Full Length Research Paper

Inheritance of cowpea resistance to flower thrips in Uganda germplasm

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Flower thrips [*Megalurothrips sjostedti* (Trybom)] is the most damaging insect pest on cowpea. However, information regarding the nature of gene action governing the inheritance of resistance to thrips is not available for cowpea genotypes in Uganda. This study was carried out to determine the inheritance pattern of cowpea resistance to flower thrips. Five resistant cowpea genotypes and three susceptible genotypes were crossed in full diallel mating design. F2 progenies were evaluated along with the parents in alpha lattice design with two replications under natural thrips infestation at Kabanyolo, Arua and Serere in Uganda. Combining ability analysis was performed using method one and model one of diallel analysis. The results showed that the environmental effects were highly significant (P<0.001). Additive, dominance and epistasis effects had major contributions. The broad sense heritability varied from 18 to 42% for thrips damage scores and from 0 to 6% for thrips counts. The estimates of narrow sense heritability were low for thrips damage score (2 to 18%) and thrips counts (0 to 9%). Genotypes TVU-1471 and TVU-1509 were identified as good transmitters of resistance to flower thrips. Crosses TVU-1509 x NE5, TVU-473 x Sanzi, TVU-123 x Sanzi, TVU-123 x TVU-473, and TVU-473 x TVU-1509 presented significant (P<0.05) and negative SCA effects for thrips damage scores and thrips counts and would be the most useful in breeding as some of their progenies would have high resistance to flower thrips. This study provides the basis of an efficient breeding program of cowpea for flower thrips resistance.

Key words: Damage score, gene action, *Megalurothrips sjostedti*, *Vigna unguiculata*.

INTRODUCTION

Cowpea [*Vigna unguiculata* (L) Walp] is one of the most important legume crops grown in semi-arid tropical regions in Africa (Afiukwa et al., 2013). The crop is majorly produced in West Africa, with Nigeria as the leading producer and consumer, accounting for 61% in Africa (FAOSTAT, 2013). Uganda is also among

the ten leading producers of cowpea and is ranked 8th in Africa (Ronner and Giller, 2012; Ddamulira et al., 2015). In Uganda, cowpea is ranked 4th after beans, groundnuts, and soybean (Ddamulira et al., 2015). Cowpea is mostly grown in the drier eastern and northern parts of Uganda (Dungu et al., 2015). This is because the crop is tolerant to drought and adapted to warm weather, hence it can produce significant yield where other legumes like beans fail to grow. However, its production is still constrained by several yield reducing factors such as thrips (*Thysanoptera; Thripidae*) which are the most important biotic stress with devastating effects on cowpea in Uganda (Hall et al., 2003). The species *Megalurothrips sjostedti* appears most destructive in Uganda, causing 20 to 100% yield losses under severe infestation (Karungi et al., 2000b).

Cultural practices recommended to limit thrips infestation include irrigation, tillage operation, planting date, crop rotation and intercropping (Dormatey et al., 2015). However, the incidence of multiple infestations in cereals, vegetables, and cowpea (Gbaguidi et al., 2013) precludes effective control through these methods. Insecticides use which has also been recommended for thrips has a major drawback such as rapid development of insecticide resistance in thrips populations rendering the chemical treatment ineffective (Dormatey et al., 2015; Sobda et al., 2017). In addition, these chemicals are expensive and sometimes need to be applied with special equipment putting them out of reach of the majority of resource-poor farmers. In order to minimize yield losses associated with thrips damage in cowpea, a major component of long lasting and affordable control package would be genetic control via host plant resistance. In fact, the identification and deployment of host-plant resistance in elite cultivars to manage thrips would minimize dependence on costly and environmentally toxic chemicals (Boukar et al., 2016). Therefore, concerted efforts are being made to develop varieties of cowpea resistant to flower thrips. Although there is evidence that low levels of resistance to flower bud thrips exists in some cowpea varieties, the desired levels of resistance have not yet been identified among available cowpea landraces and improved varieties (Omo-Ikerodah et al., 2009). In fact, this is very important as genes from resistant cowpea varieties can be incorporated through crossing with susceptible but desirable cowpea varieties to achieve more durable resistance (Muchero et al., 2008). A low level of resistance to thrips was reported in the cowpea accession TVU-1509 and its genes for resistance have been transferred to some other improved cowpea breeding lines (Omo-Ikerodah et al., 2009). Additionally, a landrace, Sanzi, from Ghana has also been identified with a high level of resistance to flower thrips in Nigeria, Mali, Cameroon and Kenya (Ngakou et al., 2008; Omo-Ikerodah et al., 2009; Domartey et al., 2015) and would be promising resistance transmitter.

In genetic analysis of cowpea resistance to flower thrips in Nigeria, Omo-Ikerodah et al. (2009) reported that more than two genes probably control the resistance to flower thrips and that additive × additive and dominance × dominance gene effects contribute to resistance to flower thrips. In addition, Domartey et al. (2015) reported that additive, dominance and epistatic gene effects made major contributions in Ghana. However, information regarding combining ability and nature of gene action governing the inheritance of thrips damage resistance is not available for cowpea genotypes in Uganda. This limits the introgression of resistance into susceptible landraces with desirable traits using resistant lines in breeding program. The knowledge of the genetic control of complex quantitative traits and the magnitude of genetic variability that exists among the available germplasm are important for selection and genetic improvement of the crop (Umar et al., 2014; Mwale et al., 2017). Furthermore, combining ability and heritability estimates are specific to germplasm being tested and the testing environments (Umar et al., 2014), highlighting the need to conduct such studies on the available germplasm under local environments. Therefore, the objective of this study was to elucidate the mode of inheritance of genes controlling the resistance to flower thrips among the resistance sources in order to set a breeding program of cowpea for integrated pest management in Uganda.

MATERIALS AND METHODS

Sites description

The evaluation of the genetic population was conducted at Makerere University Agricultural Research Institute, Kabanyolo (MUARIK), National Semi-Arid Resources Research Institute, Serere (NaSARRI), and at Abi-Zonal Agricultural Research and Development Institute, Arua (Abi ZARDI), considered as flower thrips hotspots in Uganda. The description of study sites is detailed in Table 1. The experiment was carried out during the long rainy season of 2017 (15 March - 30 June 2017).

Development of genetic populations

Three thrips-susceptible genotypes (WC36, MU9 and NE5 originated from Uganda) and five resistant genotypes (TVU-1471, TVU-473, TVU-123 and TVU-1509 from Nigeria and Sanzi a landrace from Ghana) were crossed using the full diallel mating design to generate 56 sets of F1s and reciprocals crosses and 8 selfed parents. The F1s and the reciprocal plants were selfed to generate F2 seeds. The F2 seeds were harvested from individual plant in bag.

Experimental design

The F2 seeds and the parents seeds were field grown in the three locations during the first rainy season of 2017 (15 March -30 June 2017) in alpha lattice design (8 blocks x 8 genotypes per block) with two replications. Seeds from each F1 plant were planted on a 2-row plot, 2 m long with inter-row and intra-row spacing as 0.75 and 0.30 m, respectively leaving one plant per hill. To ensure high infestation pressure of flower thrips, spreader rows of susceptible genotype,

Table 1. Description of study locations.

Locations	Geographical coordinates[a]		Altitude (m.a.s.l)[a]	Averageannual temperature[a] (°C)	Average annual rainfall[a] (mm)	Soils[b]
	Latitude	Longitude				
MUARIK (Wakiso)	0°28'N	32°37'E	1200	21.50	1150	Sandy clay loam
Abi-ZARDI (Arua)	3°4.58'N	30°56'E	1206	24	1250	Sandy clay loams
NaSARRI (Serere)	1°35'N	33°35'E	1140	26.05	1419	Black clays

m.a.s. l, Meters above sea level; [a&b], source: [a]Sserumaga et al .(2015); [b] Fungo et al. (2011).

Table 2. Scale for rating flower bud thrips damage on cowpea.

Rating	Appearance
1	no browning/drying (i.e scaling) of stipules, leaf or flower buds; no bud abscission
3	initiation of browning of stipules, leaf or flower buds; no bud abscission
5	distinct browning/drying of stipules and leaf or flower buds; some bud abscission
7	serious bud abscission accompanied by browning/drying of stipules and buds; non-elongation of peduncles
9	very severe bud abscission, heavy browning, drying of stipules and buds; distinct non-elongation of (most or all) peduncles

Source: Jackai and Singh (1988).

WC36 were planted around the experimental plot and between the rows two weeks prior to planting of the test materials (Abudulai et al., 2006). Thirty-five days after planting, the spreader row plants were uprooted and laid down between the test plots. No pesticide was applied.

Data collection

Data collected included thrips damage rating, number of thrips per flower, number of peduncles per plant and number of pods per peduncle. The total number of pods per plant was computed from the number of pods per peduncle and the number of peduncles per plant.

The test materials were rated for damage on a scale of 1-9 from 30 to 51 days after planting according to Jackai and Singh (1988). Rating was based on a combination of varying intensities of browning of the stipules and flower buds, non-elongation of peduncles and flower bud abscission (Table 2). Populations of thrips were estimated by randomly picking 20 flowers per plot. The samples were taken early in the morning, between 7 and 9.00 am during the peak of flowering. The flowers were placed in glass vials containing 70 % ethanol solution and subsequently dissected to count the number of thrips (Abudulai et al., 2006; Omo-Ikerodah et al., 2009). The infestations were assessed four times (30, 37, 44, and 51 days after planting) during the crop phenology.

Data analyses

The data pertaining to thrips resistance; number of thrips/flower, thrips damage rating and number of pods/plant, were subjected to analysis of variance (ANOVA) using the linear mixed model procedure using Genstat software 12th edition (Payne et al., 2009). The assumptions of ANOVA (The error terms are randomly, independently, and normally distributed, with a mean of zero and a common variance) were verified before analyzing the data using

Genstat procedures. The analysis of variance per location was performed to verify the ANOVA assumptions before the across location analysis. The number of thrips/flower were transformed using logarithm base 10 function to conform to the homogeneity of the error variance. The linear mathematical model for alpha lattice experimental design used was as follows:

$$y_{ijlm} = \mu + \rho_i + \iota_j + r_l + b_{m(l)} + \rho\iota_{ji} + \varepsilon_{ijlm}$$

Where, y_{ijlm} is the observed value for the ith genotype from j^{th} location, m^{th} block nested within the lth replication; μ is the general mean effect; ρ_i the ith genotype effect (considered as fixed effect); ι_j the jth location effect (considered as fixed effect); r_l the lth replication effect (considered as random effect); $b_{m(l)}$ the effect of mth block nested within the lth replication (considered as random); $\rho\iota_{ji}$ the interaction effect of jth location and i^{th} genotype (considered as random); and ε_{ijlm} the experimental error considered as random.

The means for each trait were separated using Fisher protected Least Significant Difference (LSD) at 5% level.

Estimates were made for additive and non-additive gene effects, the coefficient of genetic determination and the number of genes governing resistance to flower thrips. The coefficient of genetic determination (CGD) is a fixed-parent analog of heritability, since heritability only strictly applies to a random population arising from random parents.

The data was analyzed in (Analysis of Genetic Designs with R for Windows (AGD-R) Version 2 (Rodríguez et al., 2015), using model one, method one of Griffing (1956) to determine the effects of general combining ability (GCA) and specific combining ability (SCA) for different parents and crosses across locations. This method is expected to provide unbiased estimates of population parameters (Griffing, 1956; Dabholkar, 1992; Singh and Chaudhary, 2004). A fixed model was used because the parents were selected purposely, based on their levels of resistance to flower thrips and other agronomic traits. The statistical model for this analysis was:

$$Y_{ijk'l} = \mu + g_i + g_j + S_{ij} + r_{ij} + l_{k'} + b_l + lg_m + ls_{ijk'} + lr_{ijk'} + e_{ijk'l}$$

Where μ is the overall mean, g_i the GCA effect of the ith parent, g_j the GCA effect of the jth parent, s_{ij} the SCA effect of the ith genotype, r_{ij} the reciprocal effect of the ith genotype, l_k the effect of kth location, b_l the effect of lth block, lg_m, the effect of mth interaction between location and genotype, $ls_{ijk'}$, the effect of the interaction between k^{th} location and SCA of the ij^{th} genotype, lr_{ijk} the effect of the interaction between k^{th} location and reciprocal of the ij^{th} genotype, and $e_{ijk'}$ the environmental effect of the $ijk'l^{th}$ observation.

The analysis of variance (ANOVA) used for the study of the combining ability was adopted from Singh and Chaudary (2004), following Griffing's (1956) method one, model one.

The ratio of GCA variance to SCA variance was also estimated according to Baker (1978) as;

$X = 2\sigma^2_g / (2\sigma^2_g + \sigma^2_s)$. This ratio suggests the relative significance of additive versus non-additive effects (Baker, 1978).

Since the parents were fixed, variance ratios were used to obtain the narrow-sense coefficient of genetic determination (NSCGD) and broad-sense coefficient of genetic determination (BSCGD), using: NSCGD = $2\sigma^2 g/ (2\sigma^2 g + \sigma^2 s + \sigma^2 e)$ and BSCGD = $(2\sigma^2 g + \sigma^2 s) / (2\sigma^2_g + \sigma^2_s + \sigma^2_e)$. Fehr (1987) referred to this estimate as "repeatability", meaning that it approximates heritability for non-random samples and the results obtained pertain only to those genotypes under study and cannot be used to infer what would be expected if random genotypes were studied.

The standard error (S.E) of the estimated general and specific combining ability and reciprocal effects were calculated using the formulas provided by Dabholkar (1992):

$$S.E_{gi} = \sqrt{\frac{p-1}{2p^2} * \delta e^2} , \quad S.E_{sij} = \sqrt{\frac{p^2-2p+2}{2p^2} * \delta e^2}, \quad S.E_{rij} = \sqrt{\frac{1}{2} * \delta e^2}$$

Where, g_i is the GCA effect of the ith parent, s_{ij} the SCA effect of the jth genotype, r^{ij} the reciprocal effect of the ith genotype, and δ^2_e the error mean square (MSE).

Segregation ratios of the F2 populations were computed to understand the nature of inheritance and to estimate the number of genes influencing flower thrips resistance. Plants or genotypes with thrips scores of 1-3 were considered resistant, 4-6 moderately susceptible, 7-9 highly susceptible. For analysis, resistant were grouped as R, and all higher ratings were grouped as S. A chi-square goodness-of-fit test was used to determine the departure of the observed frequencies from the hypothesized frequencies, using $\chi^2 = \sum_{i=1}^{classes} \frac{(N_{obs}-N_{exp})^2}{N_{exp}}$ (Rubaihayo, 1996), where N_{exp} is the expected count for a class and N_{obs} the count actually obtained. When χ^2 was significant at $P < 0.05$, the fit of a model was rejected.

Several phenotypic classes were tested: 3:1 (single dominant gene); 15:1 (duplicate dominant epistasis); 9:7 (duplicate recessive epistasis); 13:3 (dominant and recessive epistasis); 63:1 (three independent dominant genes); 57:7 (one dominant and two complementary genes); 27:37 (three complementary dominant genes); 37:27 (three complementary recessive genes); 61:3 (two dominant and one recessive gene), 49:15 (one dominant and two recessive genes); and 249:7 (two dominant and two complementary genes) (Singh and Chaudhary, 2004; Caixeta et al., 2005).

RESULTS

The analysis of variance across locations on the traits revealed that the genotypes responded differently to thrips damage from the 30 DAP to 51 DAP except the 37 DAP where the differences among genotypes for thrips damage score were not significant (P>0.05). The thrips counts per flower had no significant differences among genotypes. Significant (P<0.05) differences were also observed among genotypes for the number of pods per plant. Location significantly affected all the traits. Location significantly (P<0.05) interacted with genotype for thrips damage scores at 44 DAP and 51 DAP while the interaction effects were only significant (P<0.05) for thrips counts in flowers at 30 DAP. The number of pods per plant were significantly (P<0.001) influenced by the location by genotype interaction effects (Table 3).

Comparing the performance of crosses to the corresponding best parents, some crosses presented thrips damage scores intermediate between their resistant and susceptible parent scores. However, most of the crosses involving the genotype WC36 presented higher thrips damage scores (4-5) (Table 4). The lowest score was recorded on the resistant parent, Sanzi (2.88) while the highest value was recorded on the most susceptible parent WC36 (7.06).

On the other hand, a negative but non-significant correlation (r=-0.001) was observed between thrips damage scores and the number of thrips per flower. The highest thrips number in flowers was recorded on the progenies of MU9 x WC36 (12 thrips/flower) while the lowest value was recorded on the progenies of NE5, Sanzi x NE5, WC36 x NE5, NE5 x MU9, and TVU-1471 x Sanzi (4 thrips/flower). A negative but non-significant correlation (r= -0.03) was observed between thrips damage scores and the number of pods per plant. The resistant genotypes did not always have the highest pods number per plant. For instance, the highest pods number were recorded on the cross TVU-123 x TVU-1509 (46 pods/plant) while the lowest value was recorded on parents TVU-123 (16 pods/plant) (Table 4). Most R x S crosses and their reciprocals had pods numbers ranging from 24 to 32 pods/ plants.

The results of the full diallel analysis of variance across locations are presented in Table 5. The results showed that the mean squares for location were highly significant (P < 0.001) for all traits. There were significant (P<0.05) differences among the genotypes under investigation for thrips damage scores across locations. GCA mean squares were significant (P < 0.05) for thrips damage scores at 51DAP and not significant for thrips counts across locations. SCA mean squares were also significant (P < 0.05) for thrips damage scores at 37 DAP and 51DAP. The reciprocal mean squares were not significant for all traits. The genotypes and GCA significantly (P<0.01) interacted with the location for thrips damage scores at 37, 44 and 51 DAP. A highly significant (P<0.001) location and GCA interaction was observed for thrips counts at 37 DAP. Location and reciprocal interaction was significant (P<0.05) for thrips damage scores at 37 DAP but not significant for thrips counts (Table 5). The GCA/SCA ratio varied from 0.06 to1.5 for thrips damage scores and from 0 to 7.5 for thrips

Table 3. Analysis of variance for thrips damage scores and thrips counts in F2 generation across locations, 2017.

Source of variation	DF	Thrips damage score (1-9)				No of thrips/flower				No of pods/ plant
		30DAP	37DAP	44DAP	51DAP	30DAP	37DAP	44DAP	51DAP	
(Location)/Rep	3	0.17***	0.20^{ns}	16.82***	1.16***	188.32***	58.08***	50.50***	61.07***	82.9^{ns}
(L x Rep)/Blocks	42	0.37***	0	2.16***	1.03^{ns}	71.45^{ns}	121.41***	60.84***	82.02*	309**
Location (L)	2	0.29**	184.59***	145.19***	309.64***	2112.98***	174.26***	309.19***	116.47*	15942.6^{***}
Genotypes	63	0.09**	0.58^{ns}	0.97*	1.16**	67.44^{ns}	23.06^{ns}	8.72^{ns}	47.03^{ns}	238*
L x Genotype	126	126	0.05ns	0.52**	0.79*	0.86**	75.04^{ns}	18.03^{ns}	12.30^{ns}	224.7***
Residual	189	0.04	0.43	0.54	0.58	70	16.87	14.07	38.64	132.9
LEE	158	0.02	0.18	0.24	0.27	25.5	6.86	3.61	10.33	81.36
CV (%)		5.30	13.20	11.70	11.60	18.60	11.40	3.60	3.40	17.7

*, **, *** significant at 0.05, 0.01, 0.001 probability levels respectively; ns, not significant; L = location; G= genotype; CV= coefficient of variation; LEE = lattice effective error; DAP, number of days after planting.

Table 4. Mean performance of the parents and F2 populations to flower thrips.

Parents	Thrips damage scores (1-9)	Number of thrips/flower	Number of pods /plant
MU9	3.43	5	40.92
NE5	3.15	4	41.83
Sanzi	2.88	6	32.83
TVU-123	4.26	8	16
TVU-1471	3.7	7	24
TVU-1509	3.08	5	22.17
TVU-473	3.56	7	29.83
WC36	7.06	6	32
Crosses			
MU9 x TVU-123	3.95	5	27.33
MU9 x TVU-1471	3.6	8	29.67
MU9 x TVU-1509	4.22	6	33.83
MU9 x TVU-473	3.95	6	29.33
MU9 x WC36	4.11	12	34.25
MU9 x Sanzi	3.45	5	26
MU9xNE5	3.51	8	30
NE5 x MU9	4.39	4	35.83
NE5 x Sanzi	3.9	5	30
NE5 x TVU-123	4.23	7	43.17
NE5 x TVU-1471	3.13	7	43.33
NE5 x TVU-1509	3.62	5	29
NE5 x TVU-473	3.92	6	38.75
NE5xWC36	3.78	7	33.92
Sanzi x MU9	3.72	5	36.25
Sanzi x NE5	3.64	4	36.33
Sanzi x NE5	3.95	5	16.67
Sanzi x TVU-123	3.33	7	30.58
Sanzi x TVU-1471	3.31	6	32.08
Sanzi x TVU-1509	3.06	5	33.42
Sanzi x TVU-473	3.91	6	29.58
Sanzi x WC36	3.95	6	30.17
TVU-123 x MU9	3.71	8	29.25

Table 4. Contd.

TVU-123 x NE5	3.42	5	28.33
TVU-123 x TVU-1471	3.2	6	21.92
TVU-123 x TVU-1509	3.79	7	46.00
TVU-123 x TVU-473	3.75	7	28.92
TVU-123 x WC36	4.15	5	38.67
TVU-123 x Sanzi	3.26	6	25.92
TVU-1471 x MU9	3.78	5	27.92
TVU-1471 x NE5	3.15	7	24.17
TVU-1471 x Sanzi	3.55	4	34.67
TVU-1471 x TVU-1509	3.91	7	26.5
TVU-1471 x TVU-473	3.61	7	42.58
TVU-1471 x WC36	3.98	6	40.5
TVU-1471x TVU-123	3.78	7	29.42
TVU-1509 x MU9	3.79	5	38.08
TVU-1509 x NE5	3.23	6	36.25
TVU-1509 x Sanzi	3.32	5	30
TVU-1509 x TVU-123	3.65	6	26.08
TVU-1509 x TVU-1471	3.96	7	43
TVU-1509 x TVU-473	3.78	7	25
TVU-1509 x WC36	4.18	6	36.42
TVU-473 x MU9	3.37	7	30.42
TVU-473 x NE5	3.32	5	29.92
TVU-473 x Sanzi	3.72	5	31.92
TVU-473 x TVU-123	3.29	6	27.5
TVU-473 x TVU-1471	3.58	8	28.92
TVU-473 x TVU-1509	3.72	7	22.75
TVU-473 x WC36	4.14	5	35.75
WC36 x MU9	4.95	5	28.08
WC36 x NE5	5.03	4	30.75
WC36 x Sanzi	4.11	6	27.25
WC36 x TVU-123	4.33	5	19
WC36 x TVU-1471	3.75	7	32.17
WC36 x TVU-1509	3.59	6	37.5
WC36 x TVU-473	3.71	7	39.83
LSD	0.93	4.75	24.78

counts. The Baker ratio varied from 0.1 to 0.75 for thrips damage scores and from 0 to 0.94 for thrips counts in flowers. The narrow sense coefficient of genetic determination (NSCGD) varied from 0.02 to 0.18 for thrips damage scores and from 0 to 0.09 for thrips counts. The broad sense coefficient of genetic determination varied from 0.18 to 0.42 for thrips damage scores and from 0 to 0.06 for thrips counts (Table 5).

The estimates of parents' GCA effects are presented in Table 6. The genotypes TVU-1471 and TVU-1509 had significant ($p<0.05$) negative GCA effects for flower thrips damage scores and contributed towards resistance on average by approximately 3 thrips damage score unit while genotype WC36 had significant ($p<0.001$) positive GCA effects contributing 7 thrips damage scores towards susceptibility. For thrips counts, none of the genotypes presented significant GCA effects.

The estimated values of specific combining (SCA) ability effects showed that the crosses TVU-1509 x NE5, TVU-473 x Sanzi, TVU-123 x Sanzi and TVU-123 x TVU-473 displayed significant ($p < 0.05$) and negative SCA effects for thrips damage scores while the cross TVU-473 x TVU-1509 displayed significant ($p<0.05$) and negative SCA effects for thrips counts. The crosses Sanzi x NE5, TVU-473 x Sanzi and TVU-1471 x TVU-1509 displayed significant ($p<0.05$) and positive SCA effects for thrips damage scores while the crosses Sanzi x NE5, WC36 x TVU-1509, WC36 x MU9 and TVU-1471 x MU9 displayed significant ($p<0.05$) and positive SCA effects for thrips counts.

Table 5. Mean squares for flower thrips damage scores and thrips numbers in F2 populations across locations, 2017.

Source of variation	DF	Thrips damage score				No of Thrips			
		30 DAP	37 DAP	44 DAP	51 DAP	30 DAP	37 DAP	44 DAP	51 DAP
Rep (Location)	3	0.17***	0.20^{ns}	16.82***	1.16***	188.32***	58.08***	50.50***	61.07***
Location	2	0.29**	184.59***	145.19***	309.64***	2112.98***	174.26***	309.19***	116.47*
Cross	63	0.07*	0.46^{ns}	0.81^{ns}	1.11^{ns}	67.55^{ns}	16.96^{ns}	8.64^{ns}	44.09^{ns}
GCA	7	0.14^{ns}	0.48^{ns}	2.59^{ns}	3.36*	67.25^{ns}	45.31^{ns}	3.78^{ns}	27.19^{ns}
SCA	28	0.07^{ns}	0.64*	0.71^{ns}	1.44*	83.93^{ns}	17.04^{ns}	8.07^{ns}	41.40^{ns}
Reciprocal	28	0.08^{ns}	0.46^{ns}	0.80^{ns}	0.58^{ns}	56.57^{ns}	15.58^{ns}	10.90^{ns}	58.05^{ns}
Location x Cross	126	0.05^{ns}	0.52**	0.79*	0.86**	75.04^{ns}	18.03^{ns}	12.30^{ns}	31.73^{ns}
Location x GCA	14	0.08^{ns}	1.62***	3.68***	3.03***	92.23^{ns}	45.43*	21.60^{ns}	45.58^{ns}
Location x SCA	56	0.05^{ns}	0.37^{ns}	0.59^{ns}	0.76^{ns}	86.09^{ns}	20.13^{ns}	13.36^{ns}	29.04^{ns}
Location x Reciprocal	56	0.05^{ns}	0.58**	0.57^{ns}	0.76^{ns}	64.62^{ns}	16.16^{ns}	10.24^{ns}	37.69^{ns}
Residual	147	0.04	0.34	0.54	0.54	66.73	16.55	14.23	33.08
δ^2g (GCA)		0.003	0.004	0.06	0.09	0.02	0.9	0	0
δ^2s (SCA)		0.006	0.07	0.04	0.22	4.3	0.12	0	2.08
δ^2g (GCA)/ δ^2s (SCA)		0.5	0.06	1.5	0.40	0.005	7.5	-	0
δ^2r (Recip)		0.01	0.02	0.04	0.001	0	0	0	4.73
[a]Baker ratio		0.5	0.1	0.75	0.45	0.01	0.94	-	0
[b]NSCGD=h^2		0.09	0.02	0.16	0.18	0.0005	0.09	0	0
[c]BSCGD=H		0.18	0.19	0.21	0.42	0.06	0.1	0	0.05

*, **, *** significant at 0.05, 0.01, 0.001 probability levels respectively; ns not significant; [a] Relative importance of GCA and SCA according to Baker (1978); [b] Narrow sense coefficient of genetic determination for a fixed model (analogous to h^2); [c] broad sense coefficient of genetic determination for a fixed model (analogous to H); δ^2g, δ^2s and δ^2r are the respective additive component; (GCA); Dominance component (SCA) and Reciprocal component. All MS and CGD values were on the basis of the mean of two replications in the three locations; DAP, number of days after planting.

Table 6. GCA, SCA and reciprocal effects for thrips damage score and thrips number per flower in parents and F2 generations across locations, 2017.

Parents	Mean thrips damage scores	GCA effects of parents							
		Thrips damage score (1-9)				Number of thrips/flower			
		30 DAP	37 DAP	44 DAP	51 DAP	30 DAP	37 DAP	44 DAP	51 DAP
MU9	4	0.03^{ns}	0.08^{ns}	-0.09^{ns}	0.001^{ns}	0.44^{ns}	-0.66^{ns}	-0.06^{ns}	-0.23^{ns}
NE5	4	0.03^{ns}	-0.02^{ns}	-0.03^{ns}	-0.09^{ns}	0.11^{ns}	-0.75^{ns}	-0.12^{ns}	-0.43^{ns}
SANZI	3	-0.03^{ns}	0.08^{ns}	0.09^{ns}	0.02^{ns}	1.16^{ns}	-0.49^{ns}	-0.23^{ns}	-0.58^{ns}
TVU-1509	2	0.01^{ns}	-0.08^{ns}	-0.06^{ns}	-0.28**	0.69^{ns}	1.39**	-0.15^{ns}	-0.69^{ns}
TVU-473	2	0.04^{ns}	-0.03^{ns}	0.02^{ns}	0.11^{ns}	-0.94^{ns}	0.31^{ns}	0.11^{ns}	0.58^{ns}
TVU-123	2	-0.03^{ns}	0.04^{ns}	-0.06^{ns}	0.07^{ns}	-0.42^{ns}	0.01^{ns}	0.39^{ns}	0.39^{ns}
TVU-1471	3	-0.07**	-0.11^{ns}	-0.22**	-0.16*	-1.32^{ns}	-0.01^{ns}	0.12^{ns}	0.38^{ns}
WC36	7	0.02^{ns}	0.03^{ns}	0.34***	0.34***	0.28^{ns}	0.20^{ns}	-0.06^{ns}	0.56^{ns}
Crosses		SCA effects							
NE5 X MU9		0.03^{ns}	0.52***	0.03^{ns}	0.09^{ns}	-0.91^{ns}	0.52^{ns}	1.23^{ns}	-0.67^{ns}
SANZI X MU9		-0.02^{ns}	-0.23^{ns}	0.11^{ns}	0.34^{ns}	-0.82^{ns}	0.82^{ns}	0.31^{ns}	-1.29^{ns}
SANZI XNE5		0.11*	0.15^{ns}	0.48*	0.46*	7.88***	-0.16^{ns}	-0.51^{ns}	-0.24^{ns}
TVU-1509 X MU9		0.09^{ns}	0.28^{ns}	0.32^{ns}	0.21^{ns}	-2.5^{ns}	-0.02^{ns}	0.45^{ns}	-0.42^{ns}
TVU-1509 X NE5		-0.17**	-0.17^{ns}	0.08^{ns}	0.01^{ns}	2.10^{ns}	0.85^{ns}	-0.58^{ns}	-0.68^{ns}
TVU-1509 X Sanzi		-0.001^{ns}	-0.09^{ns}	-0.25^{ns}	-0.33^{ns}	-2.52^{ns}	-0.37^{ns}	-0.87^{ns}	-1.49^{ns}
TVU-473 X MU9		-0.08^{ns}	-0.17^{ns}	-0.19^{ns}	-0.09^{ns}	0.90^{ns}	-1.08^{ns}	0.40^{ns}	-0.16^{ns}
TVU-473 X NE5		0.04^{ns}	0.06^{ns}	0.13*	0.001^{ns}	0.70^{ns}	1.75^{ns}	0.16^{ns}	-2.06^{ns}
TVU-473 X Sanzi		-0.11*	0.10^{ns}	0.16^{ns}	0.81***	1.42^{ns}	-0.04^{ns}	-0.82^{ns}	-0.30^{ns}
TVU-473 X TVU-1509		0.09^{ns}	0.27^{ns}	-0.05^{ns}	-0.21^{ns}	-1.56^{ns}	-2.14*	0.69^{ns}	2.38^{ns}

Table 6. Contd.

Crosses								
TVU-123 X MU9	-0.04^{ns}	0.11^{ns}	0.01^{ns}	-0.28^{ns}	0.03^{ns}	1.27^{ns}	-0.59^{ns}	-1.49^{ns}
TVU-123XNE5	0.09^{ns}	0.21^{ns}	0.05^{ns}	0.02^{ns}	-2.08^{ns}	-0.32^{ns}	-1.16^{ns}	0.55^{ns}
TVU-123X Sanzi	0.03^{ns}	-0.09^{ns}	-0.21^{ns}	-0.61^{**}	2.27^{ns}	0.99^{ns}	0.12^{ns}	2.19^{ns}
TVU-123X TVU-1509	-0.04^{ns}	-0.22^{ns}	0.05^{ns}	0.20^{ns}	-2.90^{ns}	-1.73^{ns}	0.67^{ns}	-0.71^{ns}
TVU-123 X TVU-473	-0.05^{ns}	-0.03^{ns}	-0.13^{ns}	-0.44^{*}	1.06^{ns}	0.82^{ns}	-0.20^{ns}	-0.59^{ns}
TVU-1471X MU9	0.03^{ns}	0.09^{ns}	0.29^{ns}	-0.07^{ns}	1.30^{ns}	0.43^{ns}	0.74^{ns}	-0.01^{ns}
TVU-1471 X NE5	-0.07^{ns}	-0.29^{ns}	-0.21^{ns}	-0.25^{ns}	-2.0^{ns}	-0.61^{ns}	0.19^{ns}	3.83^{*}
TVU-1471X SANZI	0.03^{ns}	-0.08^{ns}	0.05^{ns}	-0.07^{ns}	-0.16^{ns}	-0.88^{ns}	-1.40^{ns}	0.27^{ns}
TVU-1471 XTVU-1509	0.01^{ns}	0.33^{*}	0.13^{ns}	0.53^{**}	-1.02^{ns}	-1.24^{ns}	0.53^{ns}	-0.27^{ns}
TVU-1471 X TVU-473	0.01^{ns}	-0.24^{ns}	0.001^{ns}	-0.03^{ns}	-0.42^{ns}	1.12^{ns}	0.24^{ns}	-0.24^{ns}
TVU-1471 X TVU-123	0.01^{ns}	-0.13^{ns}	-0.06^{ns}	-0.17^{ns}	0.50^{ns}	0.26^{ns}	-0.22^{ns}	0.13^{ns}
WC36 X MU9	-0.05^{ns}	-0.33^{*}	-0.12^{ns}	0.005^{ns}	1.97^{ns}	0.15^{ns}	-0.45^{ns}	5.84^{***}
WC36 X NE5	0.12^{*}	-0.36^{*}	-0.09^{ns}	-0.04^{ns}	-1.13^{ns}	-0.98^{ns}	0.22^{ns}	-1.01^{ns}
WC36 X SANZI	-0.01^{ns}	0.24^{ns}	0.24^{ns}	0.11^{ns}	-3.76^{ns}	-0.18^{ns}	0.77^{ns}	0.34^{ns}
WC36 X TVU-1509	0.001^{ns}	-0.20^{ns}	-0.23^{ns}	0.05^{ns}	5.87^{**}	-0.20^{ns}	0.58^{ns}	-1.36^{ns}
WC36 X TVU-473	0.08^{ns}	0.02^{ns}	-0.28^{ns}	-0.22^{ns}	-1.49^{ns}	-0.61^{ns}	-0.52^{ns}	0.28^{ns}
WC36 X TVU-123	0.07^{ns}	0.17^{ns}	0.40^{*}	0.42^{*}	0.46^{ns}	0.52^{ns}	-0.28^{ns}	-2.77^{ns}
WC36 X TVU-1471	-0.04^{ns}	0.15^{ns}	-0.24^{ns}	0.04^{ns}	0.11^{ns}	0.48^{ns}	-0.15^{ns}	-1.45^{ns}
Crosses				**Reciprocal effects**				
NE5 X MU9	-0.01^{ns}	-0.53^{***}	-0.22^{ns}	-0.13^{ns}	0.88^{ns}	-0.03^{ns}	1.52^{*}	1.97^{ns}
SANZI X MU9	0.13^{**}	0.11^{ns}	-0.08^{ns}	-0.04^{ns}	-2.56^{ns}	-0.08^{ns}	-0.52^{ns}	0.09^{ns}
SANZI X NE5	-0.06^{ns}	-0.11^{ns}	0.07^{ns}	0.28^{*}	-7.08^{***}	-0.97^{ns}	0.33^{ns}	0.22^{ns}
TVU-1509 X MU9	-0.19^{***}	-0.24^{*}	0.40^{**}	0.29^{*}	-0.26^{ns}	0.53^{ns}	0.76^{ns}	0.42^{ns}
TVU-1509 X NE5	0.03^{ns}	0.07^{*}	0.46^{**}	-0.1^{ns}	0.97^{ns}	0.96^{ns}	-0.66^{ns}	-0.42^{ns}
TVU-1509 X SANZI	0.05^{ns}	-0.11^{ns}	-0.31^{*}	-0.50^{**}	-0.48^{ns}	1.38^{ns}	-1.17^{ns}	-0.25^{ns}
TVU-473 X MU9	0.01^{ns}	0.30^{**}	0.26^{ns}	0.13^{ns}	-1.77^{ns}	-1.64^{*}	-0.73^{ns}	$0.25ns$
TVU-473 X NE5	0.08^{*}	0.21^{*}	-0.03^{ns}	-0.20^{ns}	0.29^{ns}	2.24^{**}	0.53^{ns}	-0.41^{ns}
TVU-473 X SANZI	0.02^{ns}	-0.06^{ns}	0.20^{ns}	0.22^{ns}	3.79^{*}	1.16^{ns}	0.88^{ns}	-1.38^{ns}
TVU-473 X TVU-1509	0.15^{***}	0.12^{ns}	0.08^{ns}	-0.15^{ns}	0.11^{ns}	0.23^{ns}	1.03^{ns}	-2.36^{*}
TVU-123XMU9	0.06^{ns}	0.18^{ns}	-0.01^{ns}	0.19^{ns}	-0.80^{ns}	-0.92^{ns}	-1.19^{ns}	0.28^{ns}
TVU-123 X NE5	-0.15^{***}	0.09^{ns}	0.16^{ns}	0.33^{*}	0.73^{ns}	0.44^{ns}	-0.47^{ns}	1.31^{ns}
TVU-123 X SANZI	0.10^{*}	-0.03^{ns}	-0.15^{ns}	-0.01^{ns}	3.53^{*}	-0.75^{ns}	0.32^{ns}	2.66^{*}
TVU-123 X TVU-1509	-0.02^{ns}	-0.03^{ns}	-0.05^{ns}	0.09^{ns}	-1.79^{ns}	-1.13^{ns}	1.28^{ns}	0.25^{ns}
TVU-123 X TVU-473	-0.07^{ns}	-0.22^{*}	-0.48^{**}	-0.15^{ns}	-0.53^{ns}	-1.57^{*}	0.46^{ns}	-0.67^{ns}
TVU-1471 X MU9	-0.06^{ns}	-0.01^{ns}	-0.04^{ns}	-0.08^{*}	3.94^{*}	-2.51^{**}	2.10^{**}	2.08^{*}
TVU-1471 X NE5	-0.04^{ns}	0.20^{ns}	-0.18^{ns}	-0.08^{ns}	1.34^{ns}	0.84^{ns}	-0.98^{ns}	-3.16^{**}
TVU-1471 X SANZI	0.01^{ns}	0.30^{**}	-0.23^{ns}	-0.41^{**}	-0.42^{ns}	-0.11^{ns}	-0.35^{ns}	2.03^{ns}
TVU-1471 X TVU-1509	-0.03^{ns}	0.06^{ns}	-0.05^{ns}	0.01^{ns}	-0.68^{ns}	1.09^{ns}	-1.16^{ns}	-1.37^{ns}
TVU-1471 X TVU-473	-0.11^{**}	-0.24^{*}	0.15^{ns}	0.06^{ns}	1.14^{ns}	0.71^{ns}	0.59^{ns}	-0.35^{ns}
TVU-1471 X TVU-123	-0.002^{ns}	-0.30^{**}	-0.32^{*}	-0.08^{ns}	-0.36^{ns}	0.94^{ns}	0.41^{ns}	-3.07^{**}
WC36 X MU9	0.10^{*}	0.15^{ns}	0.05^{ns}	-0.12^{ns}	-3.29^{*}	0.23^{ns}	1.08^{ns}	8.76^{***}
WC36 X NE5	-0.05^{ns}	-0.03^{ns}	0.22^{ns}	-0.02^{ns}	0.14^{ns}	0.85^{ns}	1.44^{*}	2.03^{*}
WC36 X SANZI	-0.05^{ns}	-0.05^{ns}	-0.45^{**}	-0.38^{**}	-1.18^{ns}	1.48^{*}	-0.31^{ns}	0.56^{ns}
WC36 X TVU-1509	0.08^{*}	0.27^{*}	0.14^{ns}	0.10^{ns}	-0.16^{ns}	-1.64^{*}	0.08^{ns}	-0.17^{ns}
WC36 X TVU-473	0.11^{**}	0.03^{ns}	0.19^{ns}	0.32^{*}	1.37^{ns}	-0.39^{ns}	-0.45^{ns}	-1.08^{ns}
WC36 X TVU-123	-0.07^{ns}	0.01^{ns}	0.60^{***}	0.22^{ns}	0.69^{ns}	-0.27^{ns}	1.55^{*}	-0.62^{ns}
WC36 X TVU-1471	0.04^{ns}	-0.23^{*}	0.27^{*}	0.32^{*}	2.17^{ns}	-1.55^{*}	0.90^{ns}	-1.19^{ns}

***, **, * significant at 0.001, 0.01, 0.05 probability levels, respectively; ns not significant; DAP, number of days after planting.

Table 7. Phenotypic ratios of segregating families of F2 populations against different hypothesized genetic models.

Crosses	Number of plants	χ^2 under different model ratios (df = 1)			Number of genes
		3:1	9:7	37:27	
SANZI (R) x NE 5 (S)	228	19.67***	3.37^{ns}	1.87^{ns}	2 and 3
SANZI (R) x MU9 (S)	244	2.45^{ns}	12.82***	9.63**	1
SANZI (R) x WC 36 (S)	240	22.76***	2.86^{ns}	1.46^{ns}	2 and 3
TVU-1509 (R) x WC 36 (S)	241	13.56***	7.04***	3.26^{ns}	3
TVU-1509 (R) x MU9 (S)	245	11.27***	8.92***	3.27^{ns}	3
TVU - 1509 (R) x SANZI (R)	235	3.28^{ns}	14.35***	11.03***	1
TVU- 1509 (R) x TVU- 1471(R)	234	8.67***	10.32***	3.75^{ns}	3
TVU- 1509 (R) x NE5(S)	240	0.09^{ns}	31.31***	2.24^{ns}	1 and 3
TVU- 473 (R) x MU9 (S)	243	20.08***	3.92*	2.24^{ns}	3
TVU- 473 (R) x SANZI (R)	251	1.12^{ns}	25.66***	21.04***	1
TVU- 473 (R) x TVU -1509 (R)	238	15.74***	5.61*	3.58^{ns}	3
TVU-123 (R) x NE5 (S)	238	19.50***	3.91*	2.24^{ns}	3
TVU-123(R) x SANZI(R)	245	14.43***	6.76**	3.67^{ns}	3
TVU-123(R) x TVU-1509 (R)	246	0.92^{ns}	25.94***	21.34***	1
TVU-123 (R) x TVU -473 (R)	233	8.93**	9.99**	3.54^{ns}	3
TVU-123 (R) x WC36 (S)	246	1.57^{ns}	23.38***	19.02***	1
TVU-123 (R) xMU9 (S)	216	52.25***	0.57^{ns}	1.50^{ns}	2 and 3
TVU-1471(R) x NE 5 (S)	241	0.31^{ns}	28.95***	24.14***	1
TVU-1471(R) x SANZI(R)	244	0.55^{ns}	27.65***	22.93***	1
TVU-1471 (R) x TVU - 123(R)	242	2.12^{ns}	12.13***	9.04**	1
TVU-1471 (R) x TVU - 473(R)	217	3.67^{ns}	13.59***	10.48**	1
TVU-1471(R) x WC 36 (S)	240	9.80**	9.75**	3.70^{ns}	3
TVU-1471 (R) xMU9 (S)	242	2.70^{ns}	11.24***	8.27**	1
TVU-473 (R) x NE5 (S)	248	3.63^{ns}	18.39***	14.51***	1
TVU - 473(R) x WC36 (S)	248	3.47^{ns}	17.31***	13.55**	1

***, **, * significant deviation from model ratios at 0.001, 0.01 and 0.05 probability; ns no significant deviation from model ratios.

The estimates of the reciprocal effects of the crosses revealed that some of the crosses for example NE5 x MU9, TVU-1509 x MU9, TVU-1509 x Sanzi, TVU-123 x NE5, TVU-123 x TVU-473, TV-1471 x MU9, TVU-1471 x Sanzi, TVU-1471 x TVU-473, TVU-1471 x TVU-123 and WC36 x Sanzi displayed significant (P<0.05) and negative reciprocal effects for thrips damage scores. Significant (P<0.05) and negative reciprocal effects were observed in Sanzi x NE5, TVU-473 x MU9, TVU-473 x TVU-1509, TVU-123 x TVU-473, TVU-1471 x NE5 and WC36 x TVU-1471 for thrips counts. The crosses Sanzi x MU9, Sanzi x NE5, TVU-1509 x MU9, TVU-123 x NE5, WC36 xTVU-1509 and WC36 x TVU-473 had significant (P<0.05) and positive reciprocal effects for thrips damage scores while the crosses NE5 x MU9, TVU-473 x NE5, TVU-473 x Sanzi, TVU-123 x Sanzi and WC36 x MU9 had significant (P<0.05) and positive reciprocal effects for thrips counts.

The results of the Chi-square goodness-of-fit test for F2 segregating ratios of twenty five crosses (Resistant x Resistant and Resistant x Susceptible) are presented in Table 7. The genotypic reaction to flower thrips damage

showed that twelve crosses conformed to one dominant gene inheritance (ratio of 3:1), three crosses conformed to both duplicate recessive epitasis and three complementary recessive genes inheritance (ratios of 9:7 and 37:27, respectively). One cross conformed to both one dominant gene and three complementary recessive genes inheritance (ratios of 3:1 and 37:27, respectively). Nine crosses fitted three complementary recessive genes inheritance (ratio of 37:27).

DISCUSSION

The significant (P<0.05) difference among genotypes for thrips damage scores (Table 3) indicated that there was a wide genetic variability for flower thrips resistance, and the feasibility for genetic improvement using such genetic pool of cowpea (Alghamdi, 2009). It also indicated that thrips damage on cowpea becomes more severe from 37 DAP, and consequently any thrips data record and any application of pesticide against flower thrips should begin at this time. The non-significant differences observed

among genotypes for thrips counts per flower indicated that both the susceptible and the resistant genotypes supported almost similar number of thrips suggesting that the mechanism of resistance to flower thrips in the resistant genotypes is probably tolerance. The significant location by genotype interaction for thrips damage scores (Table 3) confirmed the instability of cowpea resistance to flower thrips as found in the previous study. The lowest thrips damage scores recorded on Sanzi and on the combination of Sanzi with TVU-1509 confirmed the findings in the previous studies on these genotypes (Alabi et al., 2005; Abudulai et al., 2006; Omo-Ikerodah et al., 2009; Dormatey et al., 2015).

The significant GCA and SCA effects for thrips damage scores across locations for the damage assessment dates suggested that in this set of crosses, additive and non-additive gene effects were involved in the control of resistance to flower thrips among the selected resistant genotypes. This was confirmed by the GCA/SCA and baker ratios obtained in this study. These results clearly confirmed the great importance of additive and non-additive gene actions in the inheritance of these traits. Bi et al. (2015) reported that a large ratio between GCA and SCA effects shows the relevance of additive gene effects while a small value signifies that the trait is under dominance and/or epistatic gene effects. The higher values of GCA compared to SCA observed during the damage assessment dates (44-51DAP) in this study were a good indication as reported by Acquaah (2012) that great genetic progress could be achieved in breeding for resistance to flower thrips in cowpea by focusing on the thrips damage scores and thrips counts. Similar gene action on the resistance of cowpea to flower thrips was reported by Dormatey et al. (2015) in Ghana while evaluating the genetics of cowpea resistance to flower thrips. The estimate of Baker's ratio values were 0.75 for thrips damage score during the damage assessment dates (Table 5), implying that the performance of a single cross progeny could be predicted fairly accurately based on the GCA of its parents (Oladejo et al., 2017). However, the location and GCA interaction effects were highly significant (P<0.001) for thrips damage scores, suggesting that the additive gene effects were more influenced by the environmental effects but SCA effects of the crosses were not significant for all the thrips counts assessment dates indicating no or small proportion of non-additive genes effects controlling flower thrips counts in flower.

The non-significant reciprocal differences in this study suggested that cytoplasmic genes played a minor role in modifying flower thrips resistance across locations. This contradicted Omo-Ikerodah et al. (2009) findings where reciprocal differences were important in conditioning resistance to flower thrips among cowpea genotypes. However, the occurrence of significant reciprocal by location interaction effects (Table 5) between the reciprocal families indicated that the expression of the

cytoplasmic factors on thrips damage scores depended on the environmental factors as well as in the case of the nuclear genes (Boukar et al., 2013; Bett et al., 2017; Oladejo et al., 2017). Consequently, the use of the resistant genotype as female parent could confer superior resistance on the F2s in different locations. Therefore, in a breeding program aiming at improving resistance to flower thrips in cowpea, resistant genotypes should be used as female parent in crosses when the location favors the expression of the maternal effect.

The broad sense coefficient of genetic determination varied from 0.18 to 0.42 for thrips damage scores across the assessment dates and indicated a low genetic contribution towards the phenotypic variance. The results of 82 and 58% for the phenotypic variation for thrips damage score were due to environmental variance implying that genotypes response to thrips damage was highly influenced by environmental factors (temperature, rainfall). These results confirmed the findings from the screening done in the previous study conducted in the same locations. The estimates of narrow sense coefficient of genetic determination were low for thrips damage score (2 to18%) and thrips counts (0 to 9%) suggesting that early-generation selection would be expected to be ineffective. This low narrow sense coefficient of genetic determination was expected since the resistance of cowpea to flower thrips is a quantitative trait (Omo-Ikerodah et al., 2009), however, different findings were reported by Dormatey et al. (2015) in Ghana where the broad sense heritability were relatively high among all the crosses for thrips damage scores (54.28%) and thrips counts (55.13%) coupled with moderately high narrow sense heritability, averaging 17.55 and 20.04% for thrips damage rating and number of thrips, respectively. These differences among results could probably be due to the differences in the parental lines used for crossing. Estimates of heritability value depend on the population in consideration, environmental conditions and the genetic complexity of the trait under study (Singh and Miklas, 2015).

In general, genotypes TVU-1471 and TVU-1509 were revealed as good transmitters of resistance to flower thrips in cowpea as compared to the other parents and could be very useful for introgressing flower thrips resistance into local susceptible genotypes. The present study confirmed the resistant status of the genotype TVU-1509 as good transmitter of resistance to flower thrips in cowpea as reported by Omo-Ikerodah et al. (2009) in Nigeria.

Crosses TVU-1509 x NE5, TVU-473 x Sanzi, TVU-123 x Sanzi and TVU-123 x TVU-473 displayed significant and negative SCA effects for thrips damage scores while the cross TVU-473 x TVU-1509 displayed significant and negative SCA effects for thrips counts (Table 7), suggesting that these crosses would be the most useful in breeding varieties for farmers because some of their progeny would have high to moderate resistances to

flower thrips and could also possess desirable market traits on the high yield components of NE5 and TVU-473 identified in the previous study. Crosses with moderate resistance could produce transgressive segregates for flower thrips resistance in subsequent generations (Acquaah, 2008). The significant and positive SCA effect presented by some R x R crosses for example TVU-473 x Sanzi and TVU-1471 x TVU-1509 could be explained by the resistance break that could accidentally happen when two more resistant genotypes are combined. However, more studies on the factors creating that situation could provide more explanation about it. Some crosses displayed significant and negative reciprocal effects for thrips damage score and thrips counts, suggesting that they were associated with maternal inheritance from the female parent (Omo-Ikerodah et al., 2009; Oladejo et al., 2017).

The 12 crosses that conformed to the ratio 3:1 suggested the presence of one dominant gene (Singh and Chaudhary, 2004), while the one cross that conformed to both the ratios 3:1 and 37:27, suggested the involvement of either one dominant gene or three complementary recessive genes. The three crosses that conformed to both the ratios 9: 7 and 37:27 indicated the involvement of either two complementary dominant genes (duplicate recessive epistasis) or three complementary recessive genes that simultaneously govern the expression of resistance to flower thrips (Caixeta et al., 2005). The nine crosses that fitted the ratio of 37:27, indicates the presence of three recessive complementary genes as reported by Caixeta et al. (2005). Similar results were reported by Bediako et al. (2012) on crosses involving the genotypes Sanzi and Bengpla in Kenya.

Conclusion

The study show that both additive and non-additive gene effects control resistance to flower thrips among the selected resistant genotypes. The cytoplasmic factors played a minor role in modifying flower thrips resistance however, significant reciprocal by location interaction effects have been reported. The estimates of heritability (broad and narrow senses) indicated that the inheritance of cowpea resistance to flower thrips is strongly influenced by the environmental effects and that early-generation selection would be expected to be ineffective. The involvement of one dominant gene, two complementary dominant genes (duplicate recessive epistasis) or three complementary recessive genes in the expression of resistance to flower thrips was identified.

However, dissection of a truly quantitative variation into its underlying Mendelian factors is difficult to achieve from phenotypic information alone, requiring a molecular technique to answer the question of number of genes and size of effects. Genotypes TVU-1471 and TVU-1509

were identified as good transmitters of resistance to flower thrips.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

ACKNOWLEDGEMENTS

REFERENCES

Abudulai M, Salifu AB, Haruna M (2006). Screening of cowpea for resistance to the flower bud thrips, *Megalurothrips sjostedti* (Trybom) (Thysanoptera: Thripidae). J. Appl. Sci. 6:1621-1624.

Acquaah G (2008). Principles of plant genetics and breeding. Blackwell publishing. ISBN 978-1-4051-3646-4. pp. 27-569.

Acquaah G (2012). Principles of Plant Genetics and Breeding, Second Edition. John Wiley & Sons, Ltd. 740p.

Afiukwa CA, Ubi BE, Kunert KJ, Titus EJ, Akusu JO (2013). Seed protein content variation in cowpea genotypes. World J. Agric. Sci. 1:094-099.

Alabi OY, Odebiyi JA, Tamo M (2005). The relationship between primary metabolites in reproductive structures of cowpea *Vigna unguiculata* (Fabaceae: Papilionidae) cultivars and field resistance to the flower bud thrips *Megalurothrips sjostedti* (Thysanoptera: Thripidae). Int. J. Trop. Insect Sci. 26:8-15.

Alghamdi SS (2009). Heterosis and combining ability in a diallel cross of eight Faba bean (*Vicia faba* L.) genotypes. Asian. J. Crop Sci. 1:66-76.

Baker RJ (1978). Issues in diallel analysis. Crop Sci. 18:533-536.

Bediako AK (2012). Inheritance of resistance to flower bud thrips (*Megalurothrips sjostedti*) in cowpea. MSc. Thesis. University of Science and Technology, Kwame Nkrumah.

Bett B, Gollasch S, Moore A, James W, Armstrong J, Walsh T, Harding R, Higgins TJV (2017). Transgenic cowpea (*Vigna unguiculata* L. Walp) expressing *Bacillus thuringiensis* Vip3Ba protein are protected against the Maruca pod borer (*Maruca vitrata*). Plant Cell Tissue Organ Cult. 131:335-345.

Bi Y, Li W, Xiao J, Lin H, Liu M, Liu M, Lai Y (2015). Heterosis and combining ability estimates in isoflavone content using different parental soybean accessions: Wild soybean, a valuable germplasm for soybean breeding. PLoS One 10(1):e0114827.

Boukar O, Bhattacharjee R, Fatokoun C, Kumar PL, Gueye B (2013). Cowpea. In Mohar S, Upadhyaga HD, Bisht IS (eds) Genetic and genomic resources of grain legume improvement. Elsevier, Amsterdam. pp. 137-156.

Boukar O, Fatokun CA, Lam Huynh B, Roberts PA, Close TJ (2016). Genomic tools in cowpea breeding programs: status and perspectives. Front. Plant Sci. 7:757.

Caixeta ET, Borém A, Alzate-Marin AL, De-Azevedo FS, De-Moraes E, Silva MG, De-Barros GE, Moreira MA (2005). Allelic relationships for genes that confer resistance to angular leaf spot in common bean. Euphytica 145:237-245.

Dabholkar AR (1992). Elements of biometrical genetics, 1st ed. Ashok, Kumat Mittal, New Delhi.

Ddamulira G, Santos CAF, Obuo P, Alanyo M, Lwanga C K (2015). Grain yield and protein content of Brazilian cowpea genotypes under diverse Ugandan environments. Americ. J. Plant Sci. 6:2074-2084.

Dormatey R, Atokple IDK, Ishiyaku MF (2015). Genetics of thrips

resistance in cowpea. Int. J. Agric. Sci. Res. 2:2348-3997.

Dungu SP, Ekere W, Bisikwa J, Kawooya R, Kalule O, Biruma M (2015). Marketing and market integration of cowpea in Uganda. J. Dev. Agric. Econ. 7:1-11.

Fehr WR (1987). Principles of cultivar development. Volume 1. Theory and technique. Iowa state university. Macmillan publishing company, London. ISBN 0-02-94- 9920-8 (v.1). 536pp.

Food and Agriculture Organization of the United Nations (FAOSTAT) (2013) Statistics Division. http://faostat3.fao.org/faostat-gateway/go/to/download/Q/QC/E

Fungo B, Grunwald S, Tenywa MM, Vanlauwe B, Nkedi-Kizza P (2011). Lunnyu soils in the Lake Victoria basin of Uganda: Link to toposequence and soil type. Afr. J. Environ. Sci. Technol. 5:15-24.

Gbaguidi AA, Dansi A, Loko LY, Dansi M, Sanni A (2013). Diversity and agronomic performances of the cowpea (*Vigna unguiculata* L. Walp.) landraces in Southern Benin. Int. J. Agron. Plant Prod. 4(5):936-949.

Griffing B (1956). Concept of general and specific combining ability in relation to diallel crossing systems. Aust. J. Biol. Sci.9:463-493.

Hall AE, Cisse N, Thiaw S, Elawad HO, Ehlers JD, Ismail AM, Fery RL, Roberts PA, Kitch LW, Murdock LL, Boukar O (2003). Development of cowpea cultivars and germplasm by the Bean/Cowpea CRSP. Field Crops Res. 82:103-134.

Jackai LEN, Singh SR (1988). Screening techniques for host plant resistance to insect pests of cowpea. Trop. Grain Leg. Bull. 35:2-18.

Karungi J, Adipala E, Kyamanywa S, Ogenga-Latigo MW, Oyobo N, Jackai LEN (2000b). Pest management in cowpeas. Part 2. Integrating planting time, plant density and insecticide application for management of cowpea field insect pests in Uganda. Crop Prot. 19:237-245.

Muchero W, Ehlers JD, Roberts PA (2008). Seedling Stage Drought-Induced Phenotypes and Drought-Responsive Genes in Diverse Cowpea Genotypes. Crop Sci. 48:541.

Mwale SE, Ssemakula MO, Sadik K, Alladassi B, Rubaihayo P, Gibson P, Singini W, Edema R. (2017). Estimates of combining ability and heritability in cowpea genotypes under drought stress and non-stress conditions in Uganda. J. Plant Breed. Crop Sci. 9:10-18.

Ngakou A, Tamò M, Parh IA, Nwaga D, Ntonifor NN, Korie S, Nebane CLN (2008). Management of cowpea flower thrips, *Megalurothrips sjostedti* (Thysanoptera: Thripidae), in Cameroon. Crop. Prot. 27:481-488.

Oladejo AS, Boukar O, Fatokoun CA, Obisesan IO (2017). Genetic analysis of thrips resistance in cowpea (*Vigna unguiculata* [L.] Walp). Euphytica. 213(9):216-227.

Omo-Ikerodah EE, Fatokun CA, Fawole I (2009). Genetic analysis of resistance to flower bud thrips (*Megalurothrips sjostedti*) in cowpea (*Vigna unguiculata* [L.] Walp.). Euphytica 165:145-154.

Payne RW (2009). The Guide to Genstat: Release 12, Part 2, Statistics. Lawes Agricultural Trust, Rothamsted, Experimental Station, Harpenden, Herts, UK.

Rodríguez F, Alvarado G, Pacheco A, Crossa J, Burgueño J (2015). "AGD-R (Analysis of Genetic Designs with R for Windows) Version 2.0". http://hdl.handle.net/11529/10202 International Maize and Wheat Improvement Center.

Ronner E, Giller KE (2012). Background information on agronomy, farming systems and on-going projects on grain legumes in Uganda, 17-18. www.N2Africa.org.

Rubaihayo PR (1996). Basic concepts of plant breeding. Longhorn Kenya Ltd. ISBN 9966 49 912 1.

Singh RK, Chaudhary BD (2004). Biometrical Methods in Quantitative Genetic Analysis. 3rd ed. Kalyani Publishers, New Delhi.

Singh SP, Miklas PN (2015). Breeding common bean for resistance to common blight: A review. Crop Sci. 55:971-984.

Sobda G, Boukar O, Tongoona PB, Ayertey J, Offei KS (2017). Quantitative trait loci (QTL) for cowpea resistance to flower bud thrips (*Megalurothrips sjostedti* Trybom). Int. J. Plant Breed. Genet. 4(6):292-299.

Sserumaga JP, Oikeh SO, Mugo S, Otim GAM, Beyene Y, Abalo G, Kikafunda J (2015). Genotype by environment interactions and agronomic performance of doubled haploids testcross maize (*Zea mays* L.) hybrids. Euphytica 205(3):1-15. doi 10.1007/s10681-015-1549-2.

Umar UU, Ado SG, Aba DA, Bugaje SM (2014). Estimates of combining ability and gene action in maize under water stress and non- stress conditions. J. Biol. Agric. Health Care 4:247-253.