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Application of Gamma Induced Mutation in Breeding Potato for Bacterial Wilt Disease Resistance

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Authors' contributions

This work was carried out in collaboration between all authors. Author EC designed the study and performed the statistical analysis. Authors EC, SB and ZK wrote the protocol and EC wrote the first draft of the manuscript. Authors MK, OK and JO managed the analyses of the study. Authors EC and ZK managed the literature searches. All authors read and approved the final manuscript.

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***Article*ABSTRACT**

Aims: Potato (*Solanum tuberosum* L.) production in Kenya has not been achieved in its full potential due to susceptibility of potato varieties to pest and diseases among others. Bacterial wilt, caused by *Ralstonia solanacearum* in potato is regarded as an important disease contributing to significant yield reduction. The disease is considered more difficult to control in field crop production using universal control measure due to pathogen's properties as a soil-borne bacterium, broad host range and the genetic variation level within the strains. The objective was to screen potato mutants at M1V4 mutant populations for resistance against bacterial wilt using pathogenicity test.
Study Design: The experimental design used was an alpha lattice with twenty three blocks each having seven plots with three replications each. Data were subjected to analysis of variance using SAS statistical package, version 9.1 and mean separation done using Duncan Multiple Range Test (DMRT) whenever there were significant differences.
Place and Duration of Study: The study was carried out at Kenya Agricultural Livestock and Research Organization (KALRO), Kabete station for one season (December 2015 to April 2016).

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Methodology: One hundred and sixty three mutants developed from three commercial varieties (Asante 72, Mpya 43 and Sherekea 47) were evaluated.
Results: The reactions of potato mutants to bacterial wilt varied from variety to variety and mutants to mutants. None of the Asante, Mpya and Sherekea mutants used was found to be resistant to bacterial wilt though Asante mutant populations showed better response. There was significant difference in some traits such as DTOW, AUDPC and PSTTN across the three potato mutant populations.
Conclusion: The variation within the potato mutants and response to bacterial wilt resistance levels could be attributed to different dose rates and the reaction of each variety to the mutagen used. Since mutation is random its effects are enormous.

Keywords: Potato; mutants; bacterial wilt; screening; pathogenicity.

1. INTRODUCTION

The cultivated potato (*Solanum tuberosum* L.) is the most important staple food second in Kenya after maize and the fourth in the world major food crop after wheat, rice and maize [1,2,3]. Potato is grown in more than 150 countries worldwide from latitudes 65 °N to 50 °S [3,4] and can grow from sea level up to 4700 metres above sea level [5]. Potato farming in Kenya employs 3.3 million people at all levels of the value chain. Potato is grown by about 800 000 farmers on about 158 000 ha per season, with an annual production of about 1.6 million tonnes in two growing seasons [3,6].

In spite of the importance of potato in Kenya, yield are still low are due to inadequate supply and untimely availability of high quality certified seeds, low soil fertility, low yielding varieties, diseases and insect pests among others [3,7]. Of the diseases, bacterial wilt [8] is a major disease found in all the potato growing areas of the country (Kenya) [9,10] affecting 77% of potato farms [11]. Bacterial wilt, caused by *Ralstonia solanacearum* strains of race 3 biovar 2A in potato is regarded as an important disease contributing to significant yield reduction of between 50 to 100% [7,12]. The disease is considered more difficult to control in field crop production owing to pathogen's properties as a soil-borne bacterium, their broad host range and the genetic variation level within the strains which makes it difficult to employ a universal control measure [13]. However, effective and long term control or management strategy could be feasible by using a combination of diverse control methods such as the use of resistant/tolerance varieties, chemical, biological and cultural practices [14,15].

Bacterial wilt resistance in potato is very complex in nature; it is probably a function of genetic and environmental adaptation [16,17,18]. Studies indicate that inheritance of resistance to bacterial wilt is dominant, polygenic and quantitative in nature, and entail genes with major and minor effects [19,20]. Interaction between genes for resistance and those for adaptation is an

essential combining ability which appears to be a substantial attribute for expression of resistance [17,18,21].

Potato breeding for bacterial wilt resistance is very demanding with limited success owing to the pathogen variability, lack of resistance sources in the species, genetic complexity involved in resistance and the tetraploid background nature of the crop making the long road even longer, complex and rather vague [22,23,24,25,26]. Field selection has been effective in identifying stable resistance in progenies derived from crosses involving resistant wild relatives. Though field selection efficiency is reduced by pathogen variability, infection and disease development variability is laborious and requires uniformly infested fields.

Potato breeding requires genetic variation of useful traits for crop improvement. In potato, most often the desired variation is lacking due to preferences of few elite local traditional cultivars for potato improvement in most parts of the world. Cultivated potato cultivars have a narrow genetic base due to common pedigrees of breeding materials [27]. This presents a serious limitation to potato crop improvement, especially with the emergence of new diseases, pests and climatic changes making it difficult for yield improvement to be realized [28]. Irradiation of planting material with suitable doses, though genetic differences could exist, can produce small effects with several important biosynthetic processes and morphological traits [29]. The use of mutation breeding could widen the genetic base for selection of specific traits of interest.

2. MATERIALS AND METHODS

2.1 Plant Materials

A total of 160 potato mutant tubers at M1V4 generation that were generated from M1V3 generation from the 3 parents were used. The 3 non irradiated parents acted as controls. The development and advancement of the Mutant population was described by [30,31,32].

2.2 Experimental Site

The experiment was carried out at National Research Laboratories (NARL), Kabete Station of the Kenya Agricultural and Livestock Research Organization (KALRO). The KALRO-Kabete station is at an altitude of 1795 m above sea level, latitude of 1°15' 31.64" S and longitude 36° 46' 17.96" E [33]. The average annual rainfall is 1295 mm with a bimodal distribution. The mean air temperature ranges from 15.3 to 28.6°C. The soil type is humic nitosol derived from quartz trachyte [34]. The experiment was carried out for one season during December 2015 to 12 April, 2016.

2.3 Field Layout and Experimental Design

A total of one hundred and sixty (160) M1V4 mutant potato genotypes and three controls were planted for screening for bacterial wilt resistance. The experimental design used was an alpha lattice with twenty four blocks of seven plots each and replicated three times.

The linear model for alpha design, Latinized by block was:

$$y_{ijtl} = \mu + g_i + r_j + \alpha_t +, \alpha(r)_{jl} + \varepsilon_{ijtl}$$

The y_{ijtl} represent the observations, μ is the population mean, g_i the genotypic effects, r_j the resolvable replicate effects, α_t the Latinized block effects, $\alpha(r)_{jl}$ the incomplete block effects within replicates and ε_{ijtl} the random errors.

2.4 Inoculum Collection

Ralstonia solanacearum inoculums were obtained from naturally infected potato plants in farmer's field in Kitale, Trans-Nzoia County, Kenya. The wilted plants were collected from the field and preliminary diagnostic test carried out in the field to preclude the existences of other bacteria. Diagnosis in the field was easily accomplished through the vascular flow test [35]. A piece of stem about 2-3 cm long were cut from the base of a wilting potato plant and suspending in clear water in a glass container. The cut stem is held with an opened paper clip to maintain a vertical position. After some few minutes, the presence *R. solanacearum* within the vascular system will be confirmed by the smoke-like milky threads streaming downward from the cut stem [36,37,14]. Positive plants were taken to the laboratory where resistance assay of *R. solanacearum* isolates were obtained as described by [38].

2.5 Inoculum Preparation

The infected potato tubers was washed with water to remove soil particles and later immersed in 70% ethanol for 2 to 3 minutes to remove any other bacteria from the plant surface. It was cut aseptically and left for 5 minutes for the bacterial exudates to ooze. Culturing was done by streaking the oozing bacterial exudates onto a SMSA agar plates. The agar plates were then incubated at 28–30°C or at room temperature for 5 days. Populations of *R. solanacearum* were determined using a modified Semi-Selective Media South Africa (SMSA) method [39] before inoculating the field during the time of planting of the crop. The *Ralstonia solanacearum* colonies were then grown on Triphenyl Tetrazolium Chloride (TTC) medium to obtain pure cultures [40]. The stock inoculum solution was prepared and serial dilutions was prepared (10^{-3}, 10^{-5} and 10^{-7}) and plated on semi-selective media for *R. solanacearum* and was replicated twice. The plates were incubated at 30°C for 48 hour after which the bacterial colonies were counted and used for inoculation.

2.6 Planting, Inoculation and Crop Management

Planting was done on ridges spaced at 75 cm inter-row and 30 cm intra row for each genotype (mutant/control). Five plants were planted per plot/clone in an alpha lattice design with seven blocks each having twenty one plots with three replications. Di-ammonium phosphate (DAP) fertilizer (18:46:0) were applied as recommended and thoroughly mixed with soil before planting. Bacterial suspensions concentrated at 3.0×10^9 cfu/ml were poured into the planting furrows to boost the innoculum concentration in the soil. All standard agronomic practices were carried out according to recommendations for potato production in Kenya [41].

2.7 Data Collection

Data were collected on days to onset of wilting (DTOW) and then done after every 7 days, final bacterial wilt incidence (BWI), Total tuber numbers (TTN), proportion of symptomatic tubers based on total tuber number (PSTTN),
Total tuber weight in tons ha^{-1} (TTW), Proportion of symptomatic tubers based on total tuber weight (PSTTW), Proportion of ware sized tubers based on total tuber weight (PWTTW) and Area under the disease progress curve (AUDPC), were calculated using the BWI scores [37,42] using the formula below:

$$\mathrm{AUDPC} = \sum (t - t)$$

Where y_i is the BWI at i^{th} days, and n is the total number of sampling times, t is the number of days after planting.

2.8 Data Analysis

Data on TTN, TTW, PWTTW, PSTTN and PSTTW were first averaged on plot basis; the average values were then used to extrapolate values per hectare. The analysis of variance showing significant differences mean separation was done using Duncan Multiple Range Test and the potato mutants were also ranked to determine resistance to bacterial wilt. Genotypes with low values were more resistant to bacterial wilt and were ranked high.

2.9 Phylogenetic Analysis

Phylogenetic trees were produced using phenotypic data of selected agronomic and bacterial wilt traits. The unweighted UPGMA [43] and the hierarchical clustering method was used based on the dissimilarity matrix calculated with Manhattan index in the DARwin software version 6.0.9.

3. RESULTS

3.1 The Response of Potato Mutant Dosage Rates to Bacterial Wilt Disease

Table 1 showed that the days to onset of wilting (DTOW) was significantly different at $p \leq 0.05$ (Asante), $p \leq 0.01$ (Kenya Mpya) and $p \leq 0.001$ (Kenya Sherekea) potato mutant dosage rates.
The area under the disease progress curve (AUDPC) was significantly different in Asante ($p \leq 0.05$), Kenya Mpya ($p \leq 0.01$) and Kenya Sherekea ($p \leq 0.01$). Total tuber number (TTN) also exhibited significant difference in Asante ($p \leq 0.01$), Kenya Mpya ($p \leq 0.05$) and non significant in Kenya Sherekea mutants. Total tuber weight (TTW) was significantly different ($p \leq 0.05$) in Kenya Sherekea and non-significant

for Asante and Kenya Mpya mutants. Kenya Mpya and Asante mutants dose rates were significantly different ($p \leq 0.05$) in percentage of symptomatic ware sized tubers of total tuber weight in t/ha (PWTTW) and percentage of symptomatic tubers of total tuber number (PSTTN) per ha while percentage of symptomatic tubers of total tuber weight per ha (PSTTW) were non-significant in Asante but significant for Kenya Mpya mutant dosage rates. Kenya Sherekea mutant dose rates were significantly different in percentage of symptomatic tubers of total tuber number (PSTTN) ($p \leq 0.01$), percentage of symptomatic tubers of total tuber weight per ha (PSTTW) ($p \leq 0.001$) and non-significant for percentage of symptomatic ware sized tubers of total tuber weight in t/ha (PWTTW) (Table 1).

3.2 Correlation Analysis

Correlations analysis between days to onset of wilting (DTOW) and area under disease pressure curve (AUDPC) were positive and significant in Asante ($p \leq 0.01$) and Kenya Sherekea ($p \leq 0.05$) and negative but non-significant in Kenya Mpya mutants (Table 2). Correlations between days to onset of wilting and percentage of symptomatic of total tuber weight in t/ha (PSTTW), percentage of symptomatic tubers of total tuber number per ha (PSTTN) and total tuber number (TTN) were positive and significant in Kenya Sherekea mutants, positive and non-significant in Asante's mutants and negative and non
significant in Kenya Mpya mutants. On the other hand, correlations between PSTTW and PSTTN; TTN and total tuber weight (TTW) were positive and significantly different in all the mutant populations. Correlations between PSTTW and with all the other traits were positive and non-significant across the mutant populations. Kenya Sherekea showed more positive significant correlation among traits versus Asante and Kenya Mpya mutants.

Table 1. Effect of different dose rates on Asante, Kenya Mpya and Kenya Sherekea potato mutants for selected agronomic and bacterial wilt resistance parameters at KALRO NARL

Mutants	Dosage (Gy)	DTOW	AUDPC	TTN	TTW	PSTTN	PSTTW	PWTTW
Asante	0	49a	560ab	24.7bc	39.3a	10.3a	6.7a	4.76
	3	50.7a	573.6ab	15.1a	37.9a	11.5ab	7.1a	17.5c
	6	51.5a	536.7a	25.9bc	40.6a	22.3c	8.6a	12.7bc
	9	55.3ab	530a	35.2d	44.3a	19.5bc	10.5a	11.4abc
	12	53.6a	546.7a	30.7cd	33.5a	9.3a	5.9a	8.3ab
	15	63.2b	662.4b	22.3ab	44.4a	19.9abc	7.6a	17.3c
	Grand mean	59.3	568	25.6	40	14.47	7.7	12
	CV %	8.4	10.2	17.1	22.9	22.2	23.9	20.5
	EMS	20.7*	3354*	19**	ns	21.6*	ns	13.4*
Kenya Mpya	0	74.7b	410a	19.7ab	61.2a	19.5ab	7.2ab	4a
	5	51.3a	561.7bc	30.5b	47a	9.1a	5.9ab	10.04b
	6	54.4a	538.6bc	27.7ab	42a	18.4ab	9.2ab	9.07b
	10	51.3a	595c	30.7b	59a	10.3a	5.1a	10.09b
	15	44.7a	501.7b	14.2a	40.6a	29.9b	10.6b	13.23b
	Grand mean	55.3	521	24.5	50	17.4	7.6	9.3
	CV %	12.7	8.7	25	13.3	14.3	27.4	16.1
	EMS	49.7**	2035**	73*	ns	59.6*	8*	11*
Kenya Sherekea	0	49a	436.7a	25a	35.5ab	9.4a	5.2a	8.2a
	3	52.5ab	560bc	25.2a	36.9ab	22.5b	5.4ab	9.3a
	5	62.3c	526.7b	28a	40.5ab	15.1ab	8.5b	9.5a
	10	56.9bc	517.6b	33.6a	44.2b	9.5ab	7.6ab	4.8a
	12	50.2ab	560bc	31.8a	42.3b	10.5ab	7.3ab	6.6a
	15	56.4abc	535.4b	26.8a	40.4ab	11.7ab	6.8ab	9.6a
	20	61.8c	525b	37.7a	32.3ab	18.6ab	6.3ab	7.8a
	30	77d	613.3c	34.7a	27.9a	37.3c	15c	10.5a
	Grand mean	58.3	534.3	30.3	37.5	16.8	7.8	8.3
	CV %	7.4	6.3	28.9	19.2	44.7	23.7	22.6
	EMS	18.7***	1121**	ns	51.8*	56.4**	3.4***	ns

*ns=not significant, *=significant at p≤0.05, **=significant at p≤0.01, ***=significant at p≤0.001; DTOW= Days to onset of wilting; PWTTW= Percentage of ware sized tubers (% of total tuber weight in t ha^{-1}); TTW= Total tuber weight (t ha^{-1}); PSTTW= Percentage of symptomatic tubers (% of total tuber weight in t ha^{-1}); TTN= Total tuber number per ha; PSTTN= Percentage of symptomatic tubers (% of total tuber number per ha); AUDPC= Area under the disease progress curve; Error mean square (EMS), Percentage Coefficient of variation (CV %), Within each column, means having the same letter are not significantly different at p≤ 0.05*

3.3 Ranking of Potato Mutants for Tolerance to Bacterial Wilt Based on Selected Agronomic and Bacterial Wilt Traits

Table 3 shows the ranking of the top five (Asante, Kenya Mpya and Kenya Sherekea) potato mutants for tolerance to bacterial wilt based on selected agronomic and bacterial wilt traits. The ranking of the mutants were based on the mean of each of the selected agronomic and bacterial wilt traits. The mutant A67 of Asante genotype was ranked first overall and in total tuber weight. Mutant A57 was ranked second overall and first in days to onset of wilting. In Kenya Mpya

mutants, M6 was ranked first overall and in percentage of symptomatic tubers of total tuber number per ha. The M4 mutant was ranked fifth overall but ranked first in days to onset of wilting and area under disease progress curve. In Kenya Sherekea mutants, mutant S20 was ranked first overall and in area under disease progress curve and total tuber weight.

3.4 Genetic Diversity of Mutant Clones Based on Selected Traits

Four groups were formed in the dendrogram based on UPGMA cluster analysis of potato mutants using DARwin software package. Group I, II and IV had equal proportionate number of Asante, Kenya Mpya and Kenya Sherekea mutant populations. Group III contain large population of Asante's mutants (Fig. 1). Group 1 was the most diverse containing sub clusters with mutant A45 being clustered alone.

Table 2. Pearson correlation coefficients for various agronomic traits in Asante, Kenya Mpya and Kenya Sherekea potato mutants

AUDPC DTOW PSTTN PSTTW PWTTW TTN TTW

Asante AUDPC 1
DTOW 0.64** 1
PSTTN 0.02 ns 0.15 ns 1
PSTTW -0.01ns 0.14 ns 0.71** 1
PWTTW 0.31 ns 0.3 ns 0.23 ns 0.24ns 1
TTN -0.23ns 0.03 ns -0.15ns -0.23ns -0.37ns 1
TTW 0.03 ns 0.16 ns -0.19ns -0.37ns 0.14 ns 0.5* 1 Kenya Mpya AUDPC 1
DTOW -0.77ns 1
PSTTN -0.27ns -0.27ns 1
PSTTW -0.09ns -0.21ns 0.43* 1
PWTTW 0.37ns 0.67 ns 0.28ns 0.02ns 1
TTN 0.35ns -0.07ns -0.56ns -0.34ns -0.07ns 1
TTW 0.11ns 0.15 ns -0.4ns 0.23ns -0.45ns 0.17* 1 Kenya Sherekea AUDPC 1
DTOW 0.46* 1
PSTTN 0.50* 0.64*** 1
PSTTW 0.62** 0.71*** 0.59** 1
PWTTW 0.12ns 0.17 ns 0.35ns 0.12ns 1
TTN 0.14ns 0.4* -0.04ns -0.03ns -0.33ns 1
TTW -0.06ns -0.16ns -0.5* -0.28ns -0.25ns 0.52** 1 **=Significant at p≤0.05; **=Significant at p≤0.01; ns=Non-significant; DTOW= Days to onset of wilting; AUDPC= Area under the disease progress curve; PSTTN= Percentage of symptomatic tubers (% of total tuber number per ha); PSTTW= Percentage of symptomatic tubers (% of total tuber weight in t ha-1); PWTTW= Percentage of ware sized tubers (% of total tuber weight in t ha^{-1}); TTN=Total tuber number per ha; TTW= Total tuber weight (t/ha)*

Table 3. Asante, Kenya Mpya and Kenya Sherekea potato mutants ranked based on some agronomic and bacterial wilt resistance parameters

Mutants	Dosage (Gy)	Clones	DTOW	AUDPC	TTN	TTW	PSTTN	PSTTW	PWTTW	Overal Rank
Asante	15	A67	18.0	3.0	2.0	1.0	1.0	3.0	40.0	1
	15	A57	1.0	2.0	5.0	9.0	11.0	28.0	16.0	2
	15	A40	7.0	27.0	10.0	11.0	2.0	1.0	52.0	3
	15	A59	3.0	7.0	14.0	47.0	9.0	32.0	7.0	4
	15	A58	18.0	40.0	3.0	8.0	9.0	25.0	50.0	5
Kenya Mpya			25.0	2.0	1.0	3.0	2.0	22.0	14	6
		M30	4.0	8.0	5.0	3.0	12.0	19.0	27.0	11
		6	M25	23.0	18.0	4.0	8.0	2.0	9.0	19.0
		6	M4	1.0	1.0	9.0	10.0	18.0	25.0	22.0
		15	10	S20	11.0	12.0	1.0	1.0	8.0	9.0
		2.0	8	5	S14	29.0	4.0	4.0	12.0	5.0
		19.0	3.0	9	10	S21	11.0	12.0	2.0	3.0
		21.0	28.0	9.0	12	12	S29	36.0	12.0	2.0
Kenya Sherekea			5.0	3.0	2.0	5	27.0	10	12	S34
		1.0	2.0	29.0	15.0	6.0	20.0	24.0	13	
5	M6	5.0	12.0	14.0	17.0	5.0	1.0	3.0	7	6
M39	17.0									

DTOW= Days to onset of wilting; AUDPC= Area under the disease progress curve; PSTTN= Percentage of symptomatic tubers (% of total tuber number per ha); PSTTW= Percentage of symptomatic tubers (% of total tuber weight in t ha-1); PWTTW= Percentage of ware sized tubers (% of total tuber weight in t ha^{-1}); TTN=Total tuber number per ha; TTW= Total tuber weight

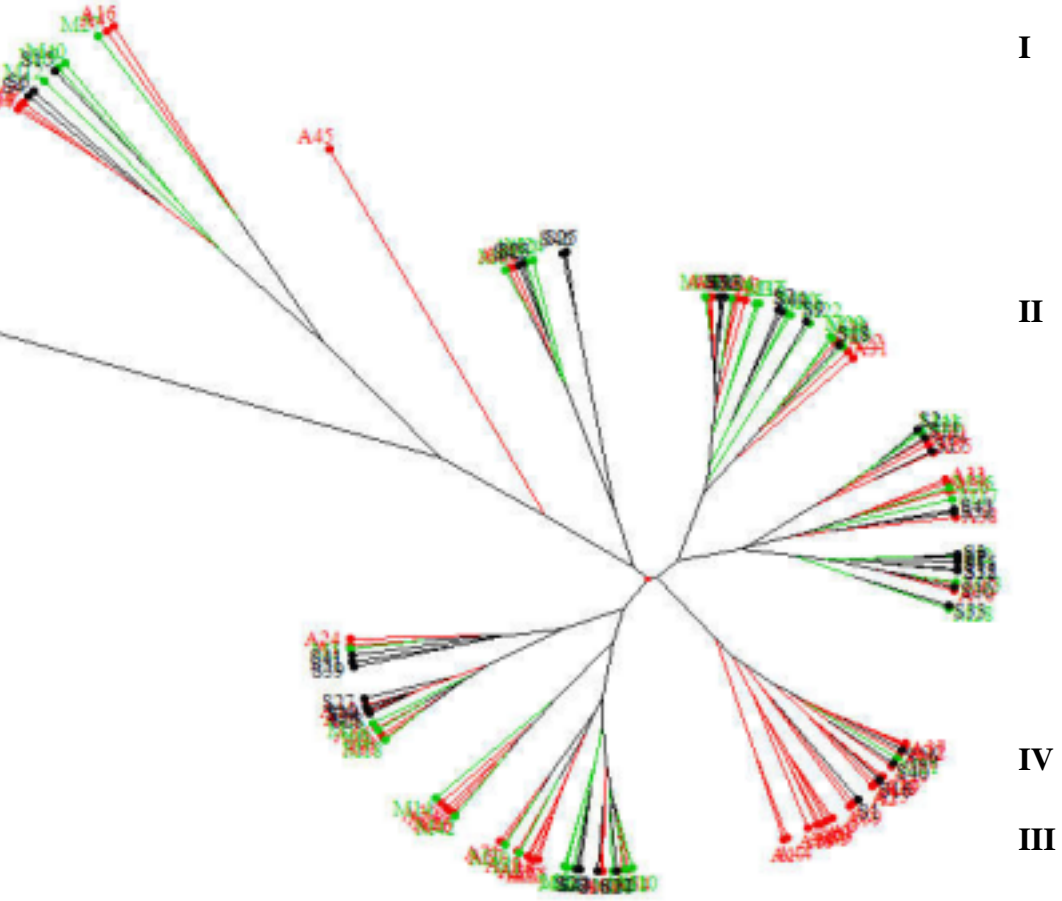

Fig. 1. Unrooted tree using Unweighted pair-group method of arithmetic averages (UPGMA) illustrating the genetic relationship 0among 163 potato mutants and 3 controls (Asante – red, Kenya Mpya – green and Kenya Sherekea – black) based on selected agronomic and bacterial wilt resistance parameters

4. DISCUSSION

This study revealed significant differences in days to onset of wilting (DTOW), area under disease progress curve (AUDPC) and percentage of symptomatic tubers of total tuber number per ha (PSTTN) in all the three potato mutant populations used. This is could be because these traits are scored directly from aerial parts and infected tubers infected with bacterial wilt disease. Similar trends have been reported by [44,45,46] for potato cultivars. The Asante, Kenya Mpya and Kenya Sherekea potato mutant populations displayed diverse resistance to bacterial wilt. The variation within each set of the potato mutants could be attributed the use of different dose rates and the reaction of each variety to the mutagen used. Since mutation is a random process, its effects are gigantic [29]. Potatoes with a broad genetic background for both bacterial wilt resistance and adaptation have a tendency to exhibit a higher level of resistance

and can be extra stable over environments [19].

Bacterial wilt incidence responded variably within the potato mutant populations in number of days after planting. The potato mutant populations with bacterial wilt incidence were observed to be significantly different at fourty days (Asante and Kenya Mpya mutants) and eighty four days (Asante and Kenya Sherekea mutants) after planting. This could be owed to the fact that resistance to bacterial wilt is dependent on the genotypes effects and the disease progress and development. Previous studies suggest that the expression resistance to bacterial wilt in potatoes is very complex and unstable in nature being attributed to high genetic variability of *R. solanacearum* strains and possibly to greater extent interaction between genes for resistance and those for adaptation [16,47,17,21,19]. In addition to the genotype effects, the observed differences could also be attributed to the changes in environmental conditions which can variably affect the entry, survival and development of the pathogen in the plant [48, 49].

Correlation analysis among most agronomic and bacterial wilt resistant traits was not consistent among the different potato mutant populations. This could be because the potato mutants were generated from different parental lines which might also have been influenced by the environmental conditions and induced mutation effects. Similar findings have been reported in the correlation between latent infection and all the other traits were not consistent [46]. Other studies have shown that plant susceptibility to bacterial wilt tuber latent infection and above ground are not correlated because the potential of clone‘s latent infection does not depend only on bacterial wilt incidence but also on other factors such as environmental conditions (soil texture, humidity and temperatures) [49,37].

The overall ranking of the three potato mutant populations with respect to selected agronomic and bacterial wilt traits showed that the best five mutants in Asante were from 15 Gy, Kenya Sherekea between 10 to 15 Gy while Kenya Mpya varied between 5 to 10 Gy. This suggests that potato mutants developed at dosages between 5 to 15 Gy could possibly result in giving better chances of obtaining potato with bacterial wilt resistance. Low dosage treatments (1 to 15 Gy) of gamma rays have been observed to stimulate growth attributed by increased cell division, and are genotype dependent [50,30] which could have an effect on any plant traits. Previous ranking of potato genotypes screened against bacterial wilt disease have been reported by [51,52,45,46].

The dendrogram generated based on the selected parameters (agronomic and bacterial wilt) did not group the potato mutants into different bacterial wilt resistant groups. The clustering pattern of the mutants revealed that mutants/lines originating from the same parents did not form a single cluster because of direct selection pressure and the random occurrence of the mutation induction. This is probably because bacterial wilt resistance is very unstable and complex due to strong host-pathogen environment interactions being involved [18,19, 46].

5. CONCLUSION

This study also sought to investigate the resistance of potato mutants as a function of induced mutation; it can be concluded that resistance of potato to bacterial wilt can be achieved through application of mutation technique. Asante mutants irradiated at dosage rates of 15 Gy gave a better response than Mpya and Sherekea mutants to bacterial wilt disease resistance.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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