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Inheritance of resistance to common bacterial blight in four selected common bean (*Phaseolus vulgaris* L.) genotypes

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Common bacterial blight (CBB) is the most serious bacterial disease of common bean in Uganda. It causes severe yield losses of up to 62%. Genetic resistance is the most effective option for controlling CBB in smallholder common bean production systems. This study was carried out to determine the inheritance pattern of CBB resistance in leaf and pod of four new resistance sources. The four resistant and four susceptible genotypes were crossed in a half-diallel mating design. F_1 individuals were advanced to F_2 and evaluated with the parents, in a randomized complete block design replicated twice. Combining ability analysis was performed according to Griffing's (1956) method IV and model 1 using Genstat 12th. General combining ability effects were significant whereas specific combining ability was not suggesting that resistance to CBB in leaf and pod was primarily controlled by additive genes effects. The estimated narrow sense coefficient of genetic determination was moderately high (0.65) for the resistance in leaf and high (0.83) for resistance in pod suggesting that early-generation selection would be effective. Baker's ratio estimates were relatively high for resistance in leaf (0.79) and pod (0.9) suggesting that hybrids' performance can be predicted based on the parents' general combining ability (GCA) effects.

Key words: *Xanthomonas axonopodis* pv. *phaseoli,* general combining ability, additive gene effects, coefficient of genetic determination.

INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) is one of the most important grain legumes for human consumption

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worldwide (Gepts et al., 2008). It provides a highly nutritious food for more than 300 million people in thetropics (CGIAR, 2014), including Uganda where it is a major source of dietary protein and calories (Broughton et al., 2003). Uganda is the second largest common bean producer in Africa, after Tanzania, with a production of 876,576 metric tons in 2014 (FAOSTAT, 2015); however, its productivity is low because the crop is stressed by various abiotic and biotic factors (Ongom, 2010). Among the stresses, common bacterial blight (CBB) caused by *Xanthomonas axonopodis* pv. phaseoli (Xap) and *X. axonopodis* pv. phaseoli var. fuscans (Xapf), is the most destructive bacterial disease of bean causing up to 62% yield losses (Opio and Namayanja, 2002). Host plant resistance, through breeding, has been suggested as the most effective measure to control the disease (Durham, 2011; Fourie et al., 2011).

Understanding the mode of inheritance and type of gene action is crucial for successful breeding (Chataika et al., 2011). In addition, choosing the appropriate breeding method requires the breeder to consider the relative contributions of the genetic (additive and non-additive) and environmental variances to phenotypic variation (Agoyi et al., 2016). Several inheritance studies have been conducted on CBB resistance and different results were reported depending on various factors such as the pathogenic variability and the genetic background of the parental lines (Fourie et al., 2011). Quantitative inheritance pattern was reported by Arnaud-Santana et al. (1994) for the leaf and pod reaction to CBB using BAC-6 and XAN-159 as genetic donors. Similarly, Miklas et al. (2003) reported that the inheritance of CBB resistance in Montana No. 5 was polygenic with at least one major-gene effect. Tryphone et al. (2012), Muimui et al. (2011) and Zapata et al. (2011) reported that CBB resistance was governed by a single dominant gene in resistant lines Wilk-2 and VAX6, VAX4 and PR 0313-58, respectively. Arnaud-Santana et al. (1994) reported low narrow sense heritability (h^2) values (0.08-0.15) for leaf and pod reactions to CBB while Tryphone et al. (2012) reported moderate narrow-sense heritability (NSH) for foliar resistance (0.32). Depending on the cross, Ariyarathne et al. (1994) found low to intermediate (0.30-0.60) and intermediate to moderately high (0.49-0.76) heritability estimates for leaf and pod reactions, respectively. A relative high h^2 value of 0.8 was reported by Ferreira et al. (2004) in an F6:7 derived lines from the cross between HAB- 52 and BAC-6.

The inheritance of resistance to CBB disease depends on the germplasm being used, thus, determining the type of gene action controlling the trait and heritability for new breeding lines is a key step in determining which breeding strategy to use for CBB resistance. The objective of this study was, therefore, to determine the mode of inheritance and estimate the coefficients of genetic determination for leaf and pod resistance to CBB in four newly selected potential sources of resistance.

MATERIALS AND METHODS

Study site

This study was carried out under screenhouse conditions at the National Crop Resources Research Institute (NaCRRI) – Namulonge of Uganda, located in Wakiso District, at an altitude of 1150 masl on latitude 0°32'N and longitude 32°53'E. The institute falls in a bimodal climate region with an average annual rainfall of 1200 mm and average annual temperature of 21 to 27°C.

Genetic material and experimental design

In 2015, a collection of one hundred and thirty-two accessions was tested for CBB resistance under greenhouse conditions at NaCRRI, Uganda. The accessions included thirty-two landraces, twenty-seven released varieties and seventy-three introduced lines. Among the introduced lines, there were fifty common bean genotypes, previously selected under CBB inoculations in Nebraska. These genotypes included 12 lines from the University of Nebraska dry bean breeding program, 27 from the Andean Diversity Panel, and 11 from the Shuttle Breeding Program between Nebraska and Puerto Rico. Based on the screening trial of 2015 in Uganda, the four most CBB resistant lines were selected for this study. These four resistant lines and four popular, locally adapted but susceptible landraces (Table 1) of common bean were crossed in a half-diallel mating design. The F1 progenies were advanced to F2 generation and the latter was evaluated along with the parental lines in a randomized complete block design experiment with two replications. Six seeds were sown in 5-L buckets and then thinned to four plants after germination. Each plot consisted of three buckets for the parental lines and six buckets for the crosses with four plants per bucket. This gives a total of 12 plants per parental line and 24 plants per cross in a plot. Each bucket contained a mixture of forest black soil, lake sand and decomposed farm yard manure in a ratio of 3:1:1. 300 g of NPK fertilizer was diluted in 10 L of water, from which 100 ml were added to the soil on a weekly basis until the reproductive stage of pod filling (Belarmino, 2015).

Inoculum

Plants were inoculated with the isolate "Kawempe 1" which is a fuscans variant of X. axonopodis pv. phaseoli. The isolate was earlier identified by CIAT-Uganda as the most prevalent and one of the most virulent pathotype of Xapf in Uganda and confirmed by Belarmino (2015). The stored culture of "Kawempe 1" was revived, grown and multiplied on Yeast Dextrose Carbonate Agar medium and 48 h after initiation of the culture, suspension of inoculum was produced and diluted with sterilized water up to the recommended concentration of 5 ×107 CFU/ml following CIAT protocol.

Inoculation

Second trifoliate leaves of 21-day old seedlings were inoculated using the razor blade method (Opio et al., 1994) by pressing the leaflet onto a sponge soaked with bacteria suspension (in a petri-dish) and making two small gentle cuts at the edge. Two pods per plant were inoculated using multiple needle sticks at pod filling stage (Opio et al., 1994). Four punctures were made on both sides of the pod, which was then pressed onto the sponge soaked with inoculum sap.

Table 1. Characteristics of the selected parental lines.

Parental lines	Seed color	Seed size	Growth habit	Source	CBB status
Masindi Yellow	Yellow	Medium	I	NaCRRI	Susceptible
Bumwufu	Red	Medium	IV	NaCRRI	Susceptible
Ocuci	Black	Small	II	NaCRRI	Susceptible
KATB1	Yellow	Medium	I	Katumani-Kenya	Susceptible
NE2-14-8	Cream + Green stripes	Small	IV	University of Nebraska	Resistant
VAX3	Red	Small	II	CIAT	Resistant
NE14-09-78	Cream + Red stripes	Medium	II	University of Nebraska	Resistant
NE17-14-29	Dark Red	Medium	IV	University of Nebraska	Resistant

NaCRRI: National Crop Resources Research Institute; CIAT: International Center of Tropical Agriculture; I: Determinate habit; II: Indeterminate bush with erect branches and stem; III: Indeterminate bush with weak stem and branches; IV: Indeterminate Climbing with weak, long and twisted stem and branches.

Data collection

Disease severity was measured on leaves at 21 and 35 days after inoculation (DAI), and on pods at 10 days after inoculation, using the CIAT 1-9 rating scale of van Schoonhoven and Pastor-Corrales (1987). The disease scores of individual plants were used to calculate an average score for each genotype per plot.Average scores of 1.0 to 3.4 were considered resistant, 3.5 to 6.4 intermediate and 6.5 to 9.0 susceptible.

Data analysis

Data were analyzed using Genstat software 12th edition (VSN International). The means of the 20 F2 family crosses and eight parental lines were compared in an analysis of variance using the following linear model for randomized complete block experimental design:

$Yij = \mu + Gi + Rj + eijk;$

where μ is the grand mean, Gi is the mean effects of the ith genotype, Rk is mean effect of the kth replication and eijk is experimental error.

Combining ability analysis was performed whereby the genetic variance component was partitioned into general and specific combining ability (GCA and SCA) variances according to Griffing's (1956) method IV, model 1. This allowed quantifying the magnitude of the additive and non-additive gene effects for common bean resistance to CBB disease. Parents were considered as fixed because they were chosen purposely considering their level of resistance to CBB. The statistical linear model used was:

$Yij = \mu + gi + gj + sij + eij$

where μ is the grand mean, gi and gj are GCA effects of the ith and jth parents respectively, sij is the SCA effect for the combination between the ith and jth parents and eij is experimental error.

Broad and narrow sense coefficient of genetic determination (BS-CGD; NS-CGD) were computed on family means basis using the formulas described by Dabholkar (1999). The relative importance of additive versus non-additive gene effects was determined according to the ratio established by Baker (1978). All negative values of estimated variance components were considered as zero in the formulas of coefficient of genetic determination (Prof Bruce Walsh, 2015; personal communication).

$BS\text{-}CGD = (2 \times \sigma^2GCA + \sigma^2SCA) / (2 \times \sigma^2GCA + \sigma^2SCA + \sigma^2e /r)$

$NS\text{-}CGD = 2 \times \sigma^2GCA / (2 \times \sigma^2GCA + \sigma^2SCA + \sigma^2e /r)$

$BR = 2 \times \sigma^2GCA / (2 \times \sigma^2GCA + \sigma^2SCA)$

where r is number of replications, σ^2GCA and σ^2SCA are variance components estimates of GCA and SCA, respectively and σ^2e is the variance due to experimental error.

A two-tailed t-test was performed to test the significance of individual parent GCA and F2 family cross SCA effects using the following formula: tGCAi = GCAi/SEGCA and tSCAi = SCAij/SESCA, where GCAi is the GCA effect of the ith parent and SCAij is the SCA effect of the combination between the ith female and jth male parents, SEGCA and SESCA are the standard errors of GCA and SCA effects, respectively.

RESULTS AND DISCUSSION

Response of F_2 family crosses and parental lines to CBB disease

The analysis of variance showed that both parents and crosses reacted significantly differently for CBB severity symptoms on leaf at 21 DAI ($p < 0.050$) and 35 DAI ($p < 0.001$) and on pod at 10 DAI ($p < 0.001$) (Table 2). This indicates that there was high genetic variability among the parental lines and their resulting F2 families. Genetic diversity is the primary condition for crop improvement (Bernado, 2010) as it provides a wide genetic base for selection to achieve high genetic gain. The high genetic diversity observed in this study will therefore favour selection among parental lines and crosses for breeding for leaf and pod resistance to CBB disease.

The disease severity mean scores of the F2 family crosses and parental lines are presented in Table 3. Parents NE2-14-8, VAX3, and NE14-09-78 had resistant reaction for both leaf and pod symptoms whereas parent NE17-14-29 had an intermediate (4.6) reaction to CBB disease on leaf. On the other hand, Masindi Yellow, Ocuci, Bumwufu and KATB1 showed a susceptible reaction both on leaf and pod. The most resistant parents to CBB disease were NE2-14-8 and VAX3 with a disease

Table 2. Analysis of variance of parents and F_2 families' resistance to CBB.

Source of variation	d.f.	Leaf_21 DAI	Leaf_35 DAI	Pod_10DAI
Rep	1	7.02**	0.08^{ns}	0.11^{ns}
Parents	7	8.98***	11.52***	10.44***
Families	19	1.28*	2.76***	6.3***
Error	27	0.6	0.6	0.66

ns: Non-significant, *, **, ***significance at 0.05, 0.01, 0.001 probability levels, respectively, d.f.: degrees of freedom

Table 3. Mean performance of the parents and F_2 families resistance to CBB.

Variable	Genotypes	Leaf_21 DAI	Leaf_35 DAI	Pod_10DAI
Parents	Masindi Yellow	6.8^{bc}	7.3^{b}	6.7^{d}
	Bumwufu	7.8^{c}	8.3^{cd}	6.9^{d}
	Ocuci	6.5^{b}	7.5^{bc}	5.9^{d}
	KATB1	8.0^{c}	8.5^{d}	6.9^{d}
	NE2-14-8	3.2^{a}	3.3^{a}	3.0^{bc}
	VAX3	3.2^{a}	3.3^{a}	2.1^{ab}
	NE14-09-78	3.3^{a}	3.4^{a}	1.3^{a}
	NE17-14-29	4.1^{a}	4.1^{a}	4.0^{c}
	LSD (0.05)	1.3	0.9	1.6
F_2 families	Ocuci/NE14-09-78	4.6^{bcde}	5.1^{cdef}	4.0^{c}
	Ocuci/NE17-14-29	5.7^{ef}	6.4^{fg}	3.1^{bc}
	Ocuci/KATB1	5.3^{def}	6.9^{g}	7.0^{d}
	Ocuci/VAX 3	4.8^{cdef}	4.8^{bcdef}	4.1^{c}
	Bumwufu/Ocuci	5.1^{def}	6.8^{g}	6.3^{d}
	Bumwufu/NE2-14-8	4.3^{dbcde}	4.3^{dbcd}	3.5^{bc}
	Bumwufu/NE14-09-78	4.3^{dbcde}	4.4^{dbcd}	4.0^{c}
	Bumwufu/KATB1	4.3^{dbcde}	4.3^{dbcd}	7.3^{d}
	Bumwufu/VAX 3	4.7^{bcdef}	4.7^{bcde}	3.4^{bc}
	NE2-14-8/NE14-09-78	2.8^{d}	2.9^{d}	2.6^{dbc}
	NE2-14-8/NE17-14-29	4.1^{dbcd}	4.7^{bcde}	2.2^{db}
	NE2-14-8/VAX 3	4.4^{dbcde}	4.6^{bcd}	2.2^{db}
	KATB1/NE14-09-78	4.3^{dbcde}	4.4^{dbcd}	3.5^{bc}
	KATB1/VAX 3	4.5^{bcde}	4.6^{bcde}	3.4^{bc}
	Masindi Yellow/Ocuci	4.9^{def}	5.8^{defg}	6.1^{d}
	Masindi Yellow/NE2-14-8	4.1^{dbcd}	4.2^{dbc}	2.7^{dbc}
	Masindi Yellow/NE14-09-78	3.2^{db}	3.3^{db}	3.6^{bc}
	Masindi Yellow/NE17-14-29	5.7^{ef}	6.2^{efg}	2.3^{db}
	Masindi Yellow/KATB1	5.2^{def}	6.8^{g}	7.4^{d}
	VAX 3/NE17-14-29	6.1^{f}	6.3^{fg}	2.2^{db}
	LSD (0.05%)	1.67	1.59	1.68

LSD: Fisher's protected least significant difference.

score of 3.3 on leaf at 35 DAI and NE14-09-78 with a score of 1.3 on pod at 10 DAI. The most susceptible parents were Bumwufu with a score of 8.3 on leaf and Bumwufu and KATB1 with a score 6.9 on pod. These cultivars behaved as expected on the basis of their CBB status in Table 1.

The F_2 family average scores for CBB severity ranged from 2.8 to 6.1 and 2.9 to 6.9 for CBB disease symptoms on leaf at 21 DAI and 35 DAI, respectively (Table 3). In both cases the cross NE2-14-8/NE14-09-78 had the highest level of resistance of 2.9 followed by the cross Masindi Yellow/NE14-09-78 with disease scores of 2.9

and 3.3, respectively. Both crosses had in common the parent NE14-09-78 suggesting that it was a good transmitter of foliar CBB resistance to its progenies. This parent would, therefore, be a promising source of CBB resistance in leaf. In the case of CBB resistance in pod, the disease severity scores ranged from 2.2 to 7.4. Three crosses VAX 3/NE17-14-29, NE2-14-8/VAX 3 and NE2-14-8/NE17-14-29 had the highest level of resistance, with a disease score of 2.2, followed by the crosses Masindi Yellow/NE17-14-29 and NE2-14-8/NE14-09-78 with disease scores of 2.3 and 2.6, respectively. These results revealed that in this set of crosses, all these four resistant parents were good transmitters of CBB resistance in pods with genotype NE17-14-29 as top. The mean scores of the crosses NE2-14-8/NE14-09-78 and NE2-14-8/NE17-14-29 were lower than either their two respective parents for resistance in leaf and pod, respectively, indicating the presence of transgressive segregation that probably resulted from the interaction of complementary resistant genes present in both parents. Transgressive segregation is a common phenomenon observed in hybrid plant population as the results of this study are consistent with the ones of Musaana et al. (1993) who also reported the presence of transgressive segregation for leaf and pod resistance to CBB in common bean. The presence of transgressive segregants among the crosses NE2-14-8/NE14-09-78 and NE2-14-8/NE17-14-29 implies that higher levels of CBB resistance can be achieved by pyramiding the resistant genes/QTLs from these parental lines (Durham, 2011).

Combining ability for leaf and pod resistance to CBB

The combining ability analysis revealed that the parents had significantly different general combining ability (GCA) effects for Leaf_21 DAI ($p < 0.01$) and Leaf_35 DAI ($p < 0.001$) and Pod_10DAI ($p < 0.001$) suggesting that additive gene effects were involved in the control of resistance to CBB disease in these genotypes. On the other hand, the specific combining ability (SCA) effects of the crosses were not significant for any of the disease assessment dates indicating that the proportion of non-additive genes effects in the control of resistance to CBB disease was not significant. These results are similar to those reported by Rodrigues et al. (1999) who observed non-significant SCA effects for resistance to CBB in leaves but differ from reports by Trindade et al. (2014) who found both GCA and SCA effects to be significant. The significant SCA effects reported by Trindade et al. (2014) could be due to the use of Griffing's (1956) diallel method 2 that involved selfs, whereby the parental lines which were genetically different (resistant versus susceptible), contributed to strong and significantly different SCA effects values. The concept of combining ability was first introduced by Sprague and Tatum (1942) who partitioned the total genetic variance observed among crosses into GCA and SCA where GCA was indicative of additive genetic effects and SCA non-additive (dominance and epistasis) effects.

Both additive and non-additive effects are important factors that breeders consider during the selection of potential parents for hybridization. For a self-pollinated crop like common bean, the additive genetic effects give a better basis for forecasting the breeding value of a parent for hybrids as they represent the transmitted effects from one generation to the next (Hallauer et al., 1988; Rubaihayo, 1996). In this study, additive genetic effects were significantly involved in the inheritance of resistance to CBB as opposed to the non-additive effects suggesting that new CBB resistant cultivars can be derived from these segregating populations. On the same note, high values of Baker's (1978) ratio of 0.8 and 0.9 were observed in this study for CBB resistance in leaf and pod, respectively, thus confirming the high relative importance of additive genetic effects over the non-additive effects in this set of crosses. High values of Baker's ratio imply high predictability of a hybrid's performance for resistance to CBB disease on the basis of the parents' GCA effects (Dabholkar, 1999). In other words, in this instance, progeny with the highest level of leaf and pod resistance to CBB would be obtained by crossing the two parents having the lowest GCA effects (Baker, 1978).

Broad and narrow coefficients of genetic determination

The estimates of broad and narrow sense heritability in form of coefficient of genetic determination are presented in Table 4. High broad sense heritability estimates (83 and 0.92% for leaf and pod, respectively) were obtained, suggesting a high genetic contribution towards the phenotypic variance of CBB resistance in this study. As a result, only 17 and 8% of the phenotypic variation for leaf and pod reaction to CBB, respectively, were due to environmental variance implying that the phenotypes reflected the genotypes.

The estimates of narrow sense heritability were moderately high (0.65) for the resistance in leaf and high (0.83) for resistance in pod suggesting that high proportion (65 and 83% for leaf and pod resistance, respectively) of the phenotypic variation observed among crosses was due to additive genetic effects. These findings are similar to results reported by Belarmino (2015) and Ferreira et al. (2004) but contrary to those of Tryphone et al. (2012) and Arnaud-Santana et al. (1994) who reported low to moderate narrow sense heritability for CBB resistance in leaf and pod. These contrasting results likely reflect differences in the parental lines used to generate the segregating populations, and indicate that estimates of heritability value depend on the population,

Table 4. Mean square, variance components and coefficients of genetic determination for F_2 families reaction to CBB disease.

Source of variation	d.f.	Leaf_21 DAI	Leaf_35 DAI	Pod_10DAI
GCA	7	1.37**	2.72***	7.39***
SCA	12	0.22^{ns}	0.60^{ns}	0.68^{ns}
Residual	27	0.30	0.30	0.33
σ^2_{GCA}	-	0.25	0.56	1.65
σ^2_{SCA}	-	-0.08	0.30	0.35
$\sigma^2_{Residual}$	-	0.30	0.30	0.33
BR	-	1.00	0.79	0.90
NS-CGD	-	0.63	0.65	0.83
BS-CGD	-	0.63	0.83	0.92

ns: Non-significant; *, **, ***Significance at 0.05, 0.01, 0.001 probability levels respectively, d.f.: degrees of freedom; NS-CGD: narrow sense coefficient of genetic determination; BS-CGD:= broad sense coefficient of genetic determination; BR: Baker's ratio.

Table 5. General combining ability (GCA) effects of the parents.

Parental lines	Leaf_21 DAI	Leaf_35 DAI	Pod_10DAI
Masindi Yellow	0.03	0.17	0.52
Bumwufu	0.10	0.05	0.97 ***
Ocuci	0.35	0.90 ***	1.19 ***
KATB1	0.12	0.43	1.65 ***
NE2-14-8	-0.77 **	-0.85 **	-0.91 **
VAX3	0.14	-0.35	-1.27 ***
NE14-09-78	-0.75 **	-1.21 ***	-1.19 ***
NE17-14-29	0.88 **	0.85 **	-1.48 ***
SE_{GCA}	0.23	0.25	0.26

**, ***Significance at 0.01, 0.001 probability levels respectively, DAI: Days after inoculation.

environmental conditions and the genetic complexity of the trait under study (Singh and Miklas, 2015). The high value of coefficient of genetic determination observed in this study suggests that the inheritance of leaf and pod resistance to CBB disease is primarily controlled by additive genetic effects. As results, since additive genetic variance represents the transmitted genetic effects and ultimately the main determinant of genetic gain from selection, breeding methods involving early-generation selection like pedigree and mass selection would be effective for breeding for CBB resistance among this set of crosses (Hallauer et al., 1988).

Combining ability effects

The estimates of parents GCA effects are presented in Table 5. Genotypes NE2-14-8 and NE14-09-78 had significant ($p < 0.01$ and $p < 0.001$) negative GCA effects for leaf resistance to CBB contributing 1.1 disease units, on average, towards resistance. Genotypes Ocuci and NE17-14-29 had significant ($p < 0.01$ and $p < 0.001$) positive GCA effects contributing, therefore, to susceptibility. In the case of pod resistance to CBB, all four resistant parents had significant negative GCA effects and contributed about 1.2 disease units, on average, towards resistance. In contrast, the susceptible genotypes Ocuci, Bumwufu and KATB1 contributed significantly ($p < 0.001$) towards susceptibility (on average, 1.25 disease score units).

These results showed that genotypes NE14-09-78 and NE2-14-8 were good transmitters of resistance to CBB both in leaf (GCA effects of -1.21 and -0.85, respectively) and pod (GCA effects of -1.19and -0.91, respectively) and can be very useful for introgressing CBB resistance into local susceptible genotypes. The parent NE17-14-29, although contributed to foliar susceptibility, had the greatest GCA effect for pod resistance and, therefore, could be utilized for transferring pod CBB resistance into susceptible materials.

The estimated values of specific combining (SCA) ability effects are presented in Table 6. None of the

Table 6. Estimates of specific combining ability effects values of the crosses.

Crosses	Leaf_21 DAI	Leaf_35 DAI	Pod_10DAI
Ocuci/NE14-09-78	0.33	0.28	-0.04
Ocuci/NE17-14-29	-0.10	-0.44	-0.64
Ocuci/KATB1	0.19	0.50	0.10
Ocuci/VAX 3	-0.31	-0.83	0.13
Bumwufu/Ocuci	0.00	0.79	0.11
Bumwufu/NE2-14-8	0.33	0.00	-0.56
Bumwufu/NE14-09-78	0.36	0.53	0.16
Bumwufu/KATB1	-0.53	0.65	0.65
Bumwufu/VAX 3	-0.16	-0.08	-0.36
NE2-14-8/NE14-09-78	-0.28	-0.11	0.64
NE2-14-8/NE17-14-29	-0.60	-0.39	0.57
NE2-14-8/VAX 3	0.36	0.68	0.33
KATB1/NE14-09-78	0.29	0.08	-0.99
KATB1/VAX 3	-0.38	-0.52	-0.97
Masindi Yellow/Ocuci	-0.12	-0.30	0.35
Masindi Yellow/NE2-14-8	0.18	-0.18	-0.98
Masindi Yellow/NE14-09-78	-0.70	-0.78	0.23
Masindi Yellow/NE17-14-29	0.21	0.09	-0.81
Masindi Yellow/KATB1	0.43	1.17 *	1.21 *
VAX 3/NE17-14-29	0.49	0.74	0.88
SE_{SCA}	0.54	0.55	0.58

*Significant at 0.05 probability level.

crosses had significant SCA effects except Masindi Yellow/KATB1 which had significant ($p < 0.05$; 1.17 and 1.21 for Leaf_35 DAI and Pod_10DAI, respectively) SCA effects. This suggests that there were no significant differences between the actual and expected (based on the parents' GCA effects) performance of the crosses, thus contributing to the low non-additive component of the genetic effects to CBB resistance in this set of crosses.

Conclusion

This study showed that leaf and pod resistance to CBB disease was mainly controlled by additive gene effects among these selected common bean genotypes. The crosses involving genotypes NE14-09-78 and NE2-14-8, just like their parents, showed good level of resistance to CBB disease and both parents had good GCA effects for both leaf and pod resistance. This indicates that these two genotypes are good sources of genetic resistance to CBB that can be utilized in bean breeding programs. The results also suggested that early-generation selection would be effective.

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CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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