BIBLIOGRAPHY

BASNGI, JAMIE BETH B. March 2006. Germplasm Evaluation for Tolerance Against Bacterial Wilt (*Ralstonia solanacearum* (E.F. Smith) Yabuuchi *et al.*). Benguet State University, La Trinidad, Benguet.

Adviser: Jocelyn C. Perez, MSc.

ABSTRACT

Nine clones: 387410.7, Warishiro, 575003, 676070, 380579.3, 720045, 573275, 285378.27 and 720071 and variety Igorota were screened to identify potato germplasm materials tolerant to bacterial wilt infection and resistant to late blight infection under green house condition.

All entries were found to be tolerant to bacterial wilt as they were able to produce marketable tubers despite the bacterial wilt infection recorded.

The clones that showed resistance to late blight infection were clones 720071, 285378.27 and 676070 respectively.

TABLE OF CONTENTS

INTRODUCTION

Nature of the Study

Potato (*Solanum tuberosum* Linn) is grown worldwide due to its nutritive and money making value. It is one of the mankind's most valuable food because it provides a source of low cost energy to human diet and a source of minerals, proteins, vitamin A and B_2, carbohydrates and some elements like potassium and phosphorus (Kipps, 1979).

A single medium-sized potato contains about half the daily adult requirement of vitamin C. Potato is very low in fat, with just 5 percent of the fat content of wheat, and one-fourth the calories of bread. When it is boiled, it has more protein than maize, and nearly twice the calcium (CIP, 1996).

In the Philippines, potato ranks third among the leading commercial vegetables in terms of peso value and ranks first among the vegetable crops in Northern Luzon (Buasen, 1978). In like manner, it ranks fourth among the major crops worldwide after wheat, maize and rice.

However, different pest and diseases affect the production of potato. Bacterial wilt caused by *Ralstonia solanacearum* (E.F. Smith) Yabuuchi *et al.* is one of the most important, widespread and lethal bacterial diseases of plants (Ma, 1990). It limits the production of potatoes, especially seed potatoes worldwide (CIP, 1992; Martin and French, 1985; Schmiediche, 1984 and Rich, 1983).

Kelman (1953) considered the earliest known record of bacterial wilt to be on tobacco in Indonesia in 1864, where entire fields were lost due to bacterial wilt. Bacterial

wilt was recorded on potatoes in the United States in 1890 (Kelman, 1953) and in Australia in 1894 (Tyron, 1895).

The disease was reported in the Philippines by Reinking (1918) affecting tobacco, pepper and tomato as cited by Valdez (1986). In 1922, Welles and Roldan reported that it caused a serious disease on solanaceous crops in Southern Luzon. Series of study conducted reveals that it became severe throughout the highland and lowland areas producing potato (Perez *et al*., 1997).

The survival of the bacterium is influenced by temperature, humidity and other physical and chemical soil factors. *R. solanacearum* may survive for many years in certain soils and may disappear from one growing season to the next (Martin and French, 1996).

Bacterial wilt can be reduced only if various control components are combined. An integrated disease management approach can lead to significant reduction or even eradication of bacterial wilt (CIP, 1996).

Among the practices recommended, the use of resistant cultivars is considered to play an important role. It is proven useful to control the potato strain, and is potentially the most effective and ideal way to manage the disease (Jyothi *et. al*., 1993; French, 1996). It also delays the development of populations of insects resistant to the pesticide currently being used (Ganga, 1992).

The study was conducted to screen potato clones and cultivars for tolerance to bacterial wilt under green house condition, and evaluate their reaction to late blight infection.

The study was conducted at the bacterial wilt nursery, the Northern Philippines Root Crops Research and Training Center (NPRCRTC) and the Department of Plant Pathology laboratory, Benguet State University from September 2005 to February 2006.

REVIEW OF LITERATURE

Causal Organism

The phytopathogen *Ralstonia solanacearum* has over 5000 genes, many of which probably facilitate bacterial wilt disease development (Brown and Allen, 2001). It is a gram-negative plant-pathogenic bacterium that causes bacterial wilt in a variety of plants (Hayward, 1994).

It is a soil-borne pathogen that naturally infects roots. It exhibits a strong and tissue-specific tropism within the host, specifically invading and highly multiplying in the xylem vessels (Smith, 1896 and Yabuuchi *et al.,* 1995). Once established in the xylem vessels, the bacteria are able to enter the intercellular spaces of the parenchyma cells in the cortex and pith in various areas of the plant. Here, *R. solanacearum* is able to dissolve the cell walls and create slimy pockets of bacteria and cell debris. Production of highly polymerized polysaccharides increases the viscosity of the xylem, which results in plugging (Shew and Lucas, 1991).

High temperatures and high soil moisture generally favors *Ralstonia solanacearum*, the exception being certain in Race 3 strains that are pathogenic on potato and are able to grow well at lower temperatures (Stevenson *et al.*, 2001). In certain soil, *R. solanacearum* may survive for many years, in others, the bacterium may disappear from one growing season to the next (Martin and French, 1996).

EPIDEMIOLOGY

Ralstonia solanacearum race 3 is a soil borne pathogen that persists in wet soils, deep soil layers (>75 cm), and reservoir plants (Van der Wolf and Perombelon, 1997). Its distribution in potato fields can be spotty, and is commonly found in areas that have poor drainage (Stevenson *et al.*, 2001). It is adapted to low temperatures, however its survival in very cold temperatures is reduced. In a study conducted in potato fields (Dirk van Elsas *et al.*, 2000), *R. solanacearum* race 3 biovar 2 population densities declined at 15 and 20°C and was severely reduced at 4°C. Severe drought negatively impacted population densities. Race 3 biovar 2 is most severe between 24-35°C (optimal temperature of 27°C) and decreases in virulence when temperatures exceed 35 °C or fall below 10 °C (Stansbury *et al.*, 2001). In regions such as Australia, England, Kenya, and Sweden the organism was not detected in previously diseased potato fields after two years, suggesting that long-term survival in temperate regions is reduced (Van der Wolf and Perombelon, 1997). In another study the bacterium persisted for 12 months in potato fields (Dirk van Elsas *et al.*, 2000). It is spread through infected potato tubers and can move plant-to-plant through the soil (Stevenson *et al.*, 2001).

Host Range and Distribution

Ralstonia solanacearum is a widely distributed pathogen found in tropical, subtropical, and some temperate regions of the world (Fegan and Prior, 2004). The species as a whole has a very broad host range and infects hundreds of species in many plant families. The majority of hosts are dicots with the major exception being bananas

and plantains. Most economically important host plants are found in the *Solanaceae* or nightshade family (Stevenson *et al.*, 2001).

Specific host range and distribution of *R. solanacearum* depends on the race and to some degree the biovar of the pathogen (Daughtrey, 2003). These host ranges and distributions have been changing in recent years. Race 1 is endemic to the southern United States. Race 3 Biovar 2 is a USDA "select agent" listed on the Agricultural Bioterrorism Act of 2002 and is subject to regulatory actions including strict quarantines since potato is extremely sensitive to this race (Ji *et al.*, 2004).

Table below illustrates the relationship of race, biovar, host range, and geographic distribution (Daughtrey, 2003).

Race	Host Range	Geographic distribution	Biovar
1	Wide	Asia, Australia	3, 4
		Americas	1
2	Banana, other *Musa* spp.	Caribbean, Brazil, Worldwide	1
3	Potato, some other Solanaceae, Geranium; few other species	Worldwide except US and Canada	2
4	Ginger	Asia	3,4
5	Mulberry		

Management

The great variability of *Ralstonia solanacearum* and the strong influence of environmental conditions on resistance make bacterial wilt a disease difficult to manage (French, 1996).

Among the practices recommended, the use of resistant cultivars is considered to play an important role. It is proven useful to control the potato strain, and is potentially

the most effective and ideal way to manage the disease (Jyothi *et al*., 1993; French, 1996). However, despite its importance, little progress has been made by either nationals or international potato breeding program in achieving stable resistance in commercial genotypes (Martin and French, 1996; Lopes and Quezado-Soarez, 1994).

Resistance is not general, but pathogen specific; a pathogen at one location may over come the resistance effective at another location. More than one pathogen may occur in a given field. Existence of different pathogen within races may reduce effectively resistance at certain location, acceptance level of resistance should be determined for definite use. When use for consumption, a certain percentage of infection may be tolerated. But if seed production, it is preferable not to tolerate any bacterial wilt, because few infested seed tubers can spread the disease over a wide area. Since the expression of resistance is pathovar, and environmental specific, an essential step in development of resistant varieties is local screening (Martin and French, 1996).

Selection for Resistance to Bacterial Wilt

The first thing to consider when planting potato must be the variety. One must choose a variety that is adaptable to the locality in order to achieve maximum production. Using the right variety ensures high yield and better quality. Series of varietal evaluation must be conducted to determine the performance of a new or previously untried variety (HARRDEC, 1996).

Clonal evaluation and selection is important in the breeding program. The standard procedure involves the selection of healthy looking and high yielding plants in the field. Tubers of each selected clones are then harvested and kept separately to be

planted in the next season. Plants are carefully inspected for any abnormalities, and if found in the first generation (F_1), their clones are rejected and removed right away from the field. Hence, successful potato production begins with long-term labor and intensive breeding (Beukema, 1985).

Kurupuaracchi (1995) conducted an on farm potato evaluation and found that not all clones were as superior as those in the on station trail. Out of the 22 clones, only 2 clones exhibited comparable level of yield ability, adaptability and stability with their popular local variety. Thus, as a role, new clones usually differ in cultural characteristics; therefore, several series of evaluations must be made at different strategic locations and seasons.

Barrozo (2001), among the twelve potato varieties screened against bacterial wilt under green house condition revealed that all varieties were resistant as no above ground symptoms were exhibited. However, belowground symptom showed that all cultivars were infected.

> The study conducted by Botangen (2003) at Bosleng, Paoay, Atok, Benguet shows that all the seven clones evaluated differed in growth, pest and disease resistibility, yield as well as processing qualities. It is important to evaluate at different locations and seasons for further screening.

Simongo (1992) found that clone LBR 1-5 was the most prolific in apical cutting and tuberlet production among the several clones evaluated. On the different on farm location trials, the performance of the individual clone varies with clone LBR 1-9 being the most promising with an average yield of 21.2t/ha and showed high resistance to late blight, good eating and tuber qualities.

Potato Breeding for Resistance to Bacterial wilt

The cultivated potato, *Solanum tuberosum* subsp. *tuberosum* (2n = 4x = 48), is one of the most important world food crops and demands continued genetic improvement to meet the needs of a changing world. Potato breeding has been a cumbersome task due to inherent biological factors: cytoplasmic nuclear sterilities, tetrasomic inheritance and inbreeding depression. In addition, yield stasis exists within the potato germplasm of North America (Douches, 1996).

The first systematic potato-breeding program for resistance to bacterial wilt started in 1967 by Rowe and Sequeira (1972) at the University of Wisconsin. These investigators began their program by intercrossing several resistant diploid clones of *Solanum phureja* from the Central Colombian Collection, with clones of S. *tuberosum* ssp. *tuberosum.* Field tests of *S. phureja* x spp. *tuberosum* hybrids revealed susceptibility of this genetic material to *Phytophthora infestans.* In a further set of crosses, the bacterial wilt-resistant hybrids were therefore crossed with Mexican late blight-resistant clones, combining the two needed resistances. The Mexican germplasm used in this crossing program consisted of ssp. *tuberosum* clones containing late blight resistance genes derived from the wild hexaploid species *S. demissum* (Schmiediche, 1986).

In 1969, the University of Wisconsin sent 369 clones, representing 10 families, to Peru where they were to be tested under natural conditions in fields heavily infected with *P. solanacearum.* French and Herrera started a screening program at Huambos in the Department of Cajamarca, Peru (Herrera, 1972). By 1974 the Wisconsin material had changed hands three times and only seven of the original 369 clones had survived. Most of the material had been lost due to causes other than bacterial wilt.

Apart from sending 369 clones of bacterial wilt resistant genetic material to Peru, the University of Wisconsin sent similar sets of material to 20 countries around the world. The success of the Wisconsin material is partly due to its high degree of heterozygosity, which results from use of unreduced gametes in the tertaploid x diploid crosses which were necessary components of this breeding program. The bacterial wilt resistance of this material comes from the diploid species *S. phureja.* Expression of this resistance at the tertaploid level becomes even more remarkable when the genomic composition of the tertaploid BR clones is considered. Only one genome, disregarding crossing over during meiosis, is *S. phureja* origin. Crossing BR clones with other tertaploid clones that are not resistant to bacterial wilt will obviously dilute the *S. phureja*-based resistance even more (Schmiediche, 1986).

The wild potato relative *Solanum commersonii* is reported to have good high-temperature resistance to brown rot of potatoes. However, *S. tuberosum* and *S. commersonii* have different ploidy and endosperm balance numbers and are therefore sexually incompatible, so their cells were fused by somatic hybridization. The resulting somatic hybrid plants were vigorous and potato-like in appearance, but their resistance level was unknown. Six of the somatic hybrids were examined, the *S. commersonii* and *S. tuberosum* parents, and cv. Atlantic with a virulent strain of *R. solanacearum* (race 3, biovar 2) at 28 ºC. The result reveals that *S. commersonii* was significantly more resistant to brown rot than the cultivated potatoes. Encouragingly, preliminary results show no significant difference in disease resistance between the somatic hybrids and the *S. commersonii* parent. The somatic hybrids are both self-compatible and fertile to *S. tuberosum* back-crosses (Laferriere *et al.*, 1997).

CIP Breeding Program

In 1977, data received from the National Potato Programs in CIP's regions had suggested that resistance based on *S. phureja* was effective against strains of *P. solanacearum* that affect potatoes, as long as the resistant material was grown under temperate climatic conditions. Such conditions can be found in tropical highland regions or in the plains of northern India during the winter.

Until 1980 the only source of resistance to bacterial wilt had been *S. phureja,* which had demonstrated its potential as well as its limitations. The narrow genetic base of this resistance had become an object of concern for breeders, pathologists and geneticists. At the end of 1980, a new clone (AVRDC 1287.19) from Taiwan developed at the Asian Vegetable Research Development Centre (AVRDC) was found out to be resistant to bacterial wilt and has heat adaptation. The source of resistance was, however, not S. *phureja* but the two wild species *S. raphanifolium* and *S. chacoense.* The latter was used in hesitation since its resistance, so effective under Taiwanese conditions, had broken down under field conditions in Peru, where a strain of *P. solanacearum* different from the Taiwan strain was present. However, the chance to introduce a new source of resistance to bacterial wilt into the existing gene pool, and combining two sources of resistance into one progeny, overrode doubts about the usefulness of this clone in CIP's breeding program (Schmiediche, 1986).

MATERIALS AND METHODS

A. Evaluation using potato seed tubers

Germplasm Materials

Healthy G_2 tubers of ten different potato clones obtained from the International Potato Center (CIP) and JICA were used in the study. The evaluation was conducted under green house condition. Treatments were arranged following the Complete Randomized Design (CRD) with 4 replications.

The clones and cultivars evaluated and their characteristics are as follows:

Clone	Cultivar's Name	Origin	Type of resistance		
			LB	Wart	BW
LBR-5	Igorota	Philippines	R		
387410.7	LBr-9	CIP	R		
Warishiro		Japan			
575003	I-931	India	R	S	S
676070	Cruza 155	Mexico	R		
380579.3	BW-III	CIP	MR	S	
720045	Atzimba	Mexico	MR	S	S
573275	ASN-69-1	Mexico	R	R	S
285378.27	None	CIP	MR	MR	
720071	Monserrate	Columbia	MR	R	S

The bacterial wilt nursery was thoroughly prepared. The area was divided into 4 blocks, each block consisting of 10 plots measuring 0.47 x 2meters each. Seven hundred grams of triple 14 and 12.5 kilograms of chicken dung were applied to each block before planting. During hilling–up, 700 grams of triple 14 was applied in each block. Dithane and Ridomil were used to protect the plant from fungal diseases.

Inoculum Preparation and Soil Inoculation

Four kilograms of infected potato tubers were cut into small cubes and were incorporated in the soil. Another 8 kilograms of infected tubers were cut into smaller pieces, allowed to ooze in 10 gallons of tap water and applied as soil drench.

To enhance the rapid multiplication of the bacteria, the inoculated soil was irrigated periodically and covered with plastic sheets to increase the soil temperature.

Data Gathered

1. Percentage Bacterial Wilt Infection. This was obtained using the formula:

$$\% \text{ infection} = \frac{\text{no. of infected plant}}{\text{total no. of plants}} \times 100$$

2. Weight of marketable tubers (kg). These are the tubers free from disease infection.
3. Weight of non-marketable tuber (kg). Marble size, rotten and diseased tubers was considered as non-marketable.
4. Total weight of tubers infected with bacterial wilt (kg). This was determined by weighing all the tubers infected with bacterial wilt.
5. Late blight infection. This was asses using the CIP rating scale:

Blight (%)	CIP Scale	Description
0	1	No blight to be seen
0.1	1	Very few plants in larger plots with a lesion, not more than two lesions per 10 m of row (30 plants)
1	2	Up to 10 small lesions

3	3	Up to 30 small lesion per plant or up to one leaflet in each 20 attacked
10	4	Most plants are visibly attacked, and one out of three leaflets infected, few multiple infections per leaflet.
25	5	Nearly every leaflet with lesion. Multiple infections per leaflet are common. Field or plants in plot are affected.
50	6	Every plant effected and half the leaf area destroyed by blight. Plots looks green, fleaked and brown; blight is very obvious.
75	7	As previous, but three quarters of each plant affected by blight. Lower branches maybe overwhelmingly killed off and only green leaves, if any, are at the top of the plant. Shape of plants maybe more spindly due to extensive foliar loss. Plot looked neither brown nor green.
91	8	Some leaves and most stems are green. Plot looks brown with some green patches.
97	9	Few green leaves, most with lesions, remain. Many stems with lesions. Plots looks brown.
100	9	All leaves and stems dead. No visible blight left to evaluate.

B. Evaluation using Stem cuttings

Two weeks old rooted stem cuttings from NPRCRTC were used in the study. Treatments were arranged following the Complete Randomized Design (CRD) with 3 replications at three plants per replicate.

The stem cuttings were planted in plastic cups with sterilized soil and maintained at the Department of Plant Pathology Green house.

The entries were as follows:

Clone	Cultivar's Name
LBR-5	Igorota
575003	I-931
676070	Cruza 155
573275	ASN-69-1
285378.27	-

Preparation of Inoculum and Inoculation Method

First Trial

Inoculum of bacterial wilt was sourced from infected tubers. The tubers were properly washed, cut and squeezed for the bacterial ooze to come out and was diluted in 10 ml distilled water. Using a sterilized wire loop, the bacterial suspension was streaked in previously plated casamino-peptone glucose agar (CPGA) consisting of the following in g/li of water:

10 g dextrose 1 g casamino acid 10 g peptone 18 g agar

Surface growth of the bacterium was scraped and diluted in the desired volume of distilled water. The soil was cultivated near the root system to create wounds before drenching 30 ml of the bacterial suspension in each plant.

After 3 days, only 2 among the 45 stem cuttings wilted.

Second Trial

In the second trial, the same germplasm materials were used but the inoculation method was modified. Before planting, roots hairs of the stem cuttings were cut and were dipped in the bacterial suspension for 3 minutes to allow entry of the bacteria in the plant system.

In addition, infected potato tubers were cut into smaller pieces and allowed to ooze out in the desired volume of distilled water. The soil was cultivated near the root system to create wounds before drenching 60 ml of the bacterial suspension in each of the plant 16 days after the first inoculation.

Bacterial population was determined using the spectrophotometer.

Data Gathered

1. Bacterial population. This was obtained by using the spectrophotometer.
2. Number of days from planting to bacterial wilt expression. Days were counted from inoculation to symptom expression.

RESULTS AND DISCUSSION

Evaluation Using Potato Seed Tubers

Percentage Bacterial Wilt Infection

Twenty three (23) days after planting (DAP), the variety Igorota and clone 387410.7 had the highest percentage of bacterial wilt infection with a mean of 27.50, and at 44 DAP, 100% plant were dead due to bacterial wilt (Table 1). On the other hand, clones 720071, 285378.27, 380579.3 and Warishiro showed a slow rate of bacterial wilt infection from 23 to 58 DAP, (Plates 1 and 2).

Among the 10 entries, only the clone 720071 still had plant that is not infected with bacterial wilt after 58 DAP.

The high incidence of bacterial wilt may have been influenced by the prevailing weather conditions. Under greenhouse condition, the temperature was relatively higher than the ambient temperature. The maximum and minimum temperatures during the last week of September (BW rating started) were 26ºC and 17ºC respectively, compared to the ambient condition with a maximum temperature of 24.5ºC and a minimum of 16.8ºC (Table 2). The prevailing weather condition during the conduct of the study favors *R. solanacearum* development as cited by Persley (1985) that the optimum temperature for *R. solanacearum* development is from 25-35ºC. Moreover, Martin and French (1996) cited that disease development of bacterial wilt is mainly influenced by temperature. High temperature promoted bacterial wilt development.

Table 1. Mean percentage bacterial wilt infection of different clones/variety evaluated

CLONE/ VARIETY	DAYS AFTER PLANTING					
	23	30	37	44	51	58
Igorota	27.50	60.00^{a}	80.00^{ab}	100^{a}	100^{a}	100
387410.7	27.50	47.50^{ab}	77.50^{a}	100^{a}	100^{a}	100
Warishiro	2.50	15.00^{c}	27.50^{bcd}	57.50^{cde}	90.00^{a}	100
575003	15.00	30.00^{bc}	52.50^{abc}	90.00^{ab}	100^{a}	100
676070	10.00	30.00^{bc}	50.00^{abc}	77.50^{abc}	97.50^{a}	100
380579.3	5.00	12.50^{c}	27.50^{cd}	52.50^{cde}	85.00^{ab}	100
720045	7.50	22.50^{bc}	37.50^{bcd}	70.00^{bcd}	95.00^{a}	100
73275	7.50	22.50^{bc}	47.50^{abc}	75.00^{abc}	92.50^{a}	100
285378.27	7.50	7.50^{c}	17.50^{cd}	45.00^{d}	72.50^{bc}	100
720071	2.50	5.00^{c}	12.50^{d}	35.00^{e}	60.00^{c}	87.50

Means followed by a common letter are not significantly different at 5% level by DMRT

Plate 1. Clone 380579.3 showing wilt symptom at 30 DAP

Plate 2. Infected tubers from clone 285378.27

Plate 3. Infected tubers from clone 676070

Table 2. Mean weekly weather data

MONTH	TEMPERATURE ºC		AVERAGE RAINFALL (mms and Tenths)	RELATIVE HUMIDITY (%)	TOTAL BRIGHT SUNSHINE (mm)
	Max	Min			
September					
Week 1	24.1	17.4	24.6	90	193.7
Week 2	25.1	17.7	7.2	89	169.7
Week 3	22.2	16.7	57.5	90	53.1
Week 4	24.5	16.8	2.3	87	293.1
October					
Week 1	24.4	16	20	89	333.4
Week 2	24	15.6	10.4	86	252.8
Week 3	25.3	16	1.0	87	363.4
Week 4	24.9	16.2	3.0	84	287.1
November					
Week 1	24	16.2	0.9	82	256.8
Week 2	25.6	15.4	4.2	82	321.1
Week 3	24.4	14.4	1.3	83	341.5
Week 4	25.6	15.3	0.0	77	322.2
February					
Week 1	23.6	14.2	0.0	79	-
Week 2	25.4	14.3	2.3	80	-
Week 3	26	13.2	0.0	80	-
Week 4	26.9	12.9	0.0	74	-

Percentage Late Blight Infection

Among the 10 entries, clone 720071 showed a slow development rate of late blight infection and was the only clone that reached 58 DAP (Table 3). Conversely, clones 676070, 720045, 573275, 285378.27 and Warishiro, reached 51 DAP and had a late blight rating ranging from 1 and 2, where no blight was seen; or very few plants in larger plots with lesions and 2 with a maximum of 10 small lesions. On the other hand,

clone 387410.7 and variety Igorota only reached 37 DAP and had late blight ratings of 2. The variety Igorota is rated as resistant to late blight. However the variety exhibited late blight infection early in its growing period but is able to regenerate new leaves and matures at 100-120 days.

Most of the entries are rated to be resistant to late blight but their resistance was not quantified due to the early infection of bacterial wilt which caused their early senescence.

The fluctuation of the prevailing weather condition played a great factor for the manifestation of resistance or susceptibility of the clones/variety to late blight infection. Thung (1974) explained that any environmental fluctuation may upset the pathogen and shift host reaction to low and higher resistance or susceptibility.

Table 3. Mean percentage late blight infection of different clones/variety evaluated

CLONE/ VARIETY	DAYS AFTER PLANTING 30	37	44	51	58
Igorota	1.25^{bc}	2.0			
387410.7	2.00^{a}	2.25			
Warishiro	1.50^{b}	1.75	3.0^{ab}	2.0	
575003	1.0^{c}	1.25	2.0^{bcd}		
676070	1.0^{c}	1.0	1.5^{d}	1.5	
380579.3	1.0^{c}	1.25	2.75^{abc}	2.75	
720045	1.0^{c}	1.50	2.50^{abcd}	2.0	
573575	1.0^{c}	1.25	1.75^{cd}	2.0	
285378.27	1.0^{c}	1.50	1.75^{cd}	1.5	
720071	1.0^{c}	1.00	2.0^{bcd}	1.75	1.5

Means followed by a common letter are not significantly different at 5% level by DMRT

Yield Parameters

Results reveal that clone 676070 had the highest marketable yield with a mean of 575g but also had the highest non marketable and bacterial wilt infected tubers (Table 4). On the other hand, the least weight of marketable tubers was obtained in clone 720071 with a mean of 36.25g which also registered the lowest in non marketable tubers and second lowest in bacterial wilt infected tubers with a mean of 21.25g.

The variety Igorota which is a late maturing variety registered one of the lowest marketable tubers with a mean of 58.75g. Its low marketable yield is associated with its early senescence due to bacterial wilt infection.

All the 10 entries evaluated were able to produce marketable tubers despite the early bacterial wilt infection making them all tolerant to bacterial wilt.

Table 4. Yield parameters of different clones/variety evaluated

CLONE/ VARIETY	MARKET ABLE (g)	NON MARKET ABLE (g)	BACTERIAL WILT INFECTED (g)
Igorota	58.75	66.25	41.25
387410.7	48.75	55.00	3.75
Warishiro	72.50	52.50	8.75
575003	95.00	126.25	51.25
676070	575.00	250.00	388.75
380579.3	218.75	167.50	72.50
720045	82.50	113.75	25.00
573275	271.25	183.75	111.25
285378.27	161.25	193.75	193.75
720071	36.25	20.00	21.25

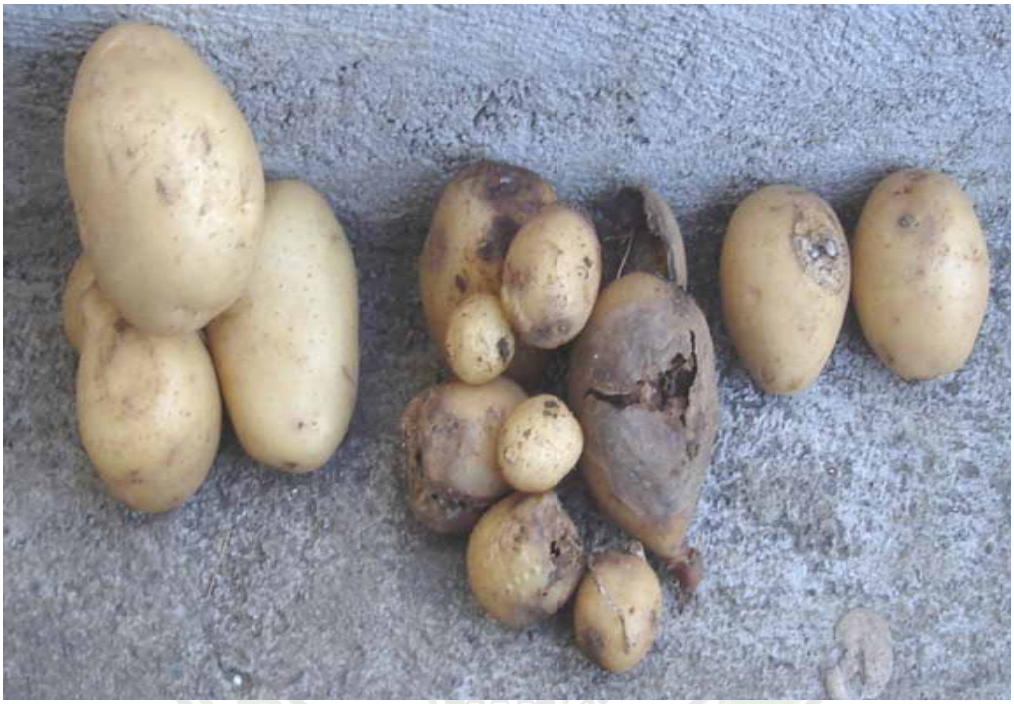

Plate 4. Marketable, non-marketable, BW infected tubers of variety Igorota

Evaluation Using Stem Cuttings

Number of days from planting to bacterial wilt expression

Table 5 shows the number of days from planting to bacterial wilt expression. Clone 285378.27 manifested bacterial wilt symptom at the shortest time having a mean of 24.210 days. The clone 575003 on the other hand exhibited the longest time of symptom expression at a mean of 25.20 days.

The recorded temperature in the greenhouse during the month of February was relatively higher than the ambient temperature. It was during the 3rd week of February that wilting was observed in the sample plants. The maximum temperature in the green house was 27ºC and minimum of 14ºC, compared to the ambient condition with a maximum of 26ºC and a minimum of 13.2ºC (Table 2). Persley (1985) cited that the optimum development of *R. solanacearum* is from 25-35ºC.

Table 5. Average number of days from transplanting to bacterial wilt symptom expression

CLONES/ CULTIVAR	MEAN NUMBER OF DAYS AFTER TRANSPLANTING
Igorota	24.887
575003	25.200
676070	24.663
573275	24.773
285378.27	24.210

Table 6 presents the bacterial population that was used during inoculation in the first and second trial. Results revealed that there is a significant difference from the bacterial population during the first and second trial.

During the termination of the study, not all the sample plants wilted. The assymptomatic cuttings were tested using the water blank test which revealed that they are infected with bacterial wilt (Plates 4 and 5).

The unsuccessful result obtained from the first trial may be due to the low bacterial population that was inoculated to the sample cuttings. Nielson and Hyness (1979), as cited by Nagpala (1986) reported that the possible variation of resistance or susceptibility of crops were affected by the age of the plant at the time of inoculation. It was reported that the best time to inoculate the plants should be about 4 weeks after planting.

Table 6. Bacterial population of inoculum used during the trials

SAMPLE	AVERAGE BACTERIAL POPULATION		
	TRIAL 1	TRIAL 2	DIFFERENCE
1	0.162	0.958	0.796
2	0.130	0.997	0.867
3	0.133	1.000	0.867

Plate 5. Wilted potato cuttings at 16 days after inoculation

Plate 6. Assymptomatic cuttings being tested for BW infection

SUMMARY, CONCLUSION AND RECOMMENDATION

Summary

Nine clones: 387410.7, Warishiro, 575003, 676070, 380579.3, 720045, 573275, 285378.27 and 720071 and the variety Igorota were evaluated to identify germplasm materials that were tolerant to bacterial wilt infection under green house condition and to determine their reaction to late blight infection.

Among the 10 entries evaluated, Igorota and clone 387410.7 showed the highest percentage bacterial wilt infection at 23 DAP. On the other hand, clone 720071 exhibited the slowest rate of bacterial wilt infection followed by clones 285378.27, 380579.3 and Warishiro respectively.

Clones 720071, 285378.27 and 676070 showed resistance to late blight infection with a rating of 1 or no blight or a maximum of 10 small lesions observed in the sample plant.

The highest marketable yield was obtained from clone 676070 which ironically registered the highest nonmarketable and bacterial wilt infected tubers.

Conclusion

Based on the results of the study, all the 10 entries evaluated were tolerant to bacterial wilt as they were able to produce marketable tubers despite bacterial wilt infection. Clones 720071, 285378.27 and 676070 confirmed their resistance to late blight infection.

Recommendation

Further evaluation must be conducted to assess the adaptability and yield of the germplasm materials.

LITERATURE CITED

BARROZO, L.S. 2001. Screening of potato varieties for resistance against bacterial wilt (*Ralstonia solanacearum*). Undergraduate Thesis. Benguet State University, La Trinidad, Benguet.

BEUKEMA, H. P. 1985. Seed quality, seed use, seed supply and seed production. Inter Agrica Center. Wageningen, Netherlands, Manual.

BOTANGEN, A.T. 2003. Evaluation and selection of potential processing potato clone grown at Atok, Benguet. MS Thesis. Benguet State University, La Trinidad, Benguet.

BROWN, D.G., and C. ALLEN, 2001. *Ralstonia solanacearum* genes induced during growth in tomato: an inside view of bacterial wilt. Department of Plant Pathology, University of Wisconsin-Madison, 1630 Linden Drive, Madison, WI 53706, USA.

BUASEN, C.T. 1978. Economic importance of Irish potato in the Philippines, First National Potato Production Course. (Mimeographed). Pp. 37.

CIP. 1992. The International Potato Center (CIP): Annual Report. Lima, Peru: Centro Internacional de la Papa.

CIP. 1996. The International Potato Center (CIP)
Posted at: www.cipotato.orgl potato/bacterial wilt/Publications.htm

DAUGHTREY, M. 2003. Southern bacterial wilt, caused by *Ralstonia solanacearum*. Society of American Florists' 19th Annual Conference on Insect and Disease Management on Ornamentals.

DIRK VAN ELSAS, J., P. KASTELEIN, P. VAN BEKKUM, J. M. VAN DER WOLF, P. M. DE VRIES, and L.S. VAN OVERBEEK. 2000. Survival of *Ralstonia solanacearum* biovar 2, the causative agent of potato brown rot, in field and microcosm soils in temperate climates. Phytopathology 90:1358-1366.

DOUCHES, D.S. 1996. Assessment of potato breeding progress in the United States over the last century. Crop Sci. (in press).

FEGAN, M. and P. PRIOR. 2004. Recent developments in the phylogeny and classification of *Ralstonia solanacearum*. Presentation at the 1st International Tomato Symposium. Orlando, FL.

FRENCH, E.R. 1996. Integrated control of bacterial wilt of potato. In: Bacterial Wilt Training Manual. International Potato Center. Lima, Peru.

GANGA, Z.N. 1992. Utilization of somaclonal variants in a backcross program for potato (*Solanum tuberusom L.)* grandula trichomes and environmental effects on their expression. Dissertation. Cornell University.

HARRDEC. 1996. Production statistics for vegetable production in the Cordillera (Draft Report). Benguet State University, La Trinidad, Benguet: Highland Agricultural Resources Research and Development Center (HARRDEC).

HAYWARD, A. C. 1994. The hosts of *Pseudomonas solanacearum*, p. 9-24. *In* A. C. Hayward, and G. L. Hartman (ed.), Bacterial wilt: the disease and its causative agent, *Pseudomonas solanacearum* CAB International Press, Wallingford, United Kingdom.

HERRERA I.A. 1972. Progresos en la seleccion de resistencia a la marchitez bacteriana en el Peru. In: French, E.R. ed., Prospects for the Potato in the Developing World. Lima, Peru, (CIP).

JI, P., T. MOMOL, S.M. OLSON, J. HONG, P. PRADHANANG, A. NARAYANAN, and J.B. JONES. 2004. New Tactics for Bacterial Wilt Management on Tomatoes in the Southern US.

JYOTHI, A.R., K. ABRAHAM and J. MATHEW. 1993. Bacterial wilt of chili in Kerala and reaction of certain chili accessions to *R. Ralstonia solanacearum.* ACIAR Bacterial Wilt Newsletter. 9:6

KELMAN, A. 1953. The bacterial wilt caused by *P. solanacearum.* A literature review and bibliography. North Carolina Agricultural experiment Station Technical Bulletin.

KIPPS, N.S. 1979. Production of Field crops. New York: Tata Mc Graw Hill Publishing Company . Ltd.

KURUPUARACCHI, S.P. 1995. Pot variety evaluation at the Agricultural Research Station. Kalpitiya, Sri Lanka. Selected Research Paper.

LAFERRIERE, L., J.P. HELGESON and C. ALLEN. 1997. *Solanum tuberosum - S. commersonii* Somatic Hybrids Resistant to Brown Rot caused by *Ralstonia solanacearum*. University of Wisconsin, 1630 Linden Dr., Madison, WI 53706 USA.

LOPES, C.A. and A.M. QUEZADO-SOARES. 1994. Bacterial wilt of two weed species of the family Labiatate, incited by *Pseudomonas solanacearum.* In: A.C. Hayward, ed., Bacterial Wilt Newsletter 11:6 (December 1994). Canberra, Australia ACIAR.

MA, QING-SHENG. 1990. Molecular biology and research on *Pseudomonas solanacearum.* In: Hayward, A. c. ed., Bacterial Wilt of Groundnut. ACIAR No Canberra, Australia; ACIAR.

MARTIN, C. and E.R. FRENCH. 1985. Bacterial wilt of potatoes: *Pseudomonas solanacearum.* Technical Bulletin 13. 2nd ed. Revised, Lima Peru: Centro Internacional de la Papa (CIP).

MARTIN, C. and E. FRENCH. 1996. Bacterial wilt of potato (*R. solanacearum).* In Bacterial Wilt Training Manual. International Potato Center. Lima, Peru, section 2. Pp. 2-8.

NAGPALA. A.L. 1986. Screening of white potato varieties for resistance to bacterial wilt caused by *P. solanacearum.* MS Thesis. Benguet State University, La Trinidad, Benguet.

NIELSON and HYNESS. 1960. Resistance of *Solanum tuberosum* to *P. solanacearum*. American potato Journal . Vol. 46: Pp451-462.

PEREZ, J.C., T.C. DICCION, G. OLBINADO, E. BUGAT, L. BALAOING, R. GAYAGAY, G. BACKIAN, C. BADEN and T. GALAP. 1997 Management of bacterial wilt(*R. solanacearum* Yabuuchi) in Potato (*Solanum tuberosum Lin)* Journal, NPRCRTC. Benguet State University, La Trinidad Benguet.

REINKING, R.B. 1918. Philippine economic plant diseases. Philippines Journal of Science, 13, Pp. 393-395.

RICH, A.E. 1983. Potato diseases. New York, New York: Academic Press.

ROWE, P.R. and L. SEQUEIRA. 1972. Development of potato clones with resistance to bacterial wilt. In: French, E.R. ed., Prospects for the Potato in the Developing World. Lima, Peru, (CIP).

SCHMIEDICHE, P. 1984. CIP's breeding strategy for bacterial wilt resistance and heat tolerance. International Potato Center. Lima, Peru.

SCHMIEDICHE, P. 1986. Breeding potatoes for resistance to bacterial wilt caused by *P. solanacearum.* In: G.J. Persley, ed., Bacterial Wilt Diseases in Asia and the South Pacific. ACIAR proceedings No. 13 Canberra, Australia.

SHEW, H.D. and G.B. LUCAS, Eds. 1991. Compendium of Tobacco Diseases. APS Press, St. Paul, MN.

SIMONGO, D. K. 1992. Assessment of potato clones using rooted apical cuttings by farmers in Benguet. MS Thesis. Benguet State University, La Trinidad, Benguet.

SMITH, E. F. 1896. A bacterial disease of tomato, pepper, eggplant and Irish potato (*Bacillus solanacearum* nov. sp.). US Dep. Agric. Div. Vegetable Physiol. Pathol. Bull. 12, 1-28.

STANSBURY, C., S. MCKIRDY, A. MACKIE, and G. POWER. 2001. Bacterial wilt: *Ralstonia solanacearum* – race 3 exotic threat to Western Australia. Fact sheet No. 7/2001. Hortguard Initiative AGWEST, The Government of Western Australia.

STEVENSON, W.R., R. LORIA, G.D. FRANC, and D.P. WEINGARTNER, Eds. 2001. Compendium of Potato Diseases, 2nd Ed. APS Press, St. Paul, MN.

THUNG, T.H. 1974. Potato diseases and hybridization. Phytopathology. 37:373-381.

TYRON, H. 1895. New potato disease. Annual report, Queensland Department of Agriculture for the year 1893-1895.

VALDEZ, R.B. 1985. Bacterial wilt in the Philippines. In: G.J. Persley, ed., Bacterial Wilt Diseases in Asia and the South Pacific. ACIAR proceedings No. 13 Canberra, Australia: ACIAR.

VAN DER WOLF, J. and M. PEROMBELON. 1997. Potato brown rot in temperate regions – a review. http://www.spud.co.uk/external/PROF/RESEARCH/scri/brownro3.htm

WELLES, C.G. and E.F. ROLDAN. 1922. Solanaceous wilt in the Philippines Islands. Philippine Agriculturist, Pp 393-404.

YABUUCHI, E., Y. KOSAKO, L. YANO, H. HOTTA and Y. NISHIUCHI. 1995. Transfer of two *Burkholderia* and an *Alcaligenes* species to *Ralstonia* gen. nov.: proposal of *Ralstonia pickettii* (Ralston, Palleroni and Doudoroff 1973) comb. nov., *Ralstonia solanacearum* (Smith 1896) comb. nov. and *Ralstonia eutropha* (Davis 1969) comb. nov. *Microbiol. Immunol. 39*, 897-904.

APPENDICES

Appendix Table 1. Percentage bacterial wilt infection at 23 DAP

CLONE/ VARIETY	BLOCK I	II	III	IV	TOTAL	MEAN
Igorota	10	10	70	20	110	27.50
387410.7	50	20	20	20	110	27.50
Warishiro	10	0	0	0	10	2.50
575003	20	20	20	0	60	15
676070	10	0	10	20	40	10
380579.3	10	0	10	0	20	5
720045	0	20	10	0	30	7.5
573275	10	20	0	0	30	7.5
285378.27	10	10	0	20	30	7.5
720071	0	0	10	0	10	2.5

Transformed data

CLONE/ VARIETY	BLOCK I	II	III	IV	TOTAL	MEAN
Igorota	3.24	3.24	8.39	4.53	19.40	4.850
387410.7	7.11	4.53	4.53	4.53	20.70	5.175
Warishiro	3.24	0.71	0.71	0.71	5.37	1.343
575003	4.53	4.53	4.53	0.71	14.30	3.575
676070	3.24	0.71	3.24	4.53	11.75	2.930
380579.3	3.24	0.71	3.24	0.71	7.90	1.975
720045	0.71	4.53	3.24	0.71	9.19	2.210
573275	3.24	4.53	0.71	0.71	9.19	2.210
285378.27	3.24	0.71	0.71	4.53	9.19	2.210
720071	0.71	0.71	3.24	0.71	5.37	1.343

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Block	3	327.478	109.159	0.6702		
Factor A	9	3112.413	345.824	2.1233^{ns}	2.25	3.14
Error	27	4397.498	162.870			
TOTAL	39	7,837.388				

^{ns}= not significant Coefficient of Variation = 62.05%

Appendix Table 2. Percentage bacterial wilt infection at 30 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	60	40	100	40	240	60
387410.7	60	40	50	40	190	47.5
Warishiro	40	0	20	0	60	15
575003	40	40	40	0	120	30
676070	30	30	10	50	120	30
380579.3	20	10	20	0	50	12.5
720045	10	40	40	0	90	22.5
573275	30	40	0	20	90	22.5
285378.27	0	0	0	30	30	7.5
720071	0	0	20	0	20	5

Transformed data

CLONE/ VARIETY	BLOCK I	II	III	IV	TOTAL	MEAN
Igorota	7.78	6.36	10.02	6.36	30.52	7.630
387410.7	7.78	6.36	7.11	6.36	27.61	6.903
Warishiro	6.36	0.71	4.53	0.71	12.31	3.078
575003	6.36	6.36	6.36	0.71	19.79	4.948
676070	5.52	5.52	3.24	7.11	21.39	5.348
380579.3	4.53	3.24	4.53	0.71	13.01	3.253
720045	3.24	6.36	6.36	0.71	16.67	4.168
573275	5.52	6.36	0.71	4.53	17.12	4.280
285378.27	0.71	0.71	0.71	5.32	7.45	1.863
720071	0.71	0.71	4.53	0.71	6.66	1.665

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Block	3	907.490	302.497	0.9879		
Factor A	9	11022.418	1224.713	3.997**	2.54	3.14
Error	27	8267.390	306.200			
TOTAL	39	20,197.298				

** = Highly Significant Coefficient of Variation = 69.30%

Appendix Table 3. Percentage bacterial wilt infection at 37 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	70	80	100	70	320	80
387410.7	80	60	90	80	310	77.5
Warishiro	50	0	40	20	110	27.5
575003	60	50	70	30	210	52.5
676070	40	60	40	60	200	50
380579.3	40	40	30	0	110	27.5
720045	20	70	60	0	150	37.5
573275	50	60	30	50	190	47.5
285378.27	20	20	0	30	70	17.5
720071	10	0	30	10	50	12.5

Transformed data

CLONE/ VARIETY	BLOCK I	II	III	IV	TOTAL	MEAN
Igorota	8.39	8.97	10.2	8.39	35.77	8.943
387410.7	8.97	7.78	9.51	8.97	35.23	8.808
Warishiro	7.11	0.71	6.36	4.53	18.71	4.678
575003	7.78	7.11	4.13	5.52	24.54	6.135
676070	6.36	7.78	6.36	7.78	28.28	7.070
380579.3	3.36	6.36	5.52	0.71	18.95	4.738
720045	4.53	8.39	7.78	0.71	21.41	5.353
573275	7.11	7.78	5.52	7.11	27.52	6.880
285378.27	4.53	4.53	0.71	5.52	15.29	3.823
720071	3.24	0.71	5.52	3.24	12.71	3.178

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Block	3	2507.50	835.833	1.8547		
Factor A	9	15162.50	1684.722	3.7384**	2.25	3.14
Error	27	1216.50	450.648			
TOTAL	39	29,837.50				

** = Highly Significant Coefficient of Variation = 37.26%

Appendix Table 4. Percentage bacterial wilt infection at 44 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	100	100	100	100	400	100
387410.7	100	100	100	100	400	100
Warishiro	70	40	60	60	230	57.5
575003	100	100	90	70	360	90
676070	60	100	70	80	310	77.5
380579.3	70	50	50	40	210	52.5
720045	40	100	100	40	280	70
573275	70	100	50	80	300	75
285378.27	40	40	40	60	180	45
720071	40	20	50	30	140	35

Transformed data

CLONE/ VARIETY	BLOCK I	II	III	IV	TOTAL	MEAN
Igorota	10.02	10.02	10.2	10.02	40.08	10.02
387410.7	10.02	10.02	10.02	10.02	40.08	10.02
Warishiro	8.39	6.36	7.78	7.78	30.31	7.578
575003	10.02	10.02	9.51	8.39	37.94	9.485
676070	7.78	10.02	8.39	8.97	35.16	8.790
380579.3	8.39	7.11	7.11	6.36	28.97	7.243
720045	6.36	10.02	10.02	6.36	32.76	8.190
573275	8.39	10.02	7.11	8.97	34.49	8.623
285378.27	6.36	6.36	6.36	7.78	26.86	6.715
720071	6.36	4.53	7.11	5.52	23.52	5.880

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Block	3	427.50	142.50	0.4998		
Factor A	9	18372.50	2041.38	7.1604**	2.54	3.14
Error	27	7697.50	285.093			
TOTAL	39	26,497.50				

**=Highly Significant Coefficient of Variation = 24.04%

Appendix Table 5. Percentage bacterial wilt infection at 51 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	100	100	100	100	400	100
387410.7	100	100	100	100	400	100
Warishiro	90	70	100	100	360	90
575003	100	100	100	100	400	100
676070	100	100	90	100	390	97.5
380579.3	100	100	70	70	340	85
720045	100	100	100	80	380	95
573275	100	100	70	100	370	92.5
285378.27	70	80	60	80	290	72.5
720071	60	50	60	70	240	60

Transformed data

CLONE/ VARIETY	BLOCK I	II	III	IV	TOTAL	MEAN
Igorota	10.02	10.02	10.02	10.02	40.08	10.02
387410.7	10.02	10.02	10.02	10.02	40.08	10.02
Warishiro	9.51	8.39	10.02	10.02	37.94	9.485
575003	10.02	10.02	10.02	10.02	40.08	10.02
676070	10.02	10.02	10.02	10.02	39.57	9.893
380579.3	10.02	10.02	9.51	8.39	36.82	9.205
720045	10.02	10.02	10.02	8.97	39.03	9.758
573275	10.02	10.02	8.39	10.02	38.45	9.613
285378.27	8.39	8.97	7.78	8.97	34.11	8.528
720071	7.78	7.11	7.78	8.39	31.06	7.765

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Block	3	267.50	89.167	0.8731		
Factor A	9	6452.50	716.944	7.0199**	2.54	3.14
Error	27	2757.50	102.130			
TOTAL	39	24,177.50				

**=Highly Significant Coefficient of Variation = 11.32%

Appendix Table 6. Percentage bacterial wilt infection at 58 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	100	100	100	100	400	100
387410.7	100	100	100	100	400	100
Warishiro	100	100	100	100	400	100
575003	100	100	100	100	400	100
676070	100	100	100	100	400	100
380579.3	100	100	100	100	400	100
720045	100	100	100	100	400	100
573275	100	100	100	100	400	100
285378.27	100	100	100	100	400	100
720071	90	80	80	100	350	87.5

Transformed data

CLONE/ VARIETY	BLOCK I	II	III	IV	TOTAL	MEAN
Igorota	10.02	10.02	10.02	10.02	40.08	10.02
387410.7	10.02	10.02	10.02	10.02	40.08	10.02
Warishiro	10.02	10.02	10.02	10.02	40.08	10.02
575003	10.02	10.02	10.02	10.02	40.08	10.02
676070	10.02	10.02	10.02	10.02	40.08	10.02
380579.3	10.02	10.02	10.02	10.02	40.08	10.02
720045	10.02	10.02	10.02	10.02	40.08	10.02
573275	10.02	10.02	10.02	10.02	40.08	10.02
285378.27	10.02	10.02	10.02	10.02	40.08	10.02
720071	9.51	8.97	8.97	10.02	37.47	9.368

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Block	3	27.50	9.167			
Factor A	9	562.50	62.50	6.8182**	2.54	3.14
Error	27	247.50	9.167			
TOTAL	39	837.50				

**=Highly Significant Coefficient of Variation = 3.07%

Appendix Table 7. Late blight infection rating at 30 DAP

CLONE/ VARIETY	BLOCK I	II	III	IV	TOTAL	MEAN
Igorota	1	1	2	1	5	1.25
387410.7	2	2	2	2	8	2.0
Warishiro	1	2	1	2	6	1.5
575003	1	1	1	1	4	1
676070	1	1	1	1	4	1
380579.3	1	1	1	1	4	1
720045	1	1	1	1	4	1
573275	1	1	1	1	4	1
285378.27	1	1	1	1	4	1
720071	1	1	1	1	4	1

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Block	3	0.075	0.025	0.4030		
Factor A	9	4.025	0.447	7.2090**	2.54	3.14
Error	27	1.675	0.062			
TOTAL	39	5.775				

**=Highly Significant Coefficient of Variation = 21.20%

Appendix Table 8. Late blight infection rating at 37 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	2	2	2	2	8	2
387410.7	2	2	3	2	9	2.25
Warishiro	2	2	1	2	7	1.75
575003	2	1	1	1	5	1.25
676070	1	1	1	1	4	1.0
380579.3	1	2	1	1	5	1.25
720045	2	1	1	2	6	1.50
573275	1	1	1	2	5	1.25
285378.27	1	2	1	2	6	1.50
720071	1	1	1	1	4	1.0

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	TABULAR F 0.01
Block	3	0.475	0.158	0.8104		
Factor A	9	6.225	0.692	3.5403**	2.54	3.14
Error	27	5.275	0.195			
TOTAL	39	11.975				

**=Highly Significant Coefficient of Variation = 29.97%

Appendix Table 9. Late blight infection rating at 44 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	3	3	3	2	11	2.75
387410.7	4	3	4	3	14	3.5
Warishiro	4	2	3	3	12	3.0
575003	3	1	3	1	8	2.0
676070	1	2	2	1	6	1.5
380579.3	3	3	3	2	11	2.75
720045	3	2	3	2	10	2.5
573275	2	2	1	2	7	1.75
285378.27	1	3	1	2	7	1.75
720071	2	2	2	2	8	2.0

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	TABULAR F 0.01
Block	3	2.10	0.70	1.5882		
Factor A	9	15.10	1.678	3.8067**	2.54	3.14
Error	27	11.90	0.441			
TOTAL	39	29.10				

**=Highly Significant Coefficient of Variation = 28.25%

Appendix Table 10. Late blight infection rating at 51 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	2	3	1	2	8	2
387410.7	4	3	4	3	14	3.5
Warishiro	2	2	2	2	8	2.0
575003	2	2	3	1	8	2.0
676070	1	2	2	1	6	1.5
380579.3	3	3	3	2	11	2.75
720045	2	2	2	2	8	2.0
573275	2	2	2	2	8	2.0
285378.27	1	2	1	2	6	1.5
720071	1	2	2	2	7	1.75

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	TABULAR F 0.01
Block	3	1.0	0.33	1.20		
Factor A	9	13.10	1.456	5.24**	2.25	3.14
Error	27	7.500	0.278			
TOTAL	39	21.60				

**=Highly Significant Coefficient of Variation = 25.10%

Appendix Table 11. Late blight infection rating at 58 DAP

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	2	1	1	1	5	1.25
387410.7	3	2	2	2	9	2.25
Warishiro	2	1	1	1	5	1.25
575003	2	2	1	1	6	1.50
676070	1	2	1	1	5	1.25
380579.3	2	2	1	1	6	1.50
720045	2	1	1	2	6	1.50
573275	1	1	1	2	5	1.25
285378.27	1	2	2	2	6	1.50
720071	1	2	2	1	6	1.50

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	TABULAR F 0.01
Block	3	1.475	0.492	1.8247		
Factor A	9	3.225	0.358	1.3299^{ns}	2.54	3.14
Error	27	7.275	0.269			
TOTAL	39	11.975				

^{ns}=Not Significant Coefficient of Variation = 35.19%

Appendix Table 12. Weight of marketable tubers (g)

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	55	180	0	0	235	58.75
387410.7	160	0	0	35	195	48.75
Warishiro	245	0	0	45	290	72.5
575003	225	0	0	155	380	95
676070	600	665	625	410	2300	575
380579.3	250	400	190	35	875	218.75
720045	125	75	90	40	330	82.5
573275	230	260	240	355	1085	271.5
285378.27	0	245	200	200	645	161.25
720071	0	145	0	0	145	63.25

Transformed data

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	7.45	13.44	0.71	0.71	22.31	5.578
387410.7	12.67	0.71	0.71	5.96	20.05	5.013
Warishiro	15.67	0.71	0.71	6.75	23.84	5.960
575003	15.02	0.71	0.71	12.47	28.91	7.228
676070	24.51	25.80	25.01	20.26	95.58	23.895
380579.3	15.83	20.01	13.80	5.96	55.6	13.900
720045	11.20	8.69	9.51	6.36	35.76	8.940
573275	15.18	16.14	15.51	18.85	65.68	16.420
285378.27	0.71	15.67	14.16	14.16	44.7	11.175
720071	0.71	12.06	0.71	0.71	14.19	3.548

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Replication	3	94.424	31.475	1.0297		
Factor A	9	1447.218	160.802	5.2609**	2.54	3.14
Error	27	825.274	30.566			
TOTAL	39	2366.915				

**=Highly Significant Coefficient of Variation = 54.39%

Appendix Table 13. Weight of non- marketable tubers (g)

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	60	105	5	95	265	66.25
387410.7	140	55	25	0	220	55
Warishiro	100	45	15	50	210	52.5
575003	100	165	110	130	505	126.25
676070	200	220	80	500	10000	250
380579.3	300	115	145	110	670	167.5
720045	150	75	35	195	455	113.75
573275	165	200	80	290	735	183.75
285378.27	370	130	120	155	775	193.5
720071	0	50	10	20	80	20

Transformed data

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	7.78	10.27	2.35	9.77	30.17	7.542
387410.7	11.85	7.45	5.05	0.71	25.06	6.265
Warishiro	10.02	6.75	3.94	7.11	27.81	6.953
575003	10.02	12.86	10.52	11.42	44.81	11.205
676070	14.16	14.85	8.97	22.37	60.35	15.088
380579.3	17.33	10.75	12.06	10.51	50.65	12.663
720045	12.27	8.69	5.96	13.98	40.9	10225
573275	12.86	14.16	8.97	17.04	53.03	13.258
285378.27	19.25	11.42	10.98	12.47	54.12	13.530
720071	0.71	7.11	3.24	4.53	15.59	3.898

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Replication	3	116.104	38.701	3.8435		
Factor A	9	496.030	55.114	5.4735**	2.54	3.14
Error	27	271.870	10.069			
TOTAL	39	884.004				

** =Highly Significant Coefficient of Variation = 31.53%

Appendix Table 14. Weight of Infected tubers (g)

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	110	45	0	10	165	41.25
387410.7	15	0	0	0	15	3.75
Warishiro	0	0	0	35	35	8.75
575003	80	40	75	10	205	51.25
676070	150	350	760	295	1555	388.75
380579.3	50	180	15	45	290	72.5
720045	30	20	40	10	100	25
573275	115	5	255	70	445	11.25
285378.27	195	205	65	310	775	193.75
720071	35	50	0	0	85	21.25

Transformed data

CLONE/ VARIETY	BLOCK				TOTAL	MEAN
	I	II	III	IV		
Igorota	10.51	6.75	0.71	3.24	21.21	5.303
387410.7	3.94	0.71	0.71	0.71	6.07	1.518
Warishiro	0.71	0.71	0.71	5.96	8.09	2.023
575003	8.97	6.36	8.69	3.24	27.26	6.815
676070	12.27	18.72	27.58	17.19	75.76	18.94
380579.3	7.11	13.44	3.94	6.75	31.24	7.810
720045	5.52	4.53	6.36	3.24	19.65	4.913
573275	10.75	2.35	15.98	8.40	37.48	9.370
285378.27	13.98	14.34	8.09	17.62	54.03	13.508
720071	5.96	7.11	0.71	0.71	14.49	3.623

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Replication	3	8.204	2.735	0.1642		
Factor A	9	1052.941	116.993	7.0271**	2.54	3.14
Error	27	449.579	16.649			
TOTAL	39	15110.665				

** =Highly Significant Coefficient of Variation = 55.27%

Appendix Table 15. Number of days from transplanting to bacterial wilt symptom expression

CLONE/ VARIETY	REPLICATE			TOTAL	MEAN
	I	II	III		
1	23.33	26.00	26.00	74.66	24.887
2	24.66	26.00	26.00	75.60	25.200
3	23.66	24.66	26.00	73.99	24.663
4	23.30	25.33	26.00	74.32	24.773
5	23.33	23.66	23.3	72.63	24.210

ANALYSIS OF VARIANCE

SOURCES OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN OF SQUARES	COMPUTED F	TABULAR F 0.05	0.01
Block	2	9.401	4.701	3.9895		
Factor A	4	7.562	0.391	0.3315^{ns}	3.84	7.01
Error	18	9.426	1.178			
TOTAL	14	20.390				

^{ns}= Not Significant Coefficient of Variation = 4.39%