**EPPO Datasheet: *Ralstonia solanacearum***

Last updated: 2021-11-29

**IDENTITY**

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| **Preferred name:** *Ralstonia solanacearum* **Authority:** (Smith) Yabuuchi et al. emend. Safni et al. **Taxonomic position:** Bacteria: Proteobacteria: Betaproteobacteria: Burkholderiales: Burkholderiaceae **Common names in English:** brown rot of potato, moko disease of banana [view more common names online...](https://gd.eppo.int/taxon/RALSSL/) **EPPO Categorization:** A2 list **EU Categorization:** A2 Quarantine pest (Annex II B) [view more categorizations online...](https://gd.eppo.int/taxon/RALSSL/categorization) **EPPO Code:** RALSSL | 18069.jpg [more photos...](https://gd.eppo.int/taxon/RALSSL/photos) |

**Notes on taxonomy and nomenclature**

*Ralstonia solanacearum* (Smith) Yabuuchi *et al.* (1995) is a species complex (RSSC) that comprises four phylotypes (Fegan & Prior, 2005). Each phylotype includes multiple phylogenetic and pathogenic variants differing in barcoding markers (including the 16S-23S rRNA gene intergenic spacer region and the, *hrpB*, *mutS* and *egl* genes), known as sequevars. Safni *et al.* (2014) reclassified the four phylotypes of the RSSC into three distinct species: *R. solanacearum* (Smith, 1896) Yabuuchi *et al.*, 1996 emend. Safni *et al.*, 2014 (Phylotype II), *Ralstonia pseudosolanacearum* Safni *et al.*, 2014 (Phylotypes I and III) and *Ralstonia syzygii* (Roberts *et al.*, 1990) Vaneechoutte *et al.*, 2004 emend Safni *et al.*, 2014 (Phylotype IV). This datasheet considers phylotype II as *R. solanacearum* *sensu stricto.*Taxonomy and nomenclature have been reviewed in detail by Paudel *et al*. (2020).

**HOSTS**

*R. solanacearum* (RSSC phylotype II) has a wide range of cultivated and wild hosts. Of major economic importance are the solanaceous crops (tomato, potato, *Capsicum* (sweet/bell/chilli) peppers, aubergine and tobacco) and the musaceous crops banana and plantain. Some genotypes with apparent host specificity were historically designated race status, i.e. on banana/plantain (race 2) and potato (race 3). Strains pathogenic to banana and plantain are now known to comprise several sequevars of phylotype II (PIIA-6, PIIA-24, PIIA-38, PIIA-41, PIIA-53, PIIB-3, PIIB-4 and PIIB-25), although only PIIA-24 and PIIA-53 appear to be restricted to banana and plantain, with the other sequevars naturally occurring on a wider host range including related *Heliconia* spp. as well as the solanaceous hosts. A variant of *R. solanacearum* PIIB-4, which is not pathogenic on banana, was termed PIIB-4NPB and was found to infect plantain, tomato, *Capsicum* peppers, aubergine, *Anthurium* and cucurbits in Martinique (Wicker *et al*., 2007). This strain has also been found in Brazil, Costa Rica, French Guiana, and Trinidad. Cellier *et al*. (2012) reported that PIIB-4 was found on potato in France.

A single sequevar of *R. solanacearum*, phylotype IIB sequevar 1 (PIIB-1; formerly referred to as race 3 biovar 2) has spread worldwide through trade in infected potatoes and has been introduced into the EPPO region (Janse, 1996). This strain has established within some river catchments in wild riparian plants (mainly *Solanum dulcamara*). This strain also infects *Pelargonium* spp. Another phylotype II sequevar (PIIA-50), which is widespread on potato in Brazil, has also been found on potato in one area of Portugal (Cruz *et al*., 2012).

Other sequevars of *R. solanacearum* (RSSC phylotype II) have a wide host range, often including solanaceous crops. Many perennial non-crop species, including nightshades of the Solanaceae, are also hosts of the pathogen worldwide and increase the potential of *R. solanacearum* to persist in the environment. New hosts are still being discovered e.g. blueberry (*Vaccinium corymbosum*) and *Hydrangea* in Florida (Ji *et al*., 2007; Norman *et al*., 2018). Natural hosts worldwide are listed below, focusing mainly on cultivated plants where isolates of the bacterium have been characterized to phylotype and sequevar of the RSSC. For historical host lists, see Kelman (1953), Bradbury (1986), Persley (1986) and Hayward (1994). Several other host lists, including wild species, have been reported, with a wider range than currently shown below, but for which the pathogen has yet to be fully characterized (e.g. Janse *et al*., 2004; Obregón *et al*., 2008; Prieto *et al.,* 2012; Lopes & Rossato, 2018).

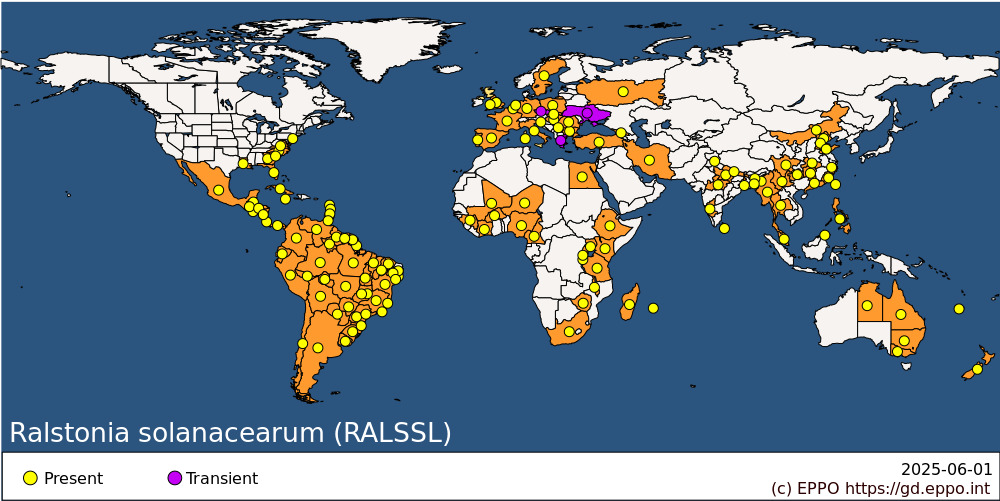
**Host list:** *Anthurium*, *Arachis hypogaea*, *Bidens mitis*, *Bidens pilosa*, *Canna indica*, *Capsicum annuum*, *Capsicum pubescens*, *Casuarina equisetifolia*, *Chenopodium album*, *Cichorium intybus*, *Citrullus lanatus*, *Cleome viscosa*, *Coleus amboinicus*, *Cucumis melo*, *Cucumis sativus*, *Cucurbita maxima*, *Cucurbita pepo*, *Datura stramonium*, *Eleusine indica*, *Emilia sonchifolia*, *Epipremnum pinnatum*, *Eucalyptus*, *Eupatorium cannabinum*, *Galinsoga parviflora*, *Galinsoga quadriradiata*, *Gliricidia sepium*, *Heliconia*, *Hydrangea macrophylla*, *Hydrangea paniculata*, *Hydrangea sp.*, *Hydrocotyle ranunculoides*, *Impatiens*, *Ipomoea hildebrandtii*, *Marsypianthes chamaedrys*, *Musa sp.*, *Musa x paradisiaca*, *Nicotiana tabacum*, *Oxalis sp.*, *Pandanus sp.*, *Pelargonium x hortorum*, *Pelargonium*, *Peperomia pellucida*, *Persicaria capitata*, *Persicaria pensylvanica*, *Phaseolus vulgaris*, *Physalis angulata*, *Piper dilatatum*, *Piper hispidum*, *Polygonum arenastrum*, *Portulaca oleracea*, *Psidium guajava*, *Salpiglossis sinuata*, *Senecio vulgaris*, *Sesbania*, *Solanum aethiopicum*, *Solanum americanum*, *Solanum betaceum*, *Solanum carolinense*, *Solanum cinereum*, *Solanum dulcamara*, *Solanum lycopersicum*, *Solanum melongena*, *Solanum nigrum*, *Solanum pseudocapsicum*, *Solanum sarrachoides*, *Solanum scabrum*, *Solanum tuberosum*, *Solanum villosum subsp. miniatum*, *Solanum villosum*, *Soliva anthemifolia*, *Tagetes*, *Urtica urens*, *Vaccinium corymbosum*, *Vaccinium hybrids*, *Xanthosoma sp.*

**GEOGRAPHICAL DISTRIBUTION**

*R. solanacearum* (RSSC phylotype II) is regarded to be of Central and South American origin where the largest diversity of genotypes is found. Certain strains have been dispersed worldwide through international trade in infected, often asymptomatic, vegetatively propagated crops e.g. banana/plantain suckers, potato tubers and ornamental host plants and plant parts. Although originally thought to originate in the rainforests of Costa Rica and the Caribbean area, the current distribution of strains causing Moko disease now covers many countries in Latin America. Furthermore, sequevar PIIB-3 has been found causing bugtok and Moko diseases of plantain (saba and cardaba ABB varieties) and dessert banana (AAA varieties) in the Philippines (Villa *et al*., 2021) and PIIB-4 has been found causing Moko disease of dessert bananas in Malaysia (Zulperi *et al*., 2016).

Originating in South America, where it is widespread, the sequevar PIIB-1 has spread worldwide to potato growing areas of Africa, Asia, Europe and Oceania. In addition to spread in infected potato, this strain has also spread through international trade of infected *Pelargonium* cuttings and entered protected horticulture in Europe and the USA (Janse *et al*., 2004; Kim *et al.,* 2003). Within the EPPO region, disease outbreaks in potato, tomato and *Pelargonium* have been strictly monitored and eradication measures imposed, although the bacterium has established in river systems in a number of countries, from which it is more difficult to eradicate entirely.

The worldwide reported distribution of *R. solanacearum*(RSSC phylotype II), is as follows:

 **EPPO Region:** Belgium, Bulgaria, Czechia, France (mainland), Georgia, Germany, Greece (mainland), Hungary, Italy (mainland, Sardegna), Netherlands, Poland, Portugal (mainland), Romania, Russian Federation (the) (Central Russia), Serbia, Slovakia, Slovenia, Spain (mainland), Sweden, Türkiye, Ukraine, United Kingdom (England, Wales) **Africa:** Burkina Faso, Burundi, Cameroon, Cote d'Ivoire, Egypt, Ethiopia, Guinea, Kenya, Madagascar, Malawi, Mali, Niger, Nigeria, Reunion, Rwanda, South Africa, Tanzania, United Republic of, Uganda, Zimbabwe **Asia:** China (Beijing, Fujian, Guangdong, Guizhou, Hebei, Hubei, Hunan, Neimenggu, Shandong, Sichuan, Yunnan, Zhejiang), India (Himachal Pradesh, Karnataka, Madhya Pradesh, Manipur, Meghalaya, Tripura, Uttar Pradesh, West Bengal), Iran, Islamic Republic of, Malaysia (Sabah, West), Myanmar, Nepal, Philippines, Sri Lanka, Taiwan, Thailand **North America:** Mexico, United States of America (Florida, Georgia, Louisiana, New Jersey, North Carolina, South Carolina, Virginia) **Central America and Caribbean:** Belize, Costa Rica, Cuba, El Salvador, Grenada, Guadeloupe, Guatemala, Honduras, Jamaica, Martinique, Nicaragua, Panama, Saint Vincent and the Grenadines, Trinidad and Tobago **South America:** Argentina, Bolivia, Brazil (Acre, Alagoas, Amapa, Amazonas, Bahia, Ceara, Distrito Federal, Espirito Santo, Goias, Maranhao, Mato Grosso, Mato Grosso do Sul, Minas Gerais, Para, Paraiba, Parana, Pernambuco, Piaui, Rio de Janeiro, Rio Grande do Sul, Rondonia, Roraima, Santa Catarina, Sao Paulo, Sergipe, Tocantins), Chile, Colombia, Ecuador, French Guiana, Guyana, Paraguay, Peru, Suriname, Uruguay, Venezuela **Oceania:** Australia (New South Wales, Northern Territory, Queensland, Victoria), New Caledonia, New Zealand

**BIOLOGY**

Although *R. solanacearum* is often described as soilborne, survival is usually short lived at low temperature in bare soil but is significant in alternative wild host plants (especially perennial nightshade species growing in waterlogged conditions or overwintering volunteers from susceptible crops). In Europe for example, infected riparian plants of *Solanum dulcamara* harbour *R. solanacearum* sequevar PIIB-1 that can then spread to susceptible potato and tomato crops when irrigated with contaminated surface water in the summer. The bacterium has been shown to survive in a viable but non-culturable (VBNC) form, but the epidemiological relevance of this is unclear (van Overbeek *et al*., 2004). Disease is usually most severe at temperatures of 24-35°C, although sequevar PIIB-1 has a lower optimum growth temperature than other strains and is therefore more suited to temperate potato-growing climates. High soil moisture or periods of wet weather or rainy seasons are associated with high disease incidence. Entry into plants is usually through root injuries from where the bacteria move by colonization of the xylem. Blocking of the vessels by bacterial biofilm is the major cause of wilting.

**DETECTION AND IDENTIFICATION**

**Symptoms**

In most hosts wilting is a common symptom of infections. The youngest leaves usually wilt first, with symptoms initially appearing at the warmest time of day. Wilting may be visible in only one stem, on one side of a plant or even sectoral in part of a leaf, depending where vascular infections occur (e.g. if they are restricted to sectors of stems and/or leaf petioles. Leaves may become bronzed or chlorotic and epinasty may occur. Wilting of the whole plant may follow rapidly if environmental conditions are favourable for pathogen growth. As the disease develops, a brown discoloration of the xylem vessels in the stem may be observed above the soil line and adventitious roots may develop. A creamy, slimy mass of bacteria exudes from vascular bundles when the stem is cut. Wilting and collapse of whole plants can lead to rapid death.

**On potato:**Foliar symptoms include rapid wilting of leaves and stems, usually first visible in single stems at the warmest time of day. Eventually, plants fail to recover and become yellow and then necrotic. As the disease develops, a streaky brown discoloration of the stem may be observed on stems above the soil line, and the leaves may have a bronze tint. Epinasty of the petioles may occur. A white, slimy mass of bacteria often exudes from vascular bundles which are cut or broken. If cut stem or tuber vascular tissue is placed in water, threads of bacterial ooze exude, distinguishing this from diseases caused by other wilting pathogens e.g. *Fusarium*, *Verticillium*, *Dickeya* and *Clavibacter*. This test is of presumptive diagnostic value in the field. Plants with foliar symptoms may bear healthy and diseased tubers, while plants that show no signs of the disease may sometimes produce infected tubers. On potato tubers, external symptoms may or may not be visible, depending on the state of development of the disease in relation to the prevailing temperature. Infection eventually results in bacterial ooze emerging from the eyes and stolon end of infected tubers. Soil may adhere to the tubers at the eyes. Cutting the diseased tuber reveals a browning and eventual necrosis of the vascular ring and immediately surrounding tissues up to 0.5 cm each side of the ring, starting from the stolon end. A creamy fluid exudate usually appears spontaneously on the vascular ring of the cut surface a few minutes after cutting. In the case of ring rot the tuber has to be squeezed in order to express a mass of yellowish decayed vascular tissue and bacterial slime. Atypical symptoms on potato (necrotic spots on the epidermis), possibly caused after lenticel infection, have been described by Rodrigues-Neto *et al*. (1984).

**On tomato, aubergine and *Capsicum* spp.:**The youngest leaves are the first to be affected and have a flaccid appearance, this usually first occurs at the warmest time of day. Wilting of the whole plant may follow rapidly if environmental conditions are favourable for the pathogen. Under less favourable conditions, the disease develops less rapidly, stunting may occur, and large numbers of adventitious roots are produced on the stem. The vascular tissues of the stem show a brown discoloration and, if the stem is cut crosswise, drops of white or cream bacterial ooze may be visible.

**On tobacco:**One of the main symptoms is unilateral wilting and premature yellowing. Leaves on one side of the plant or even a half leaf may show wilting symptoms. In severe cases, leaves wilt without changing colour and stay attached to the stem. As in tomato, the vascular tissues show a brown discoloration when cut open. The primary and secondary roots may become brown to black (Echandi, 1991).

**On cucurbits:**Symptoms on cucurbits, due to infection with the *R. solanacearum* PIIB-4NPB strain, develop rapidly from older to younger leaves that may wilt or not. Leaves turn yellow with necrotic lesions between or along major veins. Plants become flaccid and eventually collapse and die; there are no apparent symptoms on mature fruits (Wicker *et al*., 2007).

**On banana and plantain:**Moko disease first appears on young and fast-growing plants, the youngest leaves turn pale-green or yellow and wilt. Within a week all leaves may collapse. Young suckers may be blackened, stunted or twisted. The pseudostems show brown vascular discoloration (Hayward, 1983). Moko disease is easily confused with Panama disease caused by *Fusarium odoratissimum*. A clear distinction is possible when fruits are affected: brown dry rot is seen only in the case of Moko disease. In the Philippines, the symptoms of Bugtok disease on AAB type cooking banana are similar to those which cause Moko disease on AAA Cavendish banana. The two diseases are caused by the same strain of *R. solanacearum* (phylotype IIB sequevar 3), which is genetically identical to a strain from Honduras (Blomme *et al*., 2017).

**On blueberry:**Symptoms include bronzing of leaves, marginal leaf necrosis, and bacterial streaming from cut stems. Symptomatic plants resemble blueberry plants infected with *Xylella fastidiosa* (Norman *et al*., 2018)

**On *Anthurium* spp.:**Greasy, water-soaked lesions (on the lower leaf surface) turn necrotic with greasy margins (on the upper leaf surface). When the disease becomes systemic, these lesions (generally originating from the insertion point of the leaf with the petiole) develop following the main and secondary veins in a full or partial glove-shape. External infections (disseminated by water) may develop from any natural opening such as hydathodes. Leaves may turn yellow depending on the severity of the systemic invasion, and the stem may rot with abundant bacterial ooze. The plant eventually collapses and dies (Wicker *et al*. 2007).

**On geranium:**The first symptoms are wilting and subsequent chlorosis (often sectorial yellowing) of leaves. Stems may blacken and eventually become necrotic. Internally, vascular browning is often visible. Leaves later become brown and necrotic as the whole plant desiccates, collapses and dies (Janse *et al*., 2004).

**Morphology**

Gram-negative rods with a polar tuft of flagella, non-fluorescent, but diffusible brown pigment often produced. Virulent isolates develop pearly (opalescent) cream-white, flat, irregular and fluidal colonies often with characteristic whorls in the centre, which characteristically stain blood red on media containing tetazolium. A small, fluidal and round (SFR) colony-forming type is described amongst insect-transmitted Moko disease strains. Avirulent isolates form small round non-fluidal, butyrous colonies. See also Lelliott & Stead (1987) and Saddler (1994).

**Detection and inspection methods**

EPPO Standard PM 7/21 describes sampling methods, screening and identification tests for inspection and detection of the *R. solanacearum* species complex relevant for symptomatic and asymptomatic plant samples, and water samples. For field diagnosis from symptomatic tissues, bacterial slime oozing into clean water (as described above) is a simple test and there are lateral flow serological tests are commercially available. Suspected infections should be confirmed by laboratory testing. For testing asymptomatic plant material, it is advised to bulk sample and prepare extracts of vascular tissues from up to 200 stem base pieces, or in the case of potato tubers, up to 200 tissue cores from the heel ends at the point of stolon attachment. A range of screening tests are available that include isolation on semi selective and elective media, immunofluorescence microscopy (EPPO Standard PM 7/97) and a range of DNA-based tests that include conventional PCR, real-time PCR and LAMP tests. It is recommended to use more than one screening test to safeguard against false positive or false negative results. These tests can also be used to confirm the identity of bacterial colonies isolated on agar media. It may also be useful to conduct a pathogenicity test on a susceptible host, especially if the pathogen is found in a location for the first time. For accurate pathogen identification, phylotypes and sequevars are differentiated by DNA sequencing of 16S rRNA, egl, mutS and hrpB barcodes. Conventional PCR (Opina *et al*., 1997) or TaqMan qPCR (Weller *et al*., 2000) tests universally identify strains in all phylotypes whereas multiplex PCR tests identify each individual phylotype (Fegan and Prior, 2005) or host-specific strains within phylotypes (e.g. Cellier *et al.,* 2015). See EPPO Standard PM 7/21 for detailed information on the available tests.

**PATHWAYS FOR MOVEMENT**

The main pathway for international spread is via infected vegetative propagating material (e.g. seed potatoes, banana suckers and ornamental plants for planting). Asymptomatic (latent) infections, which escape visual inspections, are common at low temperatures when the rates of infection and colonization are slower, allowing host resistance mechanisms to be more effective. Similarly, latent infections also tend to occur in tolerant varieties.

Once infections are established, local spread can occur when the bacterium is transmitted mechanically during pruning operations or when cuttings are taken for propagation. Some strains of *R. solanacearum* which cause Moko disease are transmitted by insects (including pollinating flies, bees, wasps and thrips on banana) with potential for rapid spread over several kilometres. Spread to neighbouring plants can also occur through soil drainage water and by root contact. The bacterium also spreads through surface water. For example, many outbreaks of potato brown rot in Europe have been associated with spread from infected riparian *Solanum dulcamara* growing with roots in surface water which has then been used to irrigate potato or tomato crops (Janse, 1996). It is thought that initial infections of the *S. dulcamara* occurred when waste from imported ware potatoes with latent infections survived or bypassed sewage treatment, leading to contamination of watercourses inhabited by the wild host.

Within the RSSC complex natural infection of true seed has only been established for *R. pseudosolanacearum* in peanut (*Arachis hypogaea*) (Zhang *et al*., 1993). There have been findings of contaminated seed of some other hosts (including tomato, *Capsicum*, aubergine and soybean) although seed infection and transmission has not been substantiated. At present, transmission through water, soil or movement of infected vegetative plant parts is considered to be more important for most host plants than transmission via true seed.

**PEST SIGNIFICANCE**

**Economic impact**

*R. solanacearum* constitutes a serious obstacle to the culture and export of many crops in both tropical and temperate regions. Recently ranked by international phytobacteriologists as the second most important of all plant pathogenic bacteria after *Pseudomonas syringae* (Mansfield *et al*., 2012), the *Ralstonia* spp. have an extremely wide geographic distribution and host range. On potato alone, it is thought to be responsible for approximately 1 billion USD in losses each year, affecting some 3 million farmers and their families over 1.5 million ha in around 80 countries (Elphinstone, 2005). Moko disease has affected banana and plantain over thousands of km2 in Central and South America, particularly affecting small scale subsistence farmers. Also, Moko disease has caused considerable hardship to both subsistence and cash economies, where banana is a major, low input, staple source of carbohydrate, vitamins and minerals for countless communities. In many countries in which the pathogen has quarantine status, important losses result from increased surveillance, regulatory eradication measures and restricted further production on contaminated land.

**Control**

Disease management remains limited and is hampered by the faculty of the pathogen to survive for years in wet environments on plant debris or in asymptomatic weed hosts, which act as inoculum reservoirs. In the absence of any curative chemical control methods, prevention of bacterial wilt largely relies on the availability of pathogen-free planting material and effective surveillance and monitoring to protect areas which are free from the bacteria. For potato, effective disease management mainly relies on the use of limited generation seed multiplication from pathogen-free nuclear stocks with zero tolerances for the disease in official seed certification. Regular post-harvest testing of seed potato tubers is usually also necessary to avoid distribution of latent infections. Similarly, for other vegetatively propagated crops, there is a need to ensure planting material has been tested and found to be free of infection and that there are restrictions on the movement of planting material from affected to disease-free areas.

The effectiveness of strict regulatory control within Europe has been reviewed (EFSA, 2019). In relation to potato brown rot outbreaks in the EPPO region, use of healthy (tested) seed potatoes, early and reliable detection and reporting of the pathogen, quarantine measures on infected fields and farms, rotation with non-host crops for at least two years, control of weed hosts and volunteer plants (and in some cases of nematodes), prohibition of the use of contaminated surface water for irrigation, and education are key factors in control of *R. solanacearum* (EPPO Standard PM 9/3). For hydroponic glasshouse production systems, disinfection of recirculating water (e.g. using chlorine dioxide) can prevent spread of any introduced bacteria. These approaches have effectively halted international spread of *R. solanacearum* in geranium cuttings produced in Central America and East Africa for export to USA and Europe (Janse et al., 2004; USDA-APHIS, 2004).

For countries where the pathogen is widespread, various approaches to reduce disease impact through integrated cultural and biological control strategies have been reviewed (Yuliar *et al*., 2015). Disinfection of pruning and harvesting tools, e.g. using 20% solution of household bleach (3.5 % sodium hypochlorite) or less corrosive ammonia-based disinfectants, is important in preventing spread of disease e.g. in banana and plantain production. Insect transmission of Moko and banana blood diseases in commercial banana production has been successfully reduced by the now widespread practices of early male bud removal and/or bagging of the emerging florescence (Blomme *et al*., 2017). Although resistant varieties have been reported for some hosts (e.g. peanut, tobacco, tomato, aubergine) their widespread use is hampered by the broad diversity of the pathogenic strains and the difficulty of introducing resistance from related wild species without losing commercial yield and quality requirements. It is hoped that modern breeding methods will help to increase future availability of acceptable resistant varieties for the wide range of host crops (Huet, 2014).

**Phytosanitary risk**

*R. solanacearum* is a quarantine pest in many countries. The occurrence around the world of different strains of the pathogen presents an ongoing risk of the introduction of new variants capable of affecting the European and Mediterranean potato and tomato production. Absence of the bacterium is an important consideration for countries and pest free areas exporting seed potatoes.

The PIIB-1 sequevar of *R. solanacearum* (formerly known as race 3 biovar 2), causing potato brown rot, has a lower growth temperature optimum than other strains and appears to present the most important risk for the wider EPPO region. There is a definite risk that it could spread through imports of (latently) infected seed potatoes from countries where the disease now occurs. Furthermore, introduction of *R. solanacearum* by use of (latently) infected potatoes for table consumption, use as cattle fodder or for industrial processing is a potential risk if the potatoes, or wastes derived from them, are reintroduced into the agricultural system. Natural spread may take place if the bacterium is introduced via discharge of poorly or non-treated wastes into surface water, which is then used to irrigate susceptible crops. The PIIB-1 strain of *R. solanacearum* has also been previously introduced into the EPPO region in infected geranium plants for planting originating in Eastern Africa and Central America. Increased stringency of phytosanitary measures during production of the young geranium plants appears to have eliminated this risk.

Hosts other than potato are most likely to be affected in the warmer parts of the EPPO region, or under heated glasshouse conditions since strains other than PIIB-1 have higher temperature optima. Banana-infecting strains are not found in the banana-producing areas of the southern Mediterranean zone. A particular variant of the banana-infecting strain PIIB-4 of *R. solanacearum* (named PIIB-4NPB), is not pathogenic on banana but has been shown to infect cucurbits and *Anthurium* in Martinique and is also pathogenic on tomato, pepper, aubergine, *Impatiens hawkeri*, *Heliconia caribaea* and some weeds (*Portulaca oleracea*, *Cleome viscosa* and *Solanum americanum*). This strain is also present in Brazil, Costa Rica, French Guiana, and Trinidad and presents a potential risk if spread to the EPPO region in imported ornamentals (Wicker *et al*., 2007).

**PHYTOSANITARY MEASURES**

Measures should be applied to vegetative propagating material (e.g. seed potatoes, banana suckers and ornamental plants for planting) of host plants to prevent the international movement of the pathogen.

Visual inspections should be performed routinely upon export and import of host plants for planting. Laboratory checks are necessary to detect asymptomatic (latent) infections. EPPO Standard PM 8/1 recommends the phytosanitary measures which EPPO countries should use or require for seed and ware potatoes moving in international trade to prevent the introduction and spread of *R. solanacearum* species complex and other quarantine pests. EPPO Standard PM 3/21 *Post entry quarantine for potato* describes inspection and tests for the detection of pests (including *R. solanacearum*) infecting *Solanum* species or hybrids imported for germplasm conservation, breeding or research purposes, in post-entry quarantine. Plants for planting of *Musa* spp. and other host plants may be placed in post-entry quarantine to observe any symptoms and if relevant to test them to ensure their freedom from strains of the *R. solanacearum* species complex.

EPPO Standard PM 9/3 (under revision) describes a national regulatory control system for*Ralstonia solanacearum* that provides guidance on surveillance for the pathogen and its containment and eradication if found. Seed potato tubers, and other plants for planting of known hosts, should have been grown in areas found free from strains of the *R. solanacearum* species complex (RSSC) during the growing season and during the previous two growing seasons. Since *R. solanacearum* can also contaminate water courses, the irrigation of host plants with water from contaminated waterways should be prohibited.

Measures for seed are usually not needed except for peanut.

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