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Plant Pathology
REVIEW ARTICLE

Ralstonia solanacearum: A threat to potato production in Bangladesh

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ABSTRACT

Potato is one of the most important vegetable crops in Bangladesh which made it 7th producer in the world. This vegetable crop is affected by one of the most notorious soil borne pathogen, *Ralstonia solanacearum* and caused wilt symptoms in plant and brown rot in potato tuber throughout the world. The pathogen entered into the plants through different natural opening and wounds and is easily disseminated via infected biological material (seeds, vegetatively propagative materials, plants etc.), soil, contaminated irrigation water, surface water, farm equipment etc. and could survive for many years in association with alternate hosts. The bacteria is a quarantine organism being economically a serious problem for potatoes and other major crops in many tropical, subtropical and warmer areas of the world. It has an unusually wide host range including economically important crops (potato, tomato, tobacco, banana, ginger, geranium etc.) and weeds. The disease appeared as rapid and fatal wilting symptoms in the potato plants and vascular browning and/or rotting on tubers. The pathogen can be detected through the isolation of the bacteria on semi-selective TZC and/or selective SMSA (modified) media and through different biochemical tests and/or molecular test. It is a Gram-negative type bacterium, subdivided into five races based on host range and race 3 biovar III is reported in Bangladesh. Direct yield losses caused by *R. solanacearum* may vary 33 to 90% depending on the different factors such as cultivar, climate, soil type, cropping pattern, strain of the bacteria etc. Due to the latent infection of tuber by this organism, Bangladesh had been facing a temporary embargo on potato export which was imposed by Russia during 2014-15. But, latency and some other survival strategy of the pathogen created much more problems with disease detection, control and dispersal. The biological features of this pathogen makes it unusually successful against the traditional management practices. Therefore, the review focused on the biological abilities of *R. solanacearum* in relation to dispersal, survival and influences which might be important in designing effective management against the pathogen.

Keywords: *Ralstonia solanacearum*, potato, economic impact, biology, strategy, dispersal, survival, influence

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1 Introduction

Potato (*Solanum tuberosum* L.) is a tuber crop belongs to the family Solanaceae. It is the 4th important crop after wheat, rice and maize in the world, and Bangladesh is the 7th producer in the world for more than 85 lakh tons of potato production (FAOSTAT, 2015). The area of potato production is still in increasing from 4.44 to 4.62 lakh hectares in Bangladesh (FAOSTAT, 2015). It is nutritionally considered a super vegetable as well as a versatile food item and it produces more carbohydrates per unit amount than either rice or wheat. However, the yield of potato is quite low in the country as compared to the major potato growing countries like- Ireland and India (FAOSTAT, 2015). The reasons behind the lower yield of potato includes lower soil fertility, inadequate supply of certified seeds, use of low yielding varieties, different pests and diseases etc. Among them soil borne diseases are considered to cause a yield loss of as much as 10–20% annually. *Ralstonia solanacearum* formerly called *Pseudomonas solanacearum* (Yabuuchi et al., 1995) is the most destructive soil-borne pathogen (Yuliar et al., 2015) that affects potatoes in temperate, subtropical and tropical regions throughout the world by causing bacterial wilt or brown rot disease (CABI, 2017a; Champoiseau et al., 2009). It is a vascular disease, which is fatal in infected plant and has been ranked as one of the most important bacterial plant pathogens identified to date, commonly known as bacterial wilt (in case of infected plant) and brown rot (in case of infected tubers).

Geographic distributions of the pathogen are highly influenced by different factors like- availability and abundance of the host(s) and suitability of the climatic conditions etc. Its world-wide distribution, destructive nature and ability to host asymptomatically over 450 plant species (Prior et al., 1998) has resulted it to be ranked as the most important bacterial plant pathogen (Kelman, 1998). The pathogen is worldwide distributed in major host crops like- potato, tomato, banana, tobacco etc. with many weeds as alternate hosts and therefore, it can increase the potential to build up inoculum which may lead to induce a destructive economic impact (Kelman, 1998). Yield losses due to the disease varied from 33 to 90% in the potato in different potato growing areas of the world (Elphinstone, 2005). The total value of Egyptian potato exports fell from a peak of USD 102.12 million in 1995 to USD 7.7 million in 2000 mainly due to brown rot quarantine, imposed by the European Union (EU) (Kabeil et al., 2008). In India, this disease causes 50% crop loss in potato in a regular manner (Mukherjee and Dasgupta, 1989) and up to 75% losses as reported in some areas of Karnataka (Gadewar et al., 1991). Reports from Bangladesh quote some regions as having more than 30% of potato crops affected by *R. solanacearum*, with over 14% reduction in yield (Elphinstone, 2005). Nonetheless, Russia imposed a temporary ban on the entry of the potatoes from Bangladesh in May 2015 on food safety grounds after detecting this organism (Parvez, 2017).

The disease appears as rapid and fatal wilting symptoms in host plants (Yuliar et al., 2015) and infected potato plants die rapidly within 3-4 days. Older plants first show wilting of the young leaves, or partial one sided wilting of the plant and stunting, and finally the plants wilt permanently and die. The disease can be easily detected in the wilted plant stem by streaming the milky white oozes within clear water (Allen and French, 2001). The bacterial wilt is primarily tuber-borne, but infested soil also serves as a source of infection. Tuber may carry the pathogen in vascular tissues, on the tuber surface and within lenticels (Ghosh and Mandal, 2009; Martin and R, 1985; EPPO, 2004). *R. solanacearum* is gram-negative, rod-shaped bacterium measuring 0.5-0.7 $\times$ 1.5-2.0 μm in size. It grows well at 28 to 32 °C in aerobic conditions (Hayward, 1991). Disease severity caused by the pathogen mostly found to increase while associated with root nematodes. The combined pathogenic effect of *R. solanacearum* and *Meloidogyne javanica* was greater than the independent effects of either (Sitaramaiah and Sinha, 1984). The incidence of bacterial wilt is far less in the whole tubers than in cut tuber planting. The pathogen created much more problems in controlling with chemicals, which was nearly impossible to apply; antibiotics showed hardly any effect (Murakoshi and M, 1984; Farag et al., 1982); and adaptability problems of resistant varieties occurred due to the strain diversity and latent infection of the pathogen. However, biocontrol agents showed some effectiveness in the controlled condition which is still in its infancy (CABI, 2017a). Therefore, it is listed as a quarantine organism (CABI, 2017a). Hardships were observed in managing the pathogen through traditional management practices as it possesses some especial features viz. their abilities to- grow endophytically (tending to grow inward into tissues), survive in the soil especially in the deeper layers, travel along water, have VBNC (viable but non-culturable) and/or PC (phenotypic conversion) phenomena, and their relationship with weeds as asymptomatic alternate hosts (Wang and Lin, 2005) allow them to survive long in the environment and threatens the production and export businesses of potato.

Considering the facts discussed, without having prior knowledge on those behavioral pattern of the pathogen, it seems unrealistic to design an effective management strategy against the pathogen. Therefore, the review paper focuses on the deceitful biological feature(s) of *R. solanacearum* in relation to dispersal, survival and influences.

2 Description of the Pathogen

2.1 Identity

The bacteria were first named as *Bacillus solanacearum*. After several revisions, it was called for many years *Pseudomonas solanacearum*. The latest revision has settled on the name *Ralstonia solanacearum* (CABI, 2017a).

2.2 Taxonomic tree

Domain: Bacteria
Phylum: Proteobacteria
Class: Betaproteobacteria
Order: Burkholderiales
Family: Ralstoniaceae
Genus: *Ralstonia*
Species: *Ralstonia solanacearum*

2.3 Taxonomy

The bacterium is described as a non-spore forming (spores in bacteria terminology are survival structures rather than units of reproduction as in fungi), Gram-negative, rod-shaped bacterium 0.5-0.7 × 1.5-2.0 μm in size which is nitrate-reducing, ammonia-forming and grows well in aerobic conditions (Hayward, 1991). It's 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). Populations within this genus and species can be further divided into races and biovars based on differing host ranges, biochemical properties, and serological reactions. The shape and size of the causal organism was first described as a small rod with one polar flagellum with rounded ends. The size of the bacterium vary according to different growing conditions (Kelman, 1953). Bacteria isolated from infected tissues were appeared as very short rods (0.3-0.6 × 0.4-1.2μ) and those taken from young broths or cultures tend to be longer (ranging from 0.4-0.6 × 1.0-1.8μ), whereas those from old cultures have a short coccus-like form (Kelman, 1953). Yabuuchi et al. (1995) reclassified *Burkholderia solanacearum* as *Ralstonia solanacearum* which was based on the studies involving phenotypic characterization, rRNA-DNA hybridization, phylogenic analysis of 16SrDNA nucleotide sequences, and analysis of cellular lipids and fatty acids.

2.4 Subspecific classification

Several attempts have been made to find a suitable classification system for the isolates of *R. solanacearum* as they often differ in host range, geographical distribution, pathogenicity, epidemiology and physiological properties in the form of race(s), biovar(s) and/or phylotype(s).

2.5 Races and biovars

Sub-specific classification is achieved into 5 races by determining the hosts that are primarily affected. Nishat et al. (2015) reported the variation in *R. solanacearum* isolates of potato which was observed among different growing areas of Bangladesh. It was showed that the isolates were belonged to race 3 biovar III. According to Hayward (1994), five biovars can be identified based on their ability to utilize three hexose alcohols, namely mannitol, sorbitol, dulcitol; and to produce acids from the three disaccharides, lactose, maltose and cellobiose. *R. solanacearum* is considered a 'species complex', due to significant variation within the group (Fegan and Prior, 2005). It is historically subdivided into five races based loosely on host range and, five biovars based on their ability to acidify a panel of 5 to 8 carbohydrate substrates.

2.6 Phylotypes

A phylogenetically meaningful system based on DNA sequence analysis Fegan and Prior (2005); Prior and Fegan (2005) have classified *R. solanacearum* into four major genetic groups called phylotypes that reflect the geographical origin and ancestral relationships of the strains.

Box 1. Geographical origin and ancestral relationships of the strains of *R. solanacearum*

Phylotype I	Asia
Phylotype II	America
Phylotype III	Africa
Phylotype IV	Indonesia

Phylotypes are further subdivided into sequevars based on the sequence of the endoglucanase (egl) gene (Fegan and Prior, 2005; Prior and Fegan, 2005). Within each of the races or biovars there are numerous subtypes that can be associated with certain geographical regions (He, 1983) and this, together with *R. solanacearum* enjoys a world-wide distribution.

3 Distribution

Bacterial wilt affects crops of economic importance in almost all the tropical, subtropical and warmer temperate regions of the world. Biovar 2 presumed to have originated in South America (presumed site of origin of the potato) now has a wide spread distribution which 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, 1991). Biovars 1 and 2

Table 1. Characteristics of races and their relationship to biovars of *R. solanacearum*

Race	Host range	Geographic distribution	Biovar
1.	Wide (tobacco, tomato, solanaceous and nonsolanaceous weeds, diploid bananas, groundnut, potato, pepper, eggplant, olive, ginger, strawberry, geranium, Eucalyptus, other plants)	Asia, Australia, America, Bangladesh	3, 4, 1
2.	Triploid bananas, other *Musa* spp.	Caribbean, Brazil, Philippines	1
3.	Potato and tomato	Worldwide except US and Canada	2 or 2A[†]
4.	Ginger, Unknown	Australia, China, Hawaii, India, Japan, Mauritius, South Asia, India	4, 3
5.	Mulberry tree	China	5

[†] Typical race 3 strains are sometimes referred to as biovar 2A. New race 3 strains from the Amazon basin have been placed in a new biovar, designed as 2T or N2 (their relation to races is unclear). [‡] Source: Champoiseau (2008)

Table 2. Biological strategies of *R. solanacearum* contributing to management difficulties through traditional management practices

Biol. feature	Type	Condition	References
Mode of entry	i) Wounds in the root system by nematodes or other	Especially the wounds created by root-knot nematodes (*Meloidogyne* spp.); and the points of secondary root emergence.	Johnson and Schaal (1952); Kelman (1953, 1965)
	ii) Unwounded root infection	When relatively large numbers of bacteria are available it is also possible.	Kelman (1965)
Inoculum sources and dispersal	i) Infected plant materials (seeds, plant, tuber etc.)	When bacterial masses adhere to soil particles, it enhance the survival of the pathogen, tubers can carry the bacteria in three manners, namely externally on tuber surfaces, in lenticels and in the vascular tissues; during storage, bacterial populations reached a non-detectable limit within 30-60 days at 4 °C and 60-90 days at room temperature.	Shekhawat et al. (1992); Sunaina et al. (1989)
	ii) Infested soil, irrigation water, equipments etc.	Dissemination through infested water, mechanical dissemination by infested equipment both occurs in the field during irrigation and sorting of seed tubers; dissemination by chewing insects on potato (colorado potato beetle, Leptinotarsa declimlineata Say.) etc. have been reported.	Kelman (1953)
	iii) Infected host debris, alternate hosts and weeds with no visible symptom	Infected host debris is an important carrier as short-term shelter in soil which allows the survival of *R. solanacearum* between the growing seasons and serves as a transmission agent; the plant parts (eg. tubers) with no visible symptom ensures the uninterrupted dispersal of the pathogen; more than 450 species of weeds serves as symptomless alternate hosts of the pathogen.	Graham (1979); Shekhawat et al. (1992); Hayward (1991); Prior et al. (1998)
Survival	'PC'–(phenotypic conversion) phenomena	It has the ability to change from virulent to avirulent state termed as 'phenotypic conversion' (PC) by reduced production of extracellular proteins and polysaccharides; which keeps them to remain withstand and viable for a very long periods like 2 to 10 years; and can survive in various types of soils worldwide.	Poussier et al. (2003); Nesmith (1985); Denny et al. (1994)
Latency	Inoculum level and certain environmental stresses.	Latency shows when inoculum level decrease and certain environmental stresses occurs. Such as exposure to low temperatures, anaerobiosis etc. makes them symptomless and undetectable [termed as viable but non culturable (VBNC) state]; even occasionally, tubers produced on the symptomatic plants did not show infection rather diseased tubers were found on plants with no visible symptom.	Devi et al. (1982); Shekhawat and Perombelon (1991); Eddins (1941)
Favorable environment	Temperature	It cannot survive at >40 °C, becomes severe between 35∼24 °C, produces no visible symptom at <16 °C; and could survive long in lower temperature even at 4 °C, which makes it capable of dispersal and survival for long period.	Ciampi and Sequeira (1980); Seneviratne (1988); Granada and Sequeira (1983)
	Moisture and irrigation	It can survive long in the environment without doing much destruction, but moisture in the poor soil influenced the devastation of crop by the pathogen; could be disseminated with the irrigation water; and could make the disease level increased and affected synergically while in the optimum temperature.	Shekhawat et al. (1992)

are predominant in the Americas. 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 Philippines have all of biovars 1-4 and here as elsewhere in Asia, biovar 3 predominates in the lowland regions (Hayward, 1991; CABI, 2017a). However, report from Ahmed et al. (2013) and Nishat et al. (2015) showed that the *R. solanacearum* isolates causing bacterial wilt of potato in Bangladesh were belonging to Biovar III and Race 3.

4 Host Range

R. solanacearum is known to have a very extensive host range including not only economically important crop plants such as potato, tomato, tobacco and banana, but also ornamental plants, trees and weeds. Species from more than 44 plant families have been identified by Hayward (1991) and more hosts are being recognised and described. Some of the reports included onion, *Allium cepa* (Girard et al., 1992); custard apple, *Annona* spp., (Mayers and Hutton, 1987); florist geranium, *Pelagornium hortorum* (Strider, 1981); strawberry, *Fragaria* spp., (Hsu, 1991) and radish, *Raphanus sativus* L., (Hsu, 1991), etc. Cassava is cultivated in many countries where 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, 1991). *R. solanacearum*, biovar 3, has also been noted on cashew in Indonesia and the Alexandra palm in Queensland, Australia (Hayward, 1991). An alternative theory is that the pathogen hosts such crops may only 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, 1991, 1994).

All hosts of *R. solanacearum* do not necessarily express symptoms and can serve as symptomless carriers. 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) indicated that *R. solanacearum* can even survive symptomless in roots of weed-hosts and in plants considered to be non-hosts in more than 450 species which have been reported as hosts or symptomless carriers (Prior et al., 1998) of certain strains of *R. solanacearum*.

5 Symptoms and diagnosis

Symptoms *R. solanacearum* causes potato infection in two ways- i) premature wilting and plant death symptoms namely 'bacterial wilt' leading to total loss of yield; and ii) tuber rotting symptoms namely 'brown rot' occurs in the transit or storage. On potato plants, symptoms due to the blocking of the vessels (Kelman, 1953) caused by the bacteria is the major cause of wilting. The symptom starts with slight wilting of the leaves at the ends of the branches during the heating of the day which recovers at night; eventually, plants fail to recover which is soon followed by total wilting and if the base stem of the affected plant is cut transversely, the bacterial oozes comes out as milky white threads when kept in a beaker with water (Fig. 1). Such threads are not formed by other bacterial pathogens of potato. In advanced stage, epinasty of the petioles may occur and die (Fig. 1). However, under cool growing conditions, wilting and other foliar symptoms may not occur.

Symptoms in tubers, mostly occurs as vascular browning and rot and pitted lesions (Shekhawat et al., 1992). In vascular rot, the vascular tissues looks like a water soaked circle, which subsequently may turn brown. A cross section will show a brown vascular bundle ring. As the tuber is pressed, slimy drops will be out of the ring. The lesions on tuber are produced due to infection through lenticels (skin pores) (Ghosh and Mandal, 2009; Martin and R, 1985; EPPO, 2004). If potato tubers are formed in the infected plants those will possibly show the symptoms (Fig. 1). On tubers, external symptoms may or may not be visible, depending on the state of disease development. *R. solanacearum* can be distinguished by the bacterial ooze that often emerges from the eyes and stolon-end attachment of infected tubers. Soil may adhere to the tubers at the eyes (Database, 2004).

Detection *R. solanacearum* can be identified from either symptomatic or asymptomatic plants and from water or soil samples by means of several microbiological and molecular methods (Priou et al., 2006; Schaad et al., 2001; Weller et al., 2000). Screening tests can facilitate early detection of *R. solanacearum* in plants or contaminated soil and water samples, but they cannot be used to identify the race or biovar. These screening tests include bacterial streaming, plating on a semi-selective medium such as TZC medium etc. (Elphinstone et al., 1996), polymerase chain reaction (PCR) with specific primers, and pathogenicity tests using susceptible hosts such as tomato seedlings (Elphinstone et al., 1996; Schaad et al., 2001; Weller et al., 2000). Commercially-available immunostrips can be used for rapid detection of *R. solanacearum* in the field or lab. The biovar test is a biochemical assay based on the differential ability of the pathogen strains to produce acid from a panel of disaccharides and sugar alcohols, requires specialized media and may take days to several weeks. Strains of *R. solanacearum* can be sub-classified into phylotypes and then into sequevars using PCR and

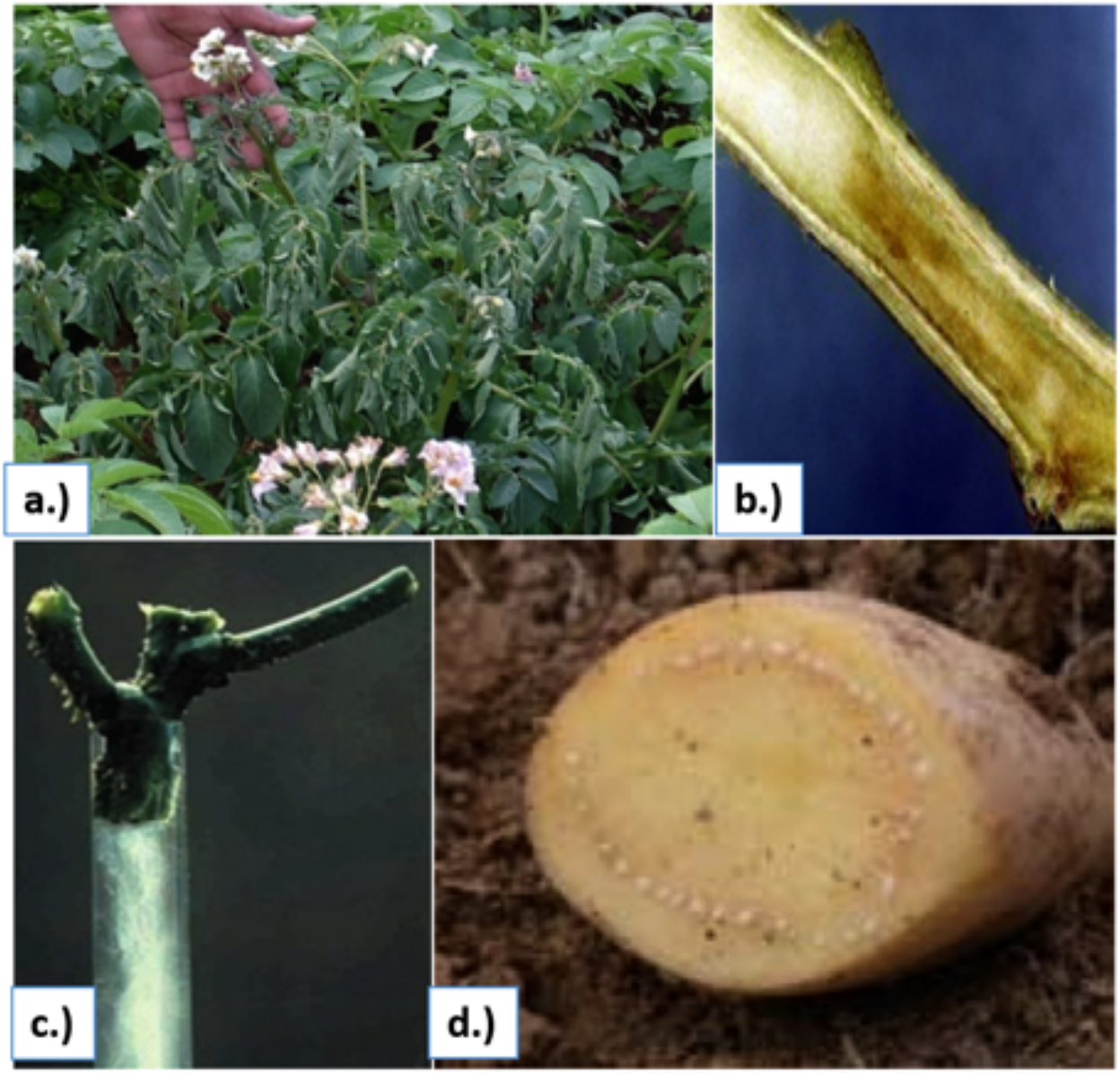

Figure 1. a) Symptoms of bacterial wilt on potatoe plants; b) bacterial streaming from the stem section; c) vascular discolouration on the infected stem; and d) bacterial ooze on vascular tissues of the tuber (Champoiseau et al., 2009)

gene sequence analysis (Champoiseau et al., 2009). Many standard methods for detection (of latent infection), identification and preparation of media for *R. solanacearum*, used in official EU testing schemes, can be found in (EU, 1998; Lelliott and Stead, 1987; Database, 1990). Detection of latent infection is by performing an immuno-fluorescence test and/or selective plating on SMSA medium eventually combined with optional PCR assays, ELISA or fluorescent in situ hybridization tests which can be performed for added sensitivity (Database, 2004). A combination of at least two different complementary tests is required to unambiguously identify the species and biovar. Unequivocal identification of R3bv2 must rely on at least two distinct methods of screening and biovar test (Champoiseau et al., 2009). SMSA medium as modified by Elphinstone et al. (1996) has been used successfully in Europe for latent infection (Elphinstone et al., 1998). Isolation from symptomatic material can easily be performed using Kelman's tetrazolium medium. In some cases when secondary infections are present and isolation on selective media is necessary. A presumptive test in the field can be the water streaming test as described under disease symptoms or a serological agglutination test using a field kit in the form of a lateral flow device (Danks and Barker, 2000).

6 Importance

R. solanacearum is the most serious pathogen of potato plants in tropical regions and can cause serious losses in temperate regions. A review of the older literature can be found in (Kelman, 1953). It is responsible for an estimated $1 billion US in losses each year Elphinstone (2005) and globally, the disease has been estimpated to affect about 1.7 million hectares of potatoes in approximately 80 countries, with global damage estimate of over USD 950 million per annum thereby contributing to yield losses in potatoes of about 75% at medium to high altitudes (1500-2800 m) (Champoiseau et al., 2009). Seed borne wilt or latent infection in potato has often been resulted in severe out breaks of bacterial wilt (French, 1985). Yield losses continue during storage and transit due to rotting and decay leading to even more revenue losses. The disease has been estimated to affect three million farm families, which accounts for about 1.5 million ha) in around 80 countries. In addition to causing yield losses in field crops, management efforts for prevention, eradication, and control of *R. solanacearum* are extremely costly, which contribute heavily to economic losses (IPDN, 2014).

Different yield loss status has been reported in several countries (Fig. 2). In Bolivia, potato yield loss at harvest ranged from 30- 90% and losses during storage were as high as 98% (IPDN, 2014; Coelho and Nutter, 2005). In Nepal, tuber rotting occurred in an average of 10% of stored potato, with a maximum of 50%. Crop losses in small farms in the Nepalese hills were up to 100%, mainly due to poor cultural practices, such as keeping seed from infected crop (IPDN, 2014; Elphinstone, 2005). Complete crop losses in small holdings in Nepal resulted from poor cultural practices including using seed from affected crops for subsequent plantings (CABI, 2017b). In Venezuela, in the period 1992-1996, *R. solanacearum* was found in most localities between 1100 and 3000 m above sea level, but was not found in localities at altitudes greater than 3000 m (CABI, 2017b). The potato production and yield losses due to bacterial wilt as high as 100 per cent have been reported in parts of tropical Africa (Shivani, 2016). In Kenya, the potato industry is threatened by bacterial wilt (BW) because soils in most production areas are infested with the wilt causing bacterium and over 50% yield losses have been reported (IPDN, 2014). The farmers reported experiencing yield losses ranging from 5% to 80% due to bacterial wilt. According to some recent studies, the disease is found in all the potato growing areas of Kenya and the country is affecting 77% of potato farms which had been introduced with tuber seeds imported from Europe (Kaguongo et al., 2010). Various reports from Kenya have indicated that there was an increase in the incidence of brown rot of potato due to the spread and build-up of the disease in the majority of the potato growing zones (IPDN, 2014; Ajanga, 1993; Barton et al., 1997; Ateka et al., 2001). Potato yield losses in Uganda estimated about 30% (IPDN, 2014; Alacho and Akimanz, 1993), with more severe losses being 100% (IPDN, 2014). In Burundi, losses of 64.1% were reported in seed potato IPDN (2014). Heavy losses of potato due to this disease were reported from the South Atlantic and Gulf Coast states of the USA (Kelman, 1953). Extensive losses of potato were reported in Greece (Zachos, 1957). In Israel, losses were heavier in the spring potato crop than the autumn crop, because of the higher growing temperatures in spring (Volcani and Palti, 1960). Kabeil et al. (2008) reported that potatoes were one of the largest exported crops in Egypt. Yet, the total value of Egyptian potato exports fell from a peak value of US$ 102.12 million in 1995 to $US 7.7 million in 2000 mainly due to this organism related quarantine restrictions imposed by the European Union (EU) which used to account for about 70-90% of Egyptian potato exports and it represented a drop from approximately 419,000 metric tons to 48,500 tons. Multiplication by cutting seed potato seriously increases the risk of high losses. Cut seed potato increased disease incidence by 250% and reduced yield by 40% (CABI, 2017b). In India, a yield loss study with one cultivar of tomato showed 10-100% mortality of plants and 0-91% yield loss (Elphinstone, 2005). In India, this disease causes 50% crop loss in potato in a regular manner and up to 75% losses as reported in some areas of

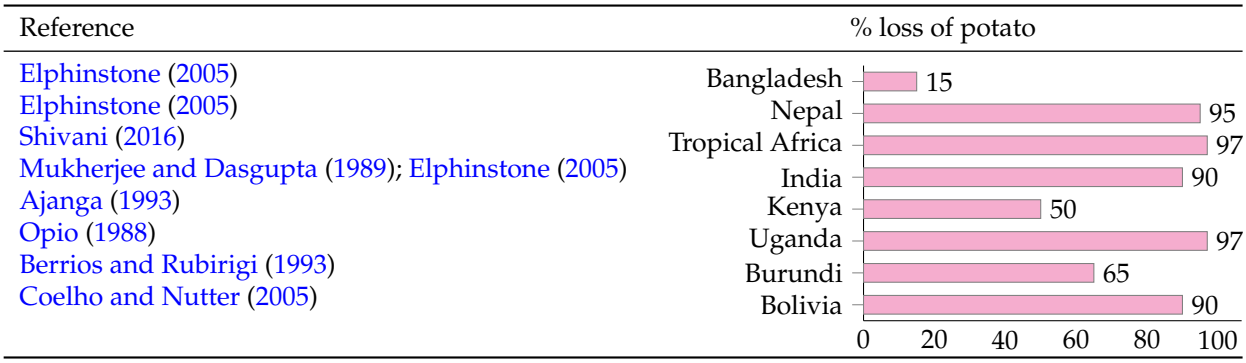

Figure 2. Loss status of potato in different countries caused by *R. solanacearum*

Karnataka (Mukherjee and Dasgupta, 1989). Reports from Bangladesh quote some regions as having more than 30% of potato crops affected by *R. solanacearum*, with over 14% reduction in yield (Elphinstone, 2005).

In Bangladesh, *R. solanacearum* incidence (Fig. 3) was recorded 9.07% in Jamalpur area, 19.98% in Nilphamari area and 22.65% in Munshigonj area. During the fiscal year (2014-2015), exports of the produced potato had been hindered because of the embargo imposed by Russia due to the infection of the pathogen. So, the potato growers and businessmen of Bangladesh had experienced much problems on the disease especially in case of export to other countries like- Malaysia, Indonesia, Sri Lanka, Thailand, Hong Kong, Vietnam, Maldives etc. (Chakraborty and Roy, 2016).

6.1 Difficulties in effective control

The biological features (Table 2) of the pathogen successfully created difficulties in effective management through traditional management practices. Soil fumigants showed either slight or no effects (Murakoshi and M, 1984) except chloropicrin among others [methyl bromide, DD MENCS (a mixture of methyl isothiocyanate, dichloropropane and dichloropropene), and metham] (Enfinger, 1979). However, one hundred years ago, chloropicrin was used during World War I as tear gas and 'vomiting gas' and scientists have concluded that chronic exposure may results in 'very high' cancer risks (Froines, 2010) and those are prohibited in some countries due to the risks posed to pesticide operators and aquatic organisms, birds, and bees. Chemical control is nearly impossible to apply and antibiotics such as streptomycin, ampicillin, tetracycline and penicillin showed hardly any effect (Farag et al., 1982); in fact, streptomycin application increased the incidence of bacterial wilt in Egypt (Farag et al., 1986). Biological control has been investigated, but is still in its infancy. Although moderate to highly resistant potato varieties have been released, but the race and strain diversity of the pathogen and high frequency of latent infection in tubers, are still in list of major problems. Besides, the resistant cultivars to be adapted in different agro-climatic zones has been faced difficulties due to different strains of the pathogen. Another concern lies in the expression of resistance for plant breeders which is strongly affected by environmental factors (Database, 2017a).

6.2 Risk Category of *R. solanacearum*

Under those circumstances, *R. solanacearum* is listed as a regulatory pathogen in A2 group (the group of quarantine pest which can be present in the location but cannot be widely distributed there and has to be officially controlled) quarantine organism (Database, 2017b) and is listed by Asia and Pacific Plant Protection Commission (APPPC) and International Association of Professional Security Consultants (IAPSC). The occurrence of different races and strains of the pathogen with varying virulence under different environmental conditions presents a serious danger to European and Mediterranean potato and tomato production. Therefore, the absence of the bacterium is an important consideration for countries exporting seed potatoes (CABI, 2017a).

7 Management difficulties

As a soil-borne pathogen, it was found to be in various types of soils worldwide. Bacterial wilt was found to survive in some fallow soil for periods of 2 to 10 years (Nesmith, 1985). Buddenhagen and Kelman (1964) reported that under certain conditions, *R. solanacearum* colonies spontaneously undergo a change from fluidal to afluidal form of morphology, linked to a great reduction in disease-inducing capacity of these cells. In this form the bacteria can conserve energy and cellular resources thereby increases its survival. When suitable host is available, the bacteria multiply. Once sufficient cell density is obtained the extracellular virulence factors are produced they become virulent and infectious. The survival of *R. solanacearum* in soil was affected by several factors such as initial inoculum concentration, whether the

land is fallow or cropped to a non-susceptible host, as well as the biological, chemical and physical properties of the soil etc. (Moffett et al., 1983). The temperature, moisture and oxygen status of the soil is further factors that influence the longevity of the pathogen. This deceitful biological features (Table 2) of the pathogen that has discussed below, contributes to management difficulties through traditional management practices and causes the pathogen unusually successful in infecting the suitable hosts.

7.1 Biology in relation to disease development and survival

Mode of entry *R. solanacearum* usually enters its hosts via wounds in the root system (Johnson and Schaal, 1952). Cultural practices such as interplanting prior to harvest often lead to increased root damage (Kelman, 1953). The role of nematodes, especially *Meloidogyne* spp. in providing the wound for bacterial entry has been mentioned by several authors (Kelman, 1953; Buddenhagen and Kelman, 1964; Hayward, 1991; Shekhawat et al., 1992). Nematodes may also modify the host tissue by making it more suitable for bacterial colonisation (Hayward, 1991). Wilt resistant cultivars have been noted to become susceptible when attacked by nematodes. 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 potato tubers. Even infection of aerial parts via wounds has been reported under field conditions (Hayward, 1991). 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 (Hayward, 1991).

Histopathology (changes in tissue due to infection) of the host Wallis and Truter (1978) studied the histopathology of tomato plants infected with *R. solanacearum*, on the spread of the pathogen within the host and the progressive destruction of its vascular tissue. In the study, it was 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 and no bacteria was 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. During the 3rd day ie. 72 hours after inoculation 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. At the 6th day ie. 144 hours after the inoculation the bacteria in the root vessel had reached a large number. Tissue collapse was observed at the 7th day ie. after 168 hours and various plugging substances were noted in the vessels and cells of diseased plants were also observed. Complete wilting of all test plants occurred at the 8th day ie. about 192 hours after inoculation (Wallis and Truter, 1978).

Sources of inoculum and modes of dispersal Two major sources of inoculum exist, namely infected planting materials (seeds/ tubers/ vegetatively propagative materials) 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. 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 of the pathogens' transmission is complicated. Tubers can carry the bacteria in three manners, namely externally on tuber surfaces, in lenticels and in the vascular tissues (Martin and R, 1985; Shekhawat et al., 1992; EPPO, 2004; Ghosh and Mandal, 2009). Although surface carried bacteria can be eliminated by chemical treatments, internal infections remain a threat. A study 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 (Sunaina et al., 1989). Irrigation water, mechanical dissemination by infested equipment, sometimes chewing insects on potato (colorado potato beetle, *Leptinotarsa declimlineata* Say.) etc. also have been reported (Kelman, 1953). Infected host debris is an important short-term shelter for *R. solanacearum* in soil (Graham, 1979) allowing survival between growing seasons. It also serves as a transmission agent. This is especially 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 and Kelman, 1964; Martin, 1981).

Presence in the virgin soils The occurrence of *R. solanacearum* in newly cleared lands or virgin soils has been cited in literature (Kelman, 1953; Sequeira and Averre, 1961; Martin, 1981) and has been attributed to the presence of wild hosts in the indigenous flora. Martin (1981) found that biovar 1 (race 1) and biovar

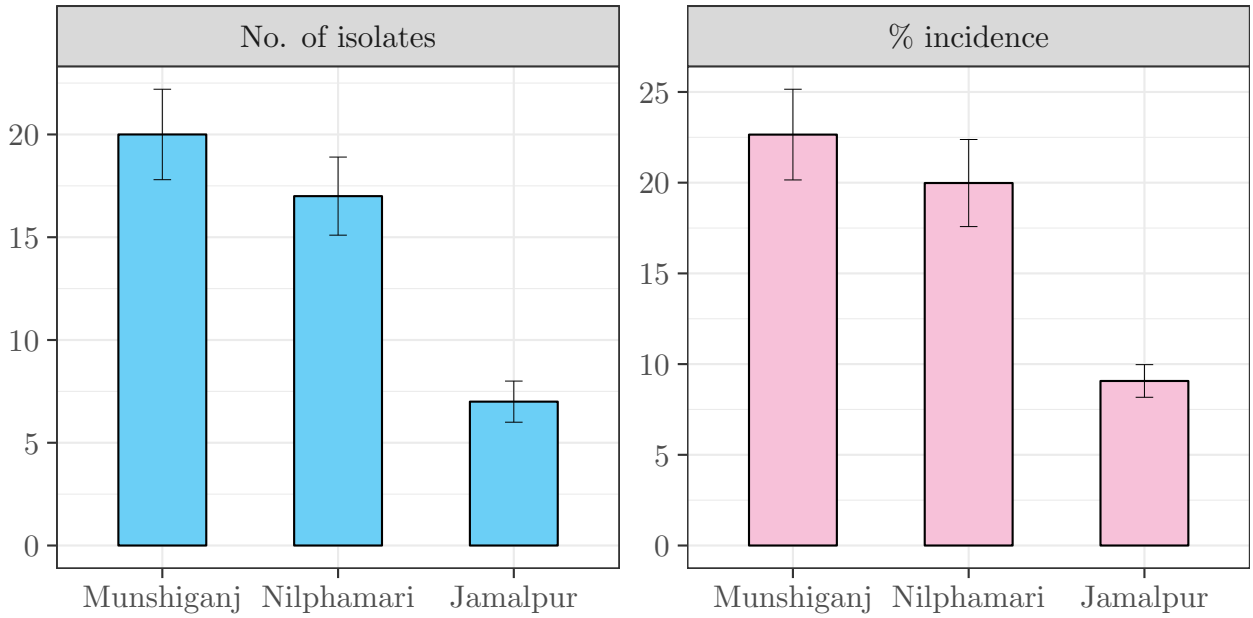

Figure 3. Incidence (%) of *R. solanacearum* recorded in different potato growing areas of Bangladesh (redrawn from Ahmed et al. (2013)

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 suggested that those strains were indigenous to the region.

Latency A primary factor contributing to the persistence of the bacteria in the potato production and export business is that this disease can exist as symptomless/ latent infections. A number of variables can determine whether or not a bacterial wilt infection will be asymptomatic. Inoculum dosages at the time of infection and environmental conditions mainly affect expression of disease symptoms (Devi et al., 1982). Additionally, the frequency of disease expression in a field may be so low that its detection in seed potato fields during field inspections is extremely difficult, if possible at all. This bacterium is known to enter a viable but not culturable (VBNC) state under some circumstances, such as exposure to low temperatures, anaerobic conditions (Shekhawat and Perombelon, 1991); this may complicate culture-based diagnostic methods (van Elsas et al., 2001). Temperature has an influence on the symptom expression of the disease. In general it may be accepted that symptoms are more intensely expressed with an increased temperature. At lower temperatures a larger extent of latent infection occurs, whilst plants and tubers show no visual symptoms (Shekhawat et al., 1992). That means, tubers look like no visual symptoms of pathogen but present in reality, which could be a potential source of disease at the next season. Even occasionally, tubers produced on the symptomatic plants did not show infection whereas, diseased tubers were found on plants with no visible symptom (Eddins, 1941). This tendency holds great danger for the seed potato production and export.

7.2 Biology in relation to influences

Temperature Temperature requirements for optimal growth are known to differ for the various strains. In case of plant infection race3 biovar2 (R3bv2) is most severe between 24 and 35 °C and decreases in aggressiveness when temperature exceeds 35 °C or fall below 16 °C. Active disease at temperatures below 16 °C is rare (Ciampi and Sequeira, 1980). R3bv2 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). Shekhawat and 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; and population decline and loss of virulence of both races was slowest between 10-30 °C. At low temperature of 5 °C, population decline was the same for both races, reaching undetectable levels within 12 weeks. Granada and Sequeira (1983) however reported that soil kept in plastic bags at 4 °C maintained bacterial wilt populations for 673 days which indicated that long-term survival in deeper soil at low temperatures is possible. However, the pathogen was able to survive in some soil samples

kept at 40 °C for seven days, but not in those kept at 43 °C (Seneviratne, 1988).

Soil moisture Moisture is an important factor of dispersal this pathogen. It may survive in the environment for long without creating much destruction, but it is moisture in the poor soil which influenced the devastation of crop by the pathogen. 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 noted the relationship between soil moisture and bacterial wilt from an early date and attributed the disease with higher level of moisture (Shekhawat and Perombelon, 1991). According to Shekhawat et al. (1992) soil moisture and temperature have a synergistic effect on disease development, which high temperatures or high soil moisture alone will not induce. However, it is very sensitive to desiccation (Champoiseau, 2008).

Soil health and soil organic matter Moffett et al. (1983) noted a greater population decline of *R. solanacearum* in the clay loam than in clay or sandy loam at higher pressure potentials while in a study on the effect of moisture and soil type on the survival of *R. solanacearum*. In the study, the increased decline of population was attributed to the higher microbial activity associated with the soil organic matter which was due to increased competition for nutrition with other soil microbes and exposure to increased microbiostasis. Tanaka (1976) reported the relation between organic matter, microbes and Ralstonia populations. In case of higher levels of organic matter and microbial activity in surface soil, the population of *R. solanacearum* decline was faster than in the subsoil with a lower content of these, and therefore, addition of manure to the subsoil reduced the populations considerably. Nesmith (1985) found that soil type influenced soil moisture and antagonistic microbial populations, which in turn affected the *Ralstonia* populations. However, in a study conducted by Shekhawat and Perombelon (1991) population decline was slower in clay than in sand even under dry conditions. In Indonesia bacterial wilt 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, 1991).

Soil pH Although the optimum pH for growth of *R. solanacearum* in vitro is about 6.8, bacterial wilt has been reported in both acidic and alkaline soils. In North Carolina a higher incidence of potato wilt occurred in soil with a pH 4.5. However, in Japan and Ceylon the occurrences were often alkaline, in one instance a soil pH of 8.5 was recorded (Kelman, 1953).

Soil layers Results of several authors (McCarter et al., 1969; Okabe, 1971; Tanaka and Noda, 1973) suggested that *R. solanacearum* can survive in deeper layers of certain soils. Once the pathogen has entered the deeper layers it can survive in localized microsites (debris or 'free soil'), even where microbial activity is likely to be low (Lloyd, 1978). Other authors (Okabe, 1971; Tanaka, 1976; Graham and Lloyd, 1979) also reported higher concentrations of *R. solanacearum* at deeper soil layers. **?** observed the distribution of the pathogen in the 0-80 cm layer of naturally infested sandy loam soil, and noted a higher population at all depths even after one year of fallow. Sunaina et al. (1989) supported 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. However, the pathogen survived at the 20-60 cm soil level even after the field had been kept fallow for 7 months. Longer survival of the pathogen was expected to be in deeper soil layers due to the undisturbed remaining root debris with bacterial exudates (Sunaina et al., 1989).

Soil anaerobiosis 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. However, anaerobic conditions favored a shift of pathogen from virulent to avirulent state (Shekhawat and Perombelon, 1991).

8 Recommendation

R. solanacearum is an important bacterial pathogen for both developing and developed countries and directly related to economic hardship to potato growers. Despite this, there are lamentably few options of control and it is urgently required to perform an advanced research on the pathogen management considering those biological manner of *R. solanacearum*.

References

Ahmed NN, Islam MR, Hossain MA, Meah MB, Hossain MM. 2013. Determination of races and biovars of *Ralstonia solanacearum* causing bacterial wilt disease of potato. J Agril Sci 5:86–93. doi: 10.5539/jas.v5n6p86.

Ajanga S. 1993. Status of bacterial wilt in Kenya. ACIAR Proceedings 45:338–340.

Alacho FO, Akimanz iDR. 1993. Progress, achievements and constraints on bacterial wilt control in Uganda. Workshop on bacterial wilt of potato caused by *Pseudomonas solanacearum* :32–41.

Allen AK C, French ER. 2001. Brown rot of potatoes. In: GDF W R Stevenson R Loria, DP Weingartner, editors. Compendium of potato Piseases, 2nd ed. APS Press, St. Paul, MN, USA. p. 11–13.

Ateka EM, Mwang'ombe AW, Kimenju JW. 2001. Reaction of potato cultivars to *Ralstonia solanacearum* in Kenya. African Crop Science Journal 9:251–256. doi: 10.4314/acsj.v9i1.27646.

Barton D, Smith J, M KZ. 1997. Socio-economic inputs to biological control of bacterial wilt disease of potato in Kenya. ODA RNRRS crop protection project R6629 :23.

Berrios DE, Rubirigi A. 1993. Integrated control of bacterial wilt in seed production by the Burundi national potato program. In: Hartman, G.L. and A.C. Hayward (eds.). Bacterial wilt. ACIAR Proceedings 45:284–288.

Buddenhagen I, Kelman A. 1964. Biological and physiological aspects of bacterial wilt caused by *Pseudomonas solanacearum*. Annual Review of Phytopathology 2:203–230. doi: 10.1146/annurev.py.02.090164.001223.

CABI. 2017a. Invasive Species Compandium (Datasheets, maps, images, abstracts and full text on invasive species of the world: Ralstonia solanacearum) .

CABI. 2017b. Plantwise Knowledge Bank (Plantwise Technical Factsheet: bacterial wilt of potato (*Ralstonia solanacearum*) .

Chakraborty R, Roy TS. 2016. Threats faced by brown rot of potato in Bangladesh. Microbiology Research 7:8–12. doi: 10.4081/mr.2016.6258.

Champoiseau GP. 2008. *Ralstonia solanacearum* race 3 biovar 2 (USDA-NRI Project- R. solanacearum race 3 biovar 2: detection, exclusion and analysis of a Select Agent Educational modules) .

Champoiseau PG, Jones JB, Allen C. 2009. *Ralstonia solanacearum* race 3 biovar 2 causes tropical losses and temperate anxieties. Plant Health Progress :1–10doi: 10.1094/php-2009-0313-01-rv.

Ciampi L, Sequeira L. 1980. Influence of temperature on virulence of race 3 strains of *Pseudomonas solanacearum*. American Potato J 57:307–317. doi: 10.1007/bf02854025.

Coelho N, Nutter F. 2005. Bacterial wilt in Amazon State (Bacterial Wilt Disease and the *Ralstonia solanacearum* Species Complex). APS Press, Minnesota, USA.

Danks C, Barker I. 2000. On-site detection of plant pathogens using lateral-flow devices. EPPO Bulletin 30:421–426. doi: 10.1111/j.1365-2338.2000.tb00922.x.

Database OG. 1990. EPPO Standards PM 3/26, *Pseudomonas solanacearum* inspection and test methods. Bull OEPP/EPPO 20:255–262.

Database OG. 2004. EPPO Standards PM 7/21, Diagnostic protocols for regulated pests. Bull OEPP/EPPO 34:155–157.

Database OG. 2017a. *Ralstonia solanacearum* (RALSSO). Normes OEPP EPPO Standards: Diagnostic protocols for regulated pests PM7/21.

Database OG. 2017b. *Ralstonia solanacearum* (RALSSO) (Associated EPPO Standards. EPPO A1 and A2 Lists of pests recommended for regulation as quarantine pests PM1/002(25):1–17.

Denny TP, Brumbley SM, Carney BF, Clough SJ, Schell MA. 1994. Bacterial wilt: the disease and its causative agent, *Pseudomonas solanacearum*. chapter Phenotype conversion of *Pseudomonas solanacearum*: its molecular basis and potential function. CAB International, Wallingford, UK.

Devi L, Menon M, Aiyer R. 1982. Population threshold of *Pseudomonas solanacearum* at the onset of first symptoms of wilt in tomato. Indian journal of microbiology 22:41–43.

Eddins AH. 1941. Investigations and control of brown rot of potatoes and related crops caused by *Bacterium solanacearum*. Florida Agric Experiment St Annu Rep 117.

Elphinstone JG. 2005. The Current Bacterial Wilt Situation: A Global Overview. APS Press, Minnesota, USA.

Elphinstone JG, Hennessy J, Wilson JK, Stead DE. 1996. Sensitivity of different methods for the detection of *Ralstonia solanacearum* in potato tuber extracts. EPPO Bulletin 26:663–678. doi: 10.1111/j.1365-2338.1996.tb01511.x.

Elphinstone JG, Stanford HM, Stead DE. 1998. Detection of *Ralstonia solanacearum* in Potato Tubers, *Solanum dulcamara* and Associated Irrigation Water. In: Bacterial Wilt Disease. Springer, Berlin, Germany. p. 133–139. doi: 10.1007/978-3-662-03592-4_19.

Enfinger JM. 1979. Evaluation of chemicals and application methods for control of bacterial wilt of tomato transplants. Phytopathology 69:637. doi: 10.1094/phyto-69-637.

EPPO. 2004. *Ralstonia solanacearum*. European and Mediterranean Plant Protection Organization. EPPO Bull 34:173–178.

EU. 1998. Council Directive 98/57/EC of 20 July 1998 on the control of *Ralstonia solanacearum* (Annex II-test scheme for the diagnosis, detection and identification of *Ralstonia solanacearum*). Official J European Commun L235:8–39.

FAOSTAT. 2015. Statistical Database 2015. Food & Agricultural Organization of the United Nations .

Farag NS, Fawzi FG, El-Said SIA, Mikhail MS. 1986. Streptomycin in relation to potato brown rot control. Acta Phytopathologica et Entomologica Hungarica 21:115–122.

Farag NS, Lashin SM, Abdel-All RS, Shatta HM, Seif-Elyazal AM. 1982. Antibiotics and control of potato black leg and brown rot diseases. Agril Res Rev 60:149–166.

Fegan M, Prior P. 2005. How complex is the *Ralstonia solanacearum* species complex? APS press.

French ER. 1985. Interaction between strains of *Pseudomonas solanacearum*, its hosts and the environment. Bacterial Wilt Disease in Asia and the South Pacific. ACIAR Procedings 13:99–104.

Froines JR. 2010. indings of the Scientific Review Panel on 'Evaluation of Chloropicrin as a Toxic Air Contaminant' (Public Meeting). Calif Dept Pestic Reg .

Gadewar AV, Trivedi TP, Sekhawat GS. 1991. Potato in Karnataka. Technical Bulletin 17:33.

Ghosh PP, Mandal NC. 2009. Some disease management practices for bacterial wilt of potato. J Plant Protec Sci 1:51–54.

Girard JC, Nicole JF, Cheron JJ, Gaubiac AM, Huvier O, Oudard B, Suzor M. 1992. Bacterial wilt due to *Pseudomonas solanacearum* in Reunion: General situation and current trends. ACIAR Proc 45:343–347.

Graham J. 1979. Survival of *Pseudomonas solanacearum* race3 in plant debris and in latently infected potato tubers. Phytopathology 69:1100–1103. doi: 10.1094/phyto-69-1100.

Graham J, Lloyd AB. 1979. Survival of potato strain (race 3) of *Pseudomonas solanacearum* in the deeper soil layers. Australian Journal of Agricultural Research 30:489–496. doi: 10.1071/ar9790489.

Granada GA, Sequeira L. 1983. Survival of *Pseudomonas solanacearum* at low temperature. Fitopatologia 18:22–24.

Hayward AC. 1991. Biology and epidemiology of bacterial wilt caused by textitPseudomonas solanacearum. Annual Review of Phytopathology 29:65–87. doi: 10.1146/annurev.py.29.090191.000433.

Hayward AC. 1994. The hosts of *Pseudomonas solanacearum*. CAB International, Wallingford, UK.

He LY. 1983. Characteristics of strains of *Pseudomonas solanacearum* from China. Plant Disease 67:1357–1361. doi: 10.1094/pd-67-1357.

Hsu ST. 1991. Ecology and control of *Pseudomonas solanacearum* in Taiwan. Plant Prot Bull Taiwan 33:72–79.

IPDN. 2014. Bacterial Wilt Disease *Ralstonia solanacearum*: Standard Operating Procedure for Use in Diagnostic Laboratories. International Plant Diagnostic Network EA-SOP-RS1:1–24.

Johnson G, Schaal LA. 1952. Relation of chlorogenic acid to scab resistance in potatoes. Science 115:627–629. doi: 10.1126/science.115.2997.627.

Kabeil SS, Lashin SM, El-Masry MH, El-Saadani MA, Abd-Elgawad MM, Aboul-Einean AM. 2008. Potato brown rot disease in Egypt: current status and prospects. Am. Eurasian J. Agric. Environ. Sci 4:44–54.

Kaguongo WP, Ng'ang'a NM, Muthoka N, Muthami F, Maingi G. 2010. Seed potato subsector master plan for Kenya. GTZ-PSDA, USAID, CIP and Government of Kenya. Ministry of Agriculture. Kenya .

Kelman A. 1953. The bacterial wilt caused by *Pseudomonas solanacearum*. Tec Bull North Carolina Agril Exp Sta 99:1–103.

Kelman A. 1965. Root-to-root spread of *Pseudomonas solanacearum*. Phytopath 55:304–309.

Kelman A. 1998. One hundred and one years of research on bacterial wilt. In: Bacterial Wilt Disease. Springer, Berlin, Germany. p. 1–5. doi: 10.1007/978-3-662-03592-4_1.

Lelliott RA, Stead DE. 1987. Methods for the diagnosis of bacterial diseases of plants. Blackwell Scientific Publications, Oxford, UK.

Lloyd AB. 1978. Survival of the potato strain of in soil. Proceedings of the 4th international conference on plant pathogenic bacteria :875–878.

Martin C. 1981. Bacterial wilt of potatoes in the amazon basin. Plant Disease 65:246–248. doi: 10.1094/pd-65-246.

Martin C, R FE. 1985. Bacterial wilt of potatoes caused by *Pseudomonas solanacearum*. CIP Tech Information Bull 13:1–6.

Mayers PE, Hutton DG. 1987. Bacterial wilt, a new disease of custard apple: symptoms and etiology. Annals of Applied Biology 111:135–141. doi: 10.1111/j.1744-7348.1987.tb01440.x.

McCarter SM, Dukes PD, Jaworski CA. 1969. Vertical distribution of *Pseudomonas solanacearum* in several soils. Phytopath 59:1675–1677.

Moffett ML, Giles JE, Wood BA. 1983. Survival of *Pseudomonas solanacearum* biovars 2 and 3 in soil: Effect of moisture and soil type. Soil Biology and Biochemistry 15:587–591. doi: 10.1016/0038-0717(83)90054-8.

Mukherjee N, Dasgupta MK. 1989. Udvider Rog (Plant Disease). Poschimbongo Rajyo Pustak Porsad (West Bengal State Book Board) Kolkata, India.

Murakoshi S, M T. 1984. Trials of some control of tomato wilt caused by *Pseudomonas solanacearum*. Bull Kanagawa Hortic Exp St 31:50–56.

Nesmith WC. 1985. Influence of antagonists and controlled matric potential on the survival of *Pseudomonas solanacearumin* four North Carolina soils. Phytopathology 75:1182–1187. doi: 10.1094/phyto-75-1182.

Nishat S, Hamim I, Khalil MI, Ali MA, Hossain MA, Meah MB, Islam MR. 2015. Genetic diversity of the bacterial wilt pathogen *Ralstonia solanacearum* using a RAPD marker. Comptes Rendus Biologies 338:757–767. doi: 10.1016/j.crvi.2015.06.009.

Okabe N. 1971. Population changes of *Pseudomonas solanacearum* and soil organisms in artificially infested and natural field soils. Rev Plant Protec Res 4:105–108.

Opio AF. 1988. Host range and biotypes of *Pseudomonas solanacearum* EF Smith in Uganda: A preliminary study. 15th International Congress of Plant Pathology, Kyoto, Japan :20–27.

Parvez S. 2017. Potato exports fail to pick up. 14 april. The Daily Star .

Poussier S, Thoquet P, Trigalet-Demery D, Barthet S, Meyer D, Arlat M, Trigalet A. 2003. Host plant-dependent phenotypic reversion of *Ralstonia solanacearum* from non-pathogenic to pathogenic forms via alterations in the phcA gene. Molecular Microbiology 49:991–1003. doi: 10.1046/j.1365-2958.2003.03605.x.

Prior P, Allen C, Elphinstone J. 1998. Bacterial Wilt Disease. Springer, Berlin, Germany. doi: 10.1007/978-3-662-03592-4.

Prior P, Fegan M. 2005. Recent development in the phylogeny and classificaiton of *Ralstonia solanacearum* . Acta Horticulturae :127–136doi: 10.17660/actahortic.2005.695.14.

Priou S, Gutarra L, Aley P. 2006. Sensitive detection of *Ralstonia solanacearum* in soil: a comparison of different detection techniques. Plant Pathol 49:414–422.

Schaad NW, Jones JB, Chun W, et al. 2001. Laboratory guide for the identification of plant pathogenic bacteria. Ed. 3. APS Press, Minnesota, USA.

Seneviratne SN. 1988. Soil survival of *Pseudomonas solanacearum* (Bacterial Diseases of the Potato: Report of the Planning Conference on Bacterial Diseases of the Potato). CIP, Lima, Peru.

Sequeira L, Averre C. 1961. Distribution and pathogenicity of strains of *Pseudomonas solanacearum* from virgin soils in Costa Rica. Plant Disease Reporter 45:435–440.

Shekhawat GS, Chakrabarti SK, Gadewar AV. 1992. Potato bacterial wilt in India (Indian Council of Agricultural Research). Technical Bull 38:16–39.

Shekhawat GS, Perombelon MCM. 1991. Factors affecting survival in soil and virulence of *Pseudomonas solanacearum*. J Plant Diseases Protection 93:258–267.

Shivani R. 2016. Bacterial wilt disease of potato caused by *Ralstonia Solanacearum*: Plant disease .

Sitaramaiah K, Sinha SK. 1984. Interaction between *Meloidogyne javanica* and *Pseudomonas solanacearum* on brinjal. Indian J Nematol 14:1–5.

Strider DL. 1981. Southern bacterial wilt of geranium caused by *Pseudomonas solanacearum*. Plant Disease 65:52–53. doi: 10.1094/pd-65-52.

Sunaina V, Kishore V, Shekhawat GS. 1989. Latent survival of *Pseudomonas solanacearum* in potato tubers and weeds. J Plant Dis Prot 96:361–364.

Swanepoel AE. 1990. The effect of temperature on the development of wilting and on progeny tuber infection of potatoes inoculated with South

African strains of biovar 2 and 3 of *Pseudomonas solanacearum*. Potato Res 33:287–290. doi: 10.1007/bf02358459.

Tanaka Y. 1976. Factors affecting survival of *Pseudomonas solanacearum* (Proceedings of the first International Planning Conference and Workshop on the Ecology and Control of Bacterial wilt caused by *Pseudomonas solanacearum*). North Carolina State University, Raleigh, USA .

Tanaka Y, Noda N. 1973. A study of factors governing the survival of tobacco wilt disease bacteria. Bull Okayama Tobacco Experim St 32:81–93.

Thurston HD. 1963. Bacterial wilt of potatoes in Colombia. American Potato Journal 40:381–390. doi: 10.1007/bf02849446.

van Elsas JD, Kastelein P, de Vries PM, van Overbeek LS. 2001. Effects of ecological factors on the survival and physiology of *Ralstonia solanacearum* bv. 2 in irrigation water. Canadian Journal of Microbiology 47:842–854. doi: 10.1139/cjm-47-9-842.

Volcani Z, Palti J. 1960. *Pseudomonas solanacearum* in Israel. Plant Disease Reporter 44:448–449.

Wallis F, Truter SJ. 1978. Histopathology of tomato plants infected with *Pseudomonas solanacearum*, with emphasis on ultrastructure. Physiological Plant Pathology 13:307–317. doi: 10.1016/0048-4059(78)90047-4.

Wang JF, Lin CH. 2005. Integrated Management of Tomato Bacterial Wilt. AVRDC-The World Vegetable Center, Taiwan.

Weller SA, Elphinstone JG, Smith NC, Boonham N, Stead DE. 2000. Detection of *Ralstonia solanacearum* strains with a quantitative, multiplex, real-time, fluorogenic PCR (TaqMan) assay. Applied and Environmental Microbiology 66:2853–2858. doi: 10.1128/aem.66.7.2853-2858.2000.

Yabuuchi E, Kosako Y, Yano I, Hotta H, Nishiuchi Y. 1995. Transfer of two *Burkholderia* and an *Alcaligenes* species to *Ralstonia* gen. nov. Microbiology and Immunology 39:897–904. doi: 10.1111/j.1348-0421.1995.tb03275.x.

Yuliar, Nion YA, Toyota K. 2015. Recent trends in control methods for bacterial wilt diseases caused by *Ralstonia solanacearum*. Microbes and Environments 30:1–11. doi: 10.1264/jsme2.me14144.

Zachos DG. 1957. The brown rot of potatoes in Greece. Annales de l'Institut Phytopathologique Benaki, New Series 1:115–117.

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