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Combining ability and gene action for bacterial wilt disease resistance in wild tomato (*Solanum pimpinellifolium*) and cultivated tomato (*Solanum lycopersicum*) genotypes

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Bacterial wilt caused by *Ralstonia solanacearum* is one of the most destructive and widespread diseases of tomato in Kenya. The objective of this study was to determine the combining ability effects and gene action conditioning bacterial wilt disease resistance in tomato. Eight parents were crossed in North Carolina II mating design scheme to produce sixteen F_1 hybrids. The F_1 hybrids and the parental genotypes were evaluated for bacterial wilt in an *alpha* lattice design. Among the parents, KLF acc III was the best general combiner for area under the disease progress curve (AUDPC) and disease incidence across the two cropping cycles. Red Diamond × KLF acc III, Money Maker× KK acc I, Oxyly× KLF acc III and Money Maker× KK acc II were the best specific combiners for AUDPC. Low narrow sense heritability values of 0.14, 0.16 and 0.20 were obtained for AUDPC, disease incidence and plant survival. Relative weights of additive versus non-additive gene action obtained for AUDPC, disease incidence and plant survival were 0.19, 0.20 and 0.50. General predictability ratios (GPR) values of 0.27, 0.29 and 0.50 were obtained for AUDPC, disease incidence and plant survival. These results indicated the predominance of non-additive gene action in governing the traits.

Key words: Disease resistance, bacterial wilt, combining ability, gene action, tomato.

INTRODUCTION

Tomato (*Solanum lycopersicon* L.) is one of the most widely cultivated vegetables worldwide. The area under production of this vegetable in Kenya has been on the rise due to the increase in demand (FAOSTAT, 2018; Ochilo et al*.,* 2019). The consumption outstrips the demand and this result from low production that cannot meet the need of the population. Further, there is a gap between the actual and potential yield arising from limiting factors such as lack of suitable varieties coupled with inadequate crop management strategies for control of pests and diseases. Bacterial wilt caused by *Ralstonia solanacearum* has been identified as a major biotic constraint affecting tomato production in Kenya (Laeshita and Arwiyanto, 2017).

Studies carried out on the inheritance of resistance to bacterial wilt in tomatoes reported the significance of both

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major and minor genes in regulating the resistance.

Identifying genetic loci responsible for resistance traits, linkage analysis and genome-wide association studies (GWAS) have been widely used (St. Clair, 2010). Quantitative genetic resistance controlled by several genes/Quantitative Trait Loci (QTL), shows complex multigenic inheritance, making breeding efforts challenging (Pilet-Nayel et al., 2017). In disease resistance, haplotype association analysis has been used primarily to characterize diversity at a single target locus in diverse germplasm in order to facilitate the fine mapping of genomic regions containing known resistance loci (Krattinger et al., 2013). A single gene was important for control of bacterial wilt resistance in tomato (Grimault et al., 1995; Thakur et al., 2004). In contrast, the resistance of tomato to bacterial wilt was reported to be under the control of QTLs (Ishihara et al*.,* 2012).

The difference in the results has been attributed to the use of different sources of resistance, variations in environmental conditions and different isolates of *R. solanacearum* species complex (Da-Silvia et al*.,* 2018). The RSSC strains have been classified into the *R.* solanacearum species complex, which is composed of four major phylotypes classified according to their geographical origins: I (Asia), II (America), III (Africa and the Indian Ocean), and IV (Indonesia, Australia, and Japan) based on analyses of sequence data derived from the internal transcribed spacer (ITS) region between 16S and 23S (Fegan and Prior, 2005). Recently, the RSSC was taxonomically divided into three species, with phylotypes I and III being classified as *R. pseudosolanacearum,* phylotype II being classified as *R. solanacearum*, and phylotype IV being classified as *R. syzygii* (Prior et al*.,* 2015).

Heritability is a quantitative measure of the genetic variance in phenotypic variation and has predictive value in plant breeding. It indicates the extent to which a particular set of morphogenetic traits can be transmitted through successive generations (Waqar-Ul-Haq et al., 2008). Knowledge of heritability has an effect on the selection procedures used by the plant breeder in determining which selection methods would be most beneficial in improving the traits, predicting gain from selection and determining the relative importance of genetic effects (Laghari et al., 2010). Understanding gene action involved in bacterial wilt resistance in tomato would provide a basis for planning a breeding strategy for developing breeding populations that would lead to identification of superior lines through selection. Alleles with a dominant, additive, or deleterious phenotypic effect have a different effect on heritability when they are homozygous or heterozygous. Understanding how heterozygosity and homozygosity affect gene action and interaction will aid in determining whether hybrids or inbred lines should be used as the end product of breeding programs (Fasoula and Fasoula, 1997). Additive gene action is the mode of gene action in which each of two alleles makes an equal contribution to the generation of qualitative phenotypes. Non-additive gene action is the mode of gene action in which one allele is more strongly expressed than the other (Fasoula and Fasoula, 1997). Non-additive gene action was predominant over additive gene action for the control of resistance to bacterial wilt (Singh et al*.,* 2014). In contrast, additive gene action was important in bacterial wilt resistance (Oliveira et al., 1999). Information on combining ability can help to establish an effective breeding programme. Combining ability analyses is important for facilitating the choice of suitable parents for hybridisation (Suvi et al*.,* 2021). However, combining ability analyses and genetic predictions may depend on the test populations as well as the environment (Suvi et al*.,* 2021). Studies on combining ability have been carried out in other diseases of tomato and other crops. For instance, three tomato lines were identified as potential donors for resistance to tomato yellow leaf curl virus disease in a half-diallel mating design (Pandiarana et al., 2015). Parental lines with negative general combining ability (GCA) values and families with negative specific combing ability (SCA) values were selected for breeding for resistance to rice yellow mottle virus disease (Suvi et al., 2021)

Additive, dominance, and interaction effects of genes, genetic variation in quantitative or complex traits can be partitioned into many components. The additive genetic variance is the most important since it accounts for the majority of the association between relatives and the potential for genetic change via natural or artificial selection (Hill et al., 2008). Additive genetic variance occurs when genes have an additive effect on the quantitative trait. This leads in phenotypic deviation from the mean as a result of the inheritance of a particular allele and its relative effect on the phenotype. It quantifies the degree to which individual phenotype differences may be predicted as a result of allelic substitutions additive effects. Non additive genetic variance is linked with dominant gene acts that encompass the influence of recessive alleles at a particular locus (Singh and Singh, 2018).

The North Carolina II mating design has been widely employed in parental hybridisation for population development and investigating the inheritance of important traits of various crops (Acquaah, 2009; Makanda et al., 2010; Oppong-Sekyere et al., 2019). The design, allows a breeder to estimate the General Combining Ability and Specific Combining Ability (Acquaah, 2009). GCA is defined as a genotype's average performance in a series of hybrid combinations. SCA is defined as those instances in which certain hybrid combinations outperform or underperform their parental inbred lines on an average basis (Sprague and Tatum, 1942). On the basis of SCA, observations of the performance of various cross patterns have been used to infer the gene action at work. The high SCA effects

observed in crosses where both parents are good general combiners may be attributed to additive × additive gene action (Dey et al., 2014). The high SCA effects derived from crosses between good and poor general combiner parents may be attributed to the good general combiner parent's additive effects and the poor general combiner parent's epistasis effects, which fit the favourable plant attribute (Verma and Srivastava, 2004). High SCA effects manifested by low crosses may be due to a dominance type of non-allelic gene interaction that results in over dominance, rendering the interaction unfixable (Wassimi et al., 1986). Although studies have revealed the significance of both GCA and SCA in key traits of a number crops including quality traits, disease resistance and yield, limited information exists in the estimation of GCA and SCA from crosses between cultivated and with wild species of tomato (Tyagi et al*.,* 2018). Hence, the study focused on understanding the gene action involved in the control of bacterial wilt and its inheritance. Knowledge of inheritance will be handy in developing a breeding strategy for developing bacterial wilt resistant tomato for both greenhouse and field production.

MATERIALS AND METHODS

Experimental site

The experiment was carried out in the greenhouse at Egerton University, Njoro Campus in the Department of Crops, Horticulture and Soils. The site lies approximately at 35°55'58.0"E and 0°22'11.0"S and an altitude of 223 8 m above the sea level. The area is situated in the lower highland agro-ecological zone 3 (LH 3) (Jaetzold et al., 2012).

Genetic material

Eight parental genotypes including four commercial susceptible varieties and four wild tomato genotypes with resistance to bacterial wilt were used in the study. Detailed description of these parental materials is provided in Table 1.

Mating parental genotypes

Crossing blocks having eight parents were planted in the greenhouse. Four male parents were crossed to four female parents in North Carolina Design II mating scheme. A total of 16 F_1 progenies were obtained. The planting of the parental material was done by staggering to eliminate the possibility of differential flowering time in order to ensure a synchronized flowering period to allow successful crossing. This was achieved by planting the late flowering parents first followed by the early flowering.

Collection, isolation and preservation of *R. solanacearum* inoculum

Samples of five infected tomato plants showing bacterial wilting symptoms were collected from individual farms in Subukia, Nakuru County in Kenya for isolation of the pathogen. Geographical locations of the farms were recorded using the Global Position System. A quick field ooze test was carried out to distinguish *R. solanacearum* from vascular wilts that are caused by fungal pathogens. The stems of diseased tomato plants showing typical symptoms of bacterial wilt were cut using sterilized scalpel blades. The cut ends of the stem were placed in test tubes containing sterile distilled water. The presence of the pathogen was confirmed by the proliferation of fine milky white strands when the infected tissue is placed in water. These white strands are as a result of masses of bacteria, which come out of the margins of the cut portions within few minutes (Rohini et al., 2017).

The infected tomato plants collected from the field were washed under running tap water to remove sand and soil. Vascular tissues were extracted with a new sterile scalpel blade into sections of about 10 cm in length from collar region of wilted plants (Ahmed et al*.*, 2013). The tissues were surface sterilized for thirty seconds in 1% sodium hypochlorite solution, 70% ethyl alcohol followed by three repeated washings in sterile distilled water and blot dried. The stem sections weighing one gram were macerated in a test tube containing 10 ml of clean sterile distilled water to create a stock solution. The stock solution was serially diluted by adding 1 ml of bacterial solution to eight test tubes each containing 9 ml of sterile distilled water. Each test tube was vortexed and allowed to settle for at least ten minutes.

Isolation of the bacterium was done following streak plate method as described by Grover et al*.* (2012) on to 2, 3, 5 Triphenyl Tetrazolium Chloride (Kelman's TZC agar) medium (glucose 5 g, peptone 10 g, casein hydrolysate 1 g, agar 18 g, distilled water 1000 ml), 5 ml of TZC solution filter sterilized was added to the autoclaved medium to give a final concentration of 0.005%) according to the procedure of Seleim et al. (2014). One loopful of bacterial suspension was obtained from the eight test tubes and streaked on pre sterilized moisture free plates. The plates were incubated upside down in an incubator at 28 ± 2°C for 24-48 h. Single virulent colonies from the medium were characterized by dull white colour fluid with irregular round and light pink centres and these were further streaked on TZC plates to obtain pure culture of the isolates. The pure culture was transferred to 5 mL of sterile double distilled water in screw capped bottles where they were stored for experimental use under refrigeration at -20°C for maintenance of virulence

Experimental procedure

Sixteen F_1 alongside eight parents were sowed in a nursery for a period of about 5 weeks before transplanting. The experimental design was an *alpha*-lattice design of 4 blocks and 6 units within the blocks, in two replicates. The 16F_1s with 8 parental genotypes were inoculated with the cultured pathogen 14 days after transplanting. Before inoculation, incisions were made using a sterile scalpel on either side of the main stem to a depth of 5-6 cm each to cause injury to the secondary roots (Mwangi et al*.*, 2008). Thirty millimetres of the standardized bacterial suspension containing 1×10^9 colony forming units (CFU/ml) per ml inoculation of *R. solanacearum* was poured over the roots (Singh et al., 2018). Thereafter, the plants were watered at alternative days to maintain a high soil moisture for the development of the disease.

Data collection

All plants in each experimental unit were used for data collection. The disease symptoms were observed daily from 30, 45 and 60 days after inoculation (DAI). The percent disease severity in plants was evaluated using a scale of 0-5 as described by Kempe and Sequeira (1983) (Table 2 and Figure 1).

The disease evaluation data were summarized using the percent

Table 1. Description of parental genotypes used to generate F_1s hybrids.

Genotype	Source	Bacterial wilt response	Cultivation status	Role in crosses
Cal-J	Kenya Seed Company	Susceptible	Cultivated	Female
Money Maker	Kenya Seed Company	Susceptible	Cultivated	Female
Red Diamond	Continental Seed Company	Susceptible	Cultivated	Female
Oxyly	Royal Seed Company	Susceptible	Cultivated	Female
KK acc II	Kakamega County	Resistant	Wild	Male
KK acc I	Kakamega County	Resistant	Wild	Male
KISII	Kisii County	Resistant	Wild	Male
KLF acc II	Kilifi County	Resistant	Wild	Male

KK: Kakamega, KLF: Kilifi.

Table 2. Disease rating scale for bacterial wilt.

Rating scale	Description	Disease reaction
0	No symptoms	Highly resistant
1	1 to 25% leaves wilted	Resistant
2	26 to 50% leaves wilted	Moderately resistant
3	51 to 75% leaves wilted	Moderately susceptible
4	75% but less than 100% of leaves wilted	Susceptible
5	All leaves wilted and plant dead	Highly susceptible

Source: Moussa et al. (2017).

Figure 1. Disease severity scale of Bacterial wilt on tomato (HR-Highly Resistant, R-Resistant, MR-Moderately Resistant, MS-Moderately Susceptible, S-Susceptible and HS-Highly Susceptible).

disease severity (PDS) formula as described by Sharma and Saikia (2013) and expressed as the area under the disease progress curve (AUDPC). AUDPC values of 0-150, 151-300, 301-500 and > 500 were considered to represent very low, low, moderate and high levels of resistance, respectively (Jeger et al., 2001). AUDPC was estimated following Wilcoxson et al. (1975) as:

$$\text{AUDPC}=\sum_{i=1}^{n}\left(\frac{(y_i+y_{i+1})}{2}(t_{i+1}-t_i)\right)$$

Where, y_i is the % disease severity on the i^{th} scoring; t_i is the number of days from sowing to i^{th} scoring; n is the total number of scores.

Disease incidence was calculated using the following formula described by Gashaw et al. (2014) as:

$$\text{Disease incidence} = \frac{\text{Number of infected plants}}{\text{Total number of plants assessed}} \times 100$$

Data on plant survival was calculated using the formula described

by Jyoti et al. (2015) as:

$$\text{Plant survival} = \frac{\text{Number of healthy plants}}{\text{Number of plants established}} \times 100$$

Data analyses

Data for AUDPC were log transformed while data for disease incidence and plant survival were arcsine square root transformed to obtain a normal frequency distribution. Data were subjected to analysis of variance using the computer software programme GenStat 15th edition (VSN International, Hemel Hempstead, UK). The statistical model for the analysis was;

$$Y_{ijklm} = \mu + C_j + R_l + B_{k(l)} + G_i + GC_{ij} + \varepsilon_{ijklm}$$

Where; Y_{ijkl} is the observed performance from each experimental unit; C_j is the effect due to j^{th} cropping cycle; R_l is the effect due to l^{th} replicate; $B_{k(l)}$ is the effect due to k^{th} block within the l^{th} replicate; G_i is the effect due to i^{th} genotype; GC_{ij} is the effect due to interaction between the genotype and the cycle; ε_{ijklm} is the random error component.

Genotypes, cycles and replications were considered as fixed effect while blocks were considered as random effects. Mean separation was performed using Least Significant Difference (LSD) test at $p < 0.05$ given as:

$$\text{LSD} = t_{\frac{\alpha}{2},edf} \times \text{SED}$$

Where $t_{\frac{\alpha}{2}}$, error df is the t value for a significance level of α/2, error df is the number of degrees of freedom in the error term of the analysis of variance. SED is the Standard Error of Difference Combining ability analysis was done using Line × Tester procedure developed by Kempthorne (1957) and implemented in R software package version 4.0.4 in RStudio 1.4.1106 (Team, 2014). The linear model for combining ability analysis was as follows:

$$Y_{ijk} = \mu + g_i + g_j + S_{ij} + \varepsilon_{ijk}$$

Where; Y_{ijk} is the value of the ijk^{th} observation of the cross involving i^{th} cross, and j^{th} tester in the kth replication. μ is the general mean. g_i is the GCA effect of the i^{th} line. g_j is the GCA effect of the j^{th} tester. S_{ij} is the specific combining ability (SCA) effect of the cross involving i^{th} line and j^{th} tester. ε_{ijk} is the error associated with the ijk^{th} observation.

Narrow sense heritability was estimated, after derivation of the variance components using the following formula:

$$h^2 = \frac{\sigma^2 GCA}{\sigma^2 GCA + \sigma^2 SCA + \sigma^2 e}$$

Where h^2 heritability in narrow sense, σ^2 GCA is the variance of General Combining Ability, σ^2 SCA is the variance of Specific Combining Ability.

Relative weight of additive and non-additive gene action was estimated according to Verma and Srivastava (2004) which is given as:

$$\frac{\sigma^2 GCA}{\sigma^2 SCA}$$

Where σ^2 GCA is the variance of general combining ability, σ^2 SCA is the variance of specific combining ability.

Baker's ratios were also computed to estimate the relative importance of additive and non-additive gene action in the expression of disease traits using Baker's general predicted ratio (GPR) as follows:

$$GPR = \frac{2\,\sigma^2\,\text{GCA}}{2\,\sigma^2\,\text{GCA} + \sigma^2\,\text{SCA}}$$

Where σ^2 GCA is the variance of general combining ability, σ^2 SCA is the variance of specific combining ability.

A ratio of >0.5 implies that GCA is more important than SCA in the inheritance of the character and a ratio of < 0.5 implies that SCA is more important than GCA in the inheritance of the character (Baker, 1978).

RESULTS

Analysis of variance and phenotypic performance for AUDPC, disease incidence and plant survival

Significant ($p < 0.001$) variation among the genotypes was recorded across the cropping cycles for AUDPC and plant survival at 30 days and for AUDPC, disease incidence and plant survival at 45 and 60 days after inoculation (DAI) (Table 3). Cropping cycles effects were significant $p < 0.001$) for plant survival at 30 DAI, disease incidence and plant survival at 45 DAI and AUDPC, disease incidence and plant survival at 60 DAI. Effects due to interaction between genotypes and cropping cycles were significant ($p < 0.05$) for plant survival at 60 DAI, ($p < 0.01$) for plant survival at 30 and 45 DAI and ($p < 0.001$) for AUDPC at 60 DAI.

Genotypes expressed variation for AUDPC, disease incidence and plant survival in the two cropping cycles. There was a trend of high disease pressure in the first cropping cycle with mean AUDPC of 543 and 940 at 45 and 60 DAI compared to the second cropping cycle with mean AUDPC of 543 and at 45 and 563 at 60 DAI. In contrast, the plant survival was higher in the second cropping cycle at 45 and 60 DAI with 72 and 58% of the plants surviving compared to the first cropping cycle when only 56 and 38% of the plants survived at 45 and 60 DAI (Table 4).

In general, the crosses recorded lower values for AUDPC and disease incidence and high values of plant survival as compared to the parents. Three crosses Cal-J × KLF acc III, Oxyly × KLF acc III and Red Diamond × KLF acc III and four wild parental genotypes KK acc II, KK acc I, KISII and KLF acc III with AUDPC and disease incidence of 0 values and 100% plant survival were highly resistant compared to commercial varieties which displayed a susceptible reaction to bacterial wilt across cropping cycles (Tables 5 and 6). Apparently all the resistant F_1s were progenies of KLF acc III parent.

Combining ability analyses for parents and crosses

Means squares due to parents and crosses were

Table 3. Mean squares for AUDPC, disease incidence and plant survival of tomato genotypes at 30, 45 and 60 days after inoculation evaluated for two cropping cycles in the greenhouse at Egerton University, Njoro in 2020.

Source of variation	df	30 days after inoculation			45 days after inoculation			60 days after inoculation		
		AUDPC	DI	PS	AUDPC	DI	PS	AUDPC	DI	PS
Cycle	1	0.00	0.18	1.70***	0.00	0.19***	0.65***	0.06***	0.45***	1.60***
Rep(Cropping cycle)	1	0.01	0.03	0.02	0.00	0.02	0.01	0.00	0.30	0.00
Genotype	23	1.72***	0.14***	0.39***	3.04***	0.35***	0.62***	2.77***	0.73***	1.20***
Cycle ×Genotype	23	0.00	0.02	0.07**	0.00	0.01	0.02**	0.01***	0.04	0.06*
Residual	47	0.00	0.02	0.02	0.00	0.01	0.01	0.00	0.03	0.03
CV %		0.70	23.20	1.60	0.20	4.50	1.60	0.00	12.90	0.50

*, **, *** Significant at, (p< 0.05), (p< 0.01), (p< 0.001) respectively AUDPC Area under disease progress curve, PS: Plant Survival, DI: Disease Incidence, CV: Coefficient of variation.

Table 4. Range and mean values of AUDPC, Disease incidence and Plant survival at 45 and 60 days after inoculation for thirty-six tomato.

Cycle	45 days after inoculation						60 days after inoculations					
	AUDPC		Disease incidence		Plant survival		AUDPC		Disease incidence		Plant survival	
	Range	Mean± SE	Range	Mean± SE	Range	Mean± SE	Range	Mean± SE	Range	Mean± SE	Range	Mean± SE
1st cycle	0-945	543.±15.25	0-71	27±1.00	20-100	56±1.38	0-1575	940±26.23	0-93	48±1.38	0-100	38±1.76
2nd cycle	0-906	534±15.50	0-50	19±0.61	29-100	72±0.95	0-1352	564±23.48	0-79	39 ±1.17	0-100	58±1.38

Genotypic variation was displayed among the parents and the crosses for AUDPC, AUDPC: Area Under Disease Progress Curve, SE: Standard Error disease incidence and plant survival.

significant (p<0.001) for AUDPC, disease incidence and plant survival. Means squares of Parents× Crosses was significant (p<0.001) for AUDPC and disease incidence. Means squares due to Crosses were significant (p<0.001) for AUDPC, disease incidence and plant survival. Means squares due to Lines× Testers interaction were significant (p<0.001) for AUDPC and disease incidence. Means squares due to Testers was significant (p<0.01) for AUDPC and disease incidence and (p<0.001) for plant survival (Table 7).

Among the parents, KLF acc III recorded the lowest negative GCA value of -1.20 for AUDPC and -0.52 for disease incidence and high GCA value of 0.72 of plant survival (Table 8). Among the F_1s, Red Diamond × KLF acc III, Money Maker× KK acc I, Oxyly× KLF acc III and Money Maker× KK acc II recorded the lowest negative SCA values of -0.41, -0.40 and -0.39. For AUDPC. Red Diamond × KLF acc III recorded the lowest negative SCA value of -0.28 for Disease incidence (Table 9).

Relative weight of additive and non-additive gene action obtained for AUDPC, disease incidence and plant survival were 0.19, 0.20 and 0.50 respectively. Narrow sense heritability values of 0.14, 0.16 and 0.20 were obtained for AUDPC, disease incidence and plant survival. General Predictability Ratios (GPR) values of 0.27, 0.29 and 0.50 were obtained for AUDPC, disease incidence and plant survival. The proportional contribution to the total variation of the testers was higher for all the disease measurements as compared to the lines and the line by testers interaction (Table 10).

DISCUSSION

Bacterial wilt resistance is a major breeding objective for tomato improvement. This is because

Table 5. Mean values of AUDPC, disease incidence and plant survival at 30, 45 and 60 days after inoculation for 8 parents evaluated for bacterial wilt resistance in the greenhouse for two cropping cycles in the greenhouse at Egerton University, Njoro in 2020.

Genotypes	AUDPC		DI		PS		AUDPC		DI		PS		AUDPC		DI		PS	
	30 DAI						45 DAI						60 DAI					
	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2
KK acc II	0	0	0	0	100	100	0	0	0	0	100	100	0	0	0	0	100	100
KK acc I	0	0	0	0	100	100	0	0	0	0	100	100	0	0	0	0	100	100
KISII	0	0	0	0	100	100	0	0	0	0	100	100	0	0	0	0	100	100
KLF acc III	0	0	0	0	100	100	0	0	0	0	100	100	0	0	0	0	100	100
Money Maker	219	235	20	20	40	80	698	784	40	20	20	60	1220	1192	71	51	0	51
Oxyly	272	259	20	20	40	71	841	841	60	40	20	40	1469	1278	79	61	0	23
Red Diamond	299	306	10	5	40	80	902	902	39	29	20	50	1504	1339	71	61	0	9
Cal-J	314	278	50	50	20	50	945	861	71	50	20	29	1575	1278	93	79	0	23
CV %	3.10	1.2	22.30	21.1	0.7	2.4	1.0	0.0	5.5	1.2	1.4	1.7	1.50	0.2	11.2	14.9	1.2	1.7
LSD(0.05)	0.32	0.32	0.30	0.30	0.31	0.31	0.08	0.08	0.19	0.19	0.19	0.19	0.06	0.06	0.32	0.32	0.35	0.35

AUDPC: Area Under Disease Progress Curve; DI: Disease Incidence; PS: Plant Survival; DAI: Days After Inoculation; CC: Cropping Cycle; KLF: Kilifi; KK: Kakamega; CV: Coefficient of Variation, LSD: Least Significant Difference. [a]LSD values based on transformed data.

of the magnitude of yield loss inflicted by the disease which impacts negatively on tomato grown either in the field or under greenhouse conditions. Screening for bacterial wilt resistance has in the past resulted in identification of resistant cultivars (Acharya et al*.,* 2018; Oussou et al., 2020). Despite the existing reports on resistance to bacterial wilt in tomato, local varieties in Kenya are largely susceptible. Introgression of novel sources of resistance from diverse sources including cultivated species and wild relatives is a necessity towards deployment of bacterial wilt resistant tomato cultivars (Kim et al*.,* 2016). Such genetic improvement not only results in reduced yield gap but also helps to reduce production costs and limits the environmental hazards caused by overuse of bactericides.

To determine differential performance among tomato germplasm, AUDPC, disease incidence and plant survival were measured. The results from the analysis of variance revealed the importance of cropping cycle on the performance of tomato against bacterial wilt (Table 3). Significant genotype-by-cropping cycle (GC) interaction for plant survival at 30 and 45 days after inoculation (DAI) and AUDPC and plant survival at 60 DAI suggested that the genotypic performance was not independent of the difference among the cropping cycles. These findings agree with earlier reports (Ganiyu et al., 2017; Guji et al*.,* 2019) and implicate the screening conditions to be key in determining the outcome of disease screening experiment. The variation arising from effects of cropping cycle may result from inconsistent temperature and humidity within the greenhouse. High temperature coupled with high relative humidity accelerate disease development (Velásquez et al., 2018).

Significant main effects due to genotypes for AUDPC, disease incidence and plant survival at 30, 45 and 60 DAI explained the presence of genetic differences among the evaluated genotypes. The trend of higher mean values for AUDPC and disease incidence and lower plant survival at 45 and 60 DAI, observed in the first cropping cycle as opposed to the second cropping cycle suggested higher disease pressure in the second cycle among the genotypes (Table 4). The differential performance may be explained by an increase in temperature during the first cropping cycle. Namisy et al*.* (2019) found that high temperatures of between 28 to 36°C triggered increased disease pressure.

The observed genetic variation and mean performance of parents and their progenies was based on AUDPC, disease incidence and plant survival which revealed mixed levels of resistance and susceptibility (Tables 5 and 6). Parents with low mean values for AUDPC and disease

Table 6. Mean values of AUDPC, disease incidence and plant survival at 30, 45 and 60 days after inoculation for 16 F_1 hybrids evaluated for bacterial wilt resistance in the greenhouse for two cropping cycles in the greenhouse at Egerton University, Njoro in 2020.

Genotype	AUDPC		DI		PS		AUDPC		DI		PS		AUDPC		DI		PS	
	30 DAI						45 DAI						60 DAI					
	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2	CC1	CC2
Cal-J × KLF acc III	0	0	0	0	100	100	0	0	0	0	100	100	0	0	0	0	100	100
Oxyly × KLF acc III	0	0	0	0	100	100	0	0	0	0	100	100	0	0	0	0	100	100
Red Diamond × KLF acc III	0	0	0	0	100	100	0	0	0	0	100	100	0	0	0	0	100	100
Cal-J × KK acc II	172	199	0	0	100	100	579	651	29	20	80	80	1037	967	61	23	42	79
Money Maker × KK acc II	190	122	0	0	50	95	636	259	40	20	29	60	1138	427	79	51	4	51
Money Maker× KLF acc III	199	224	0	0	80	100	636	714	29	20	60	80	1165	1086	61	32	32	42
Oxyly × KISII	230	214	0	0	60	100	714	698	20	29	40	60	1249	1220	42	79	4	23
Cal-J × KK acc I	235	247	0	0	95	100	749	803	20	20	71	80	1308	1220	51	23	32	79
Cal-J × KISII	235	259	0	0	61	95	714	822	29	29	39	71	1192	1220	51	51	9	23
Oxyly × KK acc II	241	253	0	0	60	100	766	731	29	20	40	80	1308	1112	71	42	23	61
Money Maker × KK acc I	247	285	5	0	50	95	766	881	40	29	29	60	1308	1308	71	51	4	51
Oxyly × KK acc I	247	224	0	0	100	95	749	651	29	20	71	71	1435	990	61	32	32	42
Red Diamond × KK acc II	253	292	29	5	39	71	749	861	50	29	29	40	1278	1278	79	61	4	42
Money Maker × KSII	265	230	5	5	50	95	803	714	29	20	29	71	1370	1086	51	32	4	42
Red Diamond × KK acc I	292	285	5.	0	61	95	881	841	40	20	39	60	1469	1278	71	42	32	51
Red Diamond × KISII	306	272	29	5	40	60	902	822	60	40	20	40	1539	1220	79	79	0	0
Cv %	3.10	0.90	22.30	21.1	0.7	2.4	1.0	0.0	5.5	1.2	1.4	1.7	1.50	0.2	11.2	14.9	1.2	1.7
LSD(0.05)	0.32	0.32	0.30	0.30	0.31	0.31	0.08	0.08	0.19	0.19	0.19	0.19	0.06	0.06	0.32	0.32	0.35	0.35

AUDPC Area Under Disease Progress Curve, DI Disease Incidence, PS Plant Survival, DAI Days After Inoculation CC Cropping Cycle, KLF Kilifi, KK Kakamega, Cv Coefficient of variation, LSD Least Significant Difference. [a]LSD values based on transformed data.

Incidence and high mean values for plant survival indicated the presence of genes for resistance and the possible potential of transmitting these genes to their progenies (Fellahi et al., 2013). The difference in performance among the parents and the crosses for AUDPC, disease incidence and plant survival indicated the existence of genotypic variation among the parents and the crosses. Suvi et al. (2021) reported genotypic variation for rice yellow mottle virus mottle disease among parents and crosses in rice.

Significant mean squares due to testers for the diseases variates suggested the prevalence of additive genetic variance among the male parents in conferring resistance to bacterial wilt (Table 7). These results concur with the earlier findings (Ajjappalavara et al., 2010; Mosa et al., 2017; Kargbo et al., 2019) and therefore indicate that the genetic advance for the disease traits can be realised through hybridisation and selection. Significant mean squares for line × tester interaction for all the traits measured demonstrated the existence of non-additive genetic variance in bacterial wilt resistance. Presence of non-additive genetic variance in the current breeding populations presents the possibility of implementing a hybrid breeding programme that would exploit heterosis in addition to additive gene action to develop new varieties. Tomato hybrids are high yielding and widely cultivated in Kenya and therefore pyramiding resistance genes in inbred lines for deployment of resistant hybrid varieties would greatly improve (Ashkani et al.,

Table 7. Combining ability mean squares for AUDPC, disease incidence and plant survival during two cropping cycles in the greenhouse at Egerton University, Njoro in 2020.

Source of variation	Df	AUDPC	DI	PS
Replications	1	0.00	0.17	0.00
Treatments	23	1.96***	0.44***	0.79 ***
Parents	7	2.69***	0.73***	1.41***
Parents vs. Crosses	1	4.28***	0.39***	0.36
Crosses	15	1.46***	0.31***	0.53***
Lines	3	0.51	0.21	0.34
Testers	3	5.14*	1.04*	1.95**
Lines× Testers	9	0.54***	0.10***	0.11
Error	23	0.00	0.01	0.04

*, **, ***, Significant at (p< 0.01), (p< 0.001) and (p< 0.000) respectively, AUDPC: Area Under Disease Progress Curve, DI: Disease Incidence, PS: Plant Survival.

Table 8. General combining ability (GCA) effects of eight parents for AUDPC, disease incidence, plant survival during two cropping cycles in the greenhouse at Egerton University, Njoro in 2020.

GCA	AUDPC	DI	PS
Lines			
Cal-J	-0.16	-0.16	0.19
Money Maker	0.38	0.20	-0.29
Oxyly	-0.12	-0.10	1.06
Red Diamond	-0.10	0.06	-0.01
SE	0.10	0.04	0.07
Testers			
KK acc II	0.36	0.28	-0.20
KK acc I	0.43	0.17	-0.11
KISII	0.42	0.07	0.40
KLF acc III	-1.20	-0.52	0.72
SE	0.10	0.04	0.07

AUDPC: Area Under Disease Progress Curve, DI: Disease Incidence, PS: Plant Survival, KK: Kakamega, KLF: Kilifi, SE: Standard error.

2015; Dormatey et al., 2020). QTL for resistance to tomato late blight was identified in a wild tomato accession (Arafa et al., 2017). QTL linked to bacterial wilt resistance in tomato have been reported by Wang et al. (2018). The QTL identified exhibited a stable and consistent expression. Kumar et al. (2018) identified QTLs linked to bacterial wilt resistance. The QTLs was found to be significantly associated with bacterial wilt resistance. However, bacterial wilt still remains a challenge in tomato production and information on stability of the identified QTLs and their utilization in breeding for resistance is limited. Negative and lower GCA effect for AUDPC and disease incidence recorded by the parent KLF acc III indicated that it was the best general combiner for resistance to bacterial wilt disease (Table 8). Similar findings were reported by Odogwu et al. (2016) bean rust resistance in common bean (*Phaseolus vulgaris*). The crosses Money Maker× KK acc II, Oxyly× KLF acc III and Red Diamond × KLF acc III recorded negative and lower SCA) effects for AUDPC which showed that these crosses were good specific combiners for resistance to bacterial wilt (Table 9). Bokmeyer et al. (2009) reported that negative SCA effects are desirable for disease resistance.

Heritability is possibly the most important statistic that can be obtained from variance components (Kearsey et al., 1996). Narrow sense heritability measures the proportion of phenotypic variation which arises from additive effects of genes in a given population. Low narrow sense heritability estimates of 0.14, 0.16 and 0.20 obtained for disease traits (Table 10) indicated that dominance gene action was critical in expression of disease resistance for the traits. Low heritability estimates imply that prediction of progeny performance would

Table 9. Specific combining ability (SCA) effects of 16 F1s for AUDPC, disease incidence, plant survival during two cropping cycles in the greenhouse at Egerton University, Njoro in 2020.

Genotype	AUDPC	DI	PS
Cal-J× KK acc II	0.01	0.02	0.09
Cal-J× KK acc I	0.13	0.02	-0.10
Cal-J× KISII	0.13	0.02	-0.05
Cal-J× KLF acc III	-0.36	-0.07	0.06
Money Maker× KK acc II	-0.39	-0.12	0.12
Money Maker× KK acc I	-0.40	-0.12	0.03
Money Maker× KISII	-0.37	-0.23	0.32
Money Maker× KLF acc III	1.17	0.47	-0.46
Oxyly× KK acc II	0.17	0.08	-0.05
Oxyly× KK acc I	0.14	0.08	-0.02
Oxyly× KISII	0.08	-0.03	-0.08
Oxyly× KLF acc III	-0.39	-0.12	0.14
Red Diamond× KK acc II	0.13	0.02	-0.16
Red Diamond × KK acc I	0.13	0.02	0.09
Red Diamond × KISII	0.15	0.24	-0.19
Red Diamond × KLF acc III	-0.41	-0.28	0.30
SE	0.02	0.08	0.14

AUDPC: Area Under Disease Progress Curve, DI: Disease Incidence, PS: Plant Survival, KK: Kakamega, KLF: Kilifi, SE: Standard Error.

Table 10. Estimates of genetic variance components and percentage contribution of the lines, testers and their interaction to the total variation for AUDPC, disease incidence and plant survival.

Parameter	AUDPC	DI	PS
GCA	0.05	0.01	0.02
SCA	0.27	0.05	0.04
GCA/SCA	0.19	0.20	0.50
(h^2)	0.16	0.14	0.20
GPR	0.27	0.29	0.50
% contribution			
Lines	7.08	13.41	13.01
Testers	70.61	66.54	73.82
Lines × testers	22.31	20.04	13.17

AUDPC: Area Under Disease Progress Curve, DI: Disease Incidence, PS: Plant Survival, GCA: General Combining Ability, SCA: Specific Combining Ability, h^2: Narrow sense heritability, GPR: General Predictability Ratio.

be difficult because of prevalence of non-heritable variation (Schmidt et al., 2019). Therefore, a selection procedure that could accumulate positive resistance genes should be adopted. Nsabiyera et al. (2013) reported similar low narrow sense heritability value of 0.16 for bacterial spot. In contrast, Da- Silva Costa et al. (2018) reported narrow sense heritability values of 0.26 and 0.53 for bacterial wilt.

Relative weights of additive and dominance gene action of 0.19, 0.20 and 0.50 respectively for disease traits indicated the superiority of non-additive gene action in their expression (Table 10). Verma and Srivastava (2004) reported the preponderance of non-additive gene action in the expression of traits. General predictability ratio of 0.27, 0.29 and 0.50 for disease traits revealed the predominance of non-additive gene action over additive gene action. This implies that the selection will not be effective and therefore the traits can be improved through use of hybrid vigour. The results are in agreement with Nsabiyera et al. (2013) who reported the predominance of non-additive gene action in the expression of disease traits. In contrast, the inheritance of bacterial wilt has been reported to be controlled by a single dominant gene (Grimault et al., 1995; Thakur et al., 2004). Oliveira et al.

(1999) reported additive gene action for resistance to bacterial wilt. Monma et al. (1997) reported the inheritance of bacterial wilt to be partially recessive. Sharma and Sharma (2015) reported the genetic control of bacterial wilt to be oligogenic. In addition, Da- Silva Costa et al. (2018) reported the predominance of additive gene action in the expression of bacterial wilt. Da- Silva Costa et al. (2018) reported the predominance of additive gene action in the expression of bacterial wilt. The proportional contribution of lines, testers and their interaction for the disease traits indicated that testers played an important role in inheritance of disease resistance. The testers contributed more positive alleles for the disease traits (Kargbo et al., 2019). Although both the gene action and both general and specific combining ability effects were evidenced, the predominance of non-additive gene action showed the presence of heterozygosity among the genotypes. From the results, all the four parents were resistant to bacterial wilt. One parent out of four was identified as the best general combiner for bacterial wilt disease. Out of the sixteen crosses, three crosses were resistant to bacterial wilt and had good specific combining ability for bacterial wilt disease resistance. The parent and the three crosses would be useful in tomato breeding programme for the development of a resistant tomato genotypes against bacterial wilt.

Conclusion

This study revealed the significance of non-additive gene action in conferring resistance to bacterial wilt. The parental genotype KLF acc III is the best general combiner for bacterial wilt disease. The cross combinations Money Maker× KK acc II, Oxyly× KLF acc III and Red Diamond × KLF acc III had good specific combining ability for resistance to bacterial wilt. From the results, a good breeding strategy would be to concentrate resistance genes in inbred lines with good genetic background through a backcrossing scheme followed by testing for general and specific combing ability for development of hybrids and potential future deployment of genetic resistance in tomato production in Kenya.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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