Data Sheets on Quarantine Pests

Ralstonia solanacearum

IDENTITY

Name: *Ralstonia solanacearum* (Smith) Yabuuchi *et al.*
Synonyms: *Bacterium solanacearum* (Smith) Chester
  Burkholderia solanacearum (Smith) Yabuuchi *et al.* (1992)
  Pseudomonas solanacearum (Smith) Smith

Taxonomic position: Bacteria: Gracilicutes
Common names: Brown rot (potato), southern bacterial wilt (tomato)
  Moko disease (banana), Granville wilt (tobacco) (English)
  Pourriture brune, Bactériose vasculaire (French)
  Braunfäule, Schleimkrankheit der Kartoffel (German)
  Podredumbre parda de la patata (Spanish)
Notes on taxonomy and nomenclature: In a taxonomic study of certain non-fluorescent species of the genus *Pseudomonas* (Yabuuchi *et al.*, 1992), the genus *Burkholderia* was proposed to encompass the variation found in this group and the name *Burkholderia solanacearum* was proposed. Subsequent study of this genus revealed that *R. solanacearum* was sufficiently distinct from other members of the genus to warrant assignment to the newly proposed genus *Ralstonia* (Yabuuchi *et al.*, 1995).
Bayer computer code: PSDMSO
EPPO A2 list: No. 58
EU Annex designation: II/A2

HOSTS

R. solanacearum as a species has an extremely wide host range, but different pathogenic varieties (races) within the species may show very limited host ranges. Within the EPPO region, the race which is now present and has potential for spread is race 3 (see below) with a limited host range, including in particular potatoes (*Solanum tuberosum*), tomatoes (*Lycopersicon esculentum*) and the weed *Solanum dulcamara*. Over 200 species, especially tropical and subtropical crops, are susceptible to one or other of the races of *R. solanacearum*. Worldwide, the most important are: tomatoes, *Musa* spp., tobacco (*Nicotiana tabacum*) and potatoes. Some minor host crops are: *Anthurium* spp., groundnuts (*Arachis hypogaea*), *Capsicum annuum*, cotton (*Gossypium hirsutum*), rubber (*Hevea brasiliensis*), cassava (*Manihot esculenta*), castor beans (*Ricinus communis*), aubergines (*Solanum melongena*) and ginger (*Zingiber officinalis*). Many weeds are also hosts of the pathogen and therefore increase the potential of *R. solanacearum* to build up inoculum. For extensive host lists, see Kelman (1953), Bradbury (1986), Persley (1986a), Hayward (1994a).

GEOGRAPHICAL DISTRIBUTION

R. solanacearum is widespread in tropical, subtropical and warm temperate areas throughout the world. For the EPPO region, it is mainly race 3 (see under Biology) which is of importance, since this so-called low-temperature strain is adapted to cooler climates in the highlands of the tropics and in the Mediterranean area. Its occurrence has now also been reported from temperate zones, and in particular race 3 has been reported from a number of European countries in the 1990s. The distribution is given below separately for *R. solanacearum* as a whole (except race 3), for confirmed or possible records of race 3, and for records of race 2 (causing Moko disease).

R. solanacearum (except race 3)

EPPO region: Denmark (found but not established in ornamental *Musa*), Netherlands (race 1 found incidentally in ornamental turmeric (*Curcuma*) in the glasshouse, imported from Thailand), Germany (intercepted only), Russia (reported on various crops, e.g. soybean, other than the hosts of race 3; status doubtful).

Asia: Armenia, Bangladesh, Bhutan, Brunei Darussalam, Cambodia, China (widespread), Georgia, Hong Kong, India (widespread), Indonesia (widespread), Iran, Japan, Korea Democratic People's Republic, Korea Republic, Malaysia (widespread), Myanmar, Nepal, Pakistan, Philippines, Russia (Far East), Singapore, Sri Lanka, Taiwan, Thailand, Viet Nam.

Africa: Angola, Burkina Faso, Burundi, Congo, Ethiopia, Gabon, Gambia, Kenya, Madagascar, Malawi, Mauritius, Mozambique, Nigeria, Réunion, Rwanda, Senegal, Seychelles, Sierra Leone, Somalia, South Africa, Swaziland, Tanzania, Tunisia, Uganda, Zaire, Zambia, Zimbabwe.

North America: Canada (found but not established on tomato and pelargonium in Ontario only), Mexico, USA (Arkansas, Florida, Georgia, Hawaii, North Carolina).

Central America and Caribbean: Belize, Costa Rica, Cuba, Dominica, Dominican Republic, El Salvador, Grenada, Guadeloupe, Guatemala, Haiti, Honduras, Jamaica, Martinique, Nicaragua, Panama, Paraguay, Puerto Rico, St. Lucia, St. Vincent and Grenadines, Trinidad and Tobago.

South America: Argentina, Belize, Brazil (widespread), Chile, Colombia, Ecuador, French Guiana, Guyana, Paraguay, Peru, Suriname, Uruguay, Venezuela.

Oceania: American Samoa, Australia (widespread), Cook Islands, Fiji, French Polynesia, Guam, Micronesia, New Caledonia, New Zealand, Papua New Guinea, Samoa, Tonga, Vanuatu.

EU: Absent.

Race 3 of R. solanacearum

(Records of unspecified races on potato in the EPPO region are treated as probable records of race 3). The bacterium is under eradication wherever it has occurred in the EU or in other EPPO countries.

EPPO region: Algeria (probable), Austria (probable, isolated incidents in 1995), Belarus (unconfirmed), Belgium (single outbreak in 1992; not found since 1994), Bulgaria (probable, found in the 1940/50s but not established), Cyprus (found in the 1950s but not established), Egypt, Finland (intercepted only), France (isolated incidents in 1995), Greece (including Crete), Israel (found at one site in the 1970s but eradicated), Italy (found in the 1950s; isolated incidents in 1995), Latvia (old unconfirmed records; now absent), Lebanon (probable), Libya (probable), Moldova (probable), Morocco (old unconfirmed records, never found on potato; now absent), Netherlands (isolated incidents in the early 1990s, several outbreaks in 1995), Poland (old unconfirmed reports from the 1940s; now absent), Portugal (isolated incidents on mainland in 1995; old unconfirmed report in Madeira, now absent), Romania (reported from symptoms only in the 1950s; now absent), Spain

(probable, found in 1981 but not established, in Canary Islands only; never found on mainland, the report in the first edition (EPPO/CABI, 1992) of an earlier, now eradicated, presence was erroneous), Sweden (probable, found on *S. dulcamara* in the 1970s and eradicated), Tunisia (old unconfirmed records; not found in recent surveys), Turkey, UK (single outbreak in potato in England in 1993; not since reported in potato, but still found in *S. dulcamara*), Ukraine (old unconfirmed records; now absent) and Yugoslavia (probable).

Asia: China (recorded on potato in Fujian, Guangdong, Guangxi, Hebei, Jiangsu and Zhejiang), Cyprus (see above), India, Indonesia (Java), Iran, Israel (see above), Japan, Nepal, Philippines (probable), Turkey.

Africa: Algeria (probable), Burundi, Egypt, Kenya, Libya (probable), Morocco (see above), South Africa, Tunisia (see above), Zambia.

North America: Mexico.

Central America and Caribbean: Costa Rica.

South America: Argentina, Brazil, Chile, Peru, Uruguay.

Oceania: Australia.

EU: Present.

Race 2 of *R. solanacearum*

(Causing Moko disease of bananas)

EPPO region: Libya.

Asia: India (West Bengal), Indonesia, Malaysia, Philippines, Sri Lanka, Thailand, Viet Nam.

Africa: Ethiopia, Libya, Malawi, Nigeria, Senegal, Sierra Leone, Somalia.

North America: Mexico, USA (Florida).

Central America and Caribbean: Belize, Costa Rica, Cuba, Dominican Republic (unconfirmed), El Salvador, Grenada, Guadeloupe, Guatemala, Haiti, Honduras, Jamaica, Nicaragua, Panama, Trinidad and Tobago.

South America: Argentina, Brazil, Colombia, Ecuador, Guyana, Paraguay, Peru, Suriname, Venezuela.

Distribution map: See CMI (1977, No. 138).

BIOLOGY

R. solanacearum does not behave as a single bacterium with a uniform biology and host range, but as a complex of variants, variously described as groups, races, biovars, biotypes, sub-races and strains. The different classifications of *R. solanacearum* have caused a considerable amount of confusion in the literature. Buddenhagen *et al.* (1962) distinguished three races on the basis of pathogenicity:

Race 1: Affecting tobacco, tomatoes, potatoes, aubergines, diploid bananas and many other (solanaceous) crops and weeds, with high growth temperature optimum (35-37°C).

Race 2: Affecting triploid bananas (causing Moko disease) and *Heliconia* spp., with high temperature optimum (35-37°C).

Race 3: Affecting mainly potatoes and tomatoes without a high virulence on other solanaceous crops, with lower temperature optimum (27°C). Other hosts are the weeds *S. dulcamara*, *S. nigrum*, *S. cinereum* (in Australia); the composite weed *Melampodium perfoliatum* (in Costa Rica); *Pelargonium hortorum*.

Two additional races affecting *Zingiber officinale* and mulberries (*Morus* spp.), respectively, were also distinguished (Buddenhagen, 1986), but their status is still unclear.

Hayward (1964) distinguished four biotypes (biovars) by their ability to produce acid from several disaccharides and sugar alcohols. Mulberry strains have been described as

biovar 5 (Buddenhagen, 1986). These biotypes do not correlate with the races of Buddenhagen *et al.* (1962). Only race 3, the potato race, is equivalent to Biotype II (Hayward, 1983). Races and biovars have been classified into two 'main groups' according to a restriction fragment length polymorphism (RFLP) analysis (Cook & Sequeira, 1988; 1994). Asian strains of race 1 (biovars 3,4,5) clustered as a group, American strains of race 1 (biovar 1), race 2 (biovar 1) and race 3 (biovar 2) as another.

Race 3 (biovar 2) has appeared in many fingerprinting studies to be very homogeneous. When studies of South American strains of race 3 (biovar 2) were made, however, more variation was observed: a) "normal" strains found east of the watershed of the Andes and all over the world, b) strains that are biochemically different, until now only found west of the watershed of the Andes and c) strains that behave almost as an intermediate between race 1 and 3 that occur in the lowlands of South America (also named biotype 2N or 2T). It is not known whether the types mentioned under b) and c) are also found in other parts of the world (Janse, 1991; Gillings & Fahy, 1994; Hayward, 1994). These findings, as well as the fact that resistance is found in the wild *Solanum phureja* (Sequiera & Rowe, 1969) indicate South America as the possible origin of race 3.

The bacterium can spread in soil, in which it survives for varying periods of time, and in irrigation (drainage) water. In tropical areas, many weeds have been shown to be alternate hosts. The slow rate of development of the bacterium on the weeds allows them to withstand infection, and so provide a bridge for the pathogen between crops.

Entry into plants is by way of injured roots, stem wounds or through stomata. Within the plant, the bacteria move in the vascular bundles, a process which is accelerated by higher temperature. Speed of movement is also dependent on the plant part colonized, for instance in tobacco bacteria move quicker in the stalk than in the roots (Ono *et al.*, 1984). This is followed by colonization of the xylem (Xiao *et al.*, 1983), where the bacteria adhere to the vessel walls or invade the lumen. They adhere by polar attraction to the cell surfaces and subsequently become localized at preferential sites of the mesophyll (Petrolini *et al.*, 1986). Blocking of the vessels by bacteria is the major cause of wilting.

The disease is most severe at 24-35°C; it is seldom found in temperate climates where the mean temperature for any winter month falls below 10°C. There are distinct temperature requirements for optimum disease development and reproduction for the different races (biovars) (Swanepol, 1990).

High soil moisture and periods of wet weather or rainy seasons are associated with high disease severity. Soil moisture is also one of the major factors affecting reproduction and survival of the pathogen; the most favourable soil moisture is -0.5 to -1 bar while -5 to -15 bar is unfavourable (Nesmith & Jenkins, 1985).

Slightly unfavourable weather conditions such as low temperatures influence symptom expression. In Kenya, certified and obviously healthy (but latently infected) potato seed tubers produced at altitudes of 1520-2120 m showed infection when planted at lower altitudes (Nyangeri *et al.*, 1984). This was due to a latent infection of the tubers grown in an environment less favourable to the pathogen.

For further information, see also Kelman (1953), OEPP/EPPO (1961), Buddenhagen & Kelman (1964), Persley (1986b), Hayward (1994b).

DETECTION AND IDENTIFICATION

Symptoms
On potatoes
Foliage: The first visible symptom is a wilting of the leaves at the ends of the branches during the heat of the day with recovery at night; eventually, plants fail to recover and die.

As the disease develops, a streaky brown discoloration of the stem may be observed on stems up to 2.5 cm or more above the soil line, and the leaves have a bronze tint. Moreover epinasty of the petioles may occur. A white, slimy mass of bacteria exudes from vascular bundles which are broken or cut. This slime oozes spontaneously from the cut surface of a potato stem in the form of threads, when kept in a beaker with water. Such threads are not formed by other bacterial pathogens of potato. This test is of presumptive diagnostic value in the field.

Tubers: External symptoms may or may not be visible, depending on the state of development of the disease; furthermore, symptoms may be confused with those of ring rot due to *Clavibacter michiganensis* subsp. *sepedonicus* (EPPO/CABI, 1996). *R. solanacearum* can be distinguished by the bacterial ooze that often emerges from the eyes and stem-end attachment of infected tubers. When this bacterial exudate dries, a mass of soil adheres to the tubers at the eyes. Cutting the diseased tuber will reveal a browning and necrosis of the vascular ring and immediately surrounding tissues up to 0.5 cm each side of the ring. 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 press out a mass of yellowish dissolved 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).

Plants with foliar symptoms caused by *R. solanacearum* may bear healthy and diseased tubers, while plants that show no signs of the disease may sometimes produce diseased tubers.

On tomatoes

The youngest leaves are the first to be affected and have a flabby appearance, usually 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 yellowish bacterial ooze may be visible (McCarter, 1991).

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 bananas

Moko disease, caused by *R. solanacearum*, is easily confused with the disease caused by *Fusarium oxysporum* f.sp. *cubense*. A clear distinction is possible when fruits are affected - a brown and dry rot is only seen in the case of Moko disease. On young and fast-growing plants, the youngest leaves turn pale-green or yellow and collapse. Within a week all leaves may collapse. Young suckers may be blackened, stunted or twisted. The pseudostems show brown vascular discolouration (Hayward, 1983).

Morphology

R. solanacearum is a Gram-negative rod, 0.5-1.5 µm in length, with a single polar flagellum. The positive staining reaction for poly-ß-hydroxybutyrate granules with Sudan Black B or Nile Blue distinguishes *R. solanacearum* from *Erwinia* species. In addition, *R. solanacearum* stains heavily at the poles with carbol fuchsin. Agar colonies are initially smooth, shining and opalescent, but become brown with age. See also Lelliott & Stead (1987) and Saddler (1994).

Detection and inspection methods

The bacterium may be obtained from infected tubers or stems for staining purposes if a small portion of tissue is pressed onto a clean glass slide. Potato tubers can be visually checked for (internal) symptoms by cutting. Tubers suspected to be (latently) infected should be diagnosed in the laboratory. Appropriate laboratory methods to detect the pathogen, also in its latent form, are indirect immunofluorescence antibody staining (IFAS) and a pathogenicity test on tomato to confirm a posititive IFAS result. Standard samples of 200 tubers per 25 t of potatoes are taken (Janse, 1988, OEPP/EPPO, 1990a). Recently a very effective selective medium has been described (Engelbrecht, 1994), that can also be applied for detection in environmental samples. ELISA and the polymerase chain reaction (PCR), based on 16S rRNA targeted primers, have also been used successfully. Biochemical tests, fatty acid analysis, RFLP and protein analysis can be used for identification purposes (Seal *et al.*, 1993; Seal & Elphinstone, 1994).

MEANS OF MOVEMENT AND DISPERSAL

The natural spread of most of the *R. solanacearum* races is very limited and slow. However, race 2, which causes Moko disease of banana, is known to be transmitted by insects and has a high potential for natural spread. Race 3 may be spread more easily with surface water when infected *S. dulcamara* grows with its roots floating in water. The bacterium may subsequently be spread to other hosts when contaminated surface water is used for irrigation (Olsson, 1976).

The main path for international spread is by (latently) infected seed potatoes and other vegetative propagating materials. Natural infection of true seed has only been firmly established for groundnut. There are a few reports of occurrence of race 1 in tomato, capsicum and aubergine seed (Persley, 1986b; Kelman *et al.*, 1994; Singh, 1995). Infections of potato tubers may be latent, due to unfavourable weather conditions, partly resistant cultivars or low virulence of certain pathogen strains; tubers with latent infection are the most probable means of introduction into a new area.

PEST SIGNIFICANCE

Economic impact

R. solanacearum constitutes a serious obstacle to the culture of many solanaceous plants in both tropical and temperate regions. The greatest economic damage has been reported on potatoes, tobacco and tomatoes in the south-eastern USA, Indonesia, Brazil, Colombia and South Africa. In the Philippines, in 1966-1968, there were average losses of 15% in tomatoes, 10% in aubergines and *Capsicum*, and 2-5% in tobacco (Zehr, 1969). In the Amazon basin in Peru, about half the banana plantations are affected and the rapid spread of the pathogen threatens to destroy plantations throughout the Peruvian jungle (French & Sequeira, 1968). In India, there are sometimes total losses in tomato crops. In the eradicated outbreak in Israel, losses occurred in potato, being heavier for the spring crop than the autumn crop, because of the high temperatures under which the former matures (Volcani & Palti, 1960). Extensive losses on potato were reported in Greece in 1951-1953 (Zachos, 1957).

Disease severity mostly increases if *R. solanacearum* is found in association with root nematodes. In tobacco, nematode infestation changes the physiology of the plants, causing susceptibility to bacterial wilt (Chen, 1984). Experiments in India showed that the

combined pathogenic effects of *R. solanacearum* and *Meloidogyne javanica* were greater than the independent effects of either (Sitaramaiah & Sinha, 1984).

Control

The control of bacterial wilt has proved to be very difficult, especially for race 1 with its broad host range. Chemical control is nearly impossible to apply. Soil fumigants showed either slight or no effects (Murakoshi & Takahashi, 1984). Antibiotics such as streptomycin, ampicillin, tetracycline and penicillin also showed hardly any effect (Farag *et al.*, 1982); in fact, streptomycin application increased the incidence of bacterial wilt in Egypt (Farag *et al.*, 1986). Biological control has been investigated, but is still in its infancy. Positive results were achieved with the antagonistic bacteria *Bacillus polymyxa* and *Pseudomonas fluorescens* which controlled bacterial blight on potato in laboratory experiments in the Philippines (Aspiras & Cruz, 1985). Experiments in Chile were successful using *P. fluorescens* to control *R. solanacearum* in potato in field and laboratory trials. Avirulent mutants of the bacterium have also been used in some studies (See Ciampi-Panno *et al.*, 1989; Gallardo & Panno, 1989; Hartman & Elphinstone, 1994).

Several resistant cultivars of potato, as well as other crops, are available, but the race and strain diversity of the pathogen make it difficult to utilize these in different countries. Potato cultivars developed in Colombia with a *Solanum phureja* and *S. demissum* background showed resistance to *R. solanacearum* in seven countries (French, 1985; Hartman & Elphistone, 1994).

Intercropping of potato with maize or *Phaseolus vulgaris* reduced inoculum density and disease development in some cases (Autrique & Potts, 1987), but microlesions were also found in these hosts, where *R. solanacearum* could persist (Granada & Sequeira, 1983). Cutting seed potato tubers should be avoided; cutting can increase disease incidence by 2.5 times and yield loss by >40% (Vijayakumar *et al.*, 1985). Crop rotation of 5-7 years without susceptible crops has been recommended. The disease may also be controlled by application of fertilizers to change soil pH. In the USA, the pathogen was eradicated by lowering the soil pH to 4-5 in summer and raising it to pH 6 in the autumn. The disease is serious on sandy, loam, clay and peat soils, but is never found in marl soils. Survival of race 3 was found to be 2-3 years in Australia under bare fallow or pasture. Host debris, latent infected tubers and deeper soil layers were found most important for survival (Graham & Lloyd, 1979; Graham *et al.*, 1979).

In relation to the current outbreak in the EPPO region, use of healthy (tested) seed potatoes, early and sure detection and reporting of the pathogen, quarantine measures on infected fields and farms, sufficient crop rotation, control of weed hosts and volunteer plants (and in some cases of nematodes), avoidance of surface water for irrigation, and education are key factors in control of race 3 of *R. solanacearum*.

Phytosanitary risk

R. solanacearum is an EPPO A2 quarantine organism (OEPP/EPPO, 1978) and also has quarantine significance for APPPC and IAPSC. The occurrence of different races and strains of the pathogen with varying virulence under different environmental conditions presents a serious danger to European and Mediterranean potato and tomato production. Absence of the bacterium is an important consideration for countries exporting seed potatoes. Hosts other than potato are most likely to be affected in the warmer parts of the EPPO region, where the bacterium already occurs. However, even in these areas, races other than race 3 have not been positively identified, and the introduction of some of the many strains not occurring in the region could have a great economic impact; for example, banana-infecting strains are not found in the banana-producing areas of the southern Mediterranean zone, and virtually have A1 quarantine status. Race 3 (biovar 2) appears to present the most important risk for the EPPO region as a whole. These is a definite risk that

it should spread through imports of (latently) infected early ware potatoes or seed potatoes from countries where the disease now occurs. Furthermore, introduction of *R. solanacearum* by use of (latently) infected potatoes 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 through contaminated surface water (the contamination originating, for example, from infected *S. dulcamara*), used for irrigation of potatoes or tomatoes.

PHYTOSANITARY MEASURES

Seed potato tubers, and other solanaceous plants for planting, should have been found free from *R. solanacearum* during the growing season and should come from a field which was found free from *R. solanacearum* during the last two growing seasons. Visual inspections should be performed routinely upon export and import. Laboratory checks for (latent) infections may be necessary. Plants for planting of *Musa* spp. should be kept in post-entry quarantine to ensure their freedom from dangerous strains of *R. solanacearum* (OEPP/EPPO, 1990b).

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