

MECHANISMS ASSOCIATED WITH INHERITANCE OF RESISTANCE TO ERGOT (*Claviceps Species*) IN SORGHUM

Dejene Kebede^{1,2*}, Patrick Rubaihayo¹, Isaac Dramadri¹, Thomas Odong¹, Richard Edema¹

¹Makerere University, College of Agricultural and Environmental Science, School of Agricultural Science, Kampala, Uganda ²Ethiopian Institute of Agricultural Research, Addis Ababa, Ethiopia

Abstract. Breeding for resistance to ergot requires reliable information on genotypic variability for hostplant resistance, inheritance of resistance to ergot and influence of non-genetic factors. Hence, this study was conducted to estimate the mode of inheritance and identify parents and crosses with good combining abilities for resistance to ergot in sorghum. Twelve genotypes including 7 resistance and 5 susceptible parental lines were used to generate the segregating populations through North Carolina II Mating Design. The crosses were evaluated at Makerere University Agricultural Research Institute Kabaynolo during 2020 and 2021 growing seasons under natural infestation conditions and data were collected based on ergot incidence and severity and other traits. Highly significant variation was observed among crosses for ergot incidence and severity. The mean ergot incidence and severity ranged between 13.4 -77.0% and 2.1 - 4.1%, respectively across years. The GCA and SCA analysis revealed highly significant differences among the F2 segregating populations for ergot incidence and severity suggesting the importance of additive and non-additive genetic effects in determining the inheritance of resistance to ergot. Heritability estimates in narrow sense ranged between 0 - 46.3% and 0.9 - 66.6% in 2020 and 2021, respectively. The variation in heritability estimates over years revealed the importance of genotype by environment interaction in the inheritance of resistance to ergot. Parents and crosses with significant negative GCA and SCA effects identified in this study could be used for improving resistance to ergot.

Keywords: Combining ability, disease incidence, disease severity, heritability.

Corresponding Author: Dejene, Kebede, Makerere University, School of Agricultural Science, Kampala, Uganda, Phone: +251913367515, e-mail: <u>dejene100@gmail.com</u>

Received: 14 October 2022; Accepted: 27 November 2022; Published: 12 December 2022.

1. Introduction

Sorghum production is constrained by a range of biotic and abiotic factors and among others is ergot caused by *Claviceps* species. Ergot is a prevalent disease in sorghum cultivation, particularly in hybrid seed productions. When environmental conditions are favorable for infection, ergot can cause a considerable yield loss in all type of sorghum genotypes (GRDC, 2017). Breeding for host resistance is an important approach for efficient management and control of ergot disease in sorghum (GRDC, 2015). Several studies indicated that resistance to ergot in sorghum is associated with floral characteristics, including the ability to pollinate and fertilize before infection occurs, mainly dependent on the availability of pollen, stigma size, start of stigma drying after pollination and resistance that hinders fungal infection or fungal spread in the gynoecium (Parh *et al.*, 2008; Miedaner & Geiger, 2015; GRDC, 2017). An extended flowering period associated with a cool and wet environment can cause a fast development and spread of the pathogen to neighboring sorghum fields (Montes-garcía *et al.*, 2008). To develop an appropriate breeding strategy for resistance to ergot, identification of resistant genotypes should be followed by the study of the mechanisms of inheritance of resistance to ergot. Ergot has for long time not been considered as a serious sorghum diseases and little information is available on genetic variance, gene action and inheritance of resistance to ergot in sorghum (Mirdita *et al.*, 2008). Therefore, the present study was conducted to estimate the inheritance and identify parents and crosses with good combining abilities for resistance to ergot in sorghum germplasm collections.

2. Materials and Methods

Experimental site

The experiment was carried out at Makerere University Agricultural Research Institute Kabaynolo (MUARIK) during 2020 and 2021. The site is located at 0°28'N and 32°37'E with an altitude of 1200 m. a. s. l. and deep ferrallitic soils with a pH range of 5.2 to 6.0 and a mean annual temperature of 15-35.5°C with an annual average rainfall of 1150 mm and humidity of 13 - 96% (Miesho *et al.*, 2019).

Plant materials and experimental procedures

The experimental materials consisted of seven resistant genotypes: PI651496, PI655972, NAROSORG2, PI597971, SESO1, SEKEDO and NAROSORG3 and five susceptible genotypes: PI656056, SNR39, PI533785, PI534124 and SQR. The materials were selected based on their resistance levels and agronomic performances from the experiments conducted during 2019 at MUARIK and NaSARRI to screen sorghum germplasm against ergot infection.

Generation of F2 segregating population

All the twelve sorghum parental lines were planted separately in a five-litter bucket (two seeds per bucket) in a screen house at MUARIK. Each line was hand emasculated before pollen shedding and crossed at flowering following 7 by 5 North Carolina II mating design approach. Flowers to be used as females were emasculated in the evening using sterile scalpel blade regularly sterilized using alcohol (95%) and hand pollinated in the morning from 9:00 - 11:00 a.m. The F1 crosses were selfed to produce F2 generation in a screen house. The F2 seeds were harvested and bulked for each of the 35 crosses.

Evaluations of segregating population

Evaluation of the F2 segregating populations for resistance to ergot was conducted at MUARIK during the second rain season (September – December) of 2020 and the first rain season (April-July) of 2021. The experiment was planted using alpha-lattice design with two replications. In 2020, the plots were planted in six rows with a row length of 3m and 0.75 m and 0.15 m spacing between rows and within rows respectively, while in 2021 the plot consisted of 3 rows due to shortage of seeds, with 3 m row length and 0.75 and 0.15 m inter and intra row spacing, respectively and the fields were weeded three times. Three crosses (PI533785 X PI651496, PI533785 X PI655972 and SNR39 X PI597971) were not included in the evaluation due to loss of their seeds.

Data collection

In 2020, planting was done in September and ergot data was collected in December for three consecutive weeks: 14 weeks after planting, (14WAP), 15 weeks after planting

(15WAP) and the final score was collected 16 weeks after planting (16WAP). In 2021, planting was done in April and data on ergot infection was recorded in July for three consecutive weeks (14WAP, 15WAP and 16WAP). Data for ergot severity was collected based on 1-5 scale visual scoring as described by Musabyimana et al., (1995): 1 = no ergot/sphacelia infection, 2 = 1-10% infection, 3 = 11-25% infection, 4 = 26-50% infection, 5 = more than 50% infection. Ergot incidence was estimated by counting all the individual plants with ergot disease symptoms in each plot and expressing the number as a ratio over the total number of plants in the plot and multiplied by 100 to express it as a percentage. Data for pollen quantity was measured on 1 - 10 scale (1 for no visible pollen and 10 for copies quantity of visible pollen) by observing the resultant clouds of pollen flicked from sorghum heads (Parh et al., 2006). Hundred seed weight was collected by weighing hundred seeds for each plot. Days to 50% flowering was recorded as the number of days from planting to when 50% of the plants shad the pollen (Awori et al., 2015). Seedling vigoursity was recorded on 1-5 scale where, 1= healthy and vigorous and 5= weak and diseased plants.

Data analysis

Analysis of variance for ergot severity, incidence and other agronomic traits were computed in R statistical package for Windows V-4.1.1. Mean separation was done using Least Significant Difference (LSD) at 0.05 level of significance. North Carolina II mating design analysis was performed using R statistical package for Windows V-4.1.1 and Genetic Designs in R (AGD-R) Version 3.0. The general combining ability (GCA) effects were analyzed for each parent. The GCA effects of the male and female parents were estimated as the difference between the grand mean and the mean of the parents for the trait. Specific combining ability (SCA) was analyzed for the F2 crosses. The SCA effects of each cross was calculated as a deviation of the cross mean from the grand mean of all the crosses adjusted for corresponding GCA effects of parents. Narrow sense and broad sense heritability were also estimated. Relative importance of GCA and SCA (Bakers' ratio) was estimated according to Baker's (1978). Pearson correlation analysis was also performed to understand the association between resistance to ergot and agronomic traits. All the analysis was done using R statistical package for Windows V-4.1.1 (R Core Team, 2020) and Genetic Designs in R (AGD-R) Version 3.0 (Rodríguez et al., 2015).

3. Results

Analysis of variance for ergot incidence, severity and other traits

In 2020, there were highly significant differences (P < 0.01) among genotypes for ergot incidence and severity at 14WAP, 15WAP and 16WAP, DTF and SV. Likewise, significant differences (P < 0.01) were observed among genotypes for ergot incidence and severity at 14WAP, 15WAP and 16WAP, DTF, SV and PQ in 2021 (Table 1).

In the combined analysis of variance (across years) significant differences were observed among genotypes for most of evaluated traits including ergot incidence and severity at 14WAP, 15WAP and 16WAP, DTF, HSW, PQ and SV. Year significantly affected ergot incidence and severity at 14WAP, 15WAP and 16WAP, HSW, DTF and SV. Year by Genotype interaction showed significant variations for ergot incidence and severity at 14WAP (Table 2).

			E	rgot inciden	ice	F	Ergot severi	ty	USW DTE			
	sov	DF	14WAP	15WAP	16WAP	14WAP	15WAP	16WAP	HSW	DTF	PQ	sv
2020	Block/Rep	6	48.7 ^{ns}	33.9 ^{ns}	52.8 ns	0.21 ns	0.06 ns	0.04 ns	0.07 ^{ns}	10.5 ns	0.07 ^{ns}	0.52**
	Rep	2	1.83 ns	303 ns	106 ns	0.00 ^{ns}	0.00 ns	0.11 ns	0.28 ns	66.0*	0.97**	0.47 ^{ns}
	Genotype	31	114**	509**	714**	0.35**	0.61**	0.67**	0.24 ^{ns}	49**	0.15 ^{ns}	0.54**
	Error	25	25.8	141	66.0	0.09	0.04	0.07	0.23	16.4	0.09	0.13
2021	Block/Rep	6	454**	330**	100**	0.40 ^{ns}	0.14 ^{ns}	0.08 ^{ns}	0.08 ^{ns}	4.29 ^{ns}	0.31 ^{ns}	0.08 ^{ns}
	Rep	2	771*	53.1 ^{ns}	56.9 ^{ns}	0.00 ^{ns}	0.01 ^{ns}	0.24 ^{ns}	0.69 ^{ns}	7.56 ^{ns}	0.09 ^{ns}	0.01 ^{ns}
	Genotype	31	509**	509**	597**	0.54**	0.35**	0.39**	0.43 ns	47**	0.46*	0.26**
	Error	25	124	87.5	63.3	0.19	0.10	0.10	0.24	15.7	0.19	0.05

 Table 1. Mean squares for ergot incidence, severity and other traits evaluated at MUARIK during 2020 and 2021

*, ** = Significant at P <0.05 and P <0.01, respectively. Abbreviations: ns – non-significant, SOV- source of variations, DF - degree of freedom, WAP – weeks after planting, DTF - days to 50% flowering, PQ - pollen quantity, HSW – hundred seed weight and SV – seedling vigour

 Table 2. Combined analysis of variance for ergot incidence, severity and other traits evaluated at MUARIK during 2020 and 2021

SOV	DF	Er	got inciden	ce	Е	rgot severi	ty	DTF	PQ	HSW	SV
		14WAP	15WAP	16WAP	14WAP	15WAP	16WAP				
Rep/Year	2	386.8*	178.4 ^{ns}	81.6 ^{ns}	0.001 ns	0.03 ^{ns}	0.17 ^{ns}	36.8 ^{ns}	0.6*	0.35 ns	0.24 ^{ns}
Year	1	15303**	2616**	2931**	18.3**	2.3**	1.02**	532**	0.2 ns	7.5**	2.1**
Genotype	31	373.2**	881.9**	1219**	0.60**	0.8**	0.9**	80.9**	0.4**	0.38*	0.66**
Year X	31	250**	137.0 ^{ns}	93.5 ^{ns}	0.29*	0.09 ^{ns}	0.11 ^{ns}	15.5 ns	0.2 ns	0.27 ns	0.14 ^{ns}
Genotype											
Error	62	109.4	127.7	66.9	0.18	0.07	0.08	14.4	0.2	0.2	0.13

*, ** = Significant at P <0.05 and P <0.01, respectively. Abbreviations: ns – non-significant, SOV- source of variations, DF - degree of freedom, WAP – weeks after planting, DTF - days to 50% flowering, PQ - pollen quantity, HSW – hundred seed weight and SV – seedling vigour

Performance of F2 genotypes

In 2020, the average ergot incidence ranged between 2.0 - 36.2% at 14WAP, 7.7 - 69.8% at 15WAP and 8.6 - 74.0% at 16WAP, and the average ergot severity varied between 1.3 - 3.0 at 14WAP, 1.9 - 4.0 at 15WAP and 2.0 - 4.0 at 16WAP. Crosses PI534124 X NAROSORG2, PI534124 X PI655972, SQR X PI651496, PI534124 X PI651496, PI656056 X NAROSORG3 showed the least ergot severity. The crosses PI656056 X PI597971 (3.7g), SQR X Seso3 (3.6g), PI656056 X Seso3 (3.3g), SQR X PI597971 (3.2g) recorded the highest hundred seed weight. Overall, PI534124 X NAROSORG2, PI656056 X PI655972, SQR X PI651496, PI656056 X NAROSORG3, PI534124 X PI655972 were the best crosses based on overall performances.

In 2021, the average ergot incidence varied between 9.6 - 63.9% at 14WAP, 15.8 – 80.0 at 15WAP and 15.8 – 80.0 % at 16WAP. The average ergot severity ranged between 1.3 – 3.5 at 14WAP, 2.3 – 4.0 at 15WAP and 2.3 – 4.0 at 16WAP. Crosses PI534124 X NAROSORG2, SQR X PI651496, PI534124 X NAROSORG3, PI656056 X PI655972, SRN39 X PI655972S had the lowest ergot severity score in 2021 and crosses PI656056 X NAROSORG3 (4.5g), PI533785 X Seso3 (4.0g), PI656056 X NAROSORG2, (4.0g), SRN39 X NAROSORG2 (4.0g), SRN39 X NAROSORG3 (4.0g) recorded the highest hundred seed weight. Crosses, PI534124 X NAROSORG2, SRN39 X NAROSORG2, SRN

PI534124 X NAROSORG3, SQR X PI651496, PI534124 X PI651496, PI656056 X PI655972 and SRN39 X PI655972 were the best crosses based on overall performance. Hundred seed weight ranged between 1.97 - 3.7g and 2.6 - 4.5g in 2020 and 2021, respectively. Days to 50% flowering varied between 69.5 - 90 days and 73 - 91 days in 2020 and 2021, respectively. The means of PQ were 6.9 in 2020, and 6.8 in 2021.

Across years, ergot incidence varied between 8.2 - 46.0% at 14WAP, 14.4 - 74.9% at 15WAP and 13.4 - 77.0% at 16WAP and ergot severity ranged between 1.3 - 3.0 at 14WAP, 2.1 - 4.0 at 15WAP and 2.1 - 4.1 at 16WAP. The crosses PI534124 X NAROSORG2 and SQR X PI651496 showed less than 10% ergot severity and had relatively lower ergot incidence across years. Crosses PI656056 X NAROSORG3 (3.7g), PI656056 X NAROSORG2 (3.6g) and PI656056 X PI597971 (3.6g) recorded the highest hundred seed weight. Crosses PI534124 X NAROSORG2, SQR X PI651496 and PI656056 X Seso3 were the best crosses based on their overall performance across the years evaluations.

Variance component analysis

The variance components (GCA and SCA) for ergot incidence and severity at 14WAP, 15WAP and 16WAP, DTF, PQ, HSW were computed from F2 segregating populations generated using 7X5 North Carolina Design II and the results revealed that the GCA effects of both female and male parents as well as the SCA effects of crosses were significant for most of the traits evaluated (Table 3). In 2020, mean squares due to GCA Female and Male were significant for ergot incidence and severity at 14WAP, 15WAP and 16WAP and DTF. Mean squares due to SCA effects were significant for ergot incidence and severity at 14WAP, 15WAP and, 16WAP and DTF. The narrow sense heritability (NSH) and broad sense heritability (BSH) ranged between 0 - 46.3% and 22.3 – 91.4%, respectively. Ergot incidence at 14WAP (46.3%) and ergot severity at 15WAP (41%) recorded relatively high NSH, while the NSH estimate of PQ was low. Baker's ratio (BR) ranged between 0 - 58.6%, with ergot incidence at 14WAP (58.6%) and 15WAP (47.6%) and ergot severity at 15WAP (44.1%) had relatively high BR (Table 3).

In 2021 season, mean squares due to GCA Female effects were highly significant (P < 0.01) for ergot incidence and severity at 14WAP, 15WAP and 16WAP, DTF and HSW. The GCA male mean squares were significant for ergot incidence and severity at 14WAP, 15WAP and 16WAP. The SCA mean squares were significant for ergot incidence at 15WAP and 16WAP, DTF and PQ. The heritability estimates in broad sense and narrow sense varied between 12.6 - 90% and 0.9 - 66.6%, respectively. Ergot incidence at 14WAP (66.6%) and 15WAP (55.3%), and ergot severity at 15WAP (64.0%) and 16WAP (55.4%) recorded high NSH estimates. The BSH estimates were high for ergot incidence at 14WAP (76.2%), 15WAP (83.3%) and 16WAP (90.0%), and ergot severity at 15WAP (74.1%) and 16WAP (78.5%) as well as DTF (72.8%). Baker's ratio ranged between 1.55 - 87.5%. The BR estimates were high for incidence at 14WAP (66.3%) and ergot severity at 14WAP (61.9%), 15WAP (86.7%) and 16WAP (70.6%) (Table 3).

Year		E	rgot inciden	ce		Ergot severity	/	DTF	PQ	
	SOV	14WAP	15WAP	16WAP	14WAP	15WAP	16WAP			HSW
	Rep	1.83 ^{ns}	303.7 ^{ns}	106 ^{ns}	0.001 ns	0.0003 ^{ns}	0.11 ns	66.0 ^{ns}	1.05**	0.03 ^{ns}
	Block/Rep	40.6 ^{ns}	147.7 ^{ns}	310**	0.178 ^{ns}	0.18**	0.16 ^{ns}	44.79*	0.141 ^{ns}	0.13 ^{ns}
	GCAFemale	297**	897**	1252**	0.50**	0.79**	1.13**	59.59*	0.109 ^{ns}	0.27 ^{ns}
	GCA Male	151**	841**	900**	0.42**	1.19**	1.06**	62.7**	0.122 ^{ns}	0.14 ^{ns}
	SCA	71.7**	307*	485**	0.31**	0.32**	0.41**	33.43*	0.142 ^{ns}	0.24 ^{ns}
2020	Residual	25.8	120.8	63.45	0.095	0.04	0.06	16.42	0.096	0.184
	σ²GCA F	19.6	31.19	41.55	0.015	0.02	0.04	1.18	0	0.004
	σ ² GCAMale	8.88	66.56	52.41	0.013	0.10	0.08	2.79	0	0
	σ²SCA	20.09	107.48	242.9	0.094	0.15	0.19	12.88	0.029	0.022
	BR (%)	58.6	47.6	27.9	23.0	44.1	38.3	23.54	0	16.3
	NSH (%)	46.3	36.8	25.5	16.5	41.0	34.8	15.82	0	3.62
	BSH (%)	79.0	77.3	91.4	72.0	93.1	90.9	67.23	37.3	22.3
	Rep	771*	53.1 ^{ns}	56.9 ^{ns}	0.001 ^{ns}	0.01 ^{ns}	0.24 ^{ns}	7.56 ^{ns}	0.10 ^{ns}	0.69 ^{ns}
	Block/Rep	611**	322**	312**	0.51*	0.24 ^{ns}	0.25*	26.1 ^{ns}	0.40 ^{ns}	0.25 ^{ns}
	GCAFemale	1983**	1935**	1589**	1.00**	1.24**	1.16**	85.6**	0.50 ^{ns}	1.1**
2021	GCA Male	485**	433**	600**	0.84**	0.41**	0.53**	12.9 ^{ns}	0.46 ^{ns}	0.13 ^{ns}
2021	SCA	190 ^{ns}	262**	347**	0.34 ^{ns}	0.13 ^{ns}	0.15 ^{ns}	43.8**	0.43*	0.34 ^{ns}
	Residual	124.8	96.86	62.99	0.20	0.098	0.089	13.31	0.196	0.212
	σ²GCA F	133.8	136.67	88.5	0.038	0.089	0.072	3.390	0	0.059
	σ ² GCAMale	40.87	24.08	32.70	0.066	0.034	0.043	0	0.002	0
	σ²SCA	25.05	81.68	162.9	0.064	0.019	0.048	14.44	0.122	0.058
	BR (%)	87.5	66.3	42.7	61.9	86.7	70.6	19.01	1.55	50.35
	NSH (%)	66.6	55.3	38.4	39.1	64.2	55.4	13.85	0.87	26.35
	BSH (%)	76.2	83.3	90.0	63.1	74.1	78.5	72.82	55.78	52.34

Table 3. Mean squares of GCA and SCA effects for ergot incidence, severity and other traits

*, ** = Significant at P <0.05 and P <0.01, respectively. Abbreviations: ns – non-significant, SOV- source of variations, WAP – weeks after planting, DTF - days to 50% flowering, PQ - pollen quantity, HSW – hundred seed weight, BR – bakers' ratio, NSH – narrow sense heritability and BSH – broad sense heritability

Variance components for combined analysis

In the combined analysis of variances, mean squares due to GCA female were significant for ergot incidence and severity at 14WAP, 15WAP and 16WAP, HSW, PQ and DTF. Mean squares due to GCA male were significant for ergot incidence and severity at 14WAP, 15WAP and 16WAP and DTF. The SCA mean squares were highly significant for ergot incidence and severity at 14WAP, 15WAP and 16WAP and DTF. The SCA mean squares were highly significant for ergot incidence and severity at 14WAP, 15WAP and 16WAP, DTF and PQ. Mean squares due to Year by GCA Female interaction were significant for ergot incidence at 14WAP, 15WAP and 16WAP. Mean squares due to Year by GCA male were significant for PQ and ergot incidence and severity 14WAP. The mean squares due to Year by SCA were significant for ergot severity at 14WAP. Heritability estimates in broad sense ranged between 27.2% and 90.3% for all recorded traits, whereas in narrow sense, heritability estimates varied from 1.7% to 49.6%. Ergot severity at 15WAP

(49.6%) and 16WAP (44.5%) had the highest heritability estimates in narrow sense, while PQ (1.7%) recorded the least heritability estimate. High BSH estimates were recorded by ergot incidence at 16WAP (90.3%), ergot severity at 15WAP (86.9%) and 16WAP (85.9%) and DTF (80.1%). Baker's ratio ranged between 4.26% and 90.8%, while HSW (90.8%) recorded the highest GCA to SCA ratio (Table 4).

	Erg	got incidence	e	1	Ergot severit	ty			
SOV	14WAP	15WAP	16WAP	14WAP	15WAP	16WAP	HSW	DTF	PQ
Year	15303**	2616**	2931**	18.3**	2.32**	1.02**	7.5**	532**	532.2**
Rep/Year	386**	178.4 ^{ns}	81.55 ^{ns}	0.001 ns	0.003 ^{ns}	0.17 ^{ns}	0.36 ^{ns}	36.8 ^{ns}	36.8 ^{ns}
Block/Rep/Year	295**	139.9 ^{ns}	60.67 ^{ns}	0.27 ^{ns}	0.05 ns	0.041 ns	0.08 ^{ns}	4.56 ^{ns}	0.211 ns
GCA Female	1529**	2389**	2631**	1.41**	1.94**	2.19**	1.1**	132**	0.42*
GCA Male	260**	1185**	1449**	0.75**	1.48**	1.48**	0.09 ^{ns}	53.1**	0.19 ^{ns}
SCA	185**	507**	884**	0.41**	0.42**	0.54**	0.34 ^{ns}	79.1**	0.42**
Year X GCA F	751**	442**	211*	0.10 ^{ns}	0.10 ^{ns}	0.09 ^{ns}	0.29 ^{ns}	12.2 ^{ns}	0.198 ^{ns}
Year X GCAM	375**	88.97 ^{ns}	51.85 ^{ns}	0.51**	0.13 ^{ns}	0.11 ^{ns}	0.18 ^{ns}	22.6 ^{ns}	0.39*
Year X SCA	94.6 ^{ns}	116.8 ^{ns}	92.1 ns	0.29*	0.11 ^{ns}	0.12 ^{ns}	0.29 ^{ns}	15.8 ^{ns}	0.19 ^{ns}
Residual	75.3	131.30	66.99	0.15	0.07	0.08	0.20	14.35	0.143
σ ² GCA Female	30.76	48.55	51.70	0.039	0.051	0.056	0.029	2.228	0.002
σ^2 GCA Male	0	44.04	43.00	0.007	0.061	0.058	0	0	0
σ²SCA	16.54	92.12	198.96	0.038	0.084	0.106	0.003	14.740	0.044
σ ² Year X GCA F	47.80	34.05	10.37	0.00	0.001	0	0	0	0
σ ² YearXσ ² GCAM	28.9	0	0	0.03	0.004	0.002	0	0.637	0.009
σ^{2} Year X SCA	5.90	0	4.45	0.04	0.004	0.016	0.037	0.000	0.024
BR (%)	65.0	50.1	32.2	54.6	57.1	51.9	90.8	13.1	4.26
NSH (%)	20.7	36.8	29.1	24.7	49.6	44.5	24.7	10.5	1.7
BSH (%)	31.8	73.4	90.3	45.2	86.9	85.9	27.2	80.1	40.1

 Table 4. Mean squares of GCA and SCA effects for ergot incidence, severity and other traits combined across years

*, ** = Significant at P <0.05 and P <0.01, respectively. abbreviations: ns – non-significant, SOV – source of variations, WAP – weeks after planting, DTF - days to 50% flowering, PQ - pollen quantity, HSW – hundred seed weight

General combining ability (GCA) effects

Higher GCA effects for ergot severity and incidence were observed by genotypes NAROSORG2, PI651496, PI655972, NAROSORG3, PI656056 and SQR in 2020. In 2021, higher GCA effects for ergot severity and incidence were observed by PI534124, SQR, PI655972, PI651496, NAROSORG2 and NAROSORG3. Genotypes, PI655972 (male), PI651496 (male) and SQR (female) showed consistently higher GCA effects in both 2020 and 2021 experiments (Table 5).

Across years, four, seven and six out twelve parents showed negative GCA effects at 14WAP, 15WAP and 16WAP, respectively for ergot incidence. Seven, eight and eight out of twelve parents showed negative GCA effects for ergot severity scored at 14WAP, 15WAP and 16WAP, respectively. Genotypes PI655972, SQR, PI651496 and NAROSORG2 recorded higher negative GCA effects across years (Table 6).

			20	20					202	1		
	I	Ergot incident	ce	1	Ergot severit	y]	Ergot incidence	e	Ergot severity		
Parents	14W AP	15W AP	16W AP	14W AP	15W AP	16W AP	14W AP	15W AP	16W AP	14W AP	15W AP	16W AP
PI656056	-2.68 ns	-0.81 ns	-0.10 ^{ns}	0.03 ns	-0.06 ns	-0.11 ns	0.48 ^{ns}	0.58 ^{ns}	2.75 ns	-0.05 ns	-0.08 ns	-0.02 ns
PI533785	6.1*	5.33 ns	5.83 ^{ns}	0.11 ^{ns}	0.14 ^{ns}	0.25 ns	17.1*	17.7*	12.8*	0.25 ns	0.5**	0.43*
PI534124	1.80 ^{ns}	1.44 ^{ns}	1.87 ^{ns}	-0.06 ^{ns}	-0.04 ns	-0.03 ns	-13.2*	-10.14 ^{ns}	-4.21 ns	-0.14 ^{ns}	-0.20 ^{ns}	-0.14 ns
SQR	-2.22 ns	-5.98 ns	-6.49 ^{ns}	0.01 ns	-0.07 ^{ns}	-0.09 ^{ns}	-3.64 ^{ns}	-7.60 ^{ns}	-7.79 ^{ns}	-0.00 ns	-0.12 ^{ns}	-0.13 ns
SRN39	-2.98 ns	0.02 ^{ns}	-1.10 ^{ns}	-0.08 ns	0.03 ns	-0.02 ns	-0.74 ^{ns}	-0.51 ns	-3.54 ^{ns}	-0.06 ns	-0.10 ^{ns}	-0.14 ns
PI597971	-2.14 ^{ns}	6.68 ^{ns}	5.32 ^{ns}	-0.06 ns	0.21 ^{ns}	0.15 ^{ns}	7.46 ^{ns}	3.91 ns	3.98 ^{ns}	0.28 ^{ns}	0.16 ^{ns}	0.22 ^{ns}
PI651496	-0.56 ns	-3.73 ns	-3.39 ns	-0.01 ns	-0.27 ns	-0.15 ^{ns}	-4.08 ^{ns}	-0.18 ^{ns}	-0.83 ns	-0.17 ^{ns}	-0.14 ^{ns}	-0.16 ^{ns}
PI655972	-0.36 ns	-9.61 ns	-7.52 ns	-0.02 ns	-0.15 ^{ns}	-0.16 ^{ns}	-5.21 ^{ns}	-5.67 ns	-7.01 ns	-0.11 ^{ns}	-0.09 ^{ns}	-0.11 ns
NAROSORG2	-2.04 ns	-2.38 ns	-2.75 ns	-0.06 ns	-0.20 ^{ns}	-0.18 ^{ns}	5.21 ^{ns}	-1.45 ns	-2.15 ^{ns}	-0.02 ^{ns}	-0.13 ^{ns}	-0.19 ^{ns}
Seso3	2.77 ^{ns}	8.07 ^{ns}	5.43 ^{ns}	0.04 ^{ns}	0.19 ^{ns}	0.17 ^{ns}	0.52 ^{ns}	3.27 ^{ns}	3.72 ^{ns}	0.05 ^{ns}	0.12 ^{ns}	0.12 ^{ns}
NAROSORG3	-0.95 ^{ns}	-3.62 ns	-0.53 ns	-0.01 ns	-0.23 ns	-0.18 ns	-5.52 ^{ns}	-1.56 ns	0.53 ^{ns}	-0.27 ns	-0.10 ^{ns}	-0.07 ns
Sekedo	3.28 ns	4.59 ^{ns}	3.44 ^{ns}	0.12 ^{ns}	0.46 ^{ns}	0.35 ns	1.62 ^{ns}	1.67 ^{ns}	1.76 ns	0.24 ^{ns}	0.18 ^{ns}	0.18 ^{ns}

Table 5. Estimates of GCA effects of parents for ergot incidence and severity

*, ** = Significant at P <0.05 and P <0.01, respectively, abbreviations: WAP – weeks after planting and ns – non-significant.

Table 6. Estimates of GCA effects of parents for ergot incidence and severity combined across years

	E	rgot incidenc	ce		Ergot severity	
Parents	14WAP	15WAP	16WAP	14WAP	15WAP	16WAP
PI656056	-0.69 ^{ns}	0.11 ^{ns}	1.09 ^{ns}	-0.01 ^{ns}	-0.08 ^{ns}	-0.08 ns
PI533785	6.83 ^{ns}	9.42 ^{ns}	8.13 ^{ns}	0.28 ^{ns}	0.34*	0.36*
PI534124	-3.06 ns	-2.81 ns	-0.51 ^{ns}	-0.15 ^{ns}	-0.13 ^{ns}	-0.08 ns
SQR	-1.69 ^{ns}	-6.10 ^{ns}	-6.69 ^{ns}	0.00 ^{ns}	-0.12 ^{ns}	-0.12 ns
SRN39	-1.39 ^{ns}	-0.62 ns	-2.03 ns	-0.13 ^{ns}	-0.01 ^{ns}	-0.07 ^{ns}
PI597971	-	5.57 ^{ns}	4.76 ^{ns}	0.01 ^{ns}	0.18 ^{ns}	0.19 ^{ns}
PI651496	-	-1.70 ^{ns}	-2.16 ^{ns}	-0.02 ns	-0.20 ^{ns}	-0.16 ^{ns}
PI655972	-	-8.30 ^{ns}	-7.59 ^{ns}	-0.02 ns	-0.11 ^{ns}	-0.13 ^{ns}
NAROSORG2	-	-2.22 ns	-2.66 ^{ns}	-0.02 ns	-0.17 ^{ns}	-0.19 ^{ns}
Seso3	-	5.53 ^{ns}	4.62 ^{ns}	0.02 ^{ns}	0.15 ^{ns}	0.15 ^{ns}
NAROSORG3	-	-2.42 ns	0.23 ^{ns}	-0.03 ns	-0.16 ^{ns}	-0.13 ^{ns}
Sekedo	-	3.54 ^{ns}	2.81 ^{ns}	0.06 ^{ns}	0.31*	0.27 ^{ns}

*, ** = Significant at P <0.05 and P <0.01, respectively, abbreviations: WAP – weeks after planting and ns – non-significant.

Specific combining ability effects

In 2020, Crosses PI534124XPI651496, PI534124XPI655972, PI534124XNAROSORG2, SQRXSeso3 and PI656056XNAROSORG3 showed significant negative SCA effects for ergot incidence. For disease severity, crosses PI534124 X PI655972, PI656056 X Seso3, SQR X PI651496 and PI656056 X Seso3 recorded significant negative SCA effects (Table 7). In 2021, Crosses PI656056 X

PI655972 and PI534124 X PI651496, PI656056 X PI655972 and PI534124 X NAROSORG2 showed significantly negative SCA effects. For DTF, seven and one crosses out of the 32 showed significant positive and negative SCA effects, respectively, while two crosses showed significantly positive SCA effects for PQ. Further, sixteen crosses showed positive SCA effects for hundred seed weight (Table 7).

Across years, the cross PI534124 X NAROSORG2 had significant negative SCA effects at 15WAP, whereas crosses including PI534124 X PI651496, PI656056 X PI655972, PI534124 X NAROSORG2 and PI656056 X NAROSORG3 showed significant negative SCA effects at 16WAP (Table 8). Crosses including PI534124 X NAROSORG2, PI534124 X PI651496, PI656056 X PI655972, PI656056 X NAROSORG3, PI534124 X PI655972, SQR X NAROSORG3 and SQR X PI651496 had generally the highest negative SCA effects for ergot incidence. For ergot severity, SQRXPI651496, SQRXPI651496 and PI656056XSeso3 crosses showed significant negative SCA effects. Crosses including SQR X PI651496, PI656056 X Seso3, PI534124 X NAROSORG2, SRN39 X PI655972 and SRN39 X NAROSORG2 generally showed the highest negative SCA effects for ergot severity (Table 8). For hundred seed weight, PI656056 X NAROSORG3, SRN39 X NAROSORG3, PI534124 X PI655972, PI533785 X Seso3 and PI656056 X NAROSORG2 had the highest SCA effects. Only one cross (PI534124 X PI651496) showed significantly positive SCA effects for PQ.

			20	020					2	021		
Crosses		Ergot incidenc	e		Ergot severity	r		Ergot inciden	ce	Ergot seve	rity	
	14WAP	15WAP	16WAP	14WAP	15WAP	16WAP	14WAP	15WAP	16WAP	14WAP	15WAP	16WAP
PI656056 X PI597971	1.08 ^{ns}	8.95 ^{ns}	19.6*	-0.16 ^{ns}	0.15 ^{ns}	0.30 ^{ns}	1.90 ns	4.37 ^{ns}	14.0*	0.19 ^{ns}	0.035 ^{ns}	0.25 ^{ns}
PI533785 X PI597971	-4.71 ns	-5.66 ^{ns}	-7.80 ^{ns}	-0.13 ^{ns}	0.38 ^{ns}	0.20 ^{ns}	-0.76 ^{ns}	4.23 ^{ms}	6.2 ^{ns}	0.17 ^{ns}	0.086 ^{ns}	0.13 ^{ns}
PI534124 X PI597971	-0.72 ns	0.51 ns	0.90 ^{ns}	0.05 ^{ns}	-0.08 ^{ns}	-0.2 ^{ns}	-1.60 ns	-5.71 ^{ns}	-12.1 ns	-0.01 ns	-0.01 ^{ns}	-0.06 ns
SQR X PI597971	-0.37 ns	6.99 ^{ns}	12.0 ^{ns}	-0.15 ^{ns}	-0.15 ^{ns}	0.07 ^{ns}	5.03 ns	10.5 ^{ns}	11.1 ^{ns}	-0.09 ns	-0.031 ns	-0.08 ns
PI656056 X PI651496	1.08 ^{ns}	8.70 ^{ns}	14.5 ns	0.19 ^{ns}	0.23 ^{ns}	0.46 ^{ns}	0.94 ^{ns}	2.78 ^{ns}	9.5 ^{ns}	-0.05 ^{ns}	-0.009 ^{ns}	0.07 ^{ns}
PI534124 X PI651496	-1.28 ns	-9.04 ns	-16.2*	0.25 ^{ns}	-0.25 ^{ns}	-0.15 ns	-0.38 ns	-7.05 ^{ns}	-15.3*	0.08 ns	0.001 ^{ns}	-0.07 ns
SQR X PI651496	-0.43 ns	-6.45 ^{ns}	-12.2 ^{ns}	-0.25 ns	-0.42 ns	-0.53*	-2.75 ns	-6.26 ^{ns}	-7.6 ^{ns}	-0.10 ^{ns}	-0.090 ^{ns}	-0.23 ns
SRN39 X PI651496	-0.61 ^{ns}	0.77 ^{ns}	-1.70 ^{ns}	-0.27 ^{ns}	0.05 ^{ns}	-0.15 ^{ns}	-0.31 ns	9.91 ^{ns}	9.4 ^{ns}	-0.08 ^{ns}	0.03 ^{ns}	0.05 ^{ns}
PI656056 X PI655972	-1.17 ^{ns}	-5.97 ^{ns}	-13.3 ns	-0.11 ^{ns}	-0.12 ^{ns}	-0.18 ns	-3.60 ns	-13.62*	-17.9*	-0.14 ^{ns}	-0.07 ^{ns}	-0.20 ns
PI534124 X PI655972	-1.27 ns	-8.28 ns	-15.7*	-0.43*	-0.35 ^{ns}	-0.47 ns	1.85 ns	-0.13 ^{ns}	-7.2 ^{ns}	0.11 ns	0.05 ns	0.05 ^{ns}
SQRXPI655972	2.41 ms	1.49 ^{ns}	-0.6 ^{ns}	0.28 ^{ns}	0.45*	0.55*	-2.35 ^{ns}	-4.49 ^{ns}	-6.0 ^{ns}	0.01 ^{ns}	0.04 ns	0.21 ^{ns}
SRN39 X PI655972	-0.77 ^{ns}	-2.76 ^{ns}	-5.2 ^{ns}	0.11 ^{ns}	-0.20 ^{ns}	-0.30 ^{ns}	0.92 ^{ns}	-1.12 ^{ns}	-2.8 ^{ns}	-0.07 ^{ns}	-0.07 ^{ns}	-0.18 ^{ns}
PI656056 X NAROSORG2	0.52 ^{ms}	6.35 ^{ns}	8.70 ^m	-0.08 ^{ns}	0.29 ^{ns}	0.16 ^{ns}	2.31 ns	4.57 ^{ns}	7.4 ^{ns}	0.06 ^{ns}	0.05 ns	0.07 ^{ns}
PI533785 X NAROSORG2	-3.62 ns	-1.52 ns	-0.6 ¹¹⁵	-0.13 ns	-0.01 ^{ns}	0.17 ns	-1.60 ns	-4.38 ^{ns}	-5.5 ^{ns}	0.24 ns	-0.06 ^{ns}	-0.10 ^{ns}
PI534124 X NAROSORG2	-1.51 ^{ns}	-9.39 ^{ns}	-20**	-0.11 ^{ns}	-0.27 ^{ns}	-0.34 ^{ns}	-2.62 ^{ns}	-9.26 ^{ns}	-18.5**	-0.19 ^{ns}	-0.07 ^{ns}	-0.21 ns
SQR X NAROSORG2	0.00 ^{ns}	0.45 ns	-0.90 ^{ns}	0.15 ^{ns}	0.07 ^{ns}	-0.08 ns	2.43 ns	3.11 ^{ms}	4.8 ^{ns}	0.05 ^{ns}	0.06 ^{ns}	0.13 ^{ns}

Table 7. Estimates of SCA effects of crosses for ergot incidence and severity

SRN39 X NAROSORG2	0.09 ns	0.27 ^{ns}	0.80 ^{ns}	-0.25 ns	-0.36 ^{ns}	-0.35 ns	2.67 ns	1.02 ns	1.4 ns	-0.18 ^{ns}	-0.04 ns	-0.10 ^{ns}
PI656056 X Seso3	-0.32 ns	-0.58 ^{ms}	2.40 ^m	0.01 ^{ns}	-0.50*	-0.58*	-0.37 ^{ms}	9.08 ^{ns}	3.8 ^{ns}	-0.07 ^{ns}	-0.05 ^{ns}	-0.18 ^{ns}
PI533785 X Seso3	6.37 ^{ms}	16.1*	24.3**	0.65**	0.87**	0.84**	5.34 ^{ns}	13.4*	16.7*	0.05 ^{ns}	0.09 ^{ns}	0.21 ^{ns}
PI534124 X Seso3	5.54 ^{ns}	7.6 ^{ns}	11.2 ^{ns}	-0.01 ns	0.17 ^{ns}	0.22 ns	-2.10 ^{ns}	-1.24 ns	9.0 ^{ns}	-0.05 ^{ns}	0.05 ns	0.23 ^{ns}
SQR X Seso3	-2.59 ^{ns}	-11.4 ^{ns}	-15.8*	-0.06 ^{ns}	-0.14 ^{ns}	-0.05 ns	-1.60 ^{ns}	-3.15 ^{ns}	-2.4 ns	0.05 ^{ns}	-0.005 ^{ns}	-0.02 ^{ns}
SRN39 X Seso3	-2.89 ns	1.35 ^{ns}	3.0 ^{ns}	-0.31 ns	-0.13 ^{ns}	0.00 ^{ns}	-0.95 ^{ns}	-6.9 ns	-9.2 ns	0.06 ^{ns}	-0.05 ns	-0.10 ^{ns}
PI656056 X NAROSORG3	-1.30 ns	-12.3 ^{ns}	-23**	-0.04 ^{ns}	-0.26 ^{ns}	-0.38 ns	-2.44 ns	-5.41 ^{ns}	-7.2 ns	-0.07 ^{ns}	0.03 ^{ns}	0.01 ^{ns}
PI533785 X NAROSORG3	-2.62 ns	-0.40 ^{ns}	2.3 ns	0.22 ^{ns}	0.03 ^{ns}	0.17 ^{ns}	0.11 ^{ns}	-4.53 ns	-2.4	0.13 ^{ns}	-0.011 ^{ns}	0.04 ^{ns}
PI534124 X NAROSORG3	1.06 ^{ns}	10.71 ^{ns}	31.8**	-0.29 ^{ns}	-0.14 ^{ns}	0.09 ^{ns}	-0.17 ^{ns}	9.15 ^{ns}	26.7**	-0.33 ^{ns}	-0.09 ^{ns}	-0.11 ns
SQR X NAROSORG3	-1.13 ns	-4.09 ^{ns}	-10.8 ^{ns}	-0.03 ns	-0.12 ^{ns}	-0.29 ns	-0.65 ns	-4.06 ns	-10.4 ^{ns}	-0.02 ^{ns}	0.0 ^{ns}	-0.05 ns
SRN39 X NAROSORG3	1.89 ^{ns}	0.23 ^{ns}	-2.7 ^{ns}	0.10 ^{ns}	0.15 ^{ns}	-0.02 ns	-0.22 ^{ns}	-0.46 ^{ns}	-4.1 ns	0.05 ^{ns}	0.029 ^{ns}	0.03 ^{ns}
PI656056 X Sekedo	-2.57 ^{ns}	-7.97 ^{ns}	-9.4 ^{ns}	0.34 ^{ns}	-0.27 ^{ns}	-0.30 ^{ns}	1.36 ^{ns}	-1.41 ^{ns}	-4.7 ns	0.0 ^{ns}	0.0 ^{ns}	-0.04 ns
PI533785 X Sekedo	10.7**	9.90 ^{ns}	15.9*	0.07 ns	-0.14 ^{ns}	-0.18 ns	0.12 ^{ns}	2.08 ms	8.4 ^{ns}	-0.20 ^{ns}	-0.033 ^{ns}	0.00 ^{ns}
PI534124 X Sekedo	-0.03 ns	12.9*	19.7*	0.17 ns	0.57**	0.71**	2.56 ns	8.07 ^{ns}	9.8 ^{ns}	0.19 ^{ns}	0.033 ^{ns}	0.09 **
SQR X Sekedo	-0.13 ^{ns}	-7.60 ¹¹⁵	-9.6 ^{ns}	0.12 ^{ns}	-0.26 ^{ns}	-0.10 ^{ns}	-0.79 ^{ns}	-0.25 ^{ns}	-3.7 ^{ns}	0.10 ^{ns}	0.009 ^{ns}	-0.04 ^{ns}
SRN39 X Sekedo	-0.70	0.22	-0.7	0.10	0.73**	0.71**	-2.25 ns	-2.78 ns	-1.2 ns	0.13 ^{ns}	0.08 ^{ns}	0.20 ^{ns}

*, ** = Significant at P <0.05 and P <0.01, respectively, abbreviations: WAP – weeks after planting and ns – non-significant.

Table 8. Estimates of	Table 8. Estimates of SCA effects of crosses for ergot incidence and severity combined								
	across years								
	Ergot incidence	Ergot severity							

]	Ergot incidence		E	Ergot severity				
Crosses	14WAP	15WAP	16WAP	14WAP	15WAP	16WAP			
PI656056 X PI597971	1.75 ^{ns}	7.66 ^{ns}	17.8**	0.07 ^{ns}	0.15 ^{ns}	0.35 ^{ns}			
PI533785 X PI597971	-2.24 ^{ns}	-1.29 ^{ns}	-0.77 ^{ns}	0.03 ^{ns}	0.28 ^{ns}	0.17 ^{ns}			
PI534124 X PI597971	-1.4 ^{ns}	-5.03 ^{ns}	-7.52 ^{ns}	0.04 ^{ns}	-0.06 ^{ns}	-0.15 ns			
SQR X PI597971	4.24 ^{ns}	11.13 ^{ns}	12.5 ns	-0.07 ns	-0.12 ^{ns}	-0.02 ns			
PI656056 X PI651496	1.04 ^{ns}	8.00 ^{ns}	13.7*	0.03 ^{ns}	0.11 ns	0.28 ^{ns}			
PI534124 X PI651496	-0.67 ^{ns}	-10.11 ns	-17.0*	0.13 ^{ns}	-0.09 ns	-0.11 ns			
SQR X PI651496	-2.46 ^{ns}	-7.26 ^{ns}	-10.6 ^{ns}	-0.14 ^{ns}	-0.35*	-0.40*			
SRN39 X PI651496	-0.68 ns	5.56 ^{ns}	3.96 ^{ns}	-0.12 ^{ns}	0.06 ^{ns}	-0.02 ns			
PI656056 X PI655972	-3.03 ns	-10.38 ns	-16.1*	-0.11 ^{ns}	-0.16 ^{ns}	-0.24 ns			
PI534124 X PI655972	0.77 ^{ns}	-5.58 ^{ns}	-12.9 ns	-0.07 ^{ns}	-0.07 ns	-0.19 ^{ns}			
SQR X PI655972	-1.15 ^{ns}	-1.05 ns	-2.78 ns	0.08 ^{ns}	0.32 ^{ns}	0.4*			
SRN39 X PI655972	0.39 ^{ns}	-1.57 ^{ns}	-3.29 ns	0.02 ^{ns}	-0.24 ^{ns}	-0.26 ns			
PI656056 X NAROSORG2	2.45 ^{ns}	7.61 ^{ns}	9.73 ^{ns}	0.02 ^{ns}	0.24 ^{ns}	0.16 ^{ns}			
PI533785 X NAROSORG2	-2.99 ^{ns}	-4.32 ns	-3.04 ^{ns}	0.07 ^{ns}	-0.10 ^{ns}	0.00 ^{ns}			
PI534124 X NAROSORG2	-2.50 ns	-12.1*	-21**	-0.12 ^{ns}	-0.27 ^{ns}	-0.34 ns			
SQR X NAROSORG2	2.28 ns	3.73 ^{ns}	2.54 ^{ns}	0.09 ^{ns}	0.17 ^{ns}	0.08 ^{ns}			
SRN39 X NAROSORG2	2.15 ns	0.06 ^{ns}	0.01 ns	-0.15 ns	-0.27 ^{ns}	-0.25 ns			

		1				
PI656056 X Seso3	-0.29 ns	3.95 ^{ns}	2.12 ns	-0.05 ^{ns}	-0.33 ns	-0.40*
PI533785 X Seso3	6.32 ^{ns}	17.4**	21.2**	0.19 ^{ns}	0.5**	0.50**
PI534124 X Seso3	0.03 ns	3.16 ^{ns}	10.7 ^{ns}	-0.02 ns	0.17 ^{ns}	0.28 ^{ns}
SQR X Seso3	-2.33 ns	-8.73 ns	-9.48 ns	0.00 ^{ns}	-0.04 ^{ns}	-0.04 ns
SRN39 X Seso3	-1.63 ns	-3.40 ns	-3.23 ns	-0.04 ^{ns}	-0.13 ns	-0.08 ns
PI656056 X NAROSORG3	-2.49 ^{ns}	-10.3 ns	-15.3*	-0.06 ns	-0.02 ns	-0.14 ns
PI533785 X NAROSORG3	-1.05 ns	-2.27 ns	0.87 ^{ns}	0.10 ^{ns}	-0.01 ns	0.10 ^{ns}
PI534124 X NAROSORG3	0.14 ^{ns}	11.45 ns	30.7**	-0.26 ns	-0.22 ns	-0.04 ns
SQR X NAROSORG3	-0.83 ns	-3.64 ns	-10.7 ns	-0.03 ns	-0.04 ns	-0.16 ^{ns}
SRN39 X NAROSORG3	0.83 ns	-0.70 ns	-4.58 ns	0.06 ns	0.07 ns	0.00 ^{ns}
PI656056 X Sekedo	0.19 ^{ns}	-6.38 ns	-7.68 ns	0.09 ns	-0.11 ns	-0.16 ^{ns}
PI533785 X Sekedo	3.65 ns	7.28 ^{ns}	13.1 ^{ns}	-0.14 ^{ns}	-0.15 ns	-0.12 ns
PI534124 X Sekedo	1.97 ^{ns}	13.2*	15.6*	0.17 ^{ns}	0.33 ^{ns}	0.39*
SQR X Sekedo	-0.66 ns	-5.07 ^{ns}	-7.26 ns	0.07 ^{ns}	-0.13 ns	-0.09 ns
SRN39 X Sekedo	-1.82 ns	-1.06 ns	-0.70 ns	0.12 ns	0.48 ^{ns}	0.48*

*, ** = Significant at P <0.05 and P <0.01, respectively, abbreviations: WAP – weeks after planting and ns – non-significant.

Association among traits

The results of Pearson's correlation analysis for ergot disease resistant traits final evaluation score, HSW, DTF, and other parameters for F2 segregating populations evaluated at MUARIK during 2020 and 2021 is presented in Table 9. Highly significant (P<0.01) positive correlation (r=0.69) was observed between ergot disease severity and incidence. Ergot disease incidence was significantly correlated with DTF (r=0.66) and SV (r=0.62). Significantly negative correlation was observed between ergot incidence and PQ (r=-0.35). Ergot severity was significantly correlated SV (r=0.37) (Table 9).

Table 9. Pearson's correlation among resistance to ergot traits evaluated during 2020 and 2021

Traits	Ergot severity	Ergot incidence	Hundred seed weight	Seedling vigour	Days to 50% flowering
Ergot severity	-				
Ergot incidence	0.69**	-			
Hundred seed weight	0.18 ^{ns}	0.06 ^{ns}	-		
Seedling vigour	0.37*	0.62**	-0.22 ^{ns}	-	
Days to 50% flowering	0.24 ^{ns}	0.66**	-0.21 ^{ns}	0.70**	-
Pollen quantity	-0.21 ns	-0.35*	0.16 ^{ns}	-0.62**	-0.52**

*, ** = Significant at P <0.05 and P <0.01, respectively, ns – non-significant.

4. Discussion

Evaluation of these genotypes for ergot resistance under natural infestation conditions for two consecutive years at MUARIK revealed highly significant difference among genotypes indicating the existence of genetic variability in response to ergot infection. Similar study by Montes-García et al. (2008) reported variability in susceptibility to ergot among crosses. Highly significant variations were observed between years and genotype by years interaction for ergot incidence and severity indicating that the evaluated genotypes performed differently across environments. Montes-García et al. (2008) indicated that the susceptibility of crosses was significantly affected by years and locations. High disease incidence (44.6%) and severity (3.0) were recorded in 2021 compared to incidence (31.2%) and severity (2.8) in 2020, suggesting environmental variations had resulted in differences in ergot disease pressure across years. The importance of environmental variability in ergot infection was reported by Parh et al. (2008) and Mikaliunaite and Dankevicius (2009). Some parents such as NAROSORG2, PI651496, PI655972 and crosses PI534124 X NAROSORG2, SQR X PI651496 and PI656056 X Seso3 had relatively lower infection rate in 2020 and 2021 but parents such as PI533785, PI656056 and Sekedo and crosses including PI533785 X Seso3, PI533785 X PI597971, PI656056 X PI597971 and PI533785 X Sekedo showed susceptible response to ergot infection across years. The consistent resistance response of these genotypes and crosses over years indicates that these genotypes can be used as a potential source of resistance genes for resistance to ergot breeding.

The variance component analysis revealed highly significant GCA and SCA effects for ergot incidence and ergot severity suggesting the importance of additive and nonadditive gene effects in determining the inheritance of resistance to ergot. Moran et al. (2000) reported that both additive and non-additive components played an important role in determining ergot vulnerability, but GDRC (2015) and Mardiata et al. (2008) reported that inheritance of ergot resistance traits were determined by simple additive dominance model. Bokmeyer and Meyer (2009) reported that negative GCA and SCA effects are desirable for disease resistance, based on a scale where the highest value corresponds to more disease attack. In this study, parents including NAROSORG2, PI651496, PI655972 showed negative GCA effects suggesting that these parents could be used for improving ergot resistance. The traits that exhibited negative significant GCA effects can be improved through selection and the parents will be expected to additively contribute their favorable alleles to develop better performing offspring. On the other hand, parents including PI533785, PI597971, Sekedo, Seso3 had relatively higher positive GCA effects, indicating these genotypes would not be good to improve ergot resistance. Pollen quantity and days to 50% flowering had lower GCA to SCA ratios suggesting that nonadditive gene effects were dominant and improvement these traits will only be effective after selection in the advanced generations.

The narrow sense heritability values were low for all traits in 2020 and across years, indicating the low genetic contribution towards the phenotypic variances, which was attributed to the high significant environment and genotype by environment interactions and suggested that inheritance resistance to ergot traits were quantitatively inherited. Reed et al. (2002) reported that resistance to ergot was not stable across the test environments and McLaren and Flett (1998) reported that ergot infection frequency and pollen traits were influenced by genotype by environment interactions.

There was positive correlation between ergo ergot incidence and severity with days to 50% flowering suggesting that delayed flowering increased ergot infection. Significantly negative association was observed between ergot resistance traits and pollen quantity, suggesting pollen quantity were important traits in ergot infection. The importance of pollen production and flowering period in ergot infection was reported by Dahlberg et al. (2001).

5. Conclusion

In conclusion, significant GCA and SCA were detected suggesting both additive and non – additive gene effects were important for inheritance of resistance to ergot. Parents and crosses with significant negative GCA and SCA were identified that could be used for improving resistance to ergot.

References

- Awori, E., Kiryowa, M., Basirika, A., Dradiku, F., Kahunza, R., Oriba, A., Edonia, C., Olupot, R., and Mukalazi, J. (2015). Performance of elite grain sorghum varieties in the West Nile Agro-ecological Zones. Uganda Journal of Agricultural Science, 16(1), 139–148.
- Baker, R.J. (1978). Issues in diallel analysis. Crop Science 18, 533-536.
- Bokmeyer, J.M., Bonos, S.A., & Meyer, W.A. (2009). Inheritance characteristics of brown patch resistance in tall fescue. *Crop Science*, 49, 2302–2308.
- Dahlberg, J.A., Bandyopadhyay, R., Rooney, W.L., Odvody, G.N., & Madera-Torres, P. (2001). Evaluation of sorghum germplasm used in US breeding programmes for sources of sugary disease resistance. *Plant Pathology*, 50(6), 681–689. https://doi.org/10.1046/j.1365-3059.2001.00636.x
- GRDC. (2015). Inheritance and mechanisms of resistance of sorghum bicolor to sorghum ergot, caused by Claviceps africana.
- GRDC. (2017). Sorghum diseases sorghum ergot (claviceps africana) | fusarium stalk rot (fusarium spp.) | charcoal rot (macrophomina phaseolina) | rust (puccinia purpurea) | johnson grass mosaic virus | head smut (sporisorium reilianum) | leaf blight (exserohilum turcic.
- McLaren, N.W., Flett, B.C. (1998). Use of weather variables to quantify sorghum ergot potential in South Africa. *Plant Disease*, 82(1), 26–29. https://doi.org/10.1094/PDIS.1998.82.1.26
- Miedaner, T., Geiger, H.H. (2015). Biology, Genetics, and Management of Ergot (Claviceps spp.) in Rye, Sorghum, and Pearl Millet. *Toxins*, *7*, 659–678. https://doi.org/10.3390/toxins7030659
- Miesho, W.B., Gebremedhin, H., Msiska, U., Bruno, A., Malinga, G.M., Ongom, P.O., Richard, E., Gibson, P., Rubaihayo, P., & Kyamanywa, S. (2019). Identification of candidate genes associated with resistance to bruchid (Callosobruchus maculatus) in cowpea. *Plant Breeding*, 1–9. https://doi.org/10.1111/pbr.12705
- Mikaliunaite, R., Dankevicius, Z. (2009). The spread of ergot (Claviceps purpurea) on Poaceae plants and incidence on cereals in Lithuania The spread of ergot (Claviceps purpurea) on Poaceae plants and incidence on cereals in Lithuania. *Zemdirbyste-Agriculture*, *96*(1392–3196), 246–259.
- Mirdita, V., Dhillon, B.S., Geiger, H.H., and Miedaner, T. (2008). Genetic variation for resistance to ergot (Claviceps purpurea [Fr.] Tul.) among full-sib families of five populations of winter rye (Secale cereale L.) Genetic variation for resistance to ergot (Claviceps purpurea [Fr.] Tul.) among full-sib fami. *Theor Appl Genet*, *118*, 85–90. https://doi.org/10.1007/s00122-008-0878-0
- Montes-garcía, N., Williams-alanís, H., Experimental, C., Bravo, R., Postal, A., Matamorosreynosa, C., Bravo, R., Cp, M., Prom, L.K., Road, B., Station, C., Isakeit, T., Station, C., Odvody, G., Christi, C., Narro-sánchez, J., Bajío, C.E., Celaya-, C., Allende, S.M. De, Station, C. (2008). Disease Severity and Susceptibility of Sorghum [Sorghum bicolor (L .) Moench] to Infection by Claviceps africana Frederickson, Mantle and de Milliano in Mexico and the United States of America. *Revista Mexicana de fitopatologia*, 26(2), 121–126.
- Moran, J.L., (2000). Differences in ergot vulnerability among sorghum genotypes and the relationship between stigma receptivity and ergot vulnerability. Master's Thesis, Texas A&M University, College Station.

- Musabyimana, T., Sehene, C., & Randyoppadhyay, R. (1995). Ergot resistance in sorghum in relation to flowering, inoculation technique and development. *Plant Pathology*, 44, 109–115.
- Parh, D.K., Jordan, D.R., Aitken, E.A.B., Gogel, B.J., McIntyre, C.L., & Godwin, I.D. (2006). Genetic components of variance and the role of pollen traits in Sorghum ergot resistance. *Crop Science*, 46(6), 2387–2395. https://doi.org/10.2135/cropsci2005.12.0476
- Parh, D.K., Jordan, D.R., Aitken, E.A.B., Mace, E.S., Jun-ai, P., McIntyre, C.L., and Godwin, I.D. 2008. QTL analysis of ergot resistance in sorghum. *Theor. Appl. Genet.* 117, 369–382. https://doi.org/10.1007/s00122-008-0781-8
- Reed, J.D., Ramundo, B.A., Claflin, L.E., and Tuinstra, M.R. (2002). Analysis of resistance to ergot in sorghum and potential alternate hosts. *Crop Science*, *42*, 1135–1138.
- Rodríguez, F., Alvarado, G., Pachec, A., Burgueño, J., and Crossa, J. (2015). AGD-R (Analysis of Genetic Designs in R). CIMMYT-Knowledge Center@cgiar.org or at Km. 45 Carretera Mexico-Veracruz, El Batán, Texcoco, Estado de México, México, C.P. 56237.
- RStudio Team (2020). RStudio: Integrated Development for R. RStudio, PBC, Boston, MA URL <u>http://www.rstudio.com/</u>.