri* has been proposed for A strains, while *X. axonopodis* pv. *aurantifolii* has been proposed for B, C and D strains. The first of these names has been accepted by Young *et al.* (1996) but the second is still regarded as invalid.

This data sheet accepts the name *X. axonopodis* pv. *citri*, but otherwise continues to regard all A, B, C and D strains as forms of a single quarantine pest. The phytosanitary risk assessment for the EPPO region extends to all. The group of strains of xanthomonads on citrus in Florida (USA) known since 1984 as *X. campestris* pv. *citrumelo*, or E strains or nursery strains of *X. campestris* pv. *citri*, are treated in a separate data sheet (EPPO/CABI, 1996), where the arguments for considering it as a different pathovar are developed.

Bayer computer code: XANTCI

EPPO A1 list: No. 1

EU Annex designation: II/A1 - as all *Xanthomonas campestris* strains pathogenic to citrus

HOSTS

Known hosts are in the family Rutaceae. *Citrus* is the main host of economic importance. Natural infections are known to occur only on *Citrus* spp., hybrids and cultivars and on *Poncirus trifoliata*, *Fortunella* spp. (*F. japonica*, *F. margarita*), *Severinia buxifolia* and *Swinglea glutinosa*. In general, grapefruits (*C. paradisi*), limes (*C. aurantiifolia*) and *Poncirus trifoliata* are highly susceptible. Sour oranges (*C. aurantium*), lemons (*C. limon*) and oranges (*C. sinensis*) are moderately susceptible. Mandarins (*C. reticulata*) are moderately resistant. Other members of the Rutaceae, including members of the sub-families Aurantioideae, Rutoideae and Toddalioideae, are susceptible to artificial infection by *X. campestris* pv. *citri*. One non-rutaceous host, *Lansium domesticum* (Meliaceae), has been reported. Cancrosis B strains have a similar host range to cancrosis A strains, but affect certain hosts much less severely (Stall & Civerolo, 1991). Canker C and D strains affect only limes (*C. aurantiifolia*).

The potential host range in the EPPO region would include any wild or cultivated rutaceous plants grown in tropical and subtropical areas where the climate is conducive to infection and disease development.

GEOGRAPHICAL DISTRIBUTION

Citrus bacterial canker probably originated in South-East Asia. Subsequently, the pathogen was disseminated throughout Asia, and then to Africa, Oceania and South America (Rossetti, 1977; Commonwealth of Australia, 1984; Stall, 1988). In recent years, the disease has occurred in islands in the Indian Ocean, in the Middle East and in North America. Strains causing a milder form of the disease, with a narrower host range, were reported in South America (cancrosis B, canker C and D). These have not been isolated from naturally infected trees since the mid-1980s.

EPPO region: Absent.

Asia: Indigenous to and widespread as A strain throughout Asia, occurring in Afghanistan, Bangladesh, Cambodia, China (Fujian, Guangdong, Guangxi, Guizhou, Hubei, Hunan, Jiangsu, Jiangxi, Sichuan, Zhejiang), Hong Kong, India (Andaman Islands, Andhra Pradesh, Assam, Haryana, Karnataka, Maharashtra, Punjab, Tamil Nadu), Indonesia (Java), Iran, Iraq (Ibrahim & Bayaa, 1989), Japan (Honshu, Kyushu, Ryukyu Archipelago, Shikoku), Korea Democratic People's Republic, Korea Republic, Lao, Malaysia (peninsular, Sabah), Maldives (Roistacher & Civerolo, 1989), Myanmar, Nepal, Oman, Pakistan, Philippines, Saudi Arabia, Singapore, Sri Lanka, Taiwan, Thailand, United Arab Emirates (El-Goorani, 1989), Viet Nam (Whittle, 1992), Yemen (Cook, 1988).

Africa: A strain in Comoros, Côte d'Ivoire, Gabon, Madagascar, Mauritius, Mozambique (eradicated), Réunion (Aubert *et al.*, 1982), Seychelles, South Africa (eradicated), Zaire.

North America: Mexico (D strain only; declared no longer to occur, the original disease being attributed to *Alternaria limicola* by Palm & Civerolo, 1994); USA (introduced into Florida in 1912 and spread to Alabama, Georgia, Louisiana, South Carolina and Texas; eradicated in Florida by 1933 and from all USA by 1947; the A strain reappeared in Florida in 1986 (Whiteside, 1988) and an eradication programme is currently being conducted to eliminate the disease and preclude establishment and dissemination of the pathogen; after a period when eradication was thought to have been successful, the disease appeared again in private gardens in the Miami area in 1995).

Central America and Caribbean: The previous version of this data sheet (EPPO/CABI, 1992) mentioned unconfirmed reports in several countries of this region. After checking, all have been found to be erroneous.
South America: Argentina (A strain along the coast, B strain only in small isolated foci on lemons in southern Entre Rios); Brazil (A and C strains; São Paulo - in the region of Presidente Prudente; Paraná - northeast, north and west central; Mato Grosso do Sul - east, southeast and south; Santa Catarina; unconfirmed reports in Mato Grosso, Minas Gerais, Rio Grande do Sul); Paraguay (A, B and C strains; east and west (Chaco central)); Uruguay (A strain under eradication; Salto - on north bank of River Uruguay; Paysandu - north; B strain eradicated since 1985).
Oceania: Australia, at one time on Thursday Island, Queensland (Jones *et al.*, 1984) but now eradicated there (Catley, 1988); since the late 1980s, at one location in Northern Territory). Christmas Island (Shivas, 1987), Cocos Islands, Fiji, Guam, Northern Mariana Islands, Micronesia, New Zealand (eradicated), Palau, Papua New Guinea.
EU: Absent.
Distribution map: See CMI (1978, No. 11).

BIOLOGY

Civerolo (1984), Stall (1988) and Goto (1992) have recently provided general reviews of citrus canker. Different forms of citrus bacterial canker disease are recognized, based on geographical distribution, primary host naturally affected and differential pathogenicity of the causal bacterium. However, all recognized forms of the disease are currently considered to be caused by variants of *X. axonopodis* pv. *citri* (see, however, Notes on taxonomy and nomenclature). The Asiatic form (A strain) is the most virulent and widespread form of the disease, affecting many rutaceous hosts. Cancrosis B (B strain) affects primarily lemons in Argentina, Uruguay and possibly Paraguay, although other *Citrus* spp. can be affected. *X. axonopodis* pv. *citri* associated with cancrosis B may be attenuated strains of the pathogen associated with the Asiatic form of the disease. In Brazil, Mexican (Gallego) lime cancrosis (C strain) primarily affects *C. aurantiifolia*. The *X. axonopodis* pv. *citri* variants associated with different forms of the disease can also be differentiated by serology, phage typing, plasmid DNA content, genomic DNA fingerprinting and restriction fragment length polymorphisms, fatty acid composition, isoenzyme content and aminopeptidase activity. Recently, new strains have been described from *C. aurantiifolia* in India, Oman and Saudi Arabia, which produce typical symptoms on this host but not on other citrus species, and also differ serologically (Vernière *et al.*, 1993).

Since the disease reported in Mexico to have been caused by the D strain is now attributed to a fungus (*Alternaria limicola*; Palm & Civerolo, 1994), the question of the identity and pathogenicity to citrus of the existing cultures of the D strain remains intriguingly open.

The primary inoculum sources for spring infections are lesions on shoots and leaves resulting from infections the previous autumn and in which the pathogen overwinters. The bacterium survives in leaf, shoot and fruit lesions that develop during the spring. Current season lesions are sources of bacteria for secondary infections.

During wet, warm weather in the spring and early summer, the bacterium oozes out of overwintering lesions when free moisture is present. Young, actively growing leaves and shoots are infected. Infection occurs through natural openings (e.g. stomatal pores) and wounds. The bacterium multiplies in the intercellular spaces while the host cells divide, producing scab-like lesions.

The pathogen has been reported to survive for various periods of time in association with citrus and non-citrus hosts, in infected plant tissue debris and in the soil. Little information is available about the nature and extent of epiphytic survival of the bacterium on citrus. Survival of the pathogen in infected plant tissue debris and in the soil is generally short-lived, but is dependent upon climatic and edaphic factors. The survival of the bacterium in the rhizosphere and on the phylloplane of weeds ranges from less than 7 days to 62 days, depending upon the host, environmental conditions and the specific site. Nevertheless, the epidemiological significance of *X. axonopodis* pv. *citri* associated with non-citrus host weeds and plant debris, or in the soil, is not completely understood. Only bacteria surviving in lesions on citrus and citrus relatives are known to be of primary epidemiological significance.

DETECTION AND IDENTIFICATION

Symptoms

X. axonopodis pv. *citri* infects all above-ground parts of susceptible hosts, particularly young, actively growing leaves, twigs, stems, trunks, thorns and fruit. Infection of young leaves and twigs usually occurs within 10-21 days after shoots begin to develop.

Lesions first appear as pin-point spots that become small, slightly raised pustules or blister-like eruptions. Initially, these appear on the lower leaf surface about 7 days after infection. Subsequently, the blisters become visible on the upper leaf surface. The young lesions are usually translucent due to water-soaking of the tissue. Lesions are initially circular but may develop irregularly. Lesions are light-coloured at first and become tan or brown. As lesions develop, the epidermis ruptures and the lesions become spongy or corky. The lesions finally become crater-like with a raised margin and sunken centre. The centre of large, old lesions may crack and/or drop out. The ability to induce corky lesions (characteristic of *X. axonopodis* pv. *citri*) has been found to be associated with a pathogenicity locus which can be transferred to other strains of *X. campestris* (Swarup *et al.*, 1991).

A yellow halo surrounding the lesions is characteristic of the disease. However, a reliable diagnostic feature of these lesions is the water-soaked, oily or greasy margin that develops around the central necrotic tissue. This margin is readily seen with transmitted light.

The lesions in young twigs and stems are superficially similar to those on leaves but on twigs and stems there may be little or no chlorosis. Moreover, the lesions are generally irregularly shaped and may be sunken. On susceptible hosts, lesions also occur on branches and trunks of mature trees.

Citrus bacterial canker disease lesions on fruit are also superficially similar to those on leaves. The yellow halo may or may not be present. Old lesions are distinctly crater-like and appear as irregularly shaped, dark-brown, scabby masses on the fruit surface. The lesions do not penetrate the rind more than 1-3 mm.

Morphology

In morphology, and colony aspect in culture, the pathogen is a typical *X. campestris* (Hayward & Waterston, 1964). In view of the need to distinguish strains and/or pathovars on citrus, it is advisable to carry out specific tests for identification.

Detection and inspection methods

Serology using polyclonal and monoclonal antibodies, bacteriophage sensitivity, plasmid DNA content analysis, genomic DNA fingerprinting, restriction fragment length polymorphism analysis and fatty acid composition analysis have been useful for disease

diagnosis and pathogen identification. DNA probes highly specific to *X. axonopodis* pv. *citri* have now been developed (Hartung, 1992). However, conclusive identification of *X. axonopodis* pv. *citri* must be based on pathogenicity to citrus.

MEANS OF MOVEMENT AND DISPERSAL

Long-distance dissemination of the pathogen occurs primarily via the movement of infected planting and propagating material, such as budwood and rootstock seedlings or budded trees from nurseries. There is no confirmed record of seed transmission. Movement of diseased fruit is a potential means of long-distance spread of the pathogen but there is no authenticated record of this being related to the epidemiology of the disease. Infected cull fruit and processed fruit pulp could facilitate long-distance spread of the pathogen. Infested personnel, clothing, equipment, tools, field boxes and other items associated with harvesting and postharvest handling of fruit are also potential means of long-distance dissemination of *X. axonopodis* pv. *citri*. Long-distance dispersal of the pathogen by animals, birds and insects has been suggested but has not been conclusively demonstrated.

Short-distance spread of the pathogen within trees and from tree to tree occurs primarily by wind-driven rain.

PEST SIGNIFICANCE

Economic impact

The primary symptoms of *X. axonopodis* pv. *citri* are leaf and twig-spotting and rind blemishes. Under conditions favourable for infection, defoliation and premature fruit drop occur on susceptible hosts. Terminal shoot dieback of highly susceptible hosts can also occur under environmental conditions favourable for infection and disease development. The internal quality of infected fruit maturing on the tree is not affected. However, fruit with lesions reduces market value as fresh fruit. Secondary rotting organisms invade lesions, causing fruit to rot.

The disease is most serious in areas of high temperature (14-38°C) and high rainfall (more than 1000 mm per year) during the growing season. Infection is also promoted by winds greater than 6.5 m s^{-1} and by wounds. It is a disease of tropical and subtropical regions but it can occur and may become established in temperate and arid regions in the absence of adequate control measures. It is regarded as one of the most serious diseases of citrus in Japan, especially on satsumas (Furuhashi & Serizawa, 1994).

Control

In countries where the pathogen has become established, disease severity can be reduced by planting more resistant species, such as mandarins and oranges. Trees growing on rootstocks promoting less vigorous growth (e.g. *Poncirus trifoliata*) are generally less affected by the disease than those on more vigorous rootstocks (e.g. rough lemons - *C. jambhiri*). Cultural practices to minimize infection and disease development include the use of properly designed and placed windbreaks (rapidly growing trees, netting) around and within blocks of trees to reduce short-distance spread of the pathogen via wind-driven rain; allowing vegetation to grow between rows of trees to reduce injury from wind-blown sand; avoidance of working in affected groves when the trees are wet from dew or rain; removal of inoculum sources by pruning infected shoots and defoliation of affected trees; disinfestation of tools and equipment; control of leaf miners to reduce leaf injuries that facilitate infection; and the timely application of copper-containing sprays (Serizawa *et al*.,

1985; McGuire, 1988; Timmer, 1988) to protect foliage and fruit during periods of susceptibility to infection by the bacterium.

Phytosanitary risk

X. axonopodis pv. *citri* is listed as an A1 quarantine pest by EPPO (OEPP/EPPO, 1977) and is also a quarantine pest for IAPSC, JUNAC and NAPPO. Australia perceives itself to be very vulnerable to the spread of citrus canker from its present very limited distribution in the north of the country (Broadbent, 1992). On the other hand, New Zealand recently decided, on the basis of a Pest Risk Analysis, that ongoing citrus canker surveillance was no longer needed. *X. axonopodis* pv. *citri* presents a risk in the EPPO region because the environmental conditions conducive to infection and disease development probably occur in all citrus-growing areas around the world. Certainly, cancrosis A presents much the greater risk, cancrosis B a lesser risk and Mexican lime cancrosis an even lesser risk (since it is specific to *C. aurantiifolia* which is hardly cultivated in the region). However, there is at present no *X. campestris* on citrus in the EPPO region, and phytosanitary measures should be applied to all strains of *X. axonopodis* pv. *citri* in the broad sense used in this data sheet.

PHYTOSANITARY MEASURES

EPPO recommends (OEPP/EPPO, 1990a) that citrus-growing countries should prohibit import of plants for planting (except seeds and tissue cultures) of Rutaceae from countries where *X. axonopodis* pv. *citri* occurs. They may also prohibit import of rutaceous fruits from the same source. Alternatively, they may require that they are free of leaves and peduncles and come from a place of production free from the pest. Plants for planting from countries where *X. axonopodis* pv. *citri* does not occur may be imported provided the plants have been tested according to EPPO Phytosanitary Procedure No. 27 (OEPP/EPPO, 1990b) or have come from an area found free by detection survey.

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