CHAPTER 1

RALSTONIA SOLANACEARUM: ASPECTS OF ITS ETIOLOGY, EPIDEMIOLOGY AND CONTROL

ABSTRACT

Bacterial wilt caused by *Ralstonia solanacearum* E.F. Smith, is a destructive seed and soil-borne disease affecting a range of economically important crops grown in temperate, subtropical and tropical climates. In South Africa the disease was first detected in 1914 on potato, thereafter several other crops such as tomato, pepper, brinjals, peanuts and tobacco have been affected. Its ability to infect a wide range of weed species enhances its survival during the non-cropping season. Bacterial wilt is difficult to control or eradicate and world-wide an integrated disease management approach is followed in reducing the severity of the disease. To develop effective control strategies it is essential to understand the organism and the disease it causes. This literature study summarises some of the findings regarding the etiology, epidemiology and control of bacterial wilt disease.

INTRODUCTION

More than a century ago Erwin F. Smith described a bacterial disease affecting potato, tomato and eggplant. The generic name for this solanaceous wilt was proposed as *Bacillus*. E.F. Smith probably did not realise that the disease he had described would become one of the most important plant diseases causing severe economic losses world-wide. Since his publication in 1896 (Smith, 1896 as reported by Kelman, 1953) a continuous stream of publications have been released internationally, each shedding some light on this unusually complex disease. Commonly known as bacterial wilt, other names include brown rot (used especially in Europe on potatoes), Granville wilt (on tobacco), southern bacterial blight (on tomato, eggplant and tobacco), and moko disease on bananas.

Its ability to affect over 450 plant species (Prior *et al.*, 1998), its world-wide distribution and its destructive ability has resulted in this disease to be ranked as the most important bacterial plant pathogen (Kelman, 1998). The economic impact can not only be measured in terms of crop losses, but must also be assessed indirectly in terms of soils becoming unsuitable for subsequent crop production. This is important to large commercial farms and to small family fields. The introduction of different strains to different geographic regions increases the risk of more crops becoming susceptible to wilt within a particular region.

Bacterial wilt disease is considered difficult to control. Knowledge of the organism and the disease it causes are prerequisites for integrated management strategies. This literature study summarises some of the findings regarding the etiology, epidemiology and control of bacterial wilt disease.

THE CAUSATIVE AGENT

The causative agent of bacterial wilt is a gram-negative, non-spore-forming, non-capsulate bacterium (Kelman, 1953). It is an aerobic organism with optimum growth temperatures ranging from 27-37 °C, depending on the strain. Maximum temperature for growth is about 39 °C and the minimum between 10-15 °C (Shekhawat *et al.*, 1992). There are conflicting reports on the amount of flagella present in a single cell. According to Bergey's Manual of Determinative Bacteriology (Holt *et al.*, 1994) this bacterium has more than one flagellum. Shekhawat *et al.* (1992) however describes virulent isolates to be non-flagellate and avirulent ones as having 1-4 polar flagella.

The shape and size of the causal organism was first described by E.F. Smith in 1896 as a small rod with rounded ends; its size varying according to growing conditions (Kelman, 1953). Bacteria isolated from infected tissues appeared as very short rods (0.3-0.6 x 0.4-1.2μ), those taken from young broths or cultures tend to be longer (ranging from 0.4-0.6 x 1.0-1.8μ), whereas those from old cultures have a short coccus-like form (Kelman, 1953).

Cultural characteristics of the colonies on tetrazolium chloride medium (Kelman, 1953) are often used to identify the bacterium. Virulent isolates produce fluidal, slightly raised colonies that are creamy white with or without pink centres. Colonies are rarely round (Shekhawat *et al.*, 1992). The pink centres often appear comma-like or half-moon in shape.

Different generic names have been used to classify the causal agent of bacterial wilt. Smith first proposed the name *Bacillus,* as he believed the bacterium to have peritrichous flagella. In 1898, Chester subsequently changed it to *Bacterium*; a name later also adopted by Smith (Kelman, 1953). With the finding that the pathogen actually had a single polar flagellum, it was placed under the classification as either *Bacterium solanacearum* or *Pseudomonas solanacearum.* Bergey (1923) reclassified the bacterium as *Phytomonas* although most continued to use the name *Bacterium.*

Studies and revision of the classification of gram-negative plant pathogens led to the provisional transferral of *P. solanacearum* to the new genus *Xanthomonas*. Since distinct cultural characteristics differed between *P. solanacearum* and the genus *Xanthomonas*, the pathogen was once again transferred back to the genus *Pseudomonas*. This agreed with the classification that was adopted in Bergey's Manual in 1948 (Kelman, 1953).

The classification of *P. solanacearum* was adopted for the next 44 years. Studies conducted by Yabuuchi *et al.* (1992) led to the proposal that *P. solanacearum* be transferred to the new genus *Burkholderia*. The seven species that were placed into this new genus differed from the type species of *Pseudomonas* in their oxidation and assimilation capabilities of several polyalcohols and disaccharides. Yabuuchi *et al.* (1992) also disagreed with the description of *P. solanacearum* given in Bergey's Manual (Holt *et al.*, 1994) in that the type strain they identified was non-motile and without any flagellum.

Yabuuchi *et al.* (1995) reclassified *Burkholderia solanacearum* as *Ralstonia solanacearum*. This reclassification was based on studies involving phenotypic characterisation, rRNA-DNA hybridisation, phylogenic analysis of 16SrDNA nucleotide sequences, and analysis of cellular lipids and fatty acids.

SUBSPECIFIC CLASSIFICATION

Several attempts have been made to find a suitable classification system for isolates of *R. solanacearum* as they often differ in host range, geographical distribution, pathogenicity, epidemiology and physiological properties. For almost thirty years two major approaches to differentiation were used, one based on hosts primarily affected resulting in the establishment of races, the other on selected biochemical properties conforming to distinct biovars. These classification systems do not abide by the Code of Nomenclature of Bacteria (Hayward, 1991a).

Classification according to race

Subspecific classification of strains into 5 races is achieved by determining the hosts that are primarily affected.

Race 1: Strains affecting tobacco, tomato, a range of other solanaceous crops, some weeds and certain diploid but not triploid bananas.

Race 2: Those affecting triploid bananas, *Heliconia* spp. and other musaceous hosts.

Race 3: Strains affecting primarily potato and to a lesser extent tomato. It is weakly pathogenic on other solanaceous crops.

Race 4: Strains affecting mainly ginger.

Race 5: This race affects mainly mulberry.

Classification according to biovar

According to Hayward (1994 b), five biovars can be identified based on ability to utilise three hexose alcohols, namely mannitol, sorbitol, dulcitol; and to produce acids from the three disaccharides, lactose, maltose and cellobiose (Table 1.1).

Table 1.1 Identification of biovars of *R. solanacearum* according to utilisation and/or oxidisation of certain carbohydrates (Hayward, 1994 b)

Biovar/Carbohydrate	Biovar 1	Biovar 2	Biovar 3	Biovar 4	Biovar 5
Lactose	-	+	+	-	+
Maltose	-	+	+	-	+
Cellobiose	-	+	+	-	+
Mannitol	-	-	+	+	+
Sorbitol	-	-	+	+	-
Dulcitol	-	-	+	+	-

The distinction between race and biovar is not always clear except in the case of race 3 and biovar 2. Generally it can be said that race 3 (potato race) is equivalent to biovar 2, although the reverse is not necessarily true. In other words not all biovar 2 strains belong to race 3 (Sequeira, 1993). Biovar 2 strains that have been isolated in the Andean highlands seem to correspond to those of race 3 (Amat *et al.*, 1978). Biovar 2 strains from the Amazon basin, however, differ in phenotypic properties regarding pathogenicity on various *Solanum* species and metabolic activities. These have been designated as biovar N2 (Gillings & Fahy, 1994). Since biovar 2 isolates were found on potato or tomato, the correspondence of race 3 and biovar 2 seems true. Biovar N2, however, is probably not equivalent to race 3, as it was isolated from other *Solanum* species such as the nightshades. Biovar N2 also does not occur in the region where potatoes originated. Biovar 2 has a more limited host range than biovar 3, and is known to contain some strains that are adapted to pathogenesis at lower temperatures. It appears that its ability to survive in fallow soil is less than for biovar 3. These generalisations about the epidemiology and control of biovar 2 (race 3) set it apart from other races and biovars (Hayward, 1991a). Strains of race 1 affecting potatoes can consist of biovars 1, 3 and 4 (He *et al.*, 1987).

Within each of these races or biovars there are numerous subtypes that can be associated with certain geographical regions (He, 1983). This, together with the fact that *R. solanacearum* enjoys a world-wide distribution and that it is associated with indigenous plants in virgin soil supports the belief that this disease has been present in tropical soil for aeons (Sequeira, 1993).

More recent attempts to classify *R. solanacearum* strains involve molecular methods such as restriction length polymorphism (RFLP) analysis. Two major divisions could be identified, division 1 consisting of race 1, biovars 3, 4 and 5; and division 2 of biovar 1 (race 1 and 2) and biovar 2 (race 3) (Cook & Sequeira, 1994). These authors recognise only three traditional races. Fegan *et al.*, (1998) does not specifically mention the separation of the races into the two divisions but, describes division 2 as consisting of biovars 1, 2 and N2.

DISEASE DEVELOPMENT AND SYMPTOMOLOGY

Mode of entry

Ralstonia solanacearum usually enters its hosts via wounds in the root system (Johnson & Schaal, 1952). Cultural practices such as interplanting prior to harvest often lead to increased root damage (Kelman, 1953). The presence of root-invading parasitic fungi such as *Phytophthora* in the soil is believed to be another factor that may influence infection, although contradicting observations have been made in this regard (Kelman, 1953). The role of nematodes, especially the root-knot nematode (*Meloidogyne* spp.), in providing a wound for bacterial entry has been mentioned by several authors (Kelman, 1953; Buddenhagen & Kelman, 1964; Hayward, 1991a; Shekhawat *et al.*, 1992). Nematodes may also modify the host tissue making it more suitable for bacterial colonisation (Hayward, 1991a). Wilt resistant cultivars have been noted to become susceptible when attacked by nematodes. It is therefore not surprising that attempts were made to combine resistance to three of the *Meloidogyne* species and to *R. solanacearum* in potato plants (Gomez *et al.*, 1983).

Root decay caused by unfavourable soil conditions is believed to provide further entrance sites for the pathogen. Invasion through insect wounds has been noted on peanut roots and on potato tubers. Even infection of aerial parts via wounds has been reported under field conditions (Kelman, 1953).

Buddenhagen & Kelman (1964) reported transmission by several genera of insects visiting inflorescences of banana plants. Bacterial invasion does not occur directly through the flower, but occurs when open xylem vessels are exposed during natural dehiscence of bracts and male flowers.

Although wounding of some kind were usually regarded as a prerequisite for infection, documentation exists where this is not the case (Kelman & Sequeira, 1965). It is postulated that the bacterium could enter the host through points of secondary root emergence (Kelman, 1953; Buddenhagen & Kelman, 1964; Kelman & Sequeira, 1965). Kelman & Sequeira (1965) observed that relatively large numbers of bacteria are needed to infect unwounded roots. They suggest that massing of bacterial cells at points of secondary root emergence is required for enzymatic digestion of the mucilaginous sheath or other barriers.

Histopathology of infected plants

The detailed study conducted by Wallis & Truter (1978) on the histopathology of tomato plants infected with *Ralstonia solanacearum*, shed considerable light on the spread of the pathogen within the host and the progressive destruction of its vascular tissue. Bacterial wilt was generally thought to be a vascular parasite confined initially to xylem vessels (Kelman, 1953; Buddenhagen & Kelman, 1964; Husain & Kelman, 1958a; Husain & Kelman, 1958b; Pegg & Sequeira, 1968). The Wallis & Truter (1978) study revealed, however, that initial colonisation of host tissue did not occur in the xylem vessels of the roots as expected, but in small diameter cells adjacent to large xylem vessels. Light microscopic examination could not identify whether these cells are tracheids, tracheid fibres or xylem parenchyma.

Wallis & Truter (1978) observed that within 24 hours after inoculation additional small diameter cells had become invaded and filled with bacteria. The bacteria within these cells showed marked pleomorphism and appeared to contain granules of storage products such as poly-B-hydroxybutric acid or volutin, indicating metabolic activity. In some cases, the bacteria were in close contact with the host cell wall and seemed to orientate themselves toward the spaces between the bars of secondary thickening. In other cases they were contained in a bag-like structure, which effectively prevented them from affecting the primary wall. According to Wallis & Truter (1978) this concentration and orientation of bacteria towards bordered pits between vessels and adjacent cells during this stage of pathogenesis indicates that bacteria are either attracted by substances diffusing into the vessel from adjacent cells, or that they are drawn along an osmotic pressure gradient. This initial spread may be influenced by the method and the site of inoculation and may also indicate that at any particular stage of development of the host plant only certain cells are physiologically predisposed to invasion.

Stimulation of tyloses formation was noted in invaded cells and less frequently in non-invaded cells about 24 hours after inoculation, an observation absent in healthy plants. This indicates that although infection stimulates the production of tyloses, the actual presence of bacteria is not required. Kelman (1953) reported similar findings. The formation of tyloses is believed to be stimulated by increased production of indole acetic acid (IAA) and other growth substances (Sequeira & Kelman, 1962; Buddenhagen & Kelman, 1964; Sequeira, 1965; Pegg & Sequeira, 1968;).

Wallis & Truter (1978) observed a slow migration of bacteria during the first 48 hours after inoculation and no bacteria could be detected at a distance greater than 3,5 cm from the cut root-tip. No bacteria could be observed in the xylem vessels. During the next 24 hrs, however, disruption and collapse of tyloses had occurred, releasing the bacteria into the xylem vessels. Bacteria spread in root vessels above the region of tylose collapse. These bacteria increased steadily and in some cases the primary wall showed signs of degradation. Only at this stage of disease development were bacteria observed in the vessels of stems. During the first 72 hours after inoculation water uptake in inoculated plants had been 15 - 20% higher than in the control plants. Thereafter, however, the inoculated plants started to wilt and fluid uptake decreased relative to that in control plants. This observation correlates with the time when tyloses, after often obstructing vessels, collapsed, became disrupted and released bacteria into the xylem vessels (Wallis & Truter, 1978). After 144 hours the bacteria in the root vessel had reached such large numbers that they became compressed into irregular shapes. Longitudinal spread in the stem was now rapid but compression of bacterial cells did not occur to such an extent as in the root vessels, nor did bacteria reach such large numbers. Tissue collapse was observed after 168 hours and various plugging substances were noted in the vessels and cells of diseased plants. Complete wilting of all test plants occurred about 192 hours after inoculation. A dense, darkly staining material, possibly of cell wall origin, accumulated in many vessels. This material was found where vessel walls had been dissolved by bacterial enzyme action and in lysigenous cavities formed by bacterial degradation of adjacent parenchyma cell walls (Wallis & Truter, 1978). It has also been reported that in some instances these parenchyma cells seem to enlarge and divide, causing partial collapse of infected vessels (Kelman, 1953).

According to Wallis & Truter (1978), the lack of damage to the walls of invaded root cells during the early stages of pathogenesis could possibly be ascribed to low levels of cellulolytic and pectinolytic enzyme activities as bacterial numbers are still relatively low. As bacterial numbers increased, degradation of wall material in the vessels was clearly visible. Kelman (1953) mentions that in solanaceous hosts, besides collapse of infected vessels, adjacent phloem areas can become infected and the cortex disorganized. The formation of a central cavity often results due to a breakdown of pith tissue. This general breakdown of walls and cells is, however, not found in stems of older plants that have well-developed secondary xylem.

Visual symptom expression

Once the pathogen has entered the host, time elapses before visual symptoms appear. This incubation period varies greatly and depends on a variety of factors such as host species, environmental conditions, age of the host and level of resistance. The symptoms characteristic of bacterial wilt include wilting, stunting and yellowing of the foliage, epinasty and vascular browning in the stems (Kelman, 1953; Buddenhagen & Kelman, 1964; Shekhawat *et al.*, 1992). It has also been noted that climatic conditions can affect the type of visual symptoms expressed. In hot dry weather, infected plants may show irregular scalded areas on their leaves, which then dry out and shatter at the edges (Kelman, 1953). Under moist conditions, however, the base of the stem, or leaf petioles may completely rot and break off (Kelman, 1953; Shekhawat *et al.*, 1992). In potatoes a slight yellowing of lower leaves is often noted as the first leaves begin to droop (Kelman, 1953). The extent to which yellowing occurs often depends on whether the onset of disease is rapid, in which case no real change in colour would occur.

Adventitious root formation on infected stems commonly occurs, especially if wilting is gradual (Kelman, 1953) or if an isolate of low virulence was responsible.

Potato tubers from infected plants do not always show external symptoms. The presence of external greyish brown discoloration indicates an advanced stage of disease, which upon further development leads to a bacterial exudate at the eyes or the stolen end of the tuber. Cross sections of infected tubers often show distinct brown discoloration and decay in the vascular ring (Shekhawat *et al.*, 1992).

DISTRIBUTION

Bacterial wilt affects crops of economic importance in almost all the tropical, subtropical and warmer temperate regions of the world. Early reports of occurrence have often been incorrect, incomplete or not confirmed by proper identification techniques, making it difficult to truly assess its distribution.

The distinct differences in geographical distributions of biovars suggest a separate evolutionary origin (Hayward, 1991a). Biovar 2 presumed to have originated in South America (presumed site of origin of the potato) now has a wide spread distribution, indicating the ease with which it can be transmitted as latent infections in potato seed tubers. In many countries of Southern Europe such as Portugal, biovar 2 is the sole biovar. This is also true for the Mediterranean area, Argentina, Chile and Uruguay (Hayward, 1991a).

Biovars 1 and 2 are predominant in the Americas with biovar 3 being rare and biovar 4 not yet being identified with the exception of one case stated by Black & Sweetmore (1993). In Australia, however, biovar 3 predominates, biovars 2 and 4 occurring to a lesser extent. Biovars 2,3 and 4 also occur in India, Indonesia, Papua New Guinea, Sri Lanka and China (together with biovar 5). Only in the Philippines have all of biovars 1-4 been found and here as elsewhere in Asia, biovar 3 predominates in the lowland regions (Hayward, 1991a).

The classification into divisions as suggested by Cook & Sequeira (1994) corresponds, with a few exceptions, to the geographic distribution of the strains. In Division I 90% of the strains came from Asia and Australia, whereas 98% of those in Division 2 were from the Americas. This suggests that in early evolution *R. solanacearum* split into two groups which then evolved in geographical isolation to give rise to the strains typical of the Old World and the New World. All race 3 strains isolated in Africa, Asia and Australia belonged to the same RFLP groups which originated in the Andean region in South America.
With the aid of RFLP techniques more data could be obtained to support the concept of geographic isolation in the pathogens evolution (Sequeira, 1993). Strains from *Heliconia* are in RFLP groups that are restricted to the Americas, whereas strains from mulberry and ginger form distinct groups that are again restricted to certain regions in Asia.

Race 3 (potato race), although being widely distributed, consists of a very compact group originating in the Andean Region. Yet strains attacking bananas in the Asian continent seem to have evolved separately from those attacking bananas in the Americas (Sequeira, 1993). *R. solanacearum* has now been identified to be the cause of an old banana disease common in Asia, the so-called blood disease. RFLP patterns of these strains are so different from all other strains, that it was concluded that this group bears very little relationship to the American race 2 strains.

So far only biovars 2 and 3 have been reported in South Africa with biovar 2 having a major impact on the potato industry. Since neighbouring countries such as Angola, Mozambique and Zimbabwe are reported to have biovar 1 of *R. solanacearum*

(De Lourdes D'Oliveira, 1967) great care needs to be taken to prevent establishment of this biovar locally.

In Table 1.2 the world-wide distribution of bacterial wilt is demonstrated with mention of the specific biovars and/ or races involved. In several instances literature sources only reveal the occurrence of *R. solanacearum* without further differentiation into biovars or races. This list may be incomplete and in some mentioned countries, such as Spain, Poland and Sweden, the disease is believed to be eradicated (Elphinstone, 1996).

Table 1.2 Distribution of bacterial wilt disease

COUNTRIES	BIOVARS	RACES	SOURCE
AMERICAS			
Argentina	2	3	Martin *et al.*, 1982
Brazil	1,2,3	1,2,3	French *et al.* 1993
Bolivia	1,2		Smith *et al.* 1998
British Honduras (Belize)	1		Black & Sweetmore, 1993
Canada	1	1	Hayward, 1991a
Chile	2		Hayward, 1991 a Ciampi-Panno, 1984
Colombia	1,2	1,3	Martin *et al.*, 1982
Costa Rica	1,2,3	1,3	Martin *et al.*, 1982
El Salvador			Kelman, 1953
Honduras		2	Woods, 1984
Mexico	2 1 1	3 1 2	Fucikovsky, 1978 Fucikovsky, 1978 Fucikovsky & Santos, 1993; Fucikovsky, 1978
Panama	2	3	Martin *et al.*, 1982
Peru	1,2,3	1,3	Martin *et al.*, 1981
United States - 1 literature source	1 4	1 1	Martin *et al.*, 1982 Black & Sweetmore, 1993
Uruguay	2	3	Hayward, 1991 a
Venezuela	2	3	Martin *et al.*, 1982
WEST INDIES			
Cuba	3		Arnat *et al.*, 1978
French West Indies	1,2,3	2,3	Prior & Steva, 1990
Grenada	1		Black & Sweetmore, 1993

COUNTRIES	BIOVARS	RACES	SOURCE
Guadeloupe			Prior *et al.*, 1993
Haiti			Kelman, 1953
Jamaica			Kelman, 1953
Martinique	2,3	1,3	
Puerto Rica	1	1	Cook & Sequeira, 1994
Tobago			Hosein & Phelps, 1997
Trinidad	1	1	Cook & Sequeira, 1994
EUROPE			
Albania			Wenneker et al., 1998
Austria			Kelman, 1953
Belgium	2	3.	Elphinstone, 1996
Denmark			Wenneker *et al., 1998*
France	2	3.	Elphinstone, 1996, Hayward *et al.*, 1998
Greece	2	3	Walker, 1992
Netherlands	2	3	Janse *et al.*, 1998
Italy	2	3	Elphinstone, 1996
Portugal	2	3	De Lourdes D'Oliveira, 1967; Elphinstone, 1996
Rumania			Kelman, 1953
Spain	2	3	Elphinstone, 1996
Sweden			Olsson, 1976
United Kingdom	2	3	Elphinstone, 1996
U.S.S.R			Walker, 1992
Yugoslavia			Walker, 1992
ASIA			
China	2,3,4,5	1,3,4,5	He, 1983
India	2,3,4	1,2,3	Shekhawat *et al.*, 1992; Sinha, 1986
Indonesia	2,3,4	1,3	Hayward, 1991; Machmud, 1986
Iran	2	3	Danesh & Bahar, 1984
Israel		3	Alvarez *et al.*, 1993
Japan	1,2,3,4	1,3	Tsuchiya & Horita, 1998
Java	2,3,4	1,2,3	Cook & Sequeira, 1994
Malaysia	3,4	.	Abdullah, 1993
Nepal	2,3	1,3	Adhikari, 1993
Pakistan	2,3		Burney & Ahmad, 1997
Philippines	1,2,3,4		Valdez, 1986
Sri Lanka	2,3,4	1,3	Velupillai, 1986; Martin

COUNTRIES	BIOVARS	RACES	SOURCE
			& Nydegger, 1982
Taiwan	3,2,4	1	Hsu, 1991
Thailand	3,4		Titatarn, 1986
Vietnam		1	Hong & Mehan, 1993
AFRICA			
Algeria			Kelman, 1953
Angola	1,2	3	De Lourdes D'Oliveira, 1967
Burundi	2	3	Berrios & Rubirigi, 1993
Egypt	2	3	Gillings & Fahy, 1994
Ethiopia			Kelman, 1953
Kenya	2,3,4	1,3	Harris, D.C. 1976
Malawi			Black *et al.* 1998
Morocco			Kelman, 1953
Mozambique	1		De Lourdes D'Oliveira, 1967
Nigeria	2	3	Cook & Sequeira, 1994
Rwanda	2	3	Vander Zaag, 1986
Somalia			Kelman, 1953
South Africa	2,3	1,3	Swanepoel, 1992
Tanzania			Black *et al.* 1998
Uganda			Tusiime *et al.*, 1998
Zaire		3	French *et al.*, 1998
Zimbabwe	1,2		Robertson, 1998
AUSTRALIA	2,3,4	1,3	Hayward, 1991b
INDIAN OCEAN			
Andaman & Nicobar Islands			Ramesh & Bandyopadhyay, 1993
Madagascar	1	1	Lallmahomed *et al.*, 1988
Mauritius	3	1	Saumtally *et al.*, 1993
Reunion	1,2,3	1,3	Girard *et al.*, 1993
PACIFIC OCEAN			
Fiji			Iqbal & Kumar, 1986
Hawaii	4	1,2	Alvarez *et al.*, 1993; Hayward, 1986
New Zealand			Kelman, 1953
Papua New Guinea	2,3,4	1,2	Tomlinson, 1985; Tomlinson & Gunther, 1986; He, 1986
ATLANTIC OCEAN			
Madeira Island	2	3	De Lourdes D'Oliveira, 1967

HOST RANGE

R. solanacearum is known to have a very extensive host range including not only economically important crop plants such as potatoes, tomatoes, tobacco and bananas, but also ornamental plants, trees and weeds. Species from more than 44 plant families have been identified by Hayward (1991a) and more hosts are being recognised and described. Some of the more recent reports include onion, *Allium cepa*, (Girard *et al.*, 1993); custard apple, *Annona* spp., (Mayers & Hutton, 1987); florist geranium, *Pelagornium hortorum*, (Strider *et al.*, 1981); strawberry, *Fragaria* L. spp., (Hsu, 1991) and radish, *Raphanus sativus* L., (Hsu, 1991). *R. solanacearum*, biovar 3, has recently also been noted on cashew in Indonesia and the Alexandra palm in Queensland, Australia (Hayward, 1991a).

There appears to be some irregularity in the distribution of bacterial wilt in certain hosts. Cassava is cultivated in many countries were bacterial wilt is endemic, yet the disease on this host appears to be confined to Indonesia. Similarly bacterial wilt on sweet potato has only been reported in China (Hayward, 1991a). Eucalyptus was first reported as a host in Brazil and China. Eucalyptus in Australia appeared to be a non-host until recently, when biovar 3 was isolated from diseased plants (Hayward, 1994b).

Specific strains pathogenic for certain hosts may have evolved only in certain parts of the world and are not found elsewhere. This theory is supported by recent RFLP studies. An alternative theory is that these hosts may only be susceptible where a number of environmental factors conducive to disease expression coincide, such as temperature regime, rainfall, soil type, inoculum potential, and other biological factors such as nematode populations (Hayward, 1991a; Hayward, 1994b).

Hosts of *R. solanacearum* do not necessarily express symptoms but can serve as symptomless carriers. This is especially true for many of the weed hosts such as common purslane (Hayward, 1991a), single leaved cleome (Shekhawat *et al.*, 1992) and the apple of Peru (Olsson, 1976). The slow rate of colonisation and disease progress in symptomless hosts allows the bacteria to stay viable longer, serving as an inoculum source for susceptible crops or wild hosts.

Studies conducted by Shekhawat *et al.* (1992) indicate that *R. solanacearum* can even survive symptomless in roots of weed-hosts and in plants considered to be non-hosts. Granada & Sequeira (1983a) have reported similar findings. Roots of bean and maize, both presumed non-hosts were invaded with bacteria. Infection was however localised and not all plants became infected.

More than 450 species have been reported as hosts or symptomless carriers (Prior *et al.*, 1998) of certain strains of *R. solanacearum*. In Table 1.3 host plants are listed with reference to the country of report and where possible the strain involved. The host range of individual strains of *R. solanacearum* differs considerably with race 1 (the solanaceous race) having the widest range. This race is more common in the sub-tropical and tropical climates where plant debris tends to decompose more rapidly thereby providing only temporary shelter to the bacteria. Alternative hosts might therefore play an important role in the pathogen's survival. Only about 30-40 species have been positively identified as hosts of biovar 2 (race 3).

Table 1.3 Known hosts or symptomless carriers of *Ralstonia solanacearum*

Weed Hosts					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
Acanthospermum hispidum DC	Upright starbur			India	Shekhawat *et al.*, 1992
Achyranthes aspera Cooke	Rough Chaff flower			India	Shekhawat *et al.*, 1992
Aclypha boehmerioides Miq.					Kelman, 1953
Aclypha hispida				India	Shekhawat *et al.*, 1992
Aclypha indica				India	Shekhawat *et al.*, 1992
Ageratum conyzoides	White weed	3		Indonesia Thailand India Andaman & Nicobar Islands Malaysia Uganda	Machmud, 1986 Titatarn, 1986 Shekhawat *et al.*, 1992 Ramesh & Bandyopadhyay, 1993 Abdullah, 1993 Akiew *et al.*, 1993
Ageratum houstonianum Mill.	Blue billy-goat weed			Australia	Akiew *et al.*, 1993
Amaranthus graecizans L.	Kaffir spinach			India	Shekhawat *et al.*, 1992
Amaranthus hybridus L.	Cape pig weed	3	1	R.S.A	Swanepoel, 1992
Amaranthus sp.	Bayam			Malaysia Andaman & Nicobar Islands Uganda	Abdullah, 1993 Ramesh & Bandyopadhyay, 1993 Tusiime *et al.*, 1998
Ambrosia artemisiifolia L.	Common ragweed				Kelman, 1953
Ambrosia trifida L.	Giant ragweed				Kelman, 1953
Anthirrhinum sp.				India	Kishore *et al.*, 1993
Artemissia sp.				India	Kishore *et al.*, 1993
Arabidopsis thaliana				India	Kishore *et al.*, 1993

Weed Hosts					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
Aritica dioca				India	Kishore *et al.*, 1993
Asclepias curassarica	Blood-flower		2	Honduras Australia	Berg, 1971 Akiew *et al.*, 1993
Atropa belladonna	Belladonna				Kelman, 1953
Bidens bipinnata L.	Spanish blackjack	3		RSA India	Swanepoel, 1992 Shekhawat *et al.*, 1992
Bidens pilosa L.	Common blackjack (Cobblers peg)	3 3	 1	Thailand India Australia Brazil Uganda	Titatarn, 1986 Shekhawat *et al.*, 1992 Akiew & Trevorrow1994 Mariano, 1998 Tusiime *et al.*, 1998
Brachiaria plantaginea				Brazil	Mariano, 1998
Calendula officinalis L.	Calendula			India	Shekhawat *et al.*, 1992
Calopogonium mucunoides Desv.	Calopo			Australia	Akiew *et al.*, 1993
Cannabis savita L. (s)	Indian Hemp			India	Shekhawat *et al.*, 1992
Capsella bursa-pastoris		2	3	Netherlands	Van Beuningen *et al*, 1998
Cardamine spp.		2	3	Netherlands	Van Beuningen *et al*, 1998
Cassia mimosoides L.	Five leaf cassia			Australia	Akiew *et al.*, 1993
Cecropia peltata			2	Honduras	Berg, 1971
Celosia argentea L.	Cockscomb			India	Shekhawat *et al.*, 1992
Cerastium fontanum		2	3	Netherlands	Van Beuningen *et al*, 1998
Cerastium glomeratum		2		Nepals	Pradhanang, 1999
Chenapodium album		2	3	Netherlands	Van Beuningen *et al*, 1998
Chenopodium ambrosoides L.	Wormseed goosefoot			India	Shekhawat *et al.*, 1992
Chenopodium murales L.	Nettle-leaved goosefoot			India	Shekhawat *et al.*, 1992
Cichorium endivia				Brazil	Mariano, 1998
Citrullua lanatus	Paddy melon			Australia	Akiew *et al.*, 1993
Cleome monophylla L.	Single leaved cleome	 2	1,3 1,3	Kenya India	Harris, 1976 Shekhawat *et al.*, 1992
Cleome speciosissima	Cat's whisker			Malaysia	Abdullah, 1993
Commelina benghalensis L.	Common signal grass			India Brazil	Shekhawat *et al.*, 1992 Mariano, 1998
Corchorus acutangulus L.	Native jute			Australia	Akiew *et al.*, 1993
Corchus olitarius	Corchorus			Malaysia	Abdullah, 1993
Cosmos caudatus	Ulam Raja			Malaysia	Abdullah, 1993
Cosmos sp.				Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993
Cosmos sulphureus			1	Andaman	Ansari, 1990
Crassocephalum crepidioides		3		Thailand Indonesia Australia, Java	Titatarn, 1986 Machmud, 1987 Hayward, 1994
Croton hirtus	Croton	3		Malaysia Indonesia Sri Lanka	Hayward, 1986 Machmud, 1986 Velupillai, 1986
Croton sperciflorus				India	Shekhawat *et al.*, 1992
Cyperus rotundus L.	Red nutgrass	3		India	Shekhawat *et al.*, 1992
Datura ferox	Large thorn apple	2,3	1,3	RSA	Swanepoel & Young, 1988
Datura metel				India	Schmiediche, 1986
Datura stramonium L.	Common thorn apple	 2,3 1,2,3 2	1 3	Kenya R.S.A India Peru Sweden Chile	Harris, 1976 Swanepoel, 1992 Kishore *et al.*, 1993 Marín & El-Nashaar, 1993 Olsson, 1976 Fernandez, 1986
Datura spp.		4		Kenya	Martin & Nydegger, 1982
Drymaria cordata		2		Nepal	Pradhanang, 1999
Dysophylla auricularia (L.) Blume					Kelman, 1953
Eclipta alba (L.)	White eclipta	3		India Australia	Shekhawat *et al.*, 1992 Akiew *et al.*, 1993

Weed Hosts					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
Eclipta prostrata		3		Thailand	Titatarn, 1986
Eleutheranthera ruderalis (Schw.)Sch.Bip					Kelman, 1953
Eragrostis curvula	Weeping love grass	3		R.S.A	Swanepoel, 1992
Erechtites hieracifolia Rafin.				Malaysia Brazil	Abdullah, 1993 Mariano, 1998
Erigeron cannadensis	Horseweed				Kelman, 1953
Erigeron floribundis (Conyza sumatrensis, C. albida)	Tall fleabane			Uganda	Tusiime *et al*., 1998
Erigeron hirta L.	Asthma plant			Australia	Akiew *et al*., 1993
Eupatorium cannabinum L.				Sweden India	Olsson, 1976 Shekhawat *et al*., 1992
Eupatorium oderatum		3 3		Thailand Costa Rica, Andaman & Nicobar Islands	Titatarn, 1986 Black & Sweetmore, 1993 Ramesh & Bandyopadhyay, 1993
Euphorbia geniculata L.	painted Euphorbia			India	Shekhawat *et al*., 1992
Euphorbia hirta L.	red Euphorbia			India Malaysia	Shekhawat *et al*., 1992 Abdullah, 1993
Euphorbia maculata L.				India	Shekhawat *et al*., 1992
Euphorbia prunifolia	Euphorbia	3		Malaysia	Hayward, 1986
Fagopyrum sagittatum				Indonesia	Machmud, 1986
Galinsoga parviflora	Small flowered quick weed			Uganda Brazil	Tusiime *et al*., 1998 Mariano, 1998
Galium aparine				India	Kishore *et al*., 1993
Galphimia gracilis L.				Malaysia	Abdullah, 1993
Gomphrena sp.				India	Shekhawat *et al*., 1992
Heliconia acuminata			2	Costa Rica	Sequeira & Averre, 1961
Heliconia caribaea			2	Costa Rica	Sequeira & Averre, 1961
Heliconia imbricata			2	Costa Rica	Sequeira & Averre, 1961
Heliconia latispatha			2	Costa Rica	Sequeira & Averre, 1961
Hibiscus cannabinus Linn.				Malaysia	Abdullah, 1993
Hyptis capitata Jacq.				Malaysia	Abdullah, 1993
Hyptis suavealens		1	1	Brazil	Quezando-Soares & Lopes, 1994
Hyptis suaveolens		 3		Sri Lanka Thailand Malaysia	Velupillai, 1986 Titatarn, 1986 Abdullah, 1993
Ipomea setosa L.				Malaysia	Abdullah, 1993
Ipomoea sp.				Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993
Jussiaea linifolia		3		Thailand Andaman & Nicobar Islands	Titatarn, 1986 Ramesh & Bandyopadhyay, 1993
Lablab purpureus L.	Lablab			Australia	Akiew *et al*., 1993
Lagosca mollis				India	Shekhawat *et al*., 1992
Lathyrus sp.				India	Kishore *et al*., 1993
Leonurus sibiricus L.				Malaysia	Abdullah, 1993
Leucas martinicensis				Uganda	Tusiime *et al*., 1998
Luzula campestris		2	3	Netherlands	Van Beuningen *et al*, 1998
Marsypianthes chamaedrys		1	1	Brazil	Mariano *et al*., 1998
Melampodium perfoliatum		1	1	Costa Rica,	Black & Sweetmore, 1993
Merremia hastata (Desr.) Hall					Kelman, 1953
Merremia umbellata (Mey.) Hall					Kelman, 1953
Merremia vitifolia (L.) Hall					Kelman, 1953
Milleria quinqueflora L.		3		Cuba	Arnat *et al*., 1978
Nepata sp.				India	Kishore *et al*., 1993
Nicandra physaloides (L.)	Apple of Peru		2	Sweden India	Olsson, 1976 Shekhawat *et al*., 1992
Nicotiana alata Link & Otto				Sweden	Kelman, 1953
Nicotiana glauca	Wild tobacco				Kelman, 1953
Nicotiana glutinosa		2	3	Peru Colombia	Martin & French, 1995 Thurston, 1963
Nicotinana rustica	Aztec tobacco	2	3	Peru Colombia	Martin & French, 1995 Thurston, 1963
Oenothera rosea				India	Kishore *et al*., 1993
Oxalis latifolia	Red garden sorrel			Uganda	Tusiime *et al*., 1998

Weed Hosts					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
Oxalis sp.				India	Kishore *et al.*, 1993
Oldenlandia corymbosa L.				India	Shekhawat *et al.*, 1992
Parthenium hysterophorus L.				Cuba	Stefanova, 1998
Physalis angulata L.	Wild gooseberry	3 2		Costa Rica R.S.A	Black & Sweetmore, 1993 Swanepoel, 1992
Physalis floridana		2	3	Chile	Fernandez, 1986
Physalis minima L.	Bladder cherry			Malaysia	Abdullah, 1993
Physalis niruri			1	India	Shekhawat *et al.*, 1992
Physalis peruviana				Colombia	Thurston, 1963
Physalis spp.	Wild gooseberry	3	1	Australia	Akiew & Trevorrow, 1994
Piper auritum			2	Honduras	Berg, 1971
Piper peltatum			2	Honduras	Berg, 1971
Polanisia viscosa (L.) DC					Kelman, 1953
Polygala				India	Kishore *et al.*, 1993
Polygonum hydropiper L.				India	Shekhawat *et al.*, 1992
Polygonum napalense				Uganda	Tusiime *et al.*, 1998
Polygonum sp.				India	Kishore *et al.*, 1993
Portulaca oleracea L.	Common purslane	 3 	1,3 	Kenya Thailand India Brazil	Harris, 1976 Titatarn, 1986 Shekhawat *et al.*, 1992 Mariano, 1998
Portulaca volarasia				Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993
Pultenaea villosa		3		Australia	Li & Hayward, 1993
Ranunculus sceleratus L.				India	Shekhawat *et al.*, 1992
Ricinus communis L.	Casteroil plant			R.S.A Phillipines India Malaysia Brazil	Swanepoel, 1992 Persley *et al.*, 1986 Shekhawat *et al.*, 1992 Abdullah, 1993 Mariano, 1998
Rumex abyssinicum				Uganda	Tusiime *et al.*, 1998
Rumex sp.		 2	 3	India Neyherlands	Shekhawat *et al.*, 1992 Van Beuningen *et al.*, 1998
Salpiglossis sinuata R & P				Sweden	Olsson, 1976
Salvia privoides Benth.					Kelman, 1953
Scoparia dulcis L.	Goutweed				Kelman, 1953
Scuttellaria scandens				India	Kishore *et al.*, 1993
Senecio sonchifolia Moench.					Kelman, 1953
Sesbania exaltata				Indonesia	Machmud, 1986
Solanum nigrum L.	Black nightshade	2 3 2 3 2 1,2,3 2	3 1,3 2 3 3	Australia Kenya South Africa Honduras Papua New Guinea Thailand India Réunion Andaman & Nicobar Islands France Sweden Peru Peru Netherlands	Graham & Lloyd, 1979 Harris, 1976 Swanepoel, 1992 Berg, 1971 Tomlinson, 1985 Titatarn, 1986 Shekhawat *et al.*, 1992 Girard *et al.*, 1993 Ramesh & Bandyopadhyay, 1993 Hayward *et al.*, 1998 Olsson, 1976 Martin & French, 1995 Marín & El-Nashaar, 1993 Van Beuningen *et al.*, 1998
Solanum capsicastrum				Sweden	Olsson, 1976
Solanum carolinense L.	Horse nettle			United States	Hayward 1991
Solanum cinereum		2	3	Australia	Graham & Lloyd, 1979
Solanum dulcamara	Bitter nightshade	2	3	Sweden Netherlands France U.K	Olsson, 1976 Janse *et al.*, 1998 Hayward *et al.*, 1998 Elphinstone *et al.* 1998
Solanum hirtum			2	Honduras	Berg, 1971
Solanum incanum l.				India	Shekhawat *et al.*, 1992
Solanum jamaicense Miller				Philippines	Valdez, 1986

Weed Hosts					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
Solanum khasianum		3		Indonesia	Machmud, 1987
Solanum khasianum Clarke				India	Shekhawat *et al.*, 1992
Solanum luteum Mill.				Sweden	Olsson, 1976
Solanum muskiana l.				India	Shekhawat *et al.*, 1992
Solanum sarrachoides		2	3	Chile	Fernandez, 1986
Solanum sisymbriifolium	Dense-thorned bitter apple			Brazil	Mariano, 1998
Solanum umbellatum			2	Honduras	Berg, 1971
Solanum verbascifolium			2	Honduras	Berg, 1971
Solanum xanthocarpum				India	Shekhawat *et al.*, 1992
Solaum indicum L.				India	Shekhawat *et al.*, 1992
Sonchus arvensis				India	Shekhawat *et al.*, 1992
Spergula arvensis L.	Corn spurry		3	India Uganda	Shekhawat *et al.*, 1992 Tusiime *et al.*, 1998
Spigela anthelmia				Inonesia	Machmud, 1986
Stachytarphita jamaicensis L.	Snake weed			Australia	Akiew *et al.*, 1993
Stellaria sennii				Uganda	Tusiime *et al.*, 1998
Stellaria media		2 2	 3	Nepal Netherlands	Pradhanang, 1999 Van Beuningen *et al.*, 1998
Synedrella nodiflora Gaertn.	Pig's weed			Malaysia	Abdullah, 1993
Tagetes minuta				Australia Uganda	Akiew *et al.*, 1993 Tusiime *et al.*, 1998
Tagetes sp.		 3		India Philippines Nepal	Shekhawat *et al.*, 1992 Persley *et al.*, 1986 Adhikari, 1993
Thaliana sp.				India	Kishore *et al.*, 1993
Thalictum javanicum				India	Kishore *et al.*, 1993
Trifolium sp.				India	Kishore *et al.*, 1993
Tropaelum lobbianum Hort.				India	Shekhawat *et al.*, 1992
Tropaeolum majus L.	Garden nasturtium			R.S.A Sweden	Swanepoel, 1992 Olsson, 1976
Urtica dioeca L.	Bush stinging nettle		 1	India, Sweden Columbia	Shekhawat *et al.*, 1992 Olsson, 1976
Valeriana hardwickii				India	Kishore *et al.*, 1993
Verbesina alata L.					Kelman, 1953
Vernonia chinense Mill.					Kelman, 1953
Vernonia cinerea		3		Thailand	Titatarn, 1986
Vicia sp.				India	Kishore *et al.*, 1993
Xanthium chinense Mill.	Cocklebur				Kelman, 1953
Xanthosomas roseum (s)			2	Honduras	Berg, 1971

Tree and Shrub Hosts:					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
Aleurites moluccana (L.) Willd.	Candlenut				Kelman, 1953
Aleurites sp.	Tung oil tree				Kelman, 1953
Anacardium occidentale L.	Cashew	3		Indonesia, Réunion	Hayward, 1994
Annona L. spp.	Custard apple	3 3	1	Australia Taiwan	Mayers & Hutton, 1987, Hayward, 1986 Hayward, 1994
Archontophoenix alexandrae	Alexandra palm	3		Australia	Arnat *et al.*, 1978
Azadirachta indica J.Juss.	Neem tree	3		Australia	Hayward, 1994
Carica papaya	Papaya			India	Shekhawat *et al.*, 1992
Casuarina equisetifolia L.	Horsetail beefwood/casuarina	3 3,4	1	China India China, India, Mauritius	He, 1983 Shekhawat *et al.*, 1992 Hayward, 1994
Cyphomandra betacea	Tree tomato	2 1,2,3	3	Peru Peru Réunion	Martin & Nydegger, 1982 Marín & El-Nashaar, 1993 Girard *et al.*, 1993
Diospyros digyna Jacq.	Black sapote	3		Australia	Hayward, 1994
Eucalyptus L'Her. spp.	Eucalyptus	1 3		Brazil, China,	Hayward, 1994 Dianese & Dristig, 1993

Tree and Shrub Hosts:

Scientific Name	Common Name	Biovar cited	Race cited	Country of Report	Literature Reference
		3		Australia	Hayward, 1994
Eucalyptus grandis				Brazil	Mariano, 1998
Eucalyptus urophylla				Brazil	Mariano, 1998
Eugenia javanica Lam.	Java/wax apple			Taiwan	Hayward, 1994
Manihot esculenta Crantz	Cassava	3,4		Indonesia	Machmud, 1986
Manihot glaziovii M. Arg.	Ceara rubber				Kelman, 1953
Moring oleifera Lam.	Horse radish tree			India	Estelitta *et al*., 1997
Morus alba L.	Mulberry	5 3,5	4	China India	He, 1983 Mathew *et al*., 1993 (b)
Musa spp.	Banana	 1 1 3	2 1 2 1	Honduras Philippines Costa Rica, Venezuela, Honduras India Malaysia Indonesia Brazil Australia Trinidad	Woods, 1984 Soguilon *et al*., 1994 French & Sequeira, 1970 French & Sequeira, 1970 Shekhawat *et al*., 1992 Abdullah, 1993 Black & Sweetmore, 1993 Mariano, 1998 Akiew & Trevorrow,1994 Black & Sweetmore, 1993
Myristica fragrans L.	Nutmeg			India	Mathew *et al*., 1993 (a)
Olea europaea L.	Olive	3,4	1	China	He, 1983
Plantago sp.	Plantain	1		Costa Rica	Martin & Nydegger, 1982
Pluchea indica Less.	(type of Indonesian shrub)			India	Shekhawat *et al*., 1992
Pogostemen patchouli L.	Patchouli (used for essential oils)			India	Mathew *et al*., 1994
Schinus terebinthifolius	Pepper tree	3		Réunion	Hayward, 1994
Syzigium aromaticum	Clove tree	3		Indonesia	Machmud, 1987
Tectona grandis L.	Teak	3		Malaysia, Indonesia	Hayward, 1994

Leguminous Hosts:

Scientific Name	Common Name	Biovar cited	Race cited	Country of Report	Literature Reference
	Snapbean	3		Philippines	Valdez, 1986
	Stringbean	2		Philippines	Valdez, 1986
Albizzia falcata Back.					Kelman, 1953
Arachis hypogaea (L.)	Groundnut	3,4 3,4, 3	1	China Vietnam, Hawaii, Australia, Philippines, Papua New Guinea, Thailand, R.S.A, Réunion, Indonesia Uganda etc. India Malaysia Andaman & Nicobar Islands Brazil	He, 1983 Persley *et al*., 1986 Persley *et al*., 1986 Persley *et al*., 1986 Persley *et al*., 1986 Persley *et al*., 1986 Shekhawat *et al*., 1992 Abdullah, 1993 Black & Sweetmore, 1993 Ramesh & Bandyopadhyay, 1993 Mariano, 1998
Canavallia ensiformis DC.	Jack bean				Kelman, 1953
Cassia tora L.	Cassia/Senna			India	Shekhawat *et al*., 1992
Cyamopsis speciousus (sic.)					Kelman, 1953
Desmodium diffusum				India	Shekhawat *et al*., 1992
Dolichos lablab L.	Indian bean/Lablab bean			India	Shekhawat *et al*., 1992
Glycine max L.	Soybean			India Vietnam Sweden	Shekhawat *et al*., 1992 Persley *et al*., 1986 Olsson, 1976
Indigoferra arrecta Hochst.	Natal indigo				Kelman, 1953
Leucaena glauca	Leucaena				Kelman, 1953
Melilotus indica				India	Shekhawat *et al*., 1992
Mucuna sp. (*capitata*)	Mucana (vegetable)				Kelman, 1953
Phaseolus aureus Roxb.				Malaysia	Abdullah, 1993
Phaseolus calcaratus Roxb.	Rice bean				Kelman, 1953
Phaseolus coccineus L.	Scarlet runner bean				Kelman, 1953
Phaseolus mungo L.	Black gram				Kelman, 1953

Leguminous Hosts:

Scientific Name	Common Name	Biovar cited	Race cited	Country of Report	Literature Reference
Phaseolus vulgaris	Bean (French/kidney) (Yellow wax bean)	 3,4 3 1,3		India Malaysia, Uganda Réunion, Brazil Sweden Colombia	Shekhawat *et al.*, 1992 Hayward, 1994 Hayward, 1994 Girard *et al.*, 1993 Melo & Takatsu, 1997 Olsson, 1976 Thurston, 1963
Phaseolus vulgaris L. var. *humulis*	bushbean			Sri Lanka	Velupillai, 1986
Pisum savitum L.	Garden Pea				Kelman, 1953
Psophocarpus tetragonolobus	Winged bean	3 3		Malaysia Philippines Sri Lanka India	Hayward, 1986 Valdez, 1986 Velupillai, 1986 Shekhawat *et al.*, 1992
Sesbania bispinosa				India	Shekhawat *et al.*, 1992
Sesbania grandiflora Pers.					Kelman, 1953
Sesbania rostrata		4		Malaysia	Hayward, 1994
Stylosanthes humulis HBK	Townsville lucerne			Australia	Hayward, 1986
Tephrosia vogellii Hook.	Vogel tephrossia				Kelman, 1953
Teraminus labialis Spreng.				India	Shekhawat *et al.*, 1992
Vicia faba	Broad bean			India Sweden	Persley *et al.*, 1986 Olsson, 1976
Vignia sinensis Savi	Cowpea, Long bean	1,2,3		Brazil India, Philippines Malaysia	Melo & Takatsu, 1997 Hayward, 1994 Persley *et al.*, 1986 Abdullah, 1993
Vignia ungiculata				India Brazil	Shekhawat *et al.*, 1992 Mariano, 1998
Voandzeia subterranea Thou.	Earthpea				Kelman, 1953

Ornamental Hosts:

Scientific Name	Common Name	Biovar cited	Race cited	Country of Report	Literature Reference
Anthurium andreanum	Anthurium	 3	 1	Sri Lanka Réunion, Australia, Mauritius	Hayward, 1986 Hayward, 1994 Banymandhub-Munbodh *et al.*, 1998
Antirhinum sp.	Snapdragon	 3	 1	India Australia	Shekhawat *et al.*, 1992 Akiew & Trevorrow, 1994
Argemone mexicana L.	Mexican poppy			India	Shekhawat *et al.*, 1992
Aster chinensis L.				India	Shekhawat *et al.*, 1992
Aster pilosus Willd.	Aster				Kelman, 1953
Canna glauca	Canna			India	Kelman, 1953
Canna indica				India	Shekhawat *et al.*, 1992
Centaurea cyanus L.	Cornflower			India	Shekhawat *et al.*, 1992
Crysanthemum coronarium				Philippines	Valdez, 1986
Crysanthemum indicum				India	Shekhawat *et al.*, 1992
Crysanthemum spp.	Crysanthemum			India	Shekhawat *et al.*, 1992
Dahlia coccinea L.				Cuba	Stefanova, 1998
Dahlia pinata				Brazil	Mariano, 1998
Dahlia rosea Cav.	Dahlia			India Malaysia	Shekhawat *et al.*, 1992 Abdullah, 1993
Dahlia spp.	Dahlia			R.S.A	Swanepoel, 1992
Dahlia variabilis				India	Shekhawat *et al.*, 1992
Ensete ventricosum	Ornamental banana	3	1	French West Indies	Prior & Steva, 1990
Euphorbia pulcherrima	Poinsettia			Réunion	Girard *et al.*, 1993
Eustoma grandiflora	Texas blue bell	4	1	Taiwan	Chao *et al.*, 1995
Gerbera spp.	Barbaton daisy				Kelman, 1953
Hedychium Koenig spp.	Ornamental ginger			Hawaii	Hayward, 1994
Helychrysum bracteatum Andr.				Cuba	Stefanova, 1998
Helinconia	Helinconia	 1,3	2 2,1	Costa Rica, Columbia Australia, Hawaii	French & Sequeira, 1970 Akiew & Trevorrow, 1994 Hayward, 1994
Helinconia caribaea Lam.				Costa Rica	Hayward, 1994

Ornamental Hosts:					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
Helinconia latispatha Benth.				Columbia	Hayward, 1994
Impatiens balsamina	Garden balsam			India	Shekhawat *et al*., 1992
Nasturtium officinale				Brazil	Mariano, 1998
Pelagornium capitatum	Rose geranium		1	Réunion	Girard *et al*., 1993
Pelagornium x asperum	Rose geranium	1		Réunion	Girard *et al*., 1993
Pelargonium hortorum	Florist geranium	1		U.S.A Australia	Strider *et al*., 1981 Strider *et al*., 1981
Petunia hybrida	Garden petunia			India	Shekhawat *et al*., 1992
Petunia spp.	Petunia			Malaysia	Abdullah, 1993
Phlox drummondii Hook	Phlox			Malaysia	Abdullah, 1993
Salvia farinacea Benth.	Blue salvia			Malaysia	Abdullah, 1993
Strelitzia reginae Banks	Bird of Paradise	3	1	Hawaii Réunion, Hawaii, Japan, Taiwan, Australia	Hayward, 1994 Hayward, 1994 Hayward, 1994
Tagetea erecta L.	African marigold	1,3		India	Shekhawat *et al*., 1992
Vinca rosea L.	Madagascar periwinkle			India	Shekhawat *et al*., 1992
Zinnia elegans Jacq.				India Malaysia	Shekhawat *et al*., 1992 Abdullah, 1993
Zinnia sp.	Zinnia			India	Shekhawat *et al*., 1992

Other Crop Hosts:					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
	Chinese cabbage	3		Philppines	Valdez, 1986
	Sweet pepper	2,3,4	1	Japan	Tsuchiya & Horita, 1998
Allium cepa	Onion			Réunion, Venezuela	Hayward, 1994
Avena savita L.	Oats			India	Shekhawat *et al*., 1992
Beta vulgaris L.	Beetroot	1,3		India Brazil	Shekhawat *et al*., 1992 Melo & Takatsu, 1997
Brassica napus L.v. *napus*	Kohlrabi			Sweden, India	Olsson, 1976 Shekhawat *et al*., 1992
Brassica oleracea var. *capitata*	Cabbage	3 1,3		India Brazil	Shekhawat *et al*., 1992 Melo & Takatsu, 1997
Capsicum annum L.	Bell pepper, chillies	3 3 3 3,4 2,3	1 1 1,3	French West Indies Papua New Guinea China Philippines Thailand India R.S.A Réunion, Andaman & Nicobar Islands Brazil Peru	Prior & Steva, 1990 Tomlinson, 1985 He, 1983 Valdez, 1986 Titatarn, 1986 Shekhawat *et al*., 1992 Swanepoel, 1992 Girard *et al*., 1993 Ramesh & Bandyopadhyay, 1993 Mariano, 1998 Martin & French, 1995
Capsicum frutescens	Chillies (Bell pepper)	3		Réunion Brazil Colombia	Girard *et al*., 1993 Mariano, 1998 Thurston, 1963
Capsicum pendulum (s)	Pepper			India	Shekhawat *et al*., 1992
Capsicum sp.				Sweden Costa Rica	Olsson, 1976 Black & Sweetmore, 1993
Centella asiatica	Indian pennywort	3	1	Sri Lanka	De Zoysa & Liyanage, 1994
Cichorium endivia	Chicory	1,3		Brazil	Melo & Takatsu, 1997
Citrullus vulgaris Schrad.	Watermelon			India	Shekhawat *et al*., 1992
Commelina nudiflora L.	Baby dewflower				Kelman, 1953
Coriandrum savitum	Coriander	1		Brazil	Melo & Takatsu, 1997
Cucumis savitus	Cucumber			Japan	Hayward, 1994
Curcuma domestica	Tumeric			Sri Lanka	Velupillai, 1986
Curcuma longa L.	Tumeric			India Sri Lanka	Shekhawat *et al*., 1992 Hayward, 1994
Curcurbita maxima x C. moschata	Pumpkin	4	1	Japan	Tsuchiya & Horita, 1998
Curcurbita moschata Poit	Pumpkin			India	Mathew *et al*., 1994b

Other Crop Hosts:

Scientific Name	Common Name	Biovar cited	Race cited	Country of Report	Literature Reference
Cucurbita pepo var. *melopepo*	Zucchini	1,2,3		Brazil	Melo & Takatsu, 1997
Daucus carota	Carrot	1,3		Brazil	Melo & Takatsu, 1997
Fragaria L. spp	Strawberry	3,4	1	Japan, Taiwan	Hayward, 1994
Gossipium sp.	Cotton			India	Shekhawat *et al.*, 1992
Helianthus annus L.	Sunflower			India	Shekhawat *et al.*, 1992
				Malaysia	Abdullah, 1993
				Cuba	Stefanova, 1998
Hibiscus cannabinus	Indian hemp			Malaysia	Abdullah, 1993
Hibiscus esculentus L.	Okra			India	Shekhawat *et al.*, 1992
Hibiscus sabdariffa L.	Roselle				Kelman, 1953
Kaempferia galanta	Medicinal plant	4		China	He, 1986
Luffa cylindrica	Loofah	3	1	Taiwan	Pan *et al.*, 1996
Lycopersicum chilense				Sweden	Olsson, 1976
Lycopersicum esculentum	Tomato	3,4		Papua New Guinea	Tomlinson, 1985
				India, Vietnam, Fiji, China	Persley *et al.*, 1986
				Sri Lanka, Thailand etc.	Persley *et al.*, 1986
		3	1	R.S.A	Engelbrecht & Hattingh, 1989
		4	1	China	He, 1983
		1,3,4		Philippines	Valdez, 1986
		3		Nepal	Adhikari, 1993
				Malaysia	Abdullah, 1993
		1,2	1,3	Réunion	Girard *et al.*, 1993
				Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993
		1,2,3,4	1	Japan	Tsuchiya & Horita, 1998
				Brazil	Mariano, 1998
				Sweden	Olsson, 1976
		2	3	Peru	Martin & French, 1995
		1,2,3		Peru	Marín & El-Nashaar, 1993
				Colombia	Thurston, 1963
Maranta arundinacea L.				Malaysia	Abdullah, 1993
Nicotiana tabacum	Tobacco			Thailand	Titatarn, 1986
				Vietnam	French & Sequeira, 1970
		3		Nepal	Adhikari, 1993
				Réunion,	Girard *et al.*, 1993
				Malaysia	Abdullah, 1993
				Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993
		2,3	1	Japan	Tsuchiya & Horita, 1998
				Brazil	Mariano, 1998
		1		Colombia	Black & Sweetmore, 1993
		1,3,4		Australia,	Swanepoel, & Young,1988
		3	2	R.S.A,	Black & Sweetmore, 1993
		4		U.S.A,	Marín & El-Nashaar, 1993
		1,2,3		Peru	Black & Sweetmore, 1993
Perilla crispa	Perilla	3		Taiwan	Hayward, 1994
Perilla ocymoides L.	Perilla			Japan	Tsuchiya & Horita, 1998
Petroselium crispum	Parsley	1,3		Brazil	Melo & Takatsu, 1997
Piper hispidinervium	Long pepper	1		Brazil	Lopes *et al.*, 1998
Pogostemon cablin	Medicinal plant	3		China	He, 1986
Pomoea batatus LAM.	Sweet potato	4	1	China	He, 1983
Raphanus sativa L.	Radish			Taiwan,	Hayward, 1994
				India	Shekhawat *et al.*, 1992
Sesamum indicum	Sesame, gingelly	3	1	China	He, 1983
				Sri Lanka	Velupillai, 1986
				Thailand	Titatarn, 1986
				India	Shekhawat *et al.*, 1992
				Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993
Setaria italica Beauv.	Indian millet			India	Shekhawat *et al.*, 1992
Solanum auriculatum	Bringellier marron			Réunion	Girard *et al.*, 1993
Solanum capsicastrum				Sweden	Olsson, 1976
Solanum gilo	(Nigerian vegetable)			Brazil	Mariano, 1998

Other Crop Hosts:

Scientific Name	Common Name	Biovar cited	Race cited	Country of Report	Literature Reference
Solanum melangena	Eggplant	3		Australia	Moffet *et al.*, 1983,
				R.S.A	Engelbrecht & Hattingh, 1989
		3	1	Papua New Guinea	Tomlinson, 1985
		4		China	He, 1983
		1,3,4		Philippines	Valdez, 1986
				Thailand	Titatarn, 1986
		3		Nepal	Adhikari, 1993
				India	Shekhawat *et al.*, 1992
				Malaysia	Abdullah, 1993
				Réunion	Girard *et al.*, 1993
			1	Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993
		2,3,4		Japan	Tsuchiya & Horita, 1998
				Brazil	Mariano, 1998
				Sweden	Olsson, 1976
		1,2,3		Peru	Marín & El-Nashaar, 1993
Solanum tuberosum	Potato	2,3,4		Philippines	Valdez, 1986
		2		Nepal	Adhikari, 1993
				Malaysia	Abdullah, 1993
		1,2	1,3	Réunion	Girard *et al.*, 1993
				Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993
		1,2		Bolivia	Smith *et al.*, 1998
		2,4	3,1	Japan	Tsuchiya & Horita, 1998
				Brazil	Mariano, 1998
				U.K., Netherlands, Italy	Hayward *et al.*, 1998
				France, Latin American	Hayward *et al.*, 1998
				countries, Australia	Hayward *et al.*, 1998
				Sweden	Olsson, 1976
		2,4		Papua New Guinea	Tomlinson, 1985
		1,2,3		Peru	Marín & El-Nashaar, 1993
		2,3		South Africa	Swanepoel & Young, 1988
		2,3		Australia	Black & Sweetmore, 1993
		1		USA	Black & Sweetmore, 1993
		1		Indonesia	Black & Sweetmore, 1993
Symphytum sp.	Forage crop	3		China	He, 1986
Tetragonia expansa	Spinach (N. Zealand)	1,3		Brazil	Melo & Takatsu, 1997
Zingiber officinale	Ginger	4		Costa Rica	Martin & Nydegger, 1982
		3		Mauritius	Hayward, 1986
		4	1	China	He, 1983
		3,4		China	Hayward, 1986
		1,3,4		Malaysia	Hayward, 1986
		3,4		Australia	Hayward, 1986
		4		Hawaii , Philippines	Hayward, 1986
		3		India	Hayward, 1986
				Sri Lanka	Velupillai, 1986
		3,4		Thailand	Hayward, 1994
				India	Shekhawat *et al.*, 1992
				Andaman & Nicobar Islands	Ramesh & Bandyopadhyay, 1993

Other plant species

Scientific Name	Common Name	Biovar cited	Race cited	Country of Report	Literature Reference
	Squash	3		Philippines	Valdez, 1986
	Fennel	4		Philippines	Valdez, 1986
	Pechay	3		Philippines	Valdez, 1986
	Plantain		2	Honduras, Colombia, Peru, Costa Rica	French & Sequeira, 1970 French & Sequeira, 1970
Hyoscyamus niger L. V. niger				Sweden	Olsson, 1976
Hyoscyamus niger L. V pallidus				Sweden	Olsson, 1976
Epipremun aureum	pathos	1	1	USA	Valdez, 1986, Norman & Yuen, 1998
Alpinia L. spp.		3		Australia	Hayward, 1994
Blainvillea rhomboidea				Brazil	Mariano, 1998
Browallia speciosa Major				India	Shekhawat *et al.*, 1992

Other plant species					
Scientific Name	**Common Name**	**Biovar cited**	**Race cited**	**Country of Report**	**Literature Reference**
Corchorus capsularis	Jute			West Bengal	Hayward, 1986
Corchorus olitorius	Jute			West Bengal	Hayward, 1986
Corchorus spp.				India	Shekhawat *et al.*, 1992
Corchorus trilocularis				India	Shekhawat *et al.*, 1992
Coriandrum savitum				India	Shekhawat *et al.*, 1992
Cosmos caudatus				Sarawak	Hayward, 1986
Cu,inum cyminum				India	Shekhawat *et al.*, 1992
Echinochloa crusgalli Beauv.				India	Shekhawat *et al.*, 1992
Eleusine coracana Gaerlin.				India	
Eustoma russellianum	Russel prairie gentian			Japan	Tsuchiya & Horita, 1998
Foeniculum vulgare				India	Shekhawat *et al.*, 1992
Galium aparine L.				India	Shekhawat *et al.*, 1992
Heliotropium indicum				India	Shekhawat *et al.*, 1992
*Hyptis suaveolens (*L.) Poit.				India Brazil	Shekhawat *et al.*, 1992 Mariano, 1998
Ipomea obscura Ker.				India	Shekhawat *et al.*, 1992
Ipomoea setosa				Malaysia	Hayward, 1986
Kalanchoe blossfeldiana v. Paellnitz				Réunion	Hayward, 1994
Kalanchoe tubiflora				Brazil	Mariano, 1998
Launea aspleniifolia DC				India	Shekhawat *et al.*, 1992
Limonium spp.	Statice	3	1	Japan	Tsuchiya & Horita, 1998
Ludwiga suffruticosa				Brazil	Mariano, 1998
Marsypianthes chamaedrys				Brazil	Mariano, 1998
Mimosa scabrella				Brazil	Mariano, 1998
Momordica charantia	Bitter Gourd	1,2,3		Philippines Sri Lanka	Valdez, 1986 Velupillai, 1986
Obetia ficifolia	Bois d'ortie			Réunion	Girard *et al.*, 1993
Parthenium hysterophorus				India	Shekhawat *et al.*, 1992
Solanum americanum				Brazil	Mariano, 1998
Trachyspermum amni				India	Shekhawat *et al.*, 1992
Urtica nivea		3	1	China	He, 1983
Verbena hybrida				India	Shekhawat *et al.*, 1992

SOURCES OF INOCULUM AND MODES OF DISPERSAL

Two major sources of inoculum exist, namely infected planting material and infested soil. Infected plants decaying in the soil can release masses of bacterial cells in a slime layer. These slime masses can adhere to soil particles and form pellets enhancing its survival (Shekhawat *et al.*, 1992). The populations in the soil can then increase or decrease depending on the presence of alternative hosts and cultural practices. The inoculum threshold for initiating disease depends highly on predisposing factors. Devi *et al.* (1982) observed that the inoculum reached a threshold of about 10^7 cfu / g soil before infection started.

Infected planting material such as potato tubers is the most effective source of inoculum and means of dispersal. Since the pathogen can be carried latently within tubers, controlling the transmission of this pathogen is complicated. Tubers can carry the bacteria in three manners, namely externally on tuber surfaces, in lenticels and in the vascular tissues (Shekhawat *et al.*, 1992). Although surface carried bacteria can be eliminated by chemical treatments, internal infections remain a threat. A study conducted by Sunaina *et al.* (1989) showed that during storage, bacterial populations decreased rapidly on the tuber surface reaching a non-detectable limit within 30-60 days at 4°C and 60-90 days at room temperature. But in lenticels and vascular tissues *R. solanacearum* could still be detected after 240 days.

Irrigation water, root contact and insects such as in the case of Moko disease of banana can also spread the disease. Mechanical dissemination occurs mainly by infested equipment both during sorting of seed tubers as well as on the field, and by movement of people and animals through infected fields. Insect dissemination plays an important role in the banana industry and has been reported in Honduras, Costa Rica and Colombia. Bees (*Trigona* spp.), wasps (*Polybia* spp.), fruit flies (*Drosophila* spp.), and flies of other genera have been identified as transmitters (Buddenhagen & Kelman, 1964). Reports where *R. solanacearum* was disseminated by chewing insects on potato (Colorado potato beetle, *Leptinotarsa declimlineata* Say.) and eggplants (green beetle, *Diabrotica graminea* Baly) have been made (Kelman, 1953). Negative results have however also been reported where insects were allowed to feed on infected plants and then on healthy ones (Kelman 1953).

Some evidence suggests that *R. solanacearum* can have an epiphytic phase in its life cycle which contributes to its survival and provides another source of inoculum. Hayward & Moffet (1978) demonstrated that leaf spot disease of capsicum was caused by *R. solanacearum*. Further studies (Moffet *et al*, 1983) revealed that under conditions with relatively high humidity, epiphytic colonisation could occur, leading to the formation of lesions on leaves. Mist inoculation of eggplant, pepper and tomato cultivated in growth-chambers caused leaf spots and wilting (Kelman *et al.*, 1994). Although leaf infection has been reported, there is no evidence that the pathogen can survive as an epiphyte on leaf and other plant surfaces (Kelman *et al.*, 1994). Aerial transmission through rain splash dispersal on tobacco has been noted in Japan (Hayward, 1991a).

Infected host debris is an important short-term shelter for *R. solanacearum* in soil (Graham *et al.*, 1979) allowing survival between growing seasons. It also serves as a transmission agent. This is especially so for race 3 which has a limited alternative host range.

Weeds serving as hosts are well-documented sources of inoculum and contribute greatly to the survival of *R. solanacearum* in the absence of a cultivated host. They may also serve as a source of infection when virgin lands are cleared for cultivation (Buddenhagen & Kelman, 1964, Martin *et al.*, 1981).

PRESENCE OF RALSTONIA SOLANACEARUM IN VIRGIN SOILS

The occurrence of *R. solanacearum* in newly cleared lands or virgin soils has been cited in literature (Kelman, 1953; Sequeira & Averre, 1961; Martin *et al.*, 1981) and has been attributed to the presence of wild hosts in the indigenous flora. In earlier reports proof was not always presented that bacterial wilt was truly a natural component of the soil microflora, and the possibility of contamination through planting material, drainage water or other means was not eliminated. Several authorities have studied the outbreak of bacterial wilt of bananas in newly planted areas in Costa Rica. A comprehensive study conducted by Sequeira & Averre (1961) involving 20000 acres of virgin woodlands in Costa Rica revealed extensive infection of *Heliconia latispatha*, *H. acuminata*, *H. imbricata* with the banana strain (B strain) of *R. solanacearum*. *Eupatorium oderatum* was found to be infected with the weed strain of the pathogen (T strain). French *et al.* (1981) who summarised the findings of Buddenhagen (1960), Sequeira (1960), Sequeira & Averre (1961) and his own, refer to the strain causing rapid wilt of bananas as race 2, strain B and those causing slow wilt and distortion on helinconias as strain D of race 2. Repeated cutting back of heliconias caused the disease to be spread mechanically to other clusters of heliconias. When bananas were planted in cleared jungle, they developed a slow wilt and distortion not typical of the banana strain.

In Costa Rica *Heliconia* were occasionally found diseased in virgin jungle showing distortion and slow wilt symptoms caused by D strains of race 2. Bananas planted in cleared jungle also developed distortion and slow wilt, symptoms not characteristic of the B strain wilt disease, which causes rapid wilting and was responsible for severe outbreaks. Continuous passage of the D strain through bananas resulted in a doubling of the disease index, leading to the conclusion that bacterial wilt of bananas arose by a selection pressure exerted by bananas upon strain D (French *et al.*, 1981).

Martin *et al.* (1981) found that biovar 1 (race 1) and biovar 2 (race 3) of the pathogen attacked potatoes grown in virgin soils in the Amazon basin. No potatoes or other wilt-susceptible crops had been planted before and infestation by contaminated water or by planting infected seed was excluded. This suggests that these strains were indigenous to the region.

Sneviratne (1969) found biovar 2 to occur in virgin soils at elevations of 1891m in Sri Lanka. He believes it unlikely that the disease could have been introduced by European seed material. In their study on persistence of *R. solanacearum* in soil in Georgia, Dukes *et al.* (1965) had cleared land of timber and brush and planted tomatoes and found a high incidence of bacterial wilt indicating that the organism was indigenous.

SURVIVAL OF RALSTONIA SOLANACEARUM IN SOIL

Ralstonia solanacearum is a soil-borne pathogen that is found in various types of soils world-wide. Reports regarding its survival period are often conflicting. Bacterial wilt was found to survive in fallow soil for periods of 2 to 10 years, yet in a different soil poorer survival rates were reported despite the presence of host plants (Nesmith &

Jenkins, 1985). Information on soil survival was often gathered indirectly or from glasshouse trials, partly because detection of the organism in field soils is difficult (Moffet & Wood, 1984).

The survival of *R. solanacearum* in soil is affected by several factors such as the initial inoculum concentration, whether the land is left fallow or cropped to a non-susceptible host, as well as the biological, chemical and physical properties of the soil (Moffet *et al.*, 1983). The temperature, moisture and oxygen status of the soil is further factors that influence the longevity of the pathogen. Survival of *R. solanacearum* in soil can be measured on the basis of two parameters, namely, the ability to withstand soil conditions to remain viable and the segregation into virulent and avirulent populations (Shekhawat & Perombelon, 1991). Several authors, amongst others Nesmith & Jenkins (1983) measures survival in terms of detection of fluidal cells on selective media. These cells are referred to as virulent. Denny *et al.* (1994) reason, however, that in soil *Ralstonia* normally exits in the "avirulent" (phenotype conversion) form, in which reduced production of extracellular proteins and extracellular polysaccharides occurs. In this form the bacteria can conserve energy and cellular resources thereby increasing its chances of surviving. Once host material is available, bacteria multiply and once sufficient cell density is obtained the extracellular virulence factors are produced. This hypothesis would greatly affect measurement of survival rate and might explain some of the discrepancies regarding the longevity of bacterial wilt in soil.

Influence of soil temperature

Temperature requirements for optimal growth are known to differ for the various strains. Biovar 2, race 3 isolates have a lower optimum growth temperature than strains of race 1 (Thurston, 1963). Disease development in terms of wilting and visible tuber infection, is known to occur at lower temperatures of 14/16 °C with biovar 2 than with biovar 3 (race 1) (Swanepoel 1990). Katayama & Kimura (1984) also found that at lower as well as intermediate temperatures of 24 °C, growth of biovar 2 (race 3) was better than that of race 1, biovar 4. Shekhawat & Perombelon

(1991) studied the survival rates of biovar 3 (race 1) and biovar 2 (race 3) at various temperatures and confirmed that race 1 is better adapted to a wider range of temperature for growth than race 3. In their study it appears that population decline and loss of virulence of both races was slowest between 10-30 °C, provided other soil factors were congenial. At 35 °C, race 1 population declined to an undetectable limit within 10 weeks, whereas populations of race 3 could not be detected after 8 weeks. At low temperature of 5 °C, population decline was the same for both races, reaching undetectable levels within 12 weeks. Granada & Sequeira (1983b) however reported that soil kept in plastic bags at 4 °C maintained bacterial wilt populations for 673 days. This indicates that long-term survival in deeper soil levels at low temperatures is possible. Soil temperatures of over 40 °C appear to be fatal to *Ralstonia* populations, depending on the period of exposure. Seneviratne (1988) found that the pathogen was able to survive in some of the soil samples kept at 40 °C for seven days, but not in those kept at 43 °C.

Survival of races 1, 2, 3 in soil kept at 28 °C and water potential of –2 bars varied with race 1 surviving longest. Race 2 and race 3 had shorter survival periods (Granada & Sequeira, 1983b).

Influence of soil moisture

Soil moisture may influence at least four aspects of the bacterial wilt disease, namely the survival of the bacterium in its free state in soil, rate of infection, disease development after infection and spread through the soil. Native farmers in India have from an early date noted the relationship between soil moisture and bacterial wilt of tobacco and attributed the disease to high moisture levels (Shekhawat *et al.*, 1992).

Studies on the effect of soil moisture on the survival of *R. solanacearum* have provided conflicting evidence. This can partly be attributed to interactions with other soil factors. According to Shekhawat *et al.* (1992) soil moisture and temperature have a synergistic effect on disease development. High temperatures or high soil moisture alone will not induce symptoms. They found that in India, potato wilt was higher after onset of the monsoon, even though high temperatures prevailed earlier in the season. Wilt incidence declined when soil moisture dropped to 8-10% of water holding capacity (WHC). Tanaka & Noda (1973) found that growth rate of *R. solanacearum* in sterile soil is higher at high soil moistures (80-100% water content) than in soil at 40% of water holding capacity. Under identical conditions but in non-sterilised soil no increase in growth was observed, regardless of water content.

Okabe (1971) found that *R. solanacearum* grew more actively in dry soil of 15-20% water content (WC) than in moist soil (40-50% WC) and reasoned that this pathogen had the specific nature to utilise small amounts of capillary water held among soil particles while growth of other micro-organisms was delayed. If, however, soil moisture were increased, re-growth of these micro-organisms would be stimulated leading to increased competitive colonisation of organic matter and antibiosis. This would reduce *R. solanacearum* populations. This theory could explain why Tanaka & Noda (1973) did not observe increase in growth in the non-sterilised soil at both moisture levels. Their moisture levels were higher than those investigated by Okabe (1971).

Shekhawat & Perombelon (1991) reported that population decline was at its lowest in a soil moisture at 60% of water holding capacity (WHC). Even after 13 weeks the pathogen could still be detected. A deviation in moisture led to an increased population decline. Greater population declines occurred at high moisture levels (80-100% of WHC) than at low moistures (20-40% of WHC). At the high moisture levels a shift towards avirulence was more pronounced when soil temperatures were low (5°C) (Shekhawat *et al.*, 1992). In dry soils the populations decreased to an undetectable limit within 6 weeks.

Hsu (1977) noted similar trends regarding high and low moisture contents. He reported a survival period of 30 days for dry soil (0.8-7.9% WC); 105 days for flooded soil; 150 days for very moist soil (43-47.4% WC); 225 days for moist soils (15.2-19.9% WC) and 390 days for moderately moist conditions (30.7-36.7% WC).

Moffet *et al.* (1983) performed a study in which pressure potential rather than moisture content was used to measure soil moisture, since they regard this a more accurate measure of the availability of water to micro-organisms. Sensitivity to degrees of drying varied with the biovar of the pathogen used and soil type, with biovar 2 decline being greater than biovar 3. Population decline for both biovars in the various soils (clay loam, sandy loam, and clay) was generally highest at -0,003 kPa.

Sensitivity to dry conditions has been reported to be a contributing factor in reducing *R. solanacearum* populations in fallow land (Sequeira, 1962). Moffet *et al.* (1983) found, however, that the rapid decline of both biovars that occurred with drying was slowed if the pressure potential was maintained at a constant value. They did note that *R. solanacearum* decreased the most in the driest soils in the initial stages of the trials, but that in the end the driest soil contained higher numbers of viable pathogens than did the wetter soils.

A rapid decline was observed in flooded or very dry soils, irrespective of soil type (Nesmith & Jenkins, 1985). Their study also revealed that antagonistic actinomycetes were most numerous in dry soil, whereas antagonistic bacteria were predominant in wet soils.

Influence of soil type

Relation between soil type and incidence of bacterial wilt is not clear and contrasting reports have been obtained. In Indonesia bacterial wilt of peanuts is most severe in heavy clay soils whereas in China it is prevalent in sandy, especially gritty soil, and not in heavy clay or loam (Hayward, 1991a).

Nesmith & Jenkins (1985) found that soil type influenced soil moisture and antagonistic microbial populations, which in turn affected the *Ralstonia* populations. Soil bacteria are active primarily in soil of higher pressure potential and reduced activity is noted at –0.03 kPa and especially at –0.15 kPa (Cook & Papendick, 1970, as quoted by Moffet *et al.*, 1983). The activity of the microbial population associated with organic matter varies with soil type and pressure potential. In a study on effect of moisture and soil type on survival of *R. solanacearum* in soil, Moffet *et al.* (1983) reported organic matter contents of 2,5% in clay loam, 1.7% in clay and 1.1% in sandy loam. They noted a greater *R. solanacearum* population decline in the clay loam than in clay or sandy loam at higher pressure potentials. The increased decline in the clay loam was attributed to the higher microbial activity associated with the organic matter. *R. solanacearum* populations thus have to compete with the soil microbes for nutrition and be exposed to increased microbiostasis. The finding that population decline was less in the clay than in the sandy loam, even though the microbial activity is expected to be higher in clay, could be due to adsorption of *Ralstonia* onto clay particles, protecting them from microbiostasis (Moffet *et al.*, 1983). The authors recorded greater decline of biovar 2 population on all of the three soil types in comparison to biovar 3 populations. This was ascribed to greater sensitivity of this biovar to microbiostasis or its poor competitive ability or to the intrinsic nature of the biovar 2 isolate.

Tanaka (1976) previously reported the relation between organic matter, microbes and *Ralstonia* populations. In surface soil with high levels of organic matter and microbial activity, the pathogen population declined faster than in the subsoil with a lower content of these, and that addition of manure to the subsoil reduced the populations considerably.

In a study conducted by Shekhawat & Perombelon (1991) population decline was slower in clay than in sand even under dry conditions. In dry clay, race 1 and race 3 were detectable after 5 weeks and 3 weeks, respectively, whereas in dry sand both races of the pathogen were undetectable within 3 weeks.

Influence of depth of soil layers

Results of several authors (Mc Carter *et al.*, 1969, Okabe, 1971, Tanaka & Noda, 1973) suggest that *R. solanacearum* can survive in deeper layers of certain soils. Once the pathogen has entered the deeper layers it can survive in localised microsites (debris or "free soil") where microbial activity is likely to be low (Lloyd, 1978). Subsequent infection of a host plant would then depend on penetration of the root system into these microsites. The survival of the pathogen in these sites would probably be limited to a few deep-rooted hosts such as potatoes and tobacco growing in favourable soil types such as loose sandy soils (Lloyd, 1978).

Vertical distribution of the bacterial wilt pathogen in the soil varies, ranging from the presence in topsoil to almost a meter deep. Martin *et al.* (1981b) investigated the presence and distribution of *R. solanacearum* at three soil depths (0-30 cm, 30-60 cm, and 60-90 cm). In one location the bacterial population were highest in the 0-30 cm soil layer, whereas in the other location the highest population was recorded in the 30-60 cm layer. The authors suggest that such varying results could be attributed to amount of rainfall, type of soil and differences in root development. Other authors (Okabe, 1971; Tanaka, 1976; Graham & Lloyd, 1979) reported highest concentrations of *R. solanacearum* at deeper soil layers. Tanaka (Tanaka, 1976) observed the distribution of the pathogen in the 0-80 cm layer of naturally infested sandy loam soil, and noted a high population at all depths even after one year of fallow. After two years, however, the pathogen had practically disappeared.

Graham & Lloyd (1979) studied the distribution of *R. solanacearum* in soil at five sites in a naturally infested field. At three sites samples were collected at soil depths of 0-15 cm, 15-30 cm, 30-45 cm, 45-60 cm and 60-75 cm, at the other two only the 30-45 cm and 60-75 cm layers were sampled. The pathogen was detected at all five sites, but not at all depths. *R. solanacearum* could not be detected in any of the 0-15 cm samples and at one site the bacteria were only present in the 15-30 cm layer. Presence was detected in the 60-75 cm layer in four of the five sampling sites. The authors regard desiccation of *R. solanacearum* cells due to dry weather prior to sampling as one of the reasons that the bacterium was absent from the 0-15 cm zone, but state that other factors could also be involved. Graham & Lloyd (1979) observed that their results contrasted with those obtained by Mc Carter *et al.* (1969) in a similar vertical distribution study. Mc Carter *et al.* (1969) recorded a high infestation in the top 30 cm layer, with markedly reduced or absent populations in the 30-45 cm zone. No *Ralstonia* populations could be detected deeper than 45-60 cm except for a low presence in one sample. Graham & Lloyd (1979) considered root development as one of the causes of variation. The work done by Mc Carter *et al.* (1969) involved soils infested by diseased tomato transplants whose roots are not likely to penetrate to great depths. Their study, however, involved soils infested with diseased potato plants.

The study conducted by Sunaina *et al.* (1892) also supports the hypothesis that the depth of root systems of hosts might govern vertical distribution. They found that during the potato season population build-up was higher in the top 30 cm than in the deeper soil layers. During the non-cropping season the population declined much quicker in the top 30 cm as compared to deeper layers, and in the top 20 cm it decreased to an undetectable level. The pathogen survived at the 20-60 cm soil level even after the field had been kept fallow for 7 months. The longer survival of the pathogen in deeper soil layers is to be expected as the roots and bacterial exudates remain undisturbed.

Influence of anaerobiosis of soil

Longevity of *R. solanacearum* is also affected by the oxygen status of the soil. Anaerobic conditions cause a more rapid population decline with undetectable levels being reached within 7 weeks, whereas 11 weeks where required under aerobic conditions to reduce the population to undetectable limits. Anaerobic conditions also favoured a shift to avirulence (Shekhawat & Perombelon, 1991).

R. solanacearum is an aerobic organism and conditions that reduce the availability of oxygen should affect its survival negatively. Flooding of soil has been reported to adversely affect the pathogen's survival (Kelman, 1953; Nesmith & Jenkins, 1985). Yet some reports indicate that flooding of rice fields for several weeks does not eradicate bacterial wilt (De S. Seneviratne, 1988; Shekhawat *et al.*, 1992). In some cases bacterial wilt incidence was higher in a potato crop that followed after paddy rotation. The soil in flooded rice paddies appears to be more oxygen-rich than flooded fallow soils. Aerobic and anaerobic microsystems exist in soil of paddies. The diffusion of oxygen through the roots of rice creates an aerobic environment in the rhizospere and rhizoplane, allowing *R. solanacearum* to survive Shekhawat *et al.* (1992).

Influence of pH of the soil

Although the optimum pH for growth of *Ralstonia solanacearum in vitro* is about 6.8, bacterial wilt has been reported in both acidic and alkaline soils. In North Carolina a high incidence of potato wilt occurred in soil with a pH 4.5. Tobacco wilt in this state was more common in moderate acid soils (pH 5-5.5) (Kelman, 1953). Similarly, in Guadeloupe bacterial wilt was reported in soils of pH 5-5.5 (Prior *et al.,* 1993) and in Mauritius it was not common in alkaline soils (Kelman, 1953). Soil conditions in which this bacterium occurred in Japan and Ceylon were often alkaline. In one instance a soil pH of 8.5 was recorded (Kelman, 1953).

Shekhawat & Perombelon (1991) investigated the impact of different pH levels on race 3 (biovar 2) and race 1 (biovar 3) grown in broth culture. At pH 4.5 growth of both race 1 and race 3 decreased and virulence was completely lost. At pH 8.5 the virulence and growth of race 3 was reduced whereas race 1 grew well although virulence was reduced. At pH 5.5 and pH 7 race 3 grew well and maintained a high level of virulence. Race 1 also grew well but could maintain high virulence only at pH 7. It would therefore seem that race 3 is better adapted to retain virulence under low pH conditions.

CONTROL OF RALSTONIA SOLANACEARUM

Chemical Control

Several chemical formulations have been evaluated for the control of bacterial wilt, with limited success. Disinfectants such as potassium sulfide, copper acetate, potassium permanganate and formalin are not effective and often damage the crop and pose a threat to the environment (Kelman, 1953; Shekhawat *et al.*, 1992). Control or eradication of bacterial wilt has been attempted with fumigants such as chloropicrin, ethylene dibromide and methyl bromide with varying results (Kelman, 1953; Enfinger *et al.*, 1979; Engelbrecht *et al.*, 1990; Melton & Powell, 1991; Chellemi *et al.*, 1997). Chloropicrin has been applied since the 1940's and in most instances crop losses could be reduced, but complete control or eradication could not be obtained (Kelman, 1953). Enfinger *et al.* (1979) evaluated the application of an array of chemical formulations, amongst them fumigants such as chloropicrin, methyl bromide, DD-MENCS (a mixture of methyl isothiocyanate, dichloropropane and dichloropropene), and Metham. Chloropicrin was the only formulation that provided significant control throughout the season. DD-MENCS and a mixture of methyl bromide and chloropicrin (67-33%) were less effective than chloropicrin on its own, but reduced wilt more than methyl bromide. Methyl bromide was found to control wilt only until midseason. Metham applied as fumigant gave moderate to poor control early in the season. Applied as fumigant it was more effective than applied as drench or when incorporated.

Dichloropropane and dichloropropene formulations have also been used since the 1940's to reduce the incidence of wilt. At high dosages where wilt was completely controlled, plants developed chlorine injury (Kelman, 1953). Engelbrecht *et al.* (1990) revealed that disease suppression was most effective if a mixture of ethylene bromide and chloropicrin (55:45 m/m) was applied at a rate of 120 L/ha. All chloropicrin treatments were found to reduce the disease incidence significantly. They also found at one of the trial sites that a chloropicrin/methyl bromide mixture was less effective than chloropicrin on its own, but that it did not differ significantly from methyl bromide application. In a trial where very high infestation levels were encountered, fumigation with ethylene bromide: chloropicrin 55:45 m/m at 120L/ha failed to suppress the disease for the entire growing season. Ethylene bromide has been banned in the United States since 1983 (Sittig, 1985).

Melton & Powell (1991) were not successful in reducing the wilt index in tobacco fields by fumigating with 1,3-dichloropropene and chloropicrin but yields were increased. Fortnum & Martin (1998) obtained both a reduction of the pathogen and an

increased yield by fumigating with 1,3-dichloropropene (78%): chloropicrin (17%) mixture with both in-row and broadcast applications. The authors suggest a waiting period of three weeks after application and warn that spring rains can interfere with the application program.
A bactericidal formulation "Terlai" which has an active ingredient similar to 2,2-dichloro-N (2 hydroxy-1(hydroxy-methyl)-2-(4-nitrophenyl) ethyl acetamide, significantly reduced the incidence of wilt, especially if applied in conjunction with *P. fluorescens* (Machmud & Machmud, 1994). Chlorosis on the leaves of tomato was noted, possibly due to high concentrations of "Terlai" applied.

Chemical compounds have also been used to treat plants to offer protection against infection with *R. solanacearum*. Infection in tobacco seedlings could be delayed slightly by treating the roots & stems with compounds such as hydroxymercuricchlorophenol, zinc sulphate plus lime, Bordeaux mixture and sulphur. Aldicarb, a systemic insecticide, was found to hasten the development of wilt in tomato plants, by altering the quality and quantity of extracellular polysaccharide (Shekhawat *et al.*, 1992). Rhizome treatment of ginger with Emisan 6 (an organomercurial) plus plantomycin for 30 minutes in addition to three spray applications resulted in 100% control of bacterial wilt. Plantomycin alone or in combination with Blitox was 95% effective (Shekhawat *et al.*, 1992).

Several studies on the use of antibiotics as control agents have been performed with varying success. Pretreatment of potato tubers with antibiotic C-6 (similar to erthromycin) followed by two foliar sprays resulted in control and a three-fold increase of yield. Dipping roots of eggplant seedlings into streptocycline prior to transplanting reduced the incidence of wilt (Shekhawat *et al.*, 1992). Engelbrecht *et al.* (1990) could not control tobacco wilt effectively with frequent application of streptomycin sulphate.

Management through Cultural Practices

The use of disease free planting material

Planting material free of bacterial wilt is vital in preventing the pathogen to be introduced into wilt-free soil. This is extremely difficult with potato where the pathogen is often harbored latently (Hartman & Elphinstone, 1994). The introduction of infected seed into cooler regions has often resulted in the production of latently infected seed on apparently disease-free fields. Such infected tubers have been known to cause severe outbreaks of the disease (French, 1994). In order to prevent the spread of the disease, cuttings from *in vitro* stocks were used on a large scale in Vietnam (Vander Zaag, 1986). Latent infections have also posed a major problem for plant breeders selecting for resistance to wilt. One example is the tolerant cultivar Cruza 148. Although plants do not show any external wilt symptoms or visible tuber infections, the bacterium is carried symptomless in the tuber (Hayward, 1991). Although such tolerance allows production of potatoes in infected regions, it assists in transmitting the disease.

R. solanacearum can be transmitted by peanut seed (Machmud & Middleton, 1991). In China groundnut seed is preserved over the winter period and Yongxiang *et al* (1993) reason that application of dry preservation measures could prevent transmission. A subsequent study showed that peanut seed with a water content of 10% or higher could transmit R. *solanacearum* (Dongfang *et al.,* 1994). These authors also suggested that under normal conditions of preservation, the pathogen might not survive. Moffet *et al.* (1981) obtained infected plants from tomato and pepper seeds that had been inoculated artificially. In order to investigate seed transmission Shakya (1993) germinated tomato seeds and found that 21% of the seedlings developed water-soaked brown discoloration on their roots. After 8-9 days these seedlings collapsed. Isolated bacteria were identified as *R. solanacearum*. Singh (1994) was able to confirm the transmission of bacterial wilt through tomato seed as well as through eggplant seed. Occurrence and survival of *R. solanacearum* biovar 3 on eggplant seed was reported by Chatterjee *et al.* (1994).

Use of whole seed versus cut seed material

Many potato crops are grown from seed pieces cut from larger tubers, mainly to reduce the cost of planting material. Emergence from cut seed is usually more uniform across a field, since sprouting from whole seed within individual hills occurs over a longer period of time (Mosley & Chase, 1993). Cutting of seed tubers increases the risk of bacterial or fungal infections. During the cutting process the pathogen can be spread from a diseased tuber to healthy ones by contaminated blades. The cut surfaces also provide ideal entry points for soilborne pathogens. In order to reduce fungal seed decay fungicides are usually applied. The impact of cutting seed material on the incidence of bacterial wilt has been noted by Shekhawat *et al.* (1988). Wilt increased up to 12 times in potato crops where cut seed had been used in comparison to uncut seed. Treatment of the cut pieces with the fungicide Dithane M-45 did not reduce the wilt.

Hilling at planting

Hilling is important to avoid sun damage of developing tubers, to protect tubers against potato tuber moth and to minimize late blight infection of tubers. In some production areas multiple hilling is performed as part of a weed control program, in others a single hilling is done after sprout emergence (Rowe & Secor, 1993). Shekhawat & Chakrabarti (1993) recommend that this procedure be completed at planting time since this minimizes injury to the potato plant. Wounds provide entry sites for bacterial wilt. Post-emergence ridging can increase injury by 10-15% (Shekhawat *et al.* 1992). Hilling three weeks after emergence does not appear to increase the incidence of wilt where low infestation levels were encountered. Where infestations were high, post-emergence hilling did increase the percentage of wilt (Kloos, 1986).

Control of nematodes and weeds

The role of root-knot nematodes in the development of bacterial wilt by providing entry points and possibly assisting tissue colonization has been reported by several authors (Hayward 1991a; Shekhawat *et al.*, 1992). Practices for the control of nematodes include the planting of resistant varieties, using chemical soil treatments, fumigation and rotation with crops that are resistant to both nematodes and bacterial wilt (Akiew *et al.*, 1993).

Weed control is vital since several species serve as hosts and allow survival in the absence of a host crop (refer to Table 6.1). In one instance, however, the presence of susceptible weeds had no influence on the severity of wilt in the next season (Akiew *et al.*, 1993). Most findings indicate that weeds promote the survival of *Ralstonia* in the soil, transmit the pathogen to the next crop, and reduce the success of rotation practices (Jackson & Conzales, 1981; Shekhawat & Perembelon 1991; Tusiime, *et al.*, 1998).

Crop rotation and fallow

Rotation with non-hosts as a means of reducing the disease incidence has been used successfully in several crops. The type of crop chosen for rotation varies greatly and depends on the region and the race of the pathogen involved. In India, a two-year rotation with wheat-lupin, wheat-maize or wheat-fallow was very effective in reducing wilt on potatoes. In other instances rotation with finger millet, horsegram, sorghum, wheat, cabbage, carrot, onion and garlic reduced wilt by more than 90% (Shekhawat *et al.*, 1992). Soybean, cowpea, velvet bean, redtop grass, maize and cotton are recommended in two to three year rotation programs to control tobacco wilt in the United States, provided no root-knot nematode infestation is present (Akiew *et al.*, 1993).

In Australia forage sorghum, signal grass and Rhodes grass are often used in the rotation program (Akiew *et al.*, 1993; Arthy & Akiew 1999). A seven-year rotation with signal grass reduced wilt of tobacco greatly, but when tobacco was cultivated for two consecutive years on these fields, wilt incidence increased again to 20 %. Combining the use of resistant or moderately resistant tobacco cultivars with a two to three year rotation sequence of grass fallow appears to be effective (Akiew & Trevorrow, 1994).

The potato strain of *Ralstonia* is reportedly brought under control with relative ease in comparison to other races. In the cool regions of Dorrigo, Australia, a 2½ -year pasture rotation was sufficient to eradicate race 3. Planting the tolerant variety Molinera or maize for several seasons after a six-month fallow eradicated the pathogen in the Peruvian highlands (French, 1994). Rotation with maize, oats or barley resulted in a 50-75% reduction in the incidence of potato wilt in India. Cowpea and cabbage are often included in rotation programs in potato cultivation (Shekhawat *et al.*, 1988).

The success of rotation in comparison to bare fallow programs varies. A rotation program using maize and bean intercrop or sweet-potato cultivation did not reduce the soil inoculum potential in Costa Rica. A five-month bare fallow with application of herbicide for weed control was more successful in reducing the incidence of wilt (Jackson & Gonzales, 1979). In another case, rice cultivation prior to a tomato crop was more successful in reducing wilt than fallow or other crops (French, 1994).

Interestingly, Shekhawat *et al.* (1992) were able to isolate *R. solanacearum* from the root tissue of maize, wheat, paddy rice and bean. Populations of the wilt organism were however not high enough to exude from the root tissue into the soil. The lack of bacterial release could explain the experienced reduction in *Ralstonia* population after a rotation with these crops. Granada & Sequeira (1983a) also found that *R. solanacearum* could infect roots of presumed non-hosts such as maize. Infections remained localized in the roots and bacterial release into the soil was less from these plants than from true host plants. Infection rate of these presumed non-hosts was also lower in that not every individual plant of a given species became infected. These findings indicate why an overall reduction of wilt is experienced when applying rotation. They do however not explain why in some cases rotation is more effective than a bare-fallow treatment.

The level of control achieved with crop rotation varies greatly and is dependent on factors affecting the survival of *Ralstonia* in the absence of a host (Akiew & Trevorrow, 1994). A short-term rotation appears in most instances, especially where race 3 is not involved, not to be effective in either eliminating or controlling wilt effectively.

Soil Amendmends

Several soil amendments have been studied in the hope of reducing wilt incidence. It was generally believed that alkalinity would favor the pathogen and acidity suppress it, but increasing soil acidity by adding potassium sulphate, nitric acid or sulphuric acid did not effect the incidence of wilt noticeably (Kelman, 1953). A range of fertilizers has also been evaluated, including superphosphate, calcium cyanamide and sodium nitrate, without much success. Urea applied at 1000kg/ha did reduce wilt significantly (Kelman 1953). Michel & Mew (1998) amended four soils with urea (200kg N/ha) and CaO (5000kg/ha) and reported that the success in reducing wilt varied with soil type. Differential reduction in wilt was also noted by Chellemi *et al.* (1992) in a trial with composted organic amendments. Site specific soil properties may be responsible for this phenomenon. A 99.9% reduction in wilt of tomato plants in glasshouses was reportedly achieved by an amendment developed by Sun & Huang (1985). The soil was amended with 1% S-H mixture which consisted of 4.4% bagasse, 8.4% rice husks, 4.25% oyster shell powder, 8.25% urea, 1.04% potassium nitrate, 13.16% calcium superphosphate and 60.5% mineral ash.

Other cultural practices

The removal of rogue plants, stubble and diseased plant material forms a vital part in disease management (Graham *et al.*, 1979; Persley, 1986). Such plant material allows the pathogen to survive in the absence of a host crop and permit infection of a subsequent crop. The use of infected farming implements, lack of sanitation after handling diseased material, or moving through infected fields contribute to the risk of transmitting the disease.

Disease avoidance revolves around the date of planting. Planting during the cool season can reduce yield losses, since high temperatures favor the development of bacterial wilt. This method has been used successfully in Australia, Taiwan and India (Shekhawat *et al.*, 1992; Akiew & Trevorrow, 1994).

Biological Control

The concern over toxicity of chemical compounds combined with their relative inefficiency in controlling bacterial wilt has motivated the search for biological agents as part of an integrated management program. Trigalet *et al.* (1994) define biological control in terms of direct microbial antagonism (competition, antibiosis) and indirect microbial antagonism (induced resistance in the host). Later the antagonistic effects of botanicals were included in this category (Trigalet & Urquhart, 1998).

Several bacterial species have been reported as being antagonistic towards *R. solanacearum*, amongst them *Pseudomonas fluorescens, P. glumae, P. cepacia, Bacillus polymyxa,* other *Bacillus* spp. and *Erwinia* spp. (Trigalet *et al.*, 1994; Shekhawat *et al.*, 1992). Avirulent mutants of *R. solanacearum* have also been identified as antagonists. These root colonizers antagonize the pathogen at the root infection site, resulting in reduced and delayed onset of wilt. Isolates of *P. fluorescens* reduced wilt by over 50 % and increased tuber yield. Similarly *Bacillus* spp. isolates have been reported to reduce wilt by almost 90% and tuber rot due to *Ralstonia* by more than 80% (Shekhawat *et al.*, 1992). Biocontrol results obtained in the laboratory with these types of antagonists are often difficult to reproduce in field conditions. Antagonism observed on agar plates could have resulted from a different set of parameters than found under natural circumstances. In field conditions, the biocontrol organisms must compete with other soil microbes and must contend with both biological and physical factors (Trigalet *et al.*, 1994). Since the plant is susceptible to infections for a long period of time, antagonists at the root infection site must be present continuously and without too many fluctuations in their population.

The development of a bio-agent that acts as an endophytic antagonist has the advantage that once it is established in the plant, it can provide continuous protection. The agent should not cause disease and must be able to colonize roots, penetrate xylem vessels and multiply within the vascular tissue (Trigalet *et al.*, 1994). For this reason several studies have been conducted on avirulent mutants of *R. solanacearum* (Trigalet *et al.*, 1998; Smith *et al.*, 1998). Problems that have been experienced with endophytic antagonism include limited systemic spread and population decline, probably due to agglutination by plant lectins or by them being bound to host cell walls (Trigalet *et al.,* 1994).

Several botanicals have been evaluated for their ability to reduce bacterial wilt infections. Terblanche & de Villiers (1998) noted that French Marigolds (*Tagetes patula* L.) not only reduced *Ralstonia* populations in the soil, but also inhibited the development of wilt symptoms on tobacco plants. Two thiophenes were extracted from the roots, which proved inhibitory to *R. solanacearum* in *in vitro* tests. Similarly it was found that sugi bark (*Crytomeria japonica* D.Don), a substrate for horticultural crops in soilless culture, reduced the incidence of wilt on tomato plants (Yu *et al.* 1997). The inhibition was mainly attributed to volatile oils, phenolics and acidic substances. Injecting the volatile oils into rockwool (another soilless substrate) also suppressed the wilt incidence. The main components of the volatile oils were identified as isophyllodecene and ferruginol. Asafoetida (*Ferula foetida*) mixed with tumeric powder (*Curcuma longa*) at ratio 1.5g: 5g: 10 liter water and 1g: 5g: 10 liter water, controlled wilt disease by 70.3 % and 69% respectively (Mazumder, 1998). Soil drenching with a formulation of asafoetida and tumeric reduced the mortality of brinjals due to bacterial wilt, especially if three drenches were performed at 20, 50 and 80 days (Pun & Das, 1997).

Resistant Cultivars

A practical and economic approach to managing bacterial wilt infested soils would be to plant resistant cultivars. According to Prior *et al.* (1998), the term resistance refers to "any measurable plant mechanism able to overcome completely or to limit the development of a pathogen or its effects". Tolerance is defined as "the overall ability for a plant to withstand development of a pathogen without major losses in yield".

Although resistance to bacterial wilt has been documented in some crop species such as tomato, obtaining stable resistance without the occurrence of latent infections is a major challenge. The specificity of the pathogen-host-environment interaction has prevented the success in obtaining a cultivar that is resistant under a range of environmental conditions and to various strains of the pathogen. It appears that the genetic basis for resistance to *R. solanacearum* is different in each resistant species. It would be beneficial to obtain a broadspectrum resistance in which maximum genetic variability for the "wilt-resistance" trait has been combined (Schmiediche, 1986).

Tung *et al.* (1990, 1992a, 1992b) demonstrated that genes affecting adaptation are involved in conferring resistance to bacterial wilt or in conditioning their expression. A heat tolerant parent was found to produce a higher frequency of resistant progenies when combined with an indisputable source of resistance. There are even some heat tolerant clones with no known resistance that could withstand wilt better under very hot conditions, than could clones known to have some specific genes for resistance. Furthermore, resistance appeared to be most stable and highest in clones where the resistance had been established by combining various specific sources of resistance (thereby producing a wide genetic background), or where a source of resistance was combined with a good source of adaptation (Tung *et al.*, 1992b).

DISCUSSION

Bacterial wilt caused by *Ralstonia solanacearum* is one of the most destructive and successful plant pathogens affecting several economically important crops. Although the disease is more common in the tropics, subtropics and the warmer temperate areas, it has also been reported in the cooler regions such as Sweden, Austria and the UK. During the last century, the pathogen has been reported in so many countries that its distribution can now be regarded as world-wide, though the strains encountered vary. The wilt organism is well adapted to survival, being able not only to infect a wide range of hosts but also being capable of remaining viable in the soil for several years. Reliable scientific data on soil survival under different circumstances is scarce. Its ability to survive in more than 450 different species has facilitated its survival even through harsh periods where soil conditions are not congenial for survival in its free state. It's success as a plant pathogen is favoured in the ready way it disseminates. Besides transmission through infected soil and infected plant material (often showing no symptoms), it can spread by mechanical contacts, contaminated water and in case of banana wilt even by insects.

Control of bacterial wilt lies in an integrated approach. Good agronomic practices such as using disease-free soils, disease-free and uncut planting material, effective weed and nematode control, incorporating good crop rotation systems and fallowing can assist greatly in reducing the incidence of wilt. Soil amendments and chemical control can be beneficial under specific circumstances. The potential of biological control as part of the management system is vast and the research input into this area has increased dramatically. Another important strategy in managing bacterial wilt infested soils lies in the use of resistant varieties. Although resistance in varieties have been globally reported over the years, most of these genotypes tend to be tolerant to bacterial populations, rather than being immune and often transmitted the disease to the progeny tubers without necessarily causing visual symptoms. Planting of such latently infected tubers could lead to infestation of previously disease-free fields or result in yield losses. Another concern for plant breeders is that the expression of resistance is strongly affected by environmental factors.

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