Vol. 12(2), pp. 175-183, April-June 2020
DOI: 10.5897/JPBCS2019.0880
Article Number: 04EDF1364081
ISSN 2006-9758

http://www.academicjournals.org/JPBCS

ACADEMIC JOURNALS

Journal of Plant Breeding and Crop Science

Full Length Research Paper

Inheritance pattern of resistance to Fusarium wilt (*Fusarium oxysporum f*. sp *sesame*) in sesame

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Received 18 February, 2020; Accepted 21 April, 2020

Fusarium wilts (*Fusarium oxysporum* f.sp. *sesame)* is among of the most destructive soil-borne disease of sesame in Uganda. The disease may cause yield loss of up to 100% if not controlled. Breeding and use of resistant varieties is the most economic and eco-friendly solution to the disease since majority of sesame growers are resource constrained. Some genotypes were reported to be moderately resistant to the disease in Uganda. However its nature of inheritance was not studied. Successful breeding requires selection of suitable parents and whose pattern of inheritance of disease resistance is known. In this study, eight parental genotypes of sesame with different levels of resistance to Fusarium wilt pathogen were used in a full diallel to produce F1 progenies. The eight parents and F1 progenies were evaluated in the screen house under high pathogen pressure through artificial infection in an Alpha Lattice Design of three replicates. The results revealed that additive and non-additive gene actions contributed to controlling resistance to Fusarium wilt. However non-additive were more predominant which were signposted by moderate Baker's ratio (53.9%) and low Coefficient of Genetic Determination narrow sense (h^2 = 45.1%). Moreover, the study indicated that maternal effects have influence toward resistance to Fusarium wilt in sesame. Among eight parents used parent Sesim 2 (with EM% GCA effect 7.32, and DI% GCA effect -4.02) and EM15-1-5 (with EM% GCA effect 3.07%, and DI% GCA effect -11.58%) were good combiner parents for transmitting resistance and are recommended for use in breeding for Fusarium wilt resistance.

Key words: Fusarium wilt, inheritance, incidence, resistance, sesame,

INTRODUCTION

Fusarium wilt (*Fusarium oxysporum* f.sp. *sesami*) is a soilborne disease in which its pathogen interacts with the host plant and when inside the plant interfere with the water supply system hence the plant wilt (Bayoumi and EL-Bramawy, 2007; Elewa et al., 2011; Joshi, 2018). Fusarium wilt is among factors responsible for low yield of sesame in Uganda. Elsewhere, the disease has been reported to cause yield loss ranging from 50 to 100%

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Table 1. Sesame parental genotypes used in the *Fusarium oxysporum f.sp. sesame i*resistance inheritance study.

S/N	Genotype	Code number	Origin	Categoriesof resistance
1	4036-1-10-2//Renner1-3-16-2	P1	Ugandan pure line	Susceptible
2	Lindi 02	P2	Tanzanian variety	Moderate susceptible
3	Sesim 3	P3	Ugandan variety	Susceptible
4	Sesim 1	P4	Ugandan variety	Susceptible
5	Renner1-3-1-17	P5	Ugandan pure line	Moderate susceptible
6	Mtwara 09	P6	Tanzanian variety	Susceptible
7	Sesim 2	P7	Ugandan variety	Moderate resistance
8	EM15-1-5	P8	Ugandan pure line	Moderate resistance

Source: Ngamba et al. (2020).

(El-Bramawy et al., 2009). Some agronomic recommendations (such as early planting, intercropping, burning of leftovers and crop rotation) have been made by researchers to manage the disease in Uganda. However these agronomic practices faces some challenges like early planting aimed at helping the plant to escape from the disease which becomes severe toward the end of the rainy season; Contrariwise, this practice expose the crop to other disease like leaf spot and waterlogging effect (Egonyu et al., 2005). Crop rotation, intercropping and burning of leftovers intended to reduce the population of the pathogen in the soil but they are not efficient due to effective survival strategies of the pathogen (Okungbowa and Shittu, 2012). The best approach for managing this disease is to grow resistant varieties. Resistant varieties are efficient, long term solution, environment friendly and affordable to smallholder resource constrained farmers (Bayoumi and EL-Bramawy, 2007; Jyothi et al., 2011; Shabana et al., 2014). Unfortunately in Uganda, there are no sesame varieties with good levels of resistance to wilt. There is great need therefore to develop resistant varieties. However, successful breeding requires selection of suitable parents whose pattern of inheritance of disease resistance is known (Chataika et al., 2011). Inheritance pattern is fundamental in breeding activities since it provides information on superior parents which can easily combine to produce an offspring with desired traits. Furthermore, it provides information on the choice of breeding methods to use in relation to the trait in question (Chandra, 2011). In a study done by Ngamba et al. (2020) some of the varieties with moderate levels of resistance to Fusarium wilt were identified. These could be useful in breeding for enhanced resistance to the disease. This study was therefore carried out todetermine the mode of inheritance of resistance to *F. oxysporum* f. sp. sesami in sesame genotypes.

MATERIALS AND METHODS

F1 progenies generation

Eight parents (Table 1) were selected and crossed in the field at NaSARRI-Serere in all possible combinations to produce F1 progenies. Full Diallel mating design method 1 was used (Griffing, 1956). The eight (8) parents and fifty six (56) F1 progenies (crosses and reciprocal crosses) were evaluated in the screen house against the isolate SEFU2 of *F. oxysporum* f.sp*. sesame* following artificial inoculation. Isolate SEFU2 was among of the isolates of *F. oxysporum f*. sp*. sesame* tested by Ngamba et al. (2020) and reported to be more aggressive compared to other tested isolates. During this study, the isolate was obtained from laboratory of Makerere University Agriculture Research Institute Kabanyolo (MUARIK). The pathogen was firstly cultured on sterilized sorghum in the laboratory for 21 days at room temperature. The fully colonized sorghum seeds were then used to inoculate the sterilized soil in plastic pots.

Experimental design

The design of the study was an Alpha Lattice Design (8 blocks x 8 genotypes) with three replicates. A block was made of 8 plastic pots (2 kg capacity). Pots were filed with sterilized soil and thereafter inoculated with Fusarium wilt pathogen (SEFU2) two days prior to planting at a ratio of 75 g of isolate per pot of 2 kg of soil. Un-inoculated controls were included in the study. Fifteen seeds from the same cross were planted in each pot. Plants were watered as the conditions necessitated, and observed daily for disease symptoms.

Data collection

Data were collected for plant stand per pot and number of diseased plants per genotype. Plant stand was collected at fourteen days after planting and was used to deduce emergence percentage (EM %) while number of diseased plants per genotype was used to compute disease incidence (DI %). The following equations were used to estimate EM% and DI%.

$$\text{EM } \% = \frac{\text{Number of emerged plants}}{\text{Total number of plant sown}} * 100$$

$$\text{DI } \% = \frac{\text{Number of diseased plants}}{\text{Total number of plant emerged}} * 100$$

The linear model used during analysis of variance was,

$$Y_{ijk} = \bar{\bar{Y}} + G_i + R_j + (R/B)_k + E_{ijk}$$

Y_{ijk} is the observation value for genotype ith and jth $\bar{\bar{Y}}$ is mean, G_i is

Table 1. Scale for classification of the genotypes.

Scale number	Infection percentage	Category of resistance
1	0.00	Immune (I)
2	0.1 -20	Resistant (R)
3	20.1 - 40	Moderately resistant (MR)
4	40.1 - 60	Moderately susceptible (MS)
5	60.1 - 80	Susceptible (S)
6	80.1 - 100	Highly susceptible (HS)

Table 3. Analysis of variance for the emergence percentage (EM %) and disease incidence (DI %) of 56 progenies of sesame with their eight parents.

SOV	df	EM%	DI%
Replication	1	50	725.96**
Parental/crosses	63	636.56***	507.66***
Residual	63	29.43	57.51
Total	127	330.77	286.08
CV%		9.26	10.96

SOV= Source of Variance, Values with **and *** represent significance at P≤0.01 and P≤0.001 respectively.

the genotype effect for ith , R_j is the replication effect for jth ,$(R/B)_k$is the replication block nested effect for the kthand E_{ijk} is the experimental error effect.

Data analysis

Data were subjected to Analysis of Variance (ANOVA) in Genstat 18th edition Software to determine significant treatment effects. Fisher Protected Least Significant Difference (LSD) test at 5% probability level was used to compare means. Progenies were then grouped according to their resistance levels using the scale developed by Kavak and Boydak (2006) with slight modification (Table 2). Combining ability analysis was carried out following Griffing (1956) method 1 model. The estimates of general combining ability (GCA) for the parents, specific combining ability (SCA) for the crosses and reciprocal effect were calculated according to the linear model,

$$Y_{ijkl} = \bar{\bar{Y}} + gca_i + gca_J + sca_{ij} + r_{ij} + P_k + (P/B)_l + e_{ijkl}$$

Where, $Y_{ijkl}$is the observed value for genotype ith and jth, $\bar{\bar{Y}}$ the grand mean, $gca_i\ and\ gca_J sca_{ij}$ and r_{ij} are the general combining and specific combining ability and reciprocal effect for ith, jth and ijth respectively, P_k replication effect for kth, *(P/B)ₗ* is the replication nested block effect for the lth and e_{ijkl} is the experimental error effect.

For gene action determination,parents were considered to be fixed. The estimated variance components (σ^2) of GCA and SCA were used to calculate the coefficient of genetic determination (CGD) both broad sense (BS (H)) and narrow sense (NS (h^2)) heritability (Equation 1 and 2). Baker's ratio (1978) which determines the fraction of genetic variation that is due to additive effects was calculated according to equation 3.

$$\text{CGDBS (H)} = \frac{2\delta^2\text{gca} + \delta^2\text{sca}}{2\delta^2\text{gca} + \delta^2\text{sca} + \delta^2\text{e}} \quad (1)$$

$$\text{CGDNS } (h^2) = \frac{2\delta^2\text{gca}}{2\delta^2\text{gca} + \delta^2\text{sca} + \delta^2\text{e}} \quad (2)$$

$$\text{BR} = \frac{2\delta^2\text{gca}}{2\delta^2\text{gca} + \delta^2\text{sca}} \quad (3)$$

RESULTS

Responses of parents and crosses on emergence and disease incidence

On analysis of data, it was realized that the block effect was not effective in an alpha lattice design, so analysis was done following the randomized complete block design model. The results showed significant differences among parent/crosses for all the traits tested (P≤0.001) (Table 3). The means of emergence ranged from 26.0 to 98.0% with a grand mean of 58.6% and disease incidence that ranged from 33.3 to 96.0% with grand mean of 69.2% (Table 4). Parents Sesim 2 (36.7%) and EM15-1-5 (39.1) continued to be moderately resistant with the rest of the parents being susceptible or highly susceptible. Only one, a reciprocal cross (EM15-1-5 x Sesim 1) (33.3%) was recorded as moderately resistant. The other crosses were ranged from susceptible to highly susceptible (Table 4).

Combining ability and gene action

Table 5 represents analysis of variance for combining ability of emergence (EM %), disease incidence (DI %),

Table 4. Means of emergence, disease incidence and categories for resistance performance of eight parents and 56 F1 progenies in response to *Fusarium oxysporum* f.sp. *sesami* in screen house.

Genotype	EM%	DI%	Category
P8 X P4	60.0	33.3	MR
P7	98.0	36.7	MR
P8	82.0	39.0	MR
P7 X P8	84.0	43.0	MS
P4 X P2	50.0	44.6	MS
P8 X P3	52.0	45.8	MS
P2 X P6	46.0	47.7	MS
P3 X P1	36.0	50.0	MS
P8 X P6	62.0	51.5	MS
P1 X P2	46.0	51.9	MS
P7 X P5	68.0	53.1	MS
P5 X P1	44.0	54.5	MS
P8 X P1	50.0	56.1	MS
P5 X P2	50.0	56.8	MS
P4 X P6	46.0	56.9	MS
P4 X P7	80.0	57.8	MS
P3 X P6	60.0	60.0	MS
P3 X P5	60.0	60.2	S
P7 X P3	60.0	60.3	S
P8 X P5	50.0	60.4	S
P3 X P2	82.0	60.8	S
P4 X P3	48.0	62.1	S
P8 X P7	64.0	62.7	S
P1 X P4	76.0	62.8	S
P4 X P8	70.0	63.1	S
P2 X P8	66.0	63.9	S
P5 X P8	56.0	64.3	S
P1 X P8	40.0	64.6	S
P3 X P8	40.0	65.0	S
P7 X P4	52.0	65.5	S
P5 X P4	64.0	65.7	S
P2 X P1	68.0	67.7	S
P7 X P6	62.0	67.7	S
P2 X P7	38.0	68.9	S
P5 X P3	54.0	70.6	S
P4 X P5	26.0	73.8	S
P5 X P7	62.0	74.2	S
P1 X P5	56.0	75.1	S
P8 X P2	66.0	75.9	S
P7 X P2	44.0	77.5	S
P3 X P7	72.0	77.8	S
P2 X P4	56.0	77.9	S
P2 X P5	46.0	78.0	S
P3	94.0	78.5	S
P6 X P5	40.0	79.2	S
P5	86.0	79.2	S
P1 X P7	60.0	79.5	S
P2 X P3	50.0	79.8	S
P6 X P4	30.0	80.6	HS

Table 4. Contd.

P5 X P6	32.0	81.7	HS
P3 X P4	60.0	83.5	HS
P4	86.0	83.5	HS
P6 X P3	26.0	84.5	HS
P1 X P3	76.0	86.9	HS
P7 X P1	80.0	87.5	HS
P4 X P1	36.0	88.7	HS
P6 X P2	36.0	88.9	HS
P2	90.0	91.0	HS
P6	78.0	92.5	HS
P6 X P7	32.0	93.8	HS
P6 X P8	62.0	93.8	HS
P1 X P6	44.0	95.8	HS
P1	92.0	95.8	HS
P6 X P1	66.0	96.9	HS
Minimum	26.0	33.3	
Maximum	98.0	96.9	
Grand Mean	58.6	69.2	

EM%-emergence percentage, DI%-disease incidence, MR-moderate resistant, S-susceptible, MS-moderate susceptible HS-highly susceptible and P-parent.

Table 5.ANOVA for combining ability for the emergence percentage and disease incidence of 56 crosses with their parents.

SOV	df	EM%	DI%
Crosses	63	318.28***	253.83***
GCA	7	354.93***	665.93***
SCA	28	419.05***	165.03***
Reciprocal	28	208.35***	239.61***
Error	63	14.72	28.76
VCgca		21.26	39.82
VCsca		202.16	68.13
H		0.94	83.71
h^2		0.16	45.12
Baker's ratio		0.17	53.90

SOV- Source of Variance, Values with *** represent significance at P≤0.001; EM%-emergence percentage; DI%-disease incidence; H- coefficient of genetic determination both broad sense; h^2- coefficient of genetic determination both narrow sense; VCgca and VCsca- variance components of general combining ability and specific combining ability respectively.

coefficient of genetic determination for broad and narrow sense ((CGD BS (H) and CGD NS (h^2)) and Baker's ratio (BR). Results showed that crosses, general combining ability (GCA), specific combine ability (SCA) and reciprocal effects were highly significant (P≤0.001) in all traits tested. Coefficient of genetic determination (broad sense) was high (>0.80) in all traits tested. The coefficient of genetic determination (narrow sense) was low (0.16 for EM% while 0.45 for DI %). Bakers' ratio was only medium (0.54) in disease incidence.

General combining ability effect of resistance to *Fusarium oxysporum* f. sp. *sesami* for parental genotypes

With exception of Lindi 02 and Sesim 1, all parents significantly influenced emergence (P≤0.001 to P≤0.05). For disease incidence, four parents (Lindi 02, Sesim 3, Sesim 1 and Renner 1-3-1-17) were not significant from each other while the remaining four parents (4036-1-10-2//Renner1-3-16-2, Mtwara, Sesim 2 and EM15-1-5) were

Table 6. General combining ability relative estimates for the emergence percentage and disease incidence of eight parents.

Parent name	EM%	DI%
4036-1-10-2//Renner1-3-16-2	1.57*	6.44***
Lindi 02	-0.82^{ns}	0.99^{ns}
Sesim3	1.69**	-0.14^{ns}
Sesim1	-0.69^{ns}	-1.465^{ns}
Renner1-3-1-17	-3.57***	-0.04^{ns}
Mtwara 09	-8.57***	9.82***
Sesim2	7.32***	-4.02***
EM15-1-5	3.07***	-11.58***

Values with *, **and *** represent significance at P≤0.05, P≤0.01 and P≤0.001 respectively while ns is non-significant; EM%-emergence percentage and DI%-disease incidence.

highly and significantly different (P≤0.001) (Table 6).

Specific combining ability effect of resistance to *F. oxysporum* f. sp*. sesami* for F1 progenies

Results for emergence indicated that the effect of seven crosses was highly significant (P≤ 0.001) and that of eleven other crosses significant (P≤ 0.01 to P≤0.05). The effect of the remaining ten crosses was not significant (Table 7). For disease incidence, 17 crosses were not significant while eleven had significant (P≤ 0.001 to P≤0.05) effects.

Reciprocal effects of resistance to *F. oxysporum* f.sp*. sesami* for F1 progenies

The reciprocal effects for emergence and disease incidence are shown in Table 8. Results showed that 18 crosses were highly significant for emergence (P≤ 0.001). For the same trait, eight crosses were significant (P≤ 0.01 and P≤ 0.05) while two crosses (EM15-1-5 x Lindi 02 and Sesim 2 x Sesim 3) were not significant. For disease incidence, 21 crosses were highly significant (P≤ 0.001) while the four crosses were not. The crosses Mtwara 09 x Sesim 1 and EM15-1-5 x Renner1-3-1-17 were significantly different at P≤ 0.05 while the cross Renner1-3-1-17 x Lindi 02 was the only highly significant one at P≤ 0.01.

DISCUSSION

Inheritance study is important on determining how gene of interest transferred from one generation to another generation, the best combiner parents for breeding program and also guide breeder to choose the best breeding methods regarding the trait of interest (Goffar et al., 2016). The study used eight promising parents of different reaction to Fusarium wilt with their 56 F1 progenies. From the results above, the parental/crosses response to EM% and DI% was highly significant suggesting that all materials responded differently to *F. oxysporum* f. sp*sesami.* This showed that there is genetic diversity within the tested materials providing a high possibility of obtaining materials with good wilt resistance. Only a cross EM15-1-5 x Sesim1 was moderate resistant (33.3%) while others ranged from moderately susceptible to highly susceptible. Combining ability analysis showed highly significant differences for crosses, GCA, SCA and reciprocal effects for all the traits tested (P≤ 0.001). This suggests that both additive and non-additive genetic variances are involved in controlling resistance to *F. oxysporum* f. sp*. sesami*. Highly significant GCA exhibited by parents is evidence that those parents had transferred their traits to the progenies. This implied that additive gene effects were involved in the transmission of the traits tested. SCA was also highly significant for all traits tested meaning that the observed and expected performance of the progenies due to allelic combination could be due to non-additive effects. Moreover, all traits observed had shown to be influenced by extra-chromosomal inheritance or maternal effects since the reciprocals effects were also significant. Different studies have shown that germination percentage is controlled by both additive and non-additive gene action and maternal effects (Donohue, 2009; Luzuriaga et al., 2006; Rix et al., 2012; Singh et al., 2017; Wanjala et al., 2017). Non-significance of SCA mean squares is a good indication that the performance of single cross progeny can be adequately predicted on the basis of GCA (Baker, 1978). From this study, it is very difficult to predict the performance of the progeny based on GCA since the SCA mean squares were significant. It was good to see that coefficient of genetic determination broad sense (H) was high (greater than 80%). This means that more than 80% of variance in phenotypic performance is genetically controlled. Coefficient of genetic determination narrow

Table 7. Specific combining ability for the emergence percentage and disease incidence of 28 crosses.

Genotype	EM%	DI%
4036-1-10-2//Renner1-3-16-2 x Lind 02	-2.31^{ns}	-16.78**
4036-1-10-2//Renner1-3-16-2 x Sesim 3	-5.81*	-7.00*
4036-1-10-2//Renner1-3-16-2 x Sesim 1	-3.44^{ns}	1.61^{ns}
4036-1-10-2//Renner1-3-16-2 x Renner1-3-1-17	-6.56**	-10.73***
4036-1-10-2//Renner1-3-16-2 x Mtwara 09	3.44^{ns}	10.92***
4036-1-10-2//Renner1-3-16-2 x Sesim 2	2.56^{ns}	11.89***
4036-1-10-2//Renner1-3-16-2 x EM15-1-5	-18.19**	-3.69^{ns}
Lindi 02 x Sesim 3	6.56**	0.31^{ns}
Lindi 02 x Sesim 1	-4.06^{ns}	-7.44*
Lindi 02 x Renner1-3-1-17	-6.19*	-2.69^{ns}
Lindi 02 x Mtwara 09	-8.19**	-11.66***
Lindi 02 x Sesim 2	-24.06***	7.06*
Lindi 02 x EM15-1-5	5.19*	11.33***
Sesim 3 x Sesim 1	-5.56*	5.25^{ns}
Sesim 3 x Renner1-3-1-17	0.31^{ns}	-3.60^{ns}
Sesim 3 x Mtwara 09	-8.69***	-6.59^{ns}
Sesim 3 x Sesim 2	-1.56^{ns}	4.01^{ns}
Sesim 3 x EM15-1-5	-17.31***	-2.03^{ns}
Sesim 1 x Renner1-3-1-17	-9.31***	2.05^{ns}
Sesim 1 x Mtwara 09	-11.31**	-8.79*
Sesim 1 x Sesim 2	0.81^{ns}	-2.07^{ns}
Sesim 1 x EM15-1-5	4.06^{ns}	-7.92*
Renner1-3-1-17 x Mtwara 09	-10.44***	1.51^{ns}
Renner1-3-1-17 x Sesim 2	2.69^{ns}	-1.47^{ns}
Renner1-3-1-17 x EM15-1-5	-5.06*	4.79 ^{ns}
Mtwara 09 x Sesim 2	-10.31***	5.76^{ns}
Mtwara 09 x EM15-1-5	8.94***	5.20^{ns}
Sesim 2 x EM15-1-5	5.06*	-0.72^{ns}

Values with *, **and *** represent significance at P≤0.05, P≤0.01 and P≤0.001 respectively while ns is non-significant; EM%-emergence percentage and DI%-disease incidence.

sense (h^2) for EM% was 0.16 while that of DI% was 45.12. This indicates that only 16% (for EM %) and 45.12% (for DI %) of the variance in the phenotypic performance is predictably transmitted, thus low predictability of progeny from parental performance. Baker's ratio was very low for EM% (0.17) while for DI% was fairly medium. This still suggests the predominance of non-additive gene action in controlling resistance to *F. oxysporum* f. sp*. sesami*in sesame. These results clearly suggest that selection during late generations would be the best breeding strategy for improving resistance in sesame to *F. oxysporum* f. sp*. sesami*. These findings correspond to other studies done under both natural and artificial inoculation using F1 progenies along with their parents. Those studies reported that gene action governing resistance to Fusarium wilt in sesame is predominantly controlled by non-additive gene action (Bayoumi and EL-Bramawy, 2007; El-Bramawy and Shaban, 2007). Furthermore, it was reported that in the late generations, resistance to Fusarium wilt is controlled by additive gene action (El-bramawy, 2006). The finding of this study could be influenced by type of genetic material used, mode of pollination, parent cross combination and method used to derive gene action (Goffar et al., 2016; Ulloa et al., 2013).

The parents 4036-1-10-2//Renner1-3-16-2, Sesim 3, Sesam 2 and EM15-1-5 showed positive desirable significant GCA effects for high germination. These could be associated with resistance to seed rot caused by *F. oxysporum* f. sp*. sesami* and are good combiners for improvement of this trait. On the other hand, Sesim 1, Mtwara 09, Renner1-3-1-17 had negative significant GCA effects for low germination. These genotypes were thus not associated with resistance to seed rot and are therefore poor combiners for improvement of sesame for this trait. Similarly, Lindi 02 had negative and non-significant GCA for germination suggesting that this parent was not associated with resistance to seed rot

Table 8. Reciprocal effect for the emergence percentage and disease incidence of 28 reciprocal crosses.

Genotype	EM%	DI%
Lindi 02 x 4036-1-10-2//Renner1-3-16-2	11.00***	7.89***
Sesim 3 x 4036-1-10-2//Renner1-3-16-2	-20.00***	-18.47***
Sesim 3 x Lindi 02	16.00***	-9.49***
Sesim 1 x 4036-1-10-2//Renner1-3-16-2	-20.00***	12.99***
Sesim 1 x Lindi 02	-3.00**	-16.70***
Sesim 1 x Lindi 02	-6.00***	-10.67***
Renner1-3-1-17 x 4036-1-10-2//Renner1-3-16-2	-6.00*	-10.29***
Renner1-3-1-17 x Lindi 02	2.00***	-10.61**
Renner1-3-1-17 x Ssesim 3	-3.00***	5.21^{ns}
Renner1-3-1-17 x Sesim 1	19.00***	-4.03^{ns}
Mtwara 09 x 4036-1-10-2//Renner1-3-16-2	11.00**	0.52***
Mtwara 09 x Lindi 02	-5.00***	20.58***
Mtwara 09 x Sesim 3	-17.00***	12.26***
Mtwara 09 x Sesim 1	-8.00***	11.82*
Mtwara 09 x Renner1-3-1-17	4.00**	-1.29^{ns}
Sesim 2 x 4036-1-10-2//Renner1-3-16-2	10.00***	4.02***
Sesim 2 x Lindi 02	3.00*	4.31***
Sesim 2 x Sesim 3	-6.00^{ns}	-8.75***
Sesim 2 x Sesim 1	-14.00***	3.85^{ns}
Sesim 2 x Renner1-3-1-17	3.00***	-10.52***
Sesim 2 x Mtwara 09	15.00***	-13.02***
EM15-1-5 x 4036-1-10-2//Renner1-3-16-2	5.00**	-4.25***
EM15-1-5 x Lindi 02	0.00^{ns}	6.02***
EM15-1-5 x Sesim 3	6.00***	-9.58***
EM15-1-5 x Sesim 1	-5.00***	-14.87***
EM15-1-5 x Renner1-3-1-17	-3.00*	-1.95*
EM15-1-5 x Mtwara 09	0.00*	-21.15***
EM15-1-5 x Sesim 2	-10.00***	9.90***

Values with *, **and *** represent significance at P≤0.05, P≤0.01 and P≤0.001 respectively while ns is non-significant; EM%-emergence percentage and DI%-disease incidence.

hence it is a poor combiner for improving this trait. Genotypes Sesim 2 and EM15-1-5 had significant negative GCA for disease incidence making them be good combiner parents for improving this trait. The susceptible parents (Mtwara 09 and 4036-1-10-2//Renner1-3-16-2) showed positive significant GCA effect indicating a possibility of these parents passing susceptibility to *F. oxysporum f.* sp*. sesami* to their progenies. Contrastingly, the susceptible parents Sesim1, Sesim 3 and Renner1-3-1-17 had non-significant negative GCA effects for disease incidence which implied that they could be good combiners and would pass resistance to *F. oxysporum* f. sp*. sesami* to their progenies. A high GCA estimate is the determinant of higher heritability with less environment effects, less gene interactions and high achievement in selection (Chigeza et al., 2013).

Although the crosses 4036-1-10-2//Renner1-3-16-2 x Lindi 02, and Lind 02 x Mtwara 09 were made from a combination of poor parents, they showed desirable highly negative significant SCA effects for disease incidence. This could be due to dominance x dominance type of non-allelic gene interaction produced over dominance (Wassimi et al., 1986). Furthermore, crosses 4036-1-10-2//Renner1-3-16-2 x Renner1-3-1-17, Sesim 3 x Renner1-3-1-17 and Sesim 1 x EM15-1-5 made from a combination of a poor and a good parent showed good SCA effects for disease incidence. This implied that favourable additive effects of the good general combiner parent contributed to the performance of these progenies (Verma and Srivastava, 2004). Therefore, the above crosses can be included in a breeding programme for developing wilt resistant sesame varieties.

Reciprocal effects showed that most of the crosses were highly and significantly influenced disease incidence. This suggests that maternal effects could be controlling resistance to *F. oxysporum* f. sp*. sesami* in sesame. This type of gene interaction is significant in a

breeding program which permits the determination of parent to be used as donor or recipients of pollen (Bahari et al., 2012). Reciprocal crosses of Renner1-3-1-17 x Lindi 02, Sesim 2 x Renner1-3-1-17, Sesim 2 x Mtwara 09 and EM15-1-5 x Mtwara 09 had positive and negative significant reciprocal effects for EM% and DI% respectively. This means that parents used as females were able to contribute much to the performance of the offspring due to maternal effects. These crosses can be used for breeding for wilt resistance in the future.

CONCLUSIONS AND RECOMMENDATIONS

Based on the results obtained from this study, inheritance of resistance to Fusarium wilt (*F. oxysporum* f. sp. *sesami*) in sesame among the studied genotypes is controlled by both additive and non-additive gene action as revealed by low coefficient of genetic determination narrow sense and moderate Baker's ratio. The study further showed that maternal effects influenced resistance to Fusarium wilt which was shown by highly significant performance among reciprocal crosses. Accordingly, selection for resistance to Fusarium wilt can be effective in late generations. The study also showed that genotypes Sesam 2 and EM15-1-5 were the best general combiner parents compared to the rest and can be used in routine breeding for resistance to Fusarium wilt. Overall, findings of this study have created the foundation for further studies upon which the sesame breeding program in Uganda can be based.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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