

Management of Bacterial Wilt [*Rhalsstonia solanacearum* Yabuuchi et al., 1995] of Potatoes: Opportunity for Host Resistance in Kenya

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Abstract

This article gives a general overview of bacterial wilt of potatoes and its management. It also highlights the potential of host resistance as an important component of integrated management of bacterial wilt in Kenya. Bacterial wilt has spread to all potato growing areas in Kenya, affecting over 70% of potato farms and causing yield losses of between 50 to 100%. This disease has no effective means of control because crop protection chemicals are ineffective and expensive and biological control agents are ineffective. In addition, phytosanitary methods such as quarantine are either expensive or difficult to apply and cultural methods such as crop rotations are largely impractical because the farms are too small to allow effective rotation, the pathogen has a wide host range, and it persists for long in the soil. Development of resistant cultivars could therefore play an important role in managing the disease. More resistant potato clones have recently been identified by CIP scientists, and this resistance needs to be incorporated into the popular but susceptible Kenyan potato cultivars so as to increase potato production in Kenya. For better results, use of high resistant varieties may be coupled with use of disease-free tubers and clean fields.

Keywords: bacterial wilt, *Rhalsstonia solanacearum*, management, host resistance, kenya, potatoes

1. Introduction

In Kenya, potato is an important food crop, second after maize in volumes produced (MoA, 2008). It is grown mainly by small scale farmers as a cash and food crop and therefore plays an important role in food security (MoA, 2005; MoA, 2008). The annual potato crop is valued at USD 6.25 million at farm gate level, and USD 12.5 million at the consumer level (ANN, 2009). Potato farming in Kenya employs 2.5 million people at all levels of the value chain (ANN, 2009). Despite its importance, potato production in Kenya has not achieved its full potential because of a number of production constraints. These include low soil fertility, inadequate supply of certified seeds, use of low yielding varieties, and diseases. The most common diseases include late blight, viral infections and bacterial wilt (Kaguongo et al., 2008). Bacterial wilt of potato was first reported in Kenya in 1945 around the Embu area, from where it spread to other parts of the country (Ajanga, 1993; Otipa et al., 2003). The disease is believed to have been introduced with tuber seeds imported from Europe (Todd, 1969). According to some recent studies, the disease is found in all the potato growing areas of the country affecting 77% of potato farms; it is followed by late blight (67%), and viral diseases (12%) (Kaguongo et al., 2010).

Bacterial wilt, caused by *Ralsstonia solanacearum* (Yabuuchi et al., 1995), is the second most important potato disease in tropical and sub-tropical regions of the world after late blight (Champoiseau et al., 2010). Globally, the disease has been estimated to affect about 1.7 million hectares of potatoes in approximately 80 countries, with global damage estimates of over USD 950 million per annum (Champoiseau et al., 2009). In addition to potatoes, the disease also affects over 200 plant species from more than 50 families (Hayward, 1991). Bacterial wilt is widely distributed in tropical, subtropical, and warm temperate climates of the world, and it occurs in about 45 countries in the southern hemisphere. In Africa, it is found in Angola, Burkina Faso, Burundi, Cameroon, Congo, Ethiopia, Gabon, Gambia, Kenya, Madagascar, Malawi, Mauritius, Mozambique, Nigeria,

Réunion, Rwanda, Senegal, Seychelles, Sierra Leone, Somalia, South Africa, Swaziland, Tanzania, Tunisia, Uganda, Zaire, Zambia, and Zimbabwe (EPPO, 2004).

2. Bacterial Wilt Symptoms on Potatoes

In the early stages of the disease, foliage symptoms include rapid wilting of the youngest leaves at the end of the branches during the hottest time of the day (Martin & French, 1985; Hayward, 1991; EPPO, 2004). At this stage, only one or half a leaflet may wilt, and plants may appear to recover at night when the temperatures are lower (Martin & French, 1985; Champoiseau et al., 2009). As the disease develops, all leaves may wilt quickly and desiccate although they remain green (Champoiseau et al., 2009). This may be followed by yellowing of the foliage, and eventual plant death; other symptoms include epinasty, chlorosis, and stunting (Martin & French, 1985; Champoiseau et al., 2009). Wilting is possibly a result of restricted water movement due to the formation of slime that surrounds the bacterial mass in the stem vascular bundles (Martin & French, 1985). Infected stem vascular bundles may become visible as long, narrow, dark-brown streaks, and the stem may also collapse in young potato plants (Champoiseau, 2008). In well-established infections, cross-sections of stems may reveal brown discoloration of infected tissues (EPPO, 2004) and a white, slimy mass of bacteria may exude from the vascular bundles of the cross-sections (Martin & French, 1985; Hayward, 1991; EPPO, 2004). This slime also streams spontaneously, in form of threads, when the cut surface of a potato stem is suspended in water (Plate 1). Such threads are not formed by other bacterial pathogens of potato (Champoiseau et al., 2009). The streaming test is of presumptive diagnostic value in the field (Martin & French, 1985; EPPO, 2004). Under cool growing conditions, wilting and other foliar symptoms may not occur (Hayward, 1991; EPPO, 2004).



Plate 1. Symptoms of bacterial wilt on potatoes. Clockwise from top; foliage wilting, discolouration of stem vascular tissues, bacterial ooze on vascular tissues of the tuber, and bacterial streaming from the stem section (Champoiseau et al., 2009)

On tubers, symptoms may be visible in the later stages of disease development (EPPO, 2004). The symptoms include bacterial ooze at the tuber eyes or at the point where the stolon attach to the tuber; and soil may adhere to the tubers at the eyes (Martin & French, 1985; EPPO, 2004). Cutting the diseased tuber may reveal browning, and eventual necrosis of the vascular ring, and the immediately surrounding tissues (Martin & French, 1985). A milky-white sticky exudate usually appears spontaneously on the vascular ring a few minutes after cutting the tuber (Champoiseau et al., 2009).

Plants with foliar symptoms may bear apparently healthy and diseased tubers, while plants that show no symptoms of the disease may sometimes produce diseased tubers (Martin & French, 1985; Hayward, 1991; EPPO, 2004). Because symptom expression is favoured by high temperatures, symptomless plants may remain latently infected for extended periods of time at low temperatures (French, 1994). In Kenya, certified and apparently healthy (but latently infected) potato seed tubers produced at altitudes of 1520-2120 meters above sea level showed infection when planted at lower altitudes (Nyangeri et al., 1984).

3. The Pathogen

The causal organism of the bacterial wilt is the bacterium *Ralstonia solanacearum* (Yabuuchi et al., 1995), which was described for the first time as *Bacillus solanacearum* by Smith in 1896 (EPPO, 2004). In the following years, five pathogenic races (based on host range under field conditions) and five biovars (based on carbon utilization patterns) were identified (Buddenhagen et al., 1962; Hayward, 1964). Race 1 occurs in the lowland tropics and warm temperate lands (French, 1994). It attacks potato, tomato, brinjals, chilli, groundnuts, tobacco, diploid bananas, and many other *solanaceous* crops, as well as many hosts in other plant families (French, 1994; Denny, 2006). It has a high temperature optimum (35-37°C), as do races 2, 4, and 5 (EPPO, 2004). Race 2 is indigenous to Central and South America, and attacks members of *Musaceae* family such as plantain, triploid bananas, and *Heliconia* (French, 1994). It causes moko disease on bananas and *Heliconia* in Central and South America, and bugtok disease on plantains in the Philippines (Martin & French, 1985; EPPO, 2004). Race 3 occurs at higher altitudes (in the tropics) and higher latitudes than race 1 (EPPO, 2004). It mainly attacks potato, tomato (especially when planted after infected potato), geranium, occasionally *Pelargonium zonale*, eggplants, capsicum, and some *solanaceous* weeds like *Solanum nigrum* and *Solanum dulcamara* (Martin & French, 1985; Janse, 1991; French, 1994). Race 3 also infects a number of non-*solanaceous* weeds asymptotically (Wenneker et al., 1999; Pradhanang et al., 2000). This race has a long association with potatoes and has an optimum temperature of 27 - 28°C (French, 1994). Race 4 affects ginger in Asia and Hawaii, while race 5 affects mulberry in China (EPPO, 2004; Table 1).

Table 1. Race determination in *Ralstonia solanacearum**

Race	1	2	3
Reaction in:			
Tomatoes/eggplants	Wilting	No reaction	Wilting
Tobacco cv. White Burley plants (stem inoculation)	Wilting	No reaction	No reaction
Tobacco cv. White Burley leaves (hypersensitivity test)	Necrosis (48 h) and wilting (7-8 days)	Hypersensitive Reaction (12-24 h)	Chlorosis(2-8 days)
<i>Musa acuminata</i>	No reaction	Wilting	No reaction

*Race 4, pathogenic to ginger and a few other hosts and race 5, pathogenic to mulberry only, not included.

Source: Janse, 1991

The bacterium has also been classified into biovars. Biovars are based on their ability to produce acid from several disaccharides and sugar alcohols (Buddenhagen, 1986; Seal et al., 1999; Denny, 2006; Table 2).

Table 2. Differentiation of *Ralstonia solanacearum* into biovars

Biochemical Test	Biovars					
		1	2A	3	4	5
Oxidation of	Mannitol	-	-	+	+	+
	Sorbitol	-	-	+	+	-
	Dulcitol	-	-	+	+	-
	Trehalose	+	-	+	+	+
Utilization of	Lactose	-	+	+	-	+
	Maltose	-	+	+	-	+
	Cellobiose	-	+	+	-	+

Source: Buddenhagen & Kelman, 1964

Biovars 3, 4, and 5 are the most versatile in terms of the range of carbon sources (Table 2). The biovars do not correlate with the races and only race 3, the potato race, is equivalent to biovar 2A (Hayward, 1983; Table 3).

Table 3. Equivalence between biovars and races of *Ralstonia solanacearum*

Race	Biovars	Hosts	Location
1	1,3,4	All <i>Solanaceous</i> crops + many other hosts	Lowland tropics (Asia, Americas and Australia)
2	1,3	Bananas and other <i>Musa</i> species	American and Asian tropics (Caribbean, Brazil, Philippines)
3	2A	Potato and tomatoes	Cool climate worldwide
4	3,4	Ginger	Asia
5	5	Mulberry	China
Not known	2T	Numerous	Amazon basin

Source: EPPO, 2004

Biovar 2 has the least host range whereas biovar 3 has the widest (Table 3). Biovar 5 is confined to mulberry in China (Hayward, 1964; Hayward, 1983; Hayward, 1991). Biovar 2 (race 3) is known as the potato or low temperature race and is found in high latitudes, and high altitudes in the tropics (Seal et al., 1999; Hayward, 2000).

Race 3/biovar 2A (R3bv2A) causes bacterial wilt of potato in over 90% of cases worldwide because potato is a cool season crop (French, 1994; EPPO, 2004). Potato is the common host for R3bv2A, but when there is high pathogen inoculum concentration in the soil, and high temperature, it can also infect tomatoes, or a few other crops, when they are grown in rotation (Buddenhagen, 1986; French, 1994; EPPO, 2004). R3bv2A occurs in the cool tropical highlands and it is widespread in the higher latitudes as far as southern Sweden and southern Argentina (Champoiseau et al., 2009). The R3bv2A is the main cause of bacterial wilt of potatoes in the Kenyan highlands (Smith et al., 1995). Although R3bv2A principally occurs in cool climates, it also occurs in potato plants grown in warmer locations from seed tubers harvested from cool climates (French, 1994). With the expansion of potatoes into warmer subtropical and tropical lands, in addition to global warming, cases of lowland bacterial wilt caused by race 1 (biovars 1, 3 and 4) have occurred (French, 1994; EPPO, 2004).

The old classification system of the pathogen into races and biovars is unsatisfactory because it is not predictive and some groups (e.g. race 1) contain very large variation. In addition, race determination is not possible, because *R. solanacearum* strains do not have race-cultivar specificity on plant hosts and, with the exception of R3b2A, the old “races” do not have phylogenetic unity (Fegan & Prior, 2005; Champoiseau et al., 2010). Recently, a more phylogenetically meaningful system has classified *R. solanacearum* into four major genetic

groups called phylotypes that reflect the geographical origin and ancestral relationships between strains (Table 4).

Table 4. Equivalences among phylotypes, biovars and races of *R. solanacearum*

Species	<i>Ralstonia solanacearum</i>													
Phylotype	I			II			III			IV				
Origin	Asia			America			Africa			Indonesia				
Biovars	3	4	5	2T	1	2	2T	1	2T	1	2	R	B	
Races	1	4	5	1	2	3								

R=*Ralstonia syzygii*; B= *Pseudomonas celebense*

Source: Prior & Fegan, 2005.

This classification scheme is based on variation of DNA sequences. Each phylotype is composed of a number of groups of strains with a highly conserved sequence termed sequevars (Prior & Fegan, 2005; Fegan & Prior, 2005). Phylotype I include all strains belonging to biovars 3, 4, and 5 and strains are isolated primarily from Asia. Phylotype II includes strains belonging to biovars 1, 2 and 2T isolated primarily from America. The R3b2A and the race 2 are both members of phylotype II. Phylotype III contains strains primarily isolated from Africa and surrounding islands. Strains in this group belong to biovars 1 and 2T. Phylotype IV contains strains isolated primarily from Indonesia belonging to biovars 1, 2 and 2T. *R. solanacearum* race 3 biovar 2A (R3b2A) belongs to Phylotype II (sequevars 1 and 2) (Figure 1).

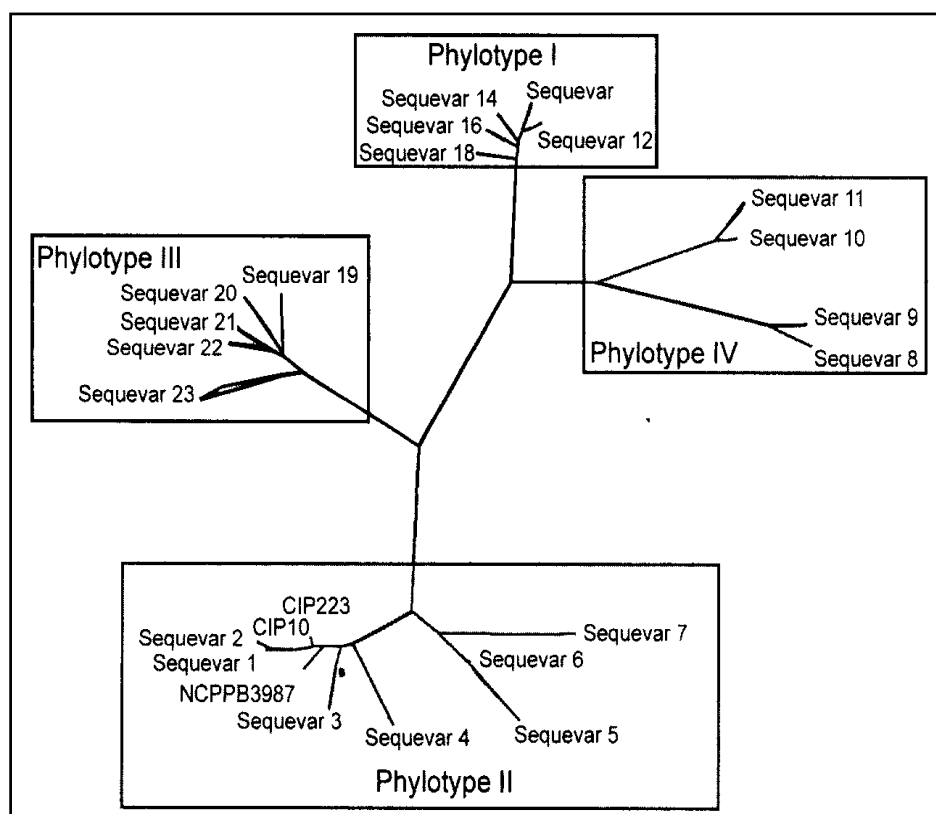


Figure 1. Phylogenetic tree showing the phylogenetic relationships of sequevars and phylotypes

Source: Fegan & Prior, 2005.

4. Simple Detection of the Bacteria

4.1 Vascular Flow Test

A diagnostic test is necessary because plant wilting caused by the bacterium *Ralstonia solanacearum* can be confused with symptoms induced by other pathogens such as *Fusarium eumartii*, *Verticillium sp.*, *Erwinia chrysanthemi*, by insect or mechanical damage at the stem base or due to drought (Champoiseau *et al.*, 2010). Diagnosis in the field is easily accomplished through the vascular flow test (Priou *et al.*, 1999). This involves cutting a piece of stem 2-3 cm long from the base of a wilting potato plant, and suspending it in clear water in a glass container. The cut stem is held with an opened paper clip to maintain a vertical position. After a few minutes, the smoke-like milky threads streaming downward from the cut stem confirms the presence *R. solanacearum* within the vascular system (French *et al.*, 1995; CIP, 2007; Champoiseau *et al.*, 2010).

4.2 The KOH Test

The *R. solanacearum* symptoms on tubers can be confused with ring rot caused by *Clavibacter michiganensis* subsp. *sepedonicus* (previously called *Corynebacterium*

sepedonicum). A quick differential test is performed directly on the bacterial exudate on the tuber to differentiate between the two bacteria. Two drops of 3% potassium hydroxide (KOH) is placed on the ooze and mixed using a laboratory loop or a wooden toothpick for 10 seconds. The formation of a milky thread upon lifting the toothpick indicates the presence of *R. solanacearum* (a Gram-negative bacterium), whereas with *C. michiganensis* subsp. *sepedonicus* (a Gram positive bacterium) the thread is not produced (Priou *et al.*, 1999).

5. Epidemiology and Survival of the Pathogen

In potatoes, the bacteria is tuber borne, and is primarily disseminated through infected seed tubers (French *et al.*, 1975; Nyangeri *et al.*, 1984; Kinyua *et al.*, 1998; Champoiseau *et al.*, 2009). Potato seed tubers carry the bacterium in the vascular tissue, lenticels, and on the surface (Kelman, 1953; Sunaina *et al.*, 1989). Infected seed tubers from bacterial wilt infested areas have mainly contributed to the spread of the disease to many potato producing areas in Kenya; this is a consequence of the informal potato seed system prevalent in the country (Kaguongo *et al.*, 2008). In cool conditions, such as tropical elevations above 2500 m, infected but symptomless plants may harbour the bacterium and then transmit the pathogen to progeny tubers as latent infection, leading to severe disease outbreaks when grown at warmer locations. The other source of inoculum is the infested soil; the bacterium is native in many tropical soils (Martin & French, 1985). Bacterial wilt is further spread through infected run-off water or soil adhering to tools and shoes (Martin & French, 1985; Pradhanang, 1999).

Under field conditions, plant infection usually occurs through the root system, especially through wounds (Kelman, 1953). The pathogen can also enter through stem wounds or stomata (EPPO, 2004). Wounds can occur due to cultivation activities, natural growth of secondary roots, attack by nematodes or other pests (Martin & French, 1985; Shekhawat & Chakrabarti, 1993).

Once introduced, the pathogen survives at soil depths of 1m or more, where microbial competition is low, or as slimy masses in the upper soil layers (Kinyua *et al.*, 1998). The pathogen can survive in soil (mostly on plant debris) and in the rooting system and rhizosphere of many hosts (weeds, other host crops, potato volunteers). Survival of the pathogen in the soil is reduced by extreme cold, and the presence of antagonistic microorganisms, while volunteer host plants enable bacterial survival across seasons (Martin & French, 1985; Hayward, 1991; Milling *et al.*, 2009). Survival depends also on the race involved; race1 usually persists for many years in the soil because of its numerous hosts, while R3bv2A tends to persist for a few years due to limited hosts (Martin & French, 1985; Champoiseau *et al.*, 2009).

The aggressiveness of the pathogen is affected mainly by temperature and moisture; high temperature, and high soil moisture promote survival, reproduction, infectivity, and spread of the bacterium, and hence disease development (Harris, 1976; Martin & French, 1985). Temperature is the most important factor affecting the host-pathogen interaction as well as survival in soils. In general, increase in ambient temperature to between 30 and 35°C increases the incidence and rate of onset of bacterial wilt on hosts such as tomato, for many but not all strains of the pathogen (Hayward, 1991). It has been shown experimentally that soils exposed to 43°C continuously for periods of four days and over were free of the pathogen (Sencviratnc, 1988). Long-term survival in deeper soil layers is likely to be a function of lower soil temperatures and decreased microbial activity owing to a paucity of indigenous soil microorganisms (Graham & Lloyd, 1979). Survival of up to 673 days in naturally infested soils stored in plastic bags at 4°C has been reported (Granada & Sequeira, 1983). Although R3bv2A survives poorly at 4°C in water or in field soil, it survives in potato tubers at this temperature indicating that the pathogen is adapted to endure constant low temperatures when sheltered in the host tissue

(Milling et al., 2009). Harris (1976) found that the optimum temperature for R3bv2A was 27°C and the minimum was 12-15°C. It was also observed that a cold climate with average soil temperatures of 14°C or below in the tropical Kenyan highlands impeded the soil survival of R3bv2A (French, 1994).

Survival of the pathogen is greatest in wet but well-drained soils, (Kinyua et al., 1998; Champoiseau et al., 2009) whereas survival is affected adversely by soil desiccation and by flooding (Buddenhagen & Kelman, 1964).

6. Management of Bacterial Wilt on Potatoes

Control of *R. solanacearum* is difficult because it is a soil borne pathogen, has wide host range, long survival in the soil, and has wide biological variation (Martin & French, 1985). No single control method has been found to be 100% effective, although in locations where the pathogen is established, some level of bacterial wilt control has been possible through use of a combination of diverse methods (EPPO, 2004; Champoiseau et al., 2010). These methods include phytosanitation and cultural practices, chemical control, biological control, and host resistance (Martin & French, 1985; Champoiseau et al., 2010).

6.1 Phytosanitation and Cultural Practices

Phytosanitation and cultural practices are the most widely used practices for controlling bacterial wilt in the field (Martin & French, 1985; Champoiseau et al., 2010). These practices can be effective in regions where bacterial wilt is endemic, or in locations where it is present but not yet established (French, 1994; Champoiseau et al., 2010). Phytosanitation practices include planting disease-free tuber seeds, and quarantine measures, while cultural practices include crop rotation, intercropping, delayed planting, soil amendments, positive selection, and negative selection (Kinyua et al., 2001; EPPO, 2004; Champoiseau et al., 2010).

Although use of disease-free seed tubers is advocated in Kenya (Wakahu et al., 2007), it is not effective because the quantities of disease-free certified seed tubers produced by the formal seed system are insufficient to meet the farmers' demands (Lung'aho et al., 1997). This complicates the management of bacterial wilt because farmers rely heavily on seed tubers from informal sources, which results in frequent re-infection of fields (Kinyua et al., 2001; Muthoni et al., 2010).

Quarantine measures on the other hand may prevent introduction of the pathogen into disease-free areas (Champoiseau et al., 2009). However, quarantine measures necessary to avoid spread of bacterial wilt to disease-free area often restrict the production of tuber seeds; this limits the commercialization of ware potatoes thus affecting the economy of the quarantined area (Martin & French, 1985). Quarantine may not be effective in Kenya due to the informal seed system, and the porous international boundaries that promote uncontrolled movement of both seed and ware potatoes (Muthoni et al., 2010).

Crop rotation with non-host plants has been reported to reduce the *Ralstonia solanacearum* concentration in the soil (Martin & French, 1985). Crop rotation of 5-7 years excluding host plants has been recommended to control the bacteria in the soil (EPPO, 2004). In Kenya, crop rotation of potatoes with maize led to higher potato yields than monocropping potatoes in the presence of bacterial wilt (Barton et al., 1997). However, it was reported that rotations of maize, cowpeas, and sweetpotatoes did not reduce the soil inoculum concentration of R3bv2A (Jackson & González, 1981). In addition, R3bv2A may also survive by infecting plant roots of non host crops grown in rotation (Saumtally et al., 1993). In Mauritius, R3bv2A was reported to survive on sugar cane roots during rotation even though sugar cane is not a host plant (Saumtally et al., 1993). In Kenya, crop rotation may not be very effective because of small farm sizes in potato producing zones leading to continuous cultivation of potatoes on the same pieces of land or very short rotations that are inadequate to reduce the disease (Lemaga, 1997; Kaguongo et al., 2008). In addition, the small scale farmers do not have sufficient land to plant anything but essential food crops.

Other cultural practices like intercropping depend on the other crop used in the intercrop. In Burundi, intercropping of potatoes with beans resulted in less disease spread than intercropping potatoes with maize, while wide within-row spacing also reduced the incidence and spread of latent infection in the progeny tubers (French, 1994).

Although delayed planting reduced bacterial wilt incidence in India and Japan, in Kenya, delay in planting time may not be the best idea because the rainy seasons are short and erratic, and farmers may not be willing to risk losing a crop.

It has been reported that bacterial wilt incidence is increased by low soil pH, and low soil fertility (Lemaga et al., 2001; Lemaga et al., 2005; Messiha, 2006). However, soil amendments to raise pH or raise soil fertility may not be practical in Kenya because it is generally expensive to the small scale potato farmers.

Positive selection is only feasible in areas where the occurrence of bacterial wilt is incidental (Gildemacher et al., 2007). The method consists of harvesting seed tubers from healthy looking plants that produce healthy looking tubers (Kinyua et al., 2001). It involves identifying healthy looking plants in the field when the crop is 6-7 weeks after planting (and before flowering), and elimination of plants that develop symptoms later in the growing season. At harvest, plants with one or more rotted tubers and those that give very low yields are eliminated. The selected healthy looking plants are harvested before the rest of the field, and the seed tubers are handled in clean disinfected containers. These seed tubers are then used for subsequent seed increase or for ware potato production. An improvement to this method is positive selection with disease indexing. This consists of harvesting seed tubers from healthy looking plants that produce healthy looking tubers, and testing tubers for latent infection. Both methods call for proper identification of disease symptoms. These methods may not be effective with potato farmers in Kenya because even if the farmers may be able to positively identify bacterial wilt symptoms, they may not be able to index for latent infection because they cannot afford ELISA kits. In addition, many farmers are semi-illiterate and may not be able to use such kits.

Negative selection involves roguing out plants with symptoms of bacterial wilt from a relatively clean seed potato plot. Atypical plants and volunteer plants in seed plots are also rogued. This is done by digging out the whole infected plant with its root system, tubers, stolons and the soil around it. The two neighbouring plants are also rogued out and burnt. The soil in the holes where the plants are rogued from is mixed with ash or lime (Gildemacher et al., 2007). Ashes and lime are known to kill the bacteria, probably due to the high soil pH it causes. Ashes have the added advantage of containing nutrients, especially potassium, and some phosphorus. The rest of the plants are later harvested and the tubers used for seed. Negative selection is the most popular method for managing bacterial wilt in Kenya (Kaguongo et al., 2010). However, it is a very tedious operation and cannot be carried out on large areas.

6.2 Use of Chemicals

The most commonly used chemical treatment has been fumigation of contaminated soil/portions of the farm with methyl bromide (Champoiseau et al., 2010). This is a very expensive and tedious exercise and cannot be used on large areas. In addition, methyl bromide has been banned in most parts in the world and is being phased out in Kenya. The other product commonly used at field level is sodium hypochlorite; it is appropriate for spot treatment of the holes left behind after roguing of the wilting plants, and for general field sanitation (Kaguongo et al., 2008). However, use of sodium hypochlorite is expensive and tedious and therefore not practical in Kenya (Kaguongo et al., 2008).

6.3 Use of Biological Control Agents

Among biological control agents, a number of soil bacteria and plant growth promoting rhizobacteria (PGPR) are currently being investigated for their role in the control of R3bv2A (Champoiseau et al., 2010). However, none is currently available commercially, and their efficacy is yet to be determined on a commercial scale (Champoiseau et al., 2010). The R3bv2A affects the vascular system of the host, and after successful colonization, the R3bv2A remains inaccessible to the biological agent that may be colonizing the root surface and rhizosphere (Gadewar et al., 1993). Search for a biological control agent for bacterial wilt from the local bacterial antagonists in Kenya was initiated in 1992; however, the biological control agents were largely ineffective (Smith et al., 1998).

6.4 Host Resistance

Use of resistant potato varieties to control bacterial wilt in Kenya is probably the cheapest and the most practical means because chemicals are generally ineffective, phytosanitation and cultural measures are difficult to apply, and biological control agents are not commercially available (Martin & French, 1985; Champoiseau et al., 2010).

6.4.1 Nature of Resistance

The best that normal breeding has achieved is moderate level of resistance to bacterial wilt on a regional level, when conditions are not excessively hot or wet; some potato cultivars are less susceptible to bacterial wilt at least in some regions (Champoiseau et al., 2010). In the 1990s, advanced potato clones were obtained from a 14-yr programme of breeding for bacterial wilt resistance at CIP. These clones were produced after various crosses with (i) clones derived from Colombian *S. phureja* genotypes produced at the University of Wisconsin's breeding programme, initiated in the 1970s by Sequeira and Rowe (1969), (ii) clone AVRDC-1287 derived from *S. chacoense* and *S. raphanifolium*, (iii) progenitors derived from wild species *S. chacoense* and *S. sparsipilum* to combine other sources of bacterial wilt resistance, (iv) *S. tuberosum* subsp. *tuberosum* genotypes that carried earliness, adaptation to heat, resistance to late blight and root-knot nematode, and immunity to potato virus X

and potato virus Y, and (v) a population of diploid cultivated species *S. stenotomum*, *S. phureja* and *S. goniocalyx* from a breeding programme at North Carolina State University for high yield and good agronomic characteristics (Schmiediche, 1985a; Schmiediche & Martin, 1986; Anguiz & Mendoza, 1997). Resistance to R3bv2A available now in *Solanum tuberosum* originated mainly from the cultivated diploid, *Solanum phureja* (Martin & French, 1985). This resistance has been found to be strain-specific and sensitive to high temperatures (Sequeira & Rowe, 1969; Sequeira, 1979; Ciampi & Sequeira, 1980; French & Lindo, 1982). This resistance is seldom expressed as immunity because it is overcome by factors that favour the disease development i.e. high temperature, high soil moisture, low soil pH, low soil fertility, and damage to the plant root system (Martin & French, 1985; Low, 1997). Resistance to R3bv2A strains is expected to be more stable than resistance to lowland strains (race 1) of *R. solanacearum*, because R3bv2A strains are a genetically homogeneous group (Priou et al., 2001). Because high level of resistance has not been identified in *S. tuberosum*, only moderately resistant cultivars are used such as cultivar *Achat'* in Brazil, *Cruza 148* (unknown origin, used in East Africa and Peru), *Molinera* and *Lopez'* in Mexico, *Prisca* in Madagascar and cultivar *Ndinamagara* in Burundi, Rwanda, and Democratic Republic of Congo (Hayward, 1991; French et al., 1998). In Uganda, clones 388575.5 and 388575.9 both from CIP are moderately resistant to bacterial wilt in the cool areas, while clones 390005.11, 388574.2B, and 388580.18A are moderately resistant to bacterial wilt in the warm areas (Kaguongo et al., 2008). In Kenya, varieties Kenya Dhamana (CIP-800228), Kenya Sifa, Kenya Karibu, Mauritius clone (89016), and Cruza-148 (CIP-720118) were rated as resistant to bacterial wilt, while varieties Asante (CIP-381381.20), Tigoni (CIP-381381.13), Nyayo, and Dutch Robyjin were highly susceptible (Ateka et al., 2001). To control bacterial wilt of potatoes better, continuous development of resistant varieties is needed (Fock et al., 2001; Champoiseau et al., 2010).

Race 3 (R3bv2A) has appeared in many fingerprinting studies to be very homogeneous (EPPO, 2004). However, studies have shown that there are differences among R3bv2A isolates from different parts of the world (Smith et al., 1995; EPPO, 2004) yet the previous breeding work in developing resistant cultivars has been taking place in centralised places. According to these studies, there are differences between South American R3bv2A isolates on the one hand and those from the rest of the world on the other (Smith et al., 1995; EPPO, 2004). Isolates from South America belong to one largely heterogeneous group, while those from the other parts of the world consist of one homogeneous group (Smith et al., 1995). Therefore, although potato clones with moderate level of resistance to R3bv2A have been developed by CIP scientists, the expression of this resistance is likely to be different in Kenya, because the resistance is race and environment specific. Thus, use of potato germplasm that conforms to regional geographic boundaries is necessary for a successful local potato breeding programme. Because a race at one location may overcome the resistance effective at another location (Grimsley & Hanson, 1998) and more than one race may occur in a given field (Martin & French, 1985) an essential step in the development of resistant varieties is local screening (Martin & French, 1985).

6.4.2 Inheritance of Resistance to Bacterial Wilt

It was reported that the resistance to bacterial wilt from *Solanum phureja* is controlled by a few genes (Martin & French, 1985); by three independent and dominant major genes, and that both additive and non-additive gene actions are important in the inheritance of the resistance (Rowe & Sequeira, 1970). Later, it was reported that this resistance was controlled by at least four major genes (French et al., 1998; Grimsley & Hanson, 1998). Other studies reported that both major and minor genes are involved in the expression of resistance to wilt; and inheritance of resistance to wilt involves both additive and non-additive gene actions (Tung et al., 1993; Tung & Schmiediche, 1995). Previous results indicated that resistance to bacterial wilt in potato is a partially dominant character, and in its inheritance, interlocus gene interactions are important (Tung et al., 1992b; 1993). Other studies indicated that the resistance is polygenic and quantitative in nature, and involve genes with major and genes with minor effects (Tung et al., 1993; Cook & Sequeira, 1994). There is also evidence that in the inheritance of resistance to wilt, non-additive gene action is important, and is largely of the epistatic type (Tung et al., 1992a; 1992b; 1993). Some other reports (Tung, 1992) showed that the non-additive variance component for disease severity was 4.5 times more than additive component and a large proportion of non-additive variance was epistasis. Therefore, breeding schemes designed to make use of both additive and non-additive gene actions seem most suitable in developing resistance. Moreover, the genetic background for adaptation is of crucial importance for expression of resistance (Schmiediche, 1985a; 1985b; Kloos & Fernandez, 1986; Tung et al., 1990; 1992a; 1992b). More evidence showed that the resistance to bacterial wilt in potatoes is very complex in nature; it is probably a function of environmental adaptation (Schmiediche, 1985a; Kloos & Fernandez, 1986) with genes for adaptation being involved (Tung et al., 1990b; 1992a; 1992b). There is a large amount of interaction between genes for resistance and those for adaptation (Tung et al., 1992a; 1992b), and combining

ability seems to be a considerable feature of the resistance (Tung et al., 1990b). Therefore, potato clones with a wide genetic background for both bacterial wilt resistance and adaptation tend to display a higher level of resistance, which is more stable over environments (Tung et al., 1993). Good adaptation of the potential host to a particular environment is likely to strengthen expression of the resistance to wilt (Tung et al., 1990; 1992a). In order to develop a stable resistance in potato populations, a wide genetic base for resistance and adaptation to the environment where the pathogen occurs would therefore be necessary (Tung et al., 1993). Incorporation of several sources of resistance into a genotype/population with wide adaptation has been thought to stabilize the resistance expression under diverse pathogenic conditions (Sequeira, 1979; Schmiediche & Martin., 1983).

Resistance to bacterial wilt both derived from *S. phureja* and in general tends to break down at high temperatures (French & Lindo, 1982; Tung et al., 1993). This strong interaction between genes for heat tolerance and those for resistance implies the presence of a large amount of favourable non-additive (epistatic) gene effects in expression of high resistance (Schmiediche & Martin., 1983; Tung et al., 1992a; 1993). Thus, breeding at the population level by incorporating multiple sources of resistance and heat tolerance should be effective in producing superior potato genotypes, suitable for production in the lowland tropics where high levels of bacterial wilt resistance and heat tolerance are much needed (Tung et al., 1993.).

For attaining a high level of stable resistance, recurrent selection employing both additive and non-additive gene actions, in a population with the widest genetic background possible for both resistance and adaptation, seems to be the most promising approach (Tung et al., 1990; 1993). In addition, testing for resistance and adaptation should be done right at the location where the cultivars are destined to be grown (Martin & French, 1985; Tung et al., 1992a; 1993; Cook & Sequeira, 1994).

6.4.3 Sources of Resistance

Some wild or cultivated species related to *Solanum tuberosum* are known to be highly tolerant to bacterial wilt, and thus are potential sources of resistance (Fock et al., 2001). Unfortunately, hybrids of potato with resistant genotypes of the wild *Solanum chacoense*, *Solanum sparsipillum*, and *Solanum multidissectum* revealed some traits of wildness such as high glycoalkaloid content in addition to moderate levels of resistance to bacterial wilt (French et al., 1998). Unlike these wild species, the cultivated *Solanum phureja* is phylogenetically close to *Solanum tuberosum*, and displays resistance traits which are dominant and readily transmitted to progeny (Fock et al., 2001). However, this resistance is overcome by high temperature (French & Lindo, 1982; French, 1994; French et al., 1998). Other sources of resistance which have been evaluated, albeit at experimental level, are *S. stenotomum* L (cultivated), and *S. commersonii* Dun, which is wild (Laferriere et al., 1999; Fock et al., 2000; 2001; Carputo et al., 2009). They too show moderate resistance and their hybrids harbour latent infection (Laferriere et al., 1999; Fock et al., 2000; 2001).

Recent developments in the search for resistance offer promise. The CIP scientists have recently developed some improved potato clones that are moderately resistant to R3bv2A, although the clones have not been tested extensively (Bonierbale Merideth, personal communication, 2010). In addition, Ateka et al. (2001) found that potato varieties, Kenya Baraka, Kenya Sifa, Kenya Karibu, Kenya Dhamana, Mauritius (CIP- 89016), and Cruza-148 (CIP-720118) were more resistant to bacterial wilt than all the other varieties grown in Kenya. They also found that the most productive and most popular potato varieties, Tigoni, Nyayo, and Dutch Robyjin were the most susceptible. Therefore, introgression of resistance from the more resistant CIP and Kenyan potato germplasm into the more productive, more popular yet more susceptible Kenyan varieties may improve potato production in Kenya.

7. Conclusions

Management of bacterial wilt in potatoes calls for an integrated approach. Plant resistance is an important component of the integrated disease management. However, although many potato varieties have been found to have some degree of resistance to the disease, they still transmit latent infection. Introgression of resistance from various sources may result in high resistant varieties. For better results, use of high resistant varieties may be coupled with use of disease-free tubers and clean fields. Because there is a strong host-pathogen-environment interaction effect in the expression of resistance, an essential step in the development of resistant varieties is local screening.

References

- Ajanga, S. (1993). Status of bacterial wilt of potato in Kenya. In G. L. Hartman, & A.C. Hayward (Eds.). *Proceedings of International Conference on Bacterial Wilt*, 28-30 October 1992, Kaohsiung, Taiwan (pp. 338-340). Australian Centre for International Agricultural Research, Canberra, Australia.

- Anguiz, R. J., & Mendoza, H. A. (1997). General and specific combining abilities for resistance to bacterial wilt (*Pseudomonas solanacearum* E.F. Smith) in PVX and PVY immune autotetraploid potatoes. *Fitopatologia*, 32, 71-80.
- ANN. (2009). Kenya to give renewed attention to potato cultivation [Online] Available at <http://www.africanagricultureblog.com/> (Accessed 5 July 2010). Africa News Network, Nairobi, Kenya.
- Ateka, E. M., Mwang'ombe, A. W., & Kimenju, J. W. (2001). Reaction of potato cultivars to *Ralstonia solanacearum* in Kenya. *African Crop Science Journal*, 9, 251-256.
- Barton, D., Smith, J., & Murimi, Z. K. (1997). *Socio-economic inputs to biological control of bacterial wilt diseases of potato in Kenya*. ODA RNRRS Crop protection: R66292 (NR International: ZA0085).
- Buddenhagen, I., & Kelman, A. (1964). Biological and physiological aspects of bacterial wilt caused by *P. solanacearum*. *Annual Review of Plant Pathology*, 2, 203-230.
- Buddenhagen, I. W. (1986). Bacterial wilt revisited. In G.J. Persley (Ed.) *Bacterial wilt disease in Asia and the South Pacific. Proceedings of an International Workshop on Bacterial wilt*, 8-10 October 1985, PCARRD, Los Banos, Philippines (pp. 126-143). Australian Centre for International Agricultural Research, Canberra, Australia.
- Buddenhagen, I. W., Sequeira, L., & Kelman, A. (1962). Designation of races of *Pseudomonas solanacearum*. *Phytopathology*, 52, 726.
- Carpato, D., Aversano, R., Barone, A., Matteo, A., Iorizzo, M., Sigillo, L., Zoina, A., & Frusciante, L. (2009). Resistance to *Ralstonia solanacearum* of sexual hybrids between *Solanum commersonii* and *Solanum tuberosum*. *American Journal of Potato Research*, 8, 196-202.
- Champoiseau, P. G. (2008). Brown rot of potato [Online] Available at http://plantpath.ias.ufl.edu/rsol/Trainingmodules/BRPotato_Module.html (Accessed 26 June 2010). United States Department of Agriculture-National Research Initiative Program. Madison, WI.
- Champoiseau, P. G., Jones, J. B., & Allen, C. (2009). *Ralstonia solanacearum* race 3 biovar 2 causes tropical losses and temperate anxieties [Online]. Available at <http://www.apsnet.org/online/feature/ralstonia/> (Accessed 25 June 2010). American Phytopathological Society. Madison, WI.
- Champoiseau, P. G., Jones, J. B., Momol, T. M., Pingsheng, J., Allen, C., Norman, D. J., ... Caldwell, K. (2010). *Ralstonia solanacearum* Race 3 biovar 2 causing brown rot of potato, bacterial wilt of tomato and southern wilt of geranium [Online]. Available at http://plantpath.ifas.ufl.edu/rsol/NRI_Project/Projectsummary.html (Accessed 25 June 2010), American Phytopathological Society. Madison, WI.
- Ciampi, L., & Sequeira, L. (1980). Influence of temperature on virulence of race 3 strains of *Pseudomonas solanacearum*. *American Potato Journal*, 57, 307-317.
- CIP. (2007). *Procedures for standard evaluation trials of advanced potato clones. An International Cooperators' Guide*. CIP, Lima, Peru. 124 pages
- Cook, D., & Sequeira, L. (1994). Strain differentiation of *Pseudomonas solanacearum* by molecular genetic methods. In A.C. Hayward & G.L. Hartman (Eds.) *Bacterial wilt: The disease and its causative agent, Pseudomonas solanacearum* (pp. 77-93). CAB International, Wallingford, UK.
- Denny, T. P. (2006). Plant pathogenic *Ralstonia* species. In S.S. Gnanamanickam (Eds.) *Plant-Associated Bacteria* (pp. 573-644). Springer Publishing, Dordrecht, The Netherlands.
- EPPO. (2004). *Ralstonia solanacearum*. *European and Mediterranean Plant Protection Organization Bulletin* 34, 173-178.
- Fegan, M., & Prior, P. (2005). How complex is the "*Ralstonia solanacearum* species complex"? In C. Allen, P. Prior, & A. C. Hayward (Eds.) *Bacterial wilt disease and the Ralstonia solanacearum species complex* (pp. 449-461). American Phytopathological Society, St. Paul, MN.
- Fock, I., Collonnier, C., Purwito, A., Luisetti, J., Souvannavong, V., Vedel, F., ... Sihachakr. D. (2000). Resistance to bacterial wilt in somatic hybrids between *Solanum tuberosum* and *Solanum phureja*. *Plant Science*, 160, 165-176.
- Fock, I., Collonnier, C., Luisetti, J., Purwito, A., Souvannavong, V., Vedel, F., ... Sihachakr. D. (2001). Use of *Solanum stenotomum* for introduction of resistance to bacterial wilt in somatic hybrids of potato. *Plant Physiology and Biochemistry*, 3, 899-908.

- French, E. R. (1994). Strategies for integrated control of bacterial wilt of potatoes. In A.C. Hayward & G.L. Hartman (Eds.) *Bacterial wilt: The disease and its causative agent, Pseudomonas solanacearum* (pp. 98-113). CAB International, UK.
- French, E. R., & Lindo, L. D. (1982). Resistance to *Pseudomonas solanacearum* in potato: Specificity and temperature sensitivity. *Phytopathology*, 72, 1408-1412.
- French, E. R., Gutarra, L., & Vilchez, G. (1975). Field survival of *Pseudomonas solanacearum* race 3 in Peru. *CIP Technical Information Bulletin*, 7, 1-6.
- French, E. R., Anguiz, R., & Aley, P. (1998). The usefulness of potato resistance to *Ralstonia solanacearum* for the integrated control of bacterial wilt. In P. H. Prior, C. Allen, & J. Elphinstone (Eds.) *Bacterial wilt disease: Molecular and ecological aspects. Report of the Second International Wilt Symposium*, 22-27 June 1997, Gosier, Guadeloupe, France (pp. 381-385). Springer-Verlag, Berlin.
- French, E. R., Gutarra, L., Aley, P., & Elphinstone, J. (1995). Culture media for *Pseudomonas solanacearum*: isolation, identification and maintenance. *Fitopatologia*, 30, 126-130.
- Gadewar, A. V., Kishore, V., Sunaina, V., Charbarti, S. K., & Shekhawat, G. S. (1993). Integrated management of bacterial wilt. In B. Hardy & E.R. French (Eds.) *Integrated management of bacterial wilt. Proceedings of an International Field Workshop*, 11-16 October 1993, New Delhi, India (pp. 137-143). Australian Centre for International Agricultural Research, Canberra.
- Gildemacher, P., Demo, P., Kinyae, P., Nyongesa, M., & Mundia, P. (2007). Selecting the best plants to improve seed potato. *LEISA*, 2, 10-11.
- Graham, J., & Lloyd, A. B. (1979). Survival of potato strain (race 3) of *Pseudomonas solanacearum* in the deeper soil layers. *Australian Journal of Agricultural Research*, 30, 489-496.
- Granada, G. A., & Sequeira, L. (1983). Survival of *Pseudomonas solanacearum* at low temperatures. *Fitopatologia*, 18, 22-24.
- Grimsley, N., & Hanson, P. (1998). Genetics of plant resistance to bacterial wilt: Round table report. p. 263-266. In P. Prior, C. Allen, & J.G. Elphinstone (Eds.) *Bacterial wilt disease: Molecular and ecological aspects. Report of the Second International Wilt Symposium*, 22-27 June 1997, Gosier, Guadeloupe, France (pp. 381-385). Springer-Verlag, Berlin.
- Harris, O. C. (1976). Bacterial wilt in Kenya with particular reference to potatoes. In L. Sequeira & A. Kelman (Eds.) *Proceedings of the first international planning conference and workshop on the ecology and control of bacterial wilt caused by Pseudomonas solanacearum*. 18-24 January 1976. Raleigh, North Carolina (pp. 84-88). Springer-Verlag, Berlin.
- Hayward, A. C. (1964). Characteristics of *P. solanacearum*. *Journal of Applied Bacteriology*, 27, 265-265.
- Hayward, A. C. (1983). *Pseudomonas solanacearum*: Bacterial wilt and moko disease. In P. C. Fahy & G. J. Persley (Eds.) *Plant bacterial diseases* (pp.129-135). Academic Press, Sydney, Australia.
- Hayward, A. C. (1991). Biology and epidemiology of bacterial wilt caused by *Pseudomonas solanacearum*. *Annual Review of Phytopathology*, 2, 65-87.
- Hayward, A. C. (2000). *Ralstonia solanacearum*. Encyclopedia of microbiology. Vol. 4, 2nd ed. Academic Press, London.
- Jackson, M. T., & González, L. C. (1981). Persistence of *Pseudomonas solanacearum* (Race 1) in a naturally infested soil in Costa Rica. *Phytopathology*, 71, 690-693.
- Janse, J. D. (1991). Infra- and intraspecific classification of *Pseudomonas solanacearum* strains using whole cell fatty acid analysis. *System. Applied Microbiol.*, 1, 335-345.
- Kaguongo, W. P., Ng'ang'a, N. M., Muthoka, N., Muthami, F., & Maingi, G. (2010). *Seed potato subsector master plan for Kenya (2009-2014)*. Seed potato study sponsored by GTZ-PSDA, USAID, CIP and Government of Kenya. Ministry of Agriculture, Kenya.
- Kaguongo, W. P., Gildemacher, P., Demo, P., Wagoire, W., Kinyae, P., Andrade, J., ... Thiele, G. (2008). *Farmer practices and adoption of improved potato varieties in Kenya and Uganda. Social Sciences Working Paper 2008-5*. International Potato Centre (CIP), Lima, Peru. 84 pages
- Kelman, A. (1953). Bacterial wilt caused by *Pseudomonas solanacearum*. A literature review and bibliography. Springer-Verlag, Berlin.

- Kinyua, Z. M., Smith, J. J., Odou, G. I., & Wachira, J. N. (1998). Increasing the availability of disease-free potato seed tubers to smallholder farmers in Kenya. In M. O. Akoroda & J. M. Ngeve (Eds.) *Proceedings of the 8th Triennial Congress of the International Symposium for Tropical Root Crops-African Branch*, 11-17 June 1998, Cotonou, Benin (pp. 494-498). ISTRC-AB, Lilongwe, Malawi.
- Kinyua, Z. M., Smith, J. J., Lung'aho, C., Olanya, M., & Priou, S. (2001). On-farm success and challenges of producing bacterial wilt free tubers in seed plots in Kenya. *African Crop Science Journal*, 9, 279-285.
- Kloos, J. P., & Fernandez, B. B. (1986). Evaluation of potato germplasm for resistance to *Pseudomonas solanacearum* (E. F. Smith) and adaptation for Mindanao. *Philippines Agriculture*, 6, 263-276.
- Laferriere, L. T., Helgeson, J. P., & Allen, C. (1999). Fertile *Solanum tuberosum* + *Solanum commersonii* somatic hybrids as sources of resistance to bacterial wilt caused by *Ralstonia solanacearum*. *Theoretical and Applied Genetics*, 98, 1272-1278.
- Lemaga, B. (1997). Integrated control of potato bacterial wilt. Literature review and work plan 1995-1997. *The African Highlands Initiative Technical Report*. Series No.3. International Centre for Research on Agroforestry (ICRAF), Nairobi, Kenya.
- Lemaga, B., Kanzikwera, R., Kakuhenzire, R., Hakiza, J. J., & Maniz, G. (2001). The effect of crop rotation on bacterial wilt incidence and potato tuber yield. *African Crop Science Journal*, 9, 257-266.
- Lemaga, B., Kakuhenzire, R., Kassa, B., Ewell, P. T., & Priou, S. (2005). Integrated control of potato bacterial wilt in eastern Africa: The experience of African Highlands Initiative. In C. Allen, P. Prior, & A.C. Hayward (Eds.) *Fate of Ralstonia solanacearum biovar 2 as affected by conditions and soil treatments in temperate climate zones* (pp. 145-157). American Phytopathological Society, St. Paul, Minnesota, USA.
- Low, J. W. (1997). Potato in south-western Uganda. Threats to sustainable production. *African Crop Science Journal*, 5, 295-412.
- Lung'aho, C., M'makwa, C., & Kidanemariam, H. M. (1997). Effect of source of mother plant, variety, and growing conditions on the production of stem cuttings and subsequent yield of mini-tubers in the Kenyan potato programme. In *Proceedings of the 4th Triennial Congress of the African Potato Association*, 23-28 February 1997, Pretoria, South Africa (pp. 72-91). African Potato Association, Kampala, Uganda.
- Martin, C., & French, E. R. (1985). Bacterial wilt of potatoes caused by *Pseudomonas solanacearum*. *CIP Technical Information Bulletin*, 13, 1-6. CIP, Lima, Peru.
- Messih, N. A. S. (2006). *Bacterial wilt of potato (Ralstonia solanacearum race 3, biovar 2): Disease management, pathogen survival, and possible eradication*. Ph.D. Thesis, Wageningen University, The Netherlands.
- Milling, A., Meng, F., Denny, T. P., & Allen, C. (2009). Interactions with hosts at cool temperatures, not cold tolerance, explain the unique epidemiology of *Ralstonia solanacearum* race 3 biovar 2. *Phytopathology*, 99, 1127-1134.
- MOA. (2005). *National policy on potato industry. Policy and reforms in the industry to improve production, research, marketing, and regulatory framework*. Ministry of Agriculture, Nairobi, Kenya.
- MOA. (2008). *National policy on potato industry. Policy reforms to revitalize the potato industry*. Ministry of Agriculture, Nairobi, Kenya.
- Muthoni, J., Mbiyu, M. W., & Nyamongo, D. O. (2010). A review of potato seed systems and germplasm conservation in Kenya. *Journal of Agricultural and Food Information*, 11, 157-167.
- Nyangeri, J. B., Gathuru, E. M., & Mukunya, D. M. (1984). Effect of latent infection and the spread of bacterial wilt of potatoes in Kenya. *Tropical Pest Management*, 30, 103-105.
- Otipa, M. J., Wakahiu, M. W., Kinyae, P. M., Thuo, D. M., & Kinoti, J. I. (2003). *Survey of the bacterial wilt of potatoes caused by Ralstonia solanacearum and its spread in major potato growing areas of Kenya*. Task Force Report. KARI, Nairobi, Kenya.
- Pradhanang, P. M. (1999). Transmission of *Ralstonia solanacearum* through drainage water. *Bacterial Wilt Newsletter*, 16, 5-7. CIP, Lima Peru.
- Pradhanang, P. M., Elphinstone, J. G., & Fox, R. T. V. (2000). Identification of crop and weed hosts of *Ralstonia solanacearum* biovar 2 in the hills of Nepal. *Plant Pathology*, 4, 403-413.

- Prior, P., & Fegan, M. (2005). Recent developments in the phylogeny and classification of *Ralstonia solanacearum*. *Acta Horticulturae*, 69, 127-136.
- Priou, S., Aley, P., Chujoy, E., Lemaga, B., & French, E. R. (1999). *Integrated management of bacterial wilt of potato*. CIP slide training series. International Potato Center, Lima, Peru.
- Priou, S., Salas, C., Mendiburu, F., Aley, P., & Gutarra, L. (2001). Assessment of latent infection frequency in progeny tubers of advanced potato clones resistant to bacterial wilt: A new selection criterion. *Potato Research*, 44, 359 - 373.
- Rowe, P. R., & Sequeira, L. (1970). Inheritance of resistance to *Pseudomonas solanacearum* in *Solanum phureja*. *Phytopathology*, 6, 1499-1501.
- Saumtally, J. S., Autrey, L. J. C., Ferre, P., & Dookun, A. (1993). Disease management strategies for the control of bacterial wilt (*Pseudomonas solanacearum*) of potato in Mauritius. In G.L. Hartman & A.C. Hayward (Eds.) *Proceedings of an International Conference on Bacterial wilt*, 28-30 October 1992, Kaohsiung, Taiwan (pp. 289-293). Australian Centre for International Agricultural Research, Canberra.
- Schmiediche, P. (1985a). Breeding bacterial wilt, *Pseudomonas solanacearum* resistant germplasm. In *Present and future strategies for potato breeding and improvement. Report of a Planning Conference* (pp. 45-55). CIP. Lima, Peru.
- Schmiediche, P. (1985b). Breeding potato for resistance to bacterial wilt caused by *Pseudomonas solanacearum*. In *Proceedings of an International Workshop on Bacterial Wilt Disease in Southeast Asia and the Pacific*, 8-10 October 1985, Los Banos, the Philippines (pp.105-111). ACIAR Canberra, Australia.
- Schmiediche, P., & Martin, C. (1983). Widening of the genetic base of the potato for resistance to bacterial wilt (*Pseudomonas solanacearum*). In *Present and future strategies for potato breeding and improvement. Report on Planning Conference*, 12-14 April 1982, CIP, Lima, Peru (pp. 172-173). CIP, Lima, Peru.
- Schmiediche, P., & Martin, C. (1986). The use of wild species in breeding for resistance to bacterial wilt (*Pseudomonas solanacearum*). *American Potato Journal*, 63,453.
- Seal, S.E., Taghavi, M., Fegan, N., Hayward, A. C., & Fegan, M. (1999). Determination of *Ralstonia (Pseudomonas) solanacearum* rDNA subgroups by PCR tests. *Plant Pathology*, 48, 115-120.
- Sencviratnc, S. N. S. (1988). Soil survival of *Pseudomonas solanacearum*. In *Bacterial diseases of the potato. Report of the planning conference on bacterial diseases of the potato* (pp.85-91). CIP, 1987.
- Sequeira, L. (1979). Development of resistance to bacterial wilt derived from *Solanum phureja*. In *Developments in control of potato bacterial diseases* (pp. 55-62). CIP. Lima, Peru.
- Sequeira, L., & Rowe, P. R. (1969). Selection and utilization of *Solanum phureja* clones with high resistance to different strains of *Pseudomonas solanacearum*. *Ame. Potato J.*, 46, 451-462.
- Shekhawat, G. S., & Chakrabarti, S. K. (1993). Integrated management of potato bacterial wilt. In B. Hardy & E.R. French (Eds.) *Integrated management of bacterial wilt. Proceedings of an International Field Workshop*, 11-16 October 1993, New Delhi, India (pp. 87-93). CIP, Lima, Peru.
- Smith, J. J., Offord, L. C., Holderness, M., & Saddler, G. S. (1995). Genetic diversity of *Bulkholderia solanacearum* (Syn. *Pseudomonas solanacearum*) race 3 in Kenya. *Applied and Environmental Microbiology.*, 61, 4263-4268.
- Smith, J. J., Offord, L. C., Holderness, M., & Saddler, G. S. (1998). The development of biological control against race 3 in Kenya. In P. Prior, C. Allen, & J. Ephinstone (Eds.) *Bacterial wilt disease: Molecular and ecological aspects. Report of the Second International Wilt Symposium*, 22-27 June 1997, Gosier, Guadeloupe, France (pp. 337-342). Springer-Verlag, Berlin, Germany.
- Sunaina, V., Kishore, V., & Shekhawat, G. S. (1989). Latent survival of *Pseudomonas* in potato tubers and weeds. *Journal of Plant Disease Protection*, 96, 361-364.
- Todd, J. M. (1969). A prospect of potato growing in Kenya. Annual Report. Ministry of Overseas Development, London.
- Tung, P. X. (1992). Genetic variation for bacterial wilt resistance in a population of tetraploid potato. *Euphytica*, 61, 73-80.
- Tung, P. X., & Schmiediche, P. (1995). Breeding for resistance to bacterial wilt caused *Pseudomonas solanacearum*: Looking for stable resistance. In B. HARDY AND E.R. FRENCH (Eds.) *Integrated*

- management of bacterial wilt. Proceeding of An International workshop*, 11-16 October 1993, New Delhi, India (pp. 173-178). CIP, Lima, Peru.
- Tung, P. X., Rasco, E. T., Zaag, P. V. & Schmiediche, P. (1990). Resistance to *Pseudomonas solanacearum* in the potato: I. Effects of sources of resistance and adaptation *Euphytica*, 45, 203-210.
- Tung, P. X., J. R., E. T. R., Zaag, P. V., & Schmiediche, P. (1990). Resistance to *Pseudomonas solanacearum* in the potato: II . Aspects of host-pathogen-environment interaction *Euphytica*, 45, 211-215.
- Tung, P. X., Hermesen, J. G. T., Zaag, P. V. D., & Schmiediche, P. (1992a). Effects of heat tolerance on expression of resistance to *Pseudomonas solanacearum* E. F. Smith in potato. *Potato Research*, 35, 321-328.
- Tung, P. X., Hermesen, J. G. T., Zaag, P. V. D., & Schmiediche, P. (1993). Inheritance of resistance to *Pseudomonas solanacearum* E.F. Smith in tetraploid potato. *Plant Breeding*, 111, 23-30.
- Tung, X. P., Hermesen, J. G. T., Zaag, P. V., & Schmiediche, P. (1992b). Effects of resistance genes, heat tolerance genes and cytoplasm on expression of resistance to *Pseudomonas solanacearum* (E. F. Smith) in potato. *Euphytica*, 60, 127-138.
- Wakahiu, M. W., Gildemacher, P. R., Kinyua, Z. M., Kabira, J. N., Kimenju, A. W., & Mutitu, E. W. (2007). Occurrence of potato bacterial wilt caused by *Ralstonia solanacearum* in Kenya and opportunities for intervention. In *7th Triennial African Potato Association Conference*, 13-18 July 2007, Alexandria, Egypt (pp. 267-271). African Potato Association, Kampala, Uganda.
- Wenneker, M., Verdel, M. S. W., Beuningen, A. R. V., Derks, J. H. J., & Janse, J. D. (1999). *Ralstonia (Pseudomonas) solanacearum* race 3 (biovar 2) in surface water and natural weed hosts: First report on stinging nettle (*Urtica dioica*). *European Journal of Plant Pathology*, 105, 307-315.
- Yabuuchi, E., Kosako, Y., Yano, I., Hotta, H., & Nishiuchi. Y. (1995). Transfer of two *Burkholderia* and an *Alcaligenes* species to *Ralstonia* genus nov.: proposal of *Ralstonia pickettii* (Ralston, Palleroni and Douderoff 1973) comb.nov., *Ralstonia solanacearum* (Smith 1896) comb. nov. and *Ralstonia eutropha* (Davis 1969) comb. nov. *Microbiology and Immunology*, 39, 897-904.