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Genetics of resistance to Fusarium stalk rot caused by *Fusarium verticilloides* in maize (*Zea mays* L.)

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Abstract

Fusarium stalk rot disease (FSR), incited by *Fusarium verticilloides*, is becoming an important biotic production constraint in many major maize growing areas causing substantial yield losses. The present investigation was conducted to understand the genetics of resistance to FSR through six generation means and variances, as a first step in addressing the problem. Five crosses were developed by crossing four FSR susceptible inbreds (VL1043, VL108867, VL121096 and VL1218) with two resistant inbreds (CM202 and CM212). Six generations of the five crosses (VL1043 × CM212, VL108867 × CM202, VL121096 × CM212, VL1218 × CM202 and VL1218 × CM212) were evaluated through artificial disease inoculation during post rainy season of 2018 and *summer*, 2019. The scaling tests and joint scaling tests indicated the inadequacy of additive-dominance model and showed the presence of epistatic gene effects in all the five crosses for FSR resistance. The study further revealed the importance of additive, dominance and additive × additive gene effects in the expression of FSR. The magnitude and direction of the additive genetic effects [a], dominance genetic effects [d], magnitudes of additive genetic variance (σ^2_A) and dominance genetic variance (σ^2_D) varied with the genetic background of the crosses over seasons. Duplicate gene interaction was evident in the inheritance of FSR resistance. Both, additive and non-additive components were found important thus reciprocal recurrent selection would be more effective in obtaining FSR resistant maize inbred lines.

Key words: Maize, Fusarium stalk rot, generation means, additive-dominance model, duplicate gene interaction

Introduction

Maize (Zea mays L.) is one of the most versatile crop grown throughout the tropical as well as temperate regions of the world providing food, livestock feed and industrial raw materials (Troyer, 2006; Archana et al. 2019). In India, about 65 diseases have been reported to affect the crop (Rahul and Singh, 2002). Among various diseases, Fusarium stalk rot, incited by *Fusarium verticillioides* (Saccardo) Nirenberg (formerly called *Fusarium moniliforme* (Sheldon) (Seifert et al. 2004), is a serious threat to maize cultivation in all continents of the world (CIMMYT, 2004). In India, the disease is prevalent in most of the maize growing areas, where water stress occurs after flowering stage of the crop (Singh et al. 2012). The FSR usually occurs after flowering stage and prior to physiological maturity. It kills plants prematurely and produce light weight ears bearing poorly filled kernels. Plants affected by FSR are easily prone to lodging which makes harvesting difficult, and ears are left in the field while harvesting. Cook (1978) reported that the disease causes a reduction of 18.7% in cob weight and 11.2% in 1000-grain weight in the infected plants. The disease incidence ranges from 10 to 42% (Desai et al. 1991; Kumar et al. 1998; Harlapur et al. 2002) in Karnataka. Very few inbreds with resistance to FSR are available (Archana et al. 2019; Lingaraj et al. 2019) and there is a rapid increase in maize area followed by increased incidence of FSR in Karnataka and other states. However, to breed a genotype with high level of resistance, the knowledge of gene action involved in the expression of trait is important. Earlier studies indicated that resistance to stalk rot is quantitatively inherited and controlled by multiple genes with additive effects (Yang et al. 2004).

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Genetic architecture and mode of inheritance of Fusarium stalk rot could be unravelled using first and/or second degree statistics. First degree statistics-based additive, dominance and their digenic interaction effects are likely to be underestimated owing to mutual cancellation of positive and negative additive effects and ambidirectional dominance effects of genes dispersed between the parents (Jayasekara and Jinks 1976; Jinks 1981). Even the second degree statistics-based gene effects are underestimated when individual gene effects are very low in magnitude (< 1.0). The use of first and second degree statistics helps in discrimination between whether underestimates of gene effects are due to internal cancellation of large/small effect ambidirectional dominant effect genes or due to smaller effects of individual genes (Kearsey and Pooni 1996). Most researchers attempting to unravel the genetics of quantitative traits use either the first or second degree statistics, or very rarely both. Hence the objective of the present study was to decipher the genetic architecture and mode of inheritance of resistance to Fusarium stalk rot in maize based on the six generation mean analysis in five maize populations to initiate breeding program to develop resistant inbreds.

Materials and methods

Plant material

Four susceptible inbreds namely VL1043, VL108867, VL121096 and VL1218 were crossed with two resistant inbreds CM202 and CM212 to produce five crosses (VL1043 × CM212, VL108867 × CM202, VL1218 × CM212, VL1218 × CM202 and VL121096 × CM212) through hand pollination during summer season, 2018. During rainy season of 2018, F_1 plants of all the crosses were raised and selfed to produce F_2 generation as well as backcrossed to corresponding parents to produce BC_1P_1 and BC_1P_2 generations. The six generations (P_1, P_2, F_1, F_2, BC_1P_1 and BC_1P_2) for each of the five maize crosses were tested for the disease response by artificial disease inoculation at the experimental plots of College of Agriculture, V. C. Farm, Mandya, which is situated at 13°57'N latitude and 76°24'E longitude , in Karnataka, India, during post rainy season, 2018 and summer, 2019.

Field layout for disease screening

The six generations of the five crosses were grown in a Randomized Complete Block Design with two replications. However, the means of different generations over replications were used for data analysis as required in the WINDOSTAT software v9.3. The parents, F_1s, F_2s and backcross generations were grown in two, four, fifty and thirty row plots of 2 m length, respectively. The entries were sown in rows spaced 0.60 m apart and with an intra-row spacing of 0.20 m.

Screening for resistance to Fusarium stalk rot

To ensure uniform disease infestation, artificial inoculation was done by following the procedure developed by the Indian Institute of Maize Research (IIMR), New Delhi (2012). Maize stalks showing symptoms typical of FSR were collected from the field. Infected stalks were cut in to small tissue and surface sterilized in 4 *per cent* sodium hydrochloride solution. The same were washed twice in sterile distilled water, dried and plated on Potato Dextrose Agar (PDA) medium. The petri plates were incubated in BOD incubator for five days to facilitate the development of pathogen colonies. The pathogen colonies were examined for morphological and fruiting body characteristics typical of *Fusarium verticillioides*. The mycelia of *Fusarium verticillioides* were placed on Potato Dextrose Agar (PDA) for pure culture. The mycelia were aseptically transferred to sterile Potato Dextrose Broth (PDB) in conical flasks for mass multiplication. These conical flasks were incubated for 15 days for the development of mycelia. On the 15^{th} day, the mycelia was grounded and filtered to obtain pathogen spore suspension and observed under microscope. The concentration was adjusted to 1×10^6 spores/ml using haemocytometer. Whenever spore concentration was high, it was diluted with sterile distilled water to maintain the desired concentration of spores. The inoculum (2 ml) was injected diagonally using the syringe after pricking and making 2 cm hole with the help of a jabber to the second inter-node from the base at 65 DAS. For disease phenotyping, the stalks were split opened 30 days after inoculation. Disease severity and intensity was recorded on individual plants using 1-9 rating scale (Table 1). The scoring pattern was developed based on the spread of inter-node discoloration inside maize stalks from the point of inoculation (Payak and Sharma 1983). Higher the discoloration, higher was the rating.

Statistical analysis

The disease score data on individual plants of the parents, their F_1s, F_2s and backcross generations were used for examining the adequacy of additive–dominance (A–D) model in the inheritance of resistance to FSR disease by employing the joint scaling test

Table 1. Disease rating scale: (IIMR, New Delhi, 2012 & Hooker, 1956)

Disease score	Symptoms	Disease reaction
1	Healthy or slight discolouration at the site of inoculation	Highly resistant
2	Up to 50% of the inoculated internode is discoloured	Resistant
3	51-75% of the inoculated internode is discoloured	Moderately resistant
4	76-100% of the inoculated internode is discoloured	Moderately susceptible
5	Less than 50% discolouration of the adjacent internode	Susceptible
6	More than 50% discolouration of the adjacent internode	Highly susceptible
7	Discolouration of three internodes	Highly susceptible
8	Discolouration of four internodes	Highly susceptible
9	Discolouration of five or more internodes and premature death of plant	Highly susceptible

(Cavalli, 1952) described by Mather and Jinks (1982). The χ^2 test for goodness of fit between observed means of six generations and those estimated based on the parameters, namely the general mean [m], the additive genetic effects [a] and the dominance genetic effects [d] was used as a criterion for examining the adequacy of A–D model. The parameters [m], [a] and [d] were estimated using weighted least square principle (Cavalli, 1952; Mather and Jinks 1982). A good and lack of fit suggested the adequacy and non-adequacy, of A–D model, respectively. In the case of inadequacy of A–D model, a perfect fit solution was used to estimate additive [a], dominance [d] and digenic epistatic effects, namely additive × additive [i], additive × dominance [j] and dominance × dominance [l] and their significance were tested using 't' test (Mather and Jinks 1982). Additive genetic variance [σ^2_A] and dominance genetic variance [σ^2_D] were estimated following the method outlined by Mather and Jinks (1982). All these biometrical genetic analyses were implemented using WINDOSTAT software v9.3 in each season and over the seasons. Bartlett's test was used to test for homogeneity (Gomez and Gomez, 1984). The minimum number of effective factors differentiating the parents was worked out using the formula given by Wright (1968) the potence ratio (PR), which indicates the degree of dominance, was computed from generation means as per Peter and Frey (1966). The genetics of resistance to FSR was interpreted based on the joint consideration of additive genetic effect [a] and additive genetic variance [σ^2_A]; dominance genetic effect [d] and dominance genetic variance [σ^2_D]; and dominance [h] and dominance × dominance [l] effects (Kearsey and Pooni 1996).

Results

The means, variances and variance of means of the six generations in the five crosses for reaction to Fusarium stalk rot are presented in Table 2. Higher expression of the disease was observed in both segregating (F_2, BC_1P_1 and BC_1P_2) and non-segregating generations (P_1, P_2, F_1) of the five crosses due to uniform artificial disease inoculation and favourable environmental conditions in both the seasons. Wide variation in FSR mean disease scores was observed among non-segregating populations and the severity score was maximum in the susceptible parent while, the F_1s recorded intermediate FSR score compared to the corresponding parents indicating quantitative nature of disease resistance. The additive-dominance model was inadequate as evident from the significant values of A, B, C and D scales (Table 3). An epistatic digenic interaction was found by performing the joint scaling test (Table 4). The opposite signs in dominance [h] and dominance × dominance interaction [l] represented the duplicate epistasis between alleles with dominance and increasing effects in the expression of FSR disease resistance in the cross VL1043 × CM212. The negative dominance [h] and positive dominance × dominance interaction [l] indicated the duplicate epistasis between alleles with dominance and decreasing effects in VL108867 × CM 202, VL121096 × CM 212, VL1218 × CM 202 and VL1218 × CM 212 crosses (Table 5).

In the presence of epistasis, estimates of additive genetic effect [a], dominance genetic effect [d] and their variances (σ^2_A and σ^2_D) are biased. Significance of both additive genetic effect [a] and additive genetic variance [σ^2_A] were noticed in all the five crosses over two seasons. The estimate of [σ^2_D] was significant in post rainy season and over the seasons while, [d] was nonsignificant in both seasons

Table 2. Estimates of means of generations with standard error, variance and variance of means for response to Fusarium stalk rot

Generations/Populations		Sample size	Mean ± SE			Variance			Variance of mean		
			Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons
VL1043 × CM212											
Parents	VL1043	14	6.07 ± 0.26	6.21±0.29	6.14±0.20	0.99	1.25	1.09	0.071	0.089	0.04
	CM212	16	3.56 ± 0.18	3.37±0.20	3.47±0.13	0.53	0.65	0.58	0.033	0.040	0.02
F_1	VL1043 × CM212	12	3.66 ± 0.22	3.91±0.31	3.79±0.19	0.61	1.17	0.87	0.050	0.097	0.04
F_2	VL1043 × CM212	220	4.51 ± 0.08	4.60±0.08	4.55±0.06	1.49	1.65	1.57	0.006	0.007	0.00
BC_1P_1	VL1043 × (VL1043 × CM212)	158	4.62 ± 0.08	4.74±0.09	4.68±0.06	1.25	1.38	1.31	0.008	0.008	0.00
BC_1P_{22}	CM212 × (VL1043 × CM212)	150	4.50 ± 0.09	4.45±0.09	4.47±0.07	1.34	1.35	1.34	0.009	0.009	0.00
VL108867 × CM202											
Parents	VL108867	16	6.12 ± 0.27	6.37±0.28	6.25±0.20	1.18	1.31	1.22	0.074	0.082	0.04
	CM202	16	3.37 ± 0.18	3.25±0.19	3.31±0.13	0.51	0.60	0.54	0.032	0.037	0.02
F_1	VL108867 × CM202	16	5.93 ± 0.35	5.62±0.36	5.78±0.25	2.06	2.11	2.05	0.128	0.132	0.06
F_2	VL108867 × CM202	324	5.51 ± 0.08	5.60±0.08	5.55±0.06	2.53	2.59	2.56	0.007	0.008	0.00
BC_1P_1	VL108867 × (VL108867 × CM202)	142	6.09 ± 0.14	6.21±0.14	6.15±0.10	2.91	2.79	2.84	0.020	0.019	0.01
BC_1P_2	CM202 × (VL108867 × CM202)	148	4.18 ± 0.10	4.01±0.09	4.10±0.07	1.58	1.38	1.49	0.011	0.009	0.01
VL121096 × CM212											
Parents	VL121096	14	6.57 ± 0.27	6.78±0.31	6.68±0.21	1.03	1.41	1.19	0.073	0.100	0.04
	CM212	12	3.58 ±0.19	3.33±0.22	3.46±0.15	0.45	0.60	0.52	0.037	0.050	0.02
F_1	VL121096 × CM212	32	4.15 ±0.11	4.25±0.12	4.20±0.08	0.41	0.51	0.45	0.012	0.016	0.01
F_2	VL121096 × CM212	220	4.81 ±0.09	4.88±0.09	4.85±0.07	1.98	2.10	2.04	0.009	0.009	0.00
BC_1P_1	VL121096 × (VL121096 × CM212)	146	4.41 ±0.07	4.50±0.08	4.46±0.06	0.89	1.06	0.98	0.006	0.007	0.00
BC_1P_2	CM212 × (VL121096 × CM212)	148	4.56 ±0.10	4.50±0.10	4.53±0.08	1.67	1.61	1.64	0.011	0.010	0.01
VL1218 × CM202											
Parents	VL1218	12	6.08 ± 0.28	6.33±0.30	6.21±0.21	0.99	1.55	1.04	0.082	0.096	0.04
	CM202	12	3.25 ±0.25	3.08±0.22	3.17±0.17	0.75	0.63	0.07	0.062	0.052	0.03
F_1	VL1218 × CM202	12	3.58 ±0.19	4.46±0.24	4.38±0.16	0.45	1.54	1.38	0.037	0.059	0.03
F_2	VL1218 × CM202	176	4.67 ±0.09	4.70±0.09	4.66±0.07	1.54	1.68	1.60	0.008	0.009	0.00
BC_1P_1	VL1218 × (VL1218 × CM202)	74	3.97 ±0.10	4.01±0.11	3.97±0.08	0.85	0.99	0.92	0.011	0.013	0.01
BC_1P_2	CM202 × (VL1218 × CM202)	116	4.25 ±0.09	4.37±0.11	4.40±0.08	1.05	1.42	1.40	0.009	0.012	0.01
VL1218 × CM212											
Parents	VL1218	14	6.07 ± 0.26	6.21±0.29	6.14±0.20	0.99	1.26	1.09	0.071	0.089	0.04
	CM212	16	3.62 ± 0.15	3.43±0.18	3.53±0.19	0.38	0.53	0.45	0.024	0.031	0.01
F1	VL1218 × CM212	26	5.03 ± 0.21	5.11±1.14	5.08±0.15	1.15	1.15	1.13	0.044	0.044	0.02
F2	VL1218 × CM212	230	4.83 ± 0.08	4.90±0.08	4.87±0.06	1.81	1.83	1.82	0.007	0.008	0.00
BC_1P_1	VL1218 × (VL1218 × CM212)	164	4.79 ± 0.09	4.85±0.09	4.83±0.07	1.46	1.54	1.50	0.008	0.009	0.00
BC_1P_2	CM212 × (VL1218 × CM212)	206	4.73 ± 0.08	4.67±0.08	4.71±0.06	1.35	1.32	1.33	0.006	0.006	0.00

“Significant at P = 0.05 and “”Significant at P = 0.01

Table 3. Estimates of scaling tests for Fusarium stalk rot scores in different generations

Cross	Scaling test											
	A			B			C			D		
	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons
VL1043 × CM212	-0.48	-0.65	-0.57	1.77**	1.61**	1.70**	1.09	0.98	1.03*	-0.10	0.01	-0.05
VL108867 × CM202	0.12	0.42	0.27	-0.93*	-0.85	-0.90*	0.67	1.54	1.11	0.74**	0.98**	0.86**
VL121096 × CM212	-1.89**	-2.02**	-1.96**	1.40**	1.43**	1.41**	0.79	0.93	0.86*	0.64*	0.76**	0.70**
VL1218 × CM202	-1.72**	-2.77**	-2.65**	1.67**	1.21**	1.26**	2.20**	0.48	0.50	1.13**	1.02**	0.95**
VL1218 × CM212	-1.52**	-1.61**	-1.57**	0.81*	0.81*	0.81**	-0.43	-0.28	-0.36	0.14	0.26	0.20

*Significant at P = 0.05 and **Significant at P = 0.01

and over the seasons in the cross VL1043 × CM212. The crosses VL108867 × CM202 and VL121096 × CM212 displayed significant negative dominance genetic effect [d] and significant positive dominance genetic variance [σ^2_D] in both seasons and over the seasons. The cross VL1218 × CM202 expressed significant negative dominance genetic effect [d] in both seasons. The dominance genetic variance [σ^2_D] was positively significant only in post rainy season. Nonsignificant dominance genetic effect [d] but significant dominance genetic variance [σ^2_D] was noticed in post rainy, summer and also over the seasons in the cross VL1218 × CM212. Our results indicated that, the magnitude and direction of additive genetic effect [a] and dominance genetic effect [d] and magnitudes of additive genetic variance [σ^2_A] and dominance genetic variance [σ^2_D] varied with the genetic background of the crosses over seasons. Predominance of dispersed genes with additive effects and their variances controlling the inheritance of FSR disease resistance was noticed (Table 6).

The number of effective factors were more in summer season compared to post rainy season while, it was intermediate over the seasons. The potence ratio in F_1 and F_2 generations indicated the preponderance of partial dominance in all the crosses except VL108867 × CM202 (Table 7). The heritability estimates indicate the progress from selection in breeding program. Moderate to high narrow sense heritability estimates were observed in all the crosses with 22.50 to 76.63 % in post rainy season, 35.31 to 72.56 % in summer season and 30.78 to 71.49 % over environments (Table 7).

Discussion

Inheritance of the resistance to FSR disease is highly complex as it is controlled by a combination of more number of genes with varying magnitude of effects and modes of action (additive, dominance and epistasis) and significant noncrossover/crossover interaction with environment (Kearsey and Pooni 1996; Yang et al. 2004). Further, modes of action of genes and their interaction with environment are a function of several factors such as frequency of genes, kinds of genetic material, history of selection and predominant mode of pollination (Bernardo 2010, 2014; Acquaah 2012). It is therefore essential to decipher the mode of action of genes controlling quantitative traits in the genetic material currently being handled by breeders to develop improved crop cultivars. Being complexly inherited, mode of action of genes can be deciphered initially using simple genetic models assuming only additive and dominance effects, and gradually increasing the complexity of the model by inclusion of parameters specifying digenic interactions and genotype × environment interaction (GEI). In our study, it was clear from the Table 2 that parents were highly diverse in disease reaction. Probably because of the diverse nature of the parents, F_2 and backcross generations also exhibited a vast difference in their reaction to FSR in all the five crosses. Significance of joint scaling test indicated inadequacy of simple additive-dominance model (A–D model) in explaining the inheritance of resistance to FSR (Table 3). Inadequacy of A–D model could be attributed to the involvement of parameters

Table 4. Estimates of components of generation means and test for adequacy of additive-dominance model in the inheritance of resistance to *Fusarium* stalk rot in maize

Crosses	(m)			(d)			(h)			χ^2 Statistic*			Probability		
	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons
VL1043 × CM212	4.80**± 0.13	4.68**± 0.15	4.74**± 0.10	0.61* ± 0.10	0.71**± 0.11	0.66**± 0.07	-0.64*± 0.26	-0.23± 0.31	-0.42*± 0.20	38.03	30.17	67.32	0.000	0.000	0.00
VL108867 × CM202	4.84**± 0.15	4.97**± 0.12	4.90**± 0.10	1.67**± 0.12	1.98**± 0.12	1.82**± 0.08	0.93**± 0.30	0.67*± 0.31	0.81**± 0.21	16.51	27.11	42.86	0.001	0.000	0.00
VL121096 × CM212	4.82**± 0.11	4.80**± 0.12	4.82**± 0.08	0.45**± 0.11	0.55**± 0.10	0.50**± 0.07	-0.62** ± 0.21	0.18-0.49*± 0.14	-0.56**±	73.47	67.80	142.03	0.000	0.000	0.00
VL1218 × CM202	4.76**± 0.14	4.37**± 0.15	4.42**± 0.11	0.37**± 0.11	0.46**± 0.12	0.37**± 0.08	-0.93**±0.09 0.27	±-0.06± 0.30	74.52 0.20	78.05	155.49	0.000	0.000	0.00	
VL1218 × CM212	4.52**± 0.12	4.49**± 0.13	4.51**± 0.09	0.52**± 0.09	0.60**± 0.10	0.57**± 0.07	0.57*± 0.24	0.68*± 0.26	0.61**± 0.17	34.97	32.18	67.86	0.000	0.000	0.00

*Simple Additive- Dominance model was not adequate in explaining the genetics of resistance to FSR

*Significant @ P = 0.05; ** Significant @ P = 0.01

Table 5. Estimates of components of generation means based on perfect fit solution (Joint Scaling Test) for reaction to *Fusarium* stalk rot

Crosses	(m)			(d)			(h)			(i)			(j)			(l)			Type of digenic epistasis		
	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons
VL1043 × CM212	4.62**± 0.45	4.80**± 0.47	4.71**± 0.33	1.25**± 0.16	1.42**± 0.18	1.34**± 0.12	0.53± 1.15	0.06± 1.23	0.30± 0.84	0.20± 0.42	-0.01± 0.44	0.09± 0.30	-2.25**± 0.41	-2.26**± 0.45	-2.26**± 0.30	-1.49± 0.82	-0.95± 0.96	-1.22± 0.63	DEDI	DEDI	DEDI
VL108867 × CM202	6.24**± 0.52	6.78**± 0.52	6.51**± 0.37	1.37**± 0.16	1.56**± 0.17	1.47**± 0.12	-2.60*± 1.41	-3.55*± 1.40	-3.08**± 0.10	-1.48**± 0.50	-1.98**± 0.49	-1.73**± 0.35	1.05* ± 0.48	1.27*± 0.49	1.16**± 0.34	2.30*± 1.12	2.40*± 1.11	2.35**± 0.78	DEDD	DEDD	DEDD
VL121096 × CM212	6.36**± 0.49	6.58**± 0.51	6.47**± 0.35	1.49**± 0.17	1.73**± 0.19	1.61**± 0.13	-3.98**± 1.21	-4.44**± 1.27	-4.21**± 0.88	-1.28*± 0.46	-1.52**± 0.47	-1.40**± 0.33	-3.28**± 0.42	-3.25**± 0.47	-3.37**± 0.32	1.78*± 0.76	2.11*± 0.81	1.94**± 0.55	DEDD	DEDD	DEDD
VL1218 × CM202	6.92**± 0.51	6.74**± 0.54	6.58**± 0.37	1.42**± 0.19	1.62**± 0.19	1.52**± 0.13	-5.65**± 1.30	-5.86**± 1.39	-5.47**± 0.96	-2.26**± 0.47	-2.03**± 0.51	-1.89**± 0.35	-3.39**± 0.48	-3.98**± 0.50	-3.91**± 0.35	2.31* ± 0.87	3.59**± 0.97	3.28**± 0.67	DEDD	DEDD	DEDD
VL1218 × CM212	5.13**± 0.46	5.35**± 0.47	5.24**± 0.33	1.22**± 0.15	1.39**± 0.17	1.31**± 0.11	-1.08± 1.15	-1.56± 1.18	-1.32± 0.82	-0.28± 0.43	-0.52± 0.44	-0.40± 0.31	-2.34**± 0.40	-2.42**± 0.43	-2.38**± 0.29	0.10± 0.80	1.32± 0.82	1.16*± 0.57	DEDD	DEDD	DEDD

* Significant @ P = 0.05; ** Significant @ P = 0.01. DEDI, duplicate epistasis between dominant increasers;DEDD, duplicate epistasis between dominant decreasers

Table 6. Estimates of additive genetic effect and variance (σ^2_A) and dominant genetic effect and variance (σ^2_D) for response to Fusarium stalk rot in maize

Crosses	[a]			(σ^2_A)			[d]			(σ^2_D)		
	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons
VL1043 × CM212	1.25**	1.42**	1.34**	0.01**	0.01**	0.01**	0.53	0.06	0.30	0.07*	0.09	0.04*
VL108867 × CM202	1.37**	1.56**	1.47**	0.01**	0.01**	0.01**	-2.60*	-3.55*	-3.08**	0.09**	0.09*	0.04**
VL121096 × CM212	1.49**	1.73**	1.61**	0.01**	0.01**	0.01**	-3.98**	-4.44**	-4.21**	0.03**	0.04*	0.02**
VL1218 × CM202	1.42**	1.62**	1.52**	0.01**	0.01**	0.01**	-5.65**	-5.87**	-5.48**	0.07**	0.09	0.04
VL1218 × CM212	1.22**	1.39**	1.31**	0.01**	0.01**	0.01**	-1.08	-1.56	-1.32	0.06*	0.07*	0.03**

*Significant at P = 0.05 and ***Significant at P = 0.01

Table 7. Number of effective factors in the genetic control of resistance to Fusarium stalk rot in F_2 generation, potence ratio (in F_1 and F_2) and heritability in narrow sense (h^2) in the crosses

Crosses	No. of effective factors in F_2			Potence ratio in F_1			Potence ratio in F_2			h^2 (%)		
	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons	Post rainy	Summer	Over seasons
VL1043 × CM212	4.66	9.25	6.08	0.92	0.62	0.76	0.48	0.23	0.37	26.29	35.31	31.16
VL108867 × CM202	3.33	5.37	3.94	0.86	0.52	0.68	1.11	1.01	1.06	22.50	38.80	30.78
VL121096 × CM212	3.11	3.80	3.45	0.62	0.47	0.54	0.35	0.20	0.27	70.18	72.56	71.49
VL1218 × CM202	4.40	30.16	18.05	0.76	0.15	0.20	0.01	0.01	0.03	76.63	56.27	54.37
VL1218 × CM212	3.07	3.63	3.26	0.15	0.21	0.18	0.02	0.11	0.05	44.59	44.00	44.37

specifying digenic epistasis and/or GEI. We included digenic epistasis parameters namely [i], [j] and [l] in the A–D model assuming the absence of GEI (Mather and Jinks 1982). Most of the parameters specifying epistasis were significant in all the crosses in both seasons and over the seasons.

Epistasis of genes controlling quantitative traits can only be classified as either predominantly duplicate or complementary, the distinction being based solely on the relative signs of [h] and [l] components. Positive estimates of both [h] and [l] lead to complementary epistasis between dominant increasing alleles, while negative estimates indicate the complementary epistasis between dominant decreasing alleles. While, positive [h] and negative [l] represents the duplicate epistasis between dominant increasing alleles, negative [h] and positive [l] indicates the duplicate epistasis between dominant decreasing alleles (Kearsey and Pooni 1996). In this study, genes controlling the FSR disease response in all the five crosses displayed duplicate epistasis in post rainy, summer and over the seasons. In the presence of epistasis, estimates of additive [a] and dominance [d] gene effects and their variances (σ^2_A and σ^2_D) are expected to be biased. However, epistasis is important in maintaining additive genetic variance [σ^2_A] even when variance attributable to epistasis (σ^2_I) is small. It has been postulated that epistasis ensures continued success of inbred recycling (Rasmusson and Philip 1997) and the conversion of σ^2_I into σ^2_A is a specific mechanism by which progress from selection can be maintained despite a narrow genetic base (Bernardo 2010, 2014). An essential assumption to obtain unbiased estimates of [a] and [d] gene effects is that in a particular homozygous parental genotype, the alleles at all the loci controlling the target trait are either having increasing or decreasing effects and display unidirectional dominance. However, in practice, each individual genotype can have a combination of both increasing and decreasing effect alleles with varying degree and direction of dominance. In extreme cases, increasing and decreasing alleles are equally dispersed. Consequently, most often, additive gene effects [a] are underestimated and the degree of underestimation depends on the degree of dispersion of alleles. Similarly, ambidirectional dominance underestimates dominance gene effects [d]. Variances

overcome the demerits associated with internal cancellation of positive and negative effects caused by gene dispersion and ambidirectional dominance. However, any level of dominance and all types of epistasis contribute to σ^2_A, σ^2_D, and σ^2_I (Bernardo 2014). Hence, it is difficult to infer predominant modes of action of genes even from second degree statistics. The joint application of both first and second degree statistics provide more comprehensive and dependable information about genetic control of quantitative traits (Kearsey and Pooni 1996).

The magnitude of additive genetic variance [σ^2_A] is not affected by gene dispersion and is orthogonal to additive genetic effect [a] and therefore is not correlated with [a]. Consequently, several combinations of [a] and [σ^2_A] are expected. For instance, [a] can be small (or zero) due to gene dispersion while, [σ^2_A] is large. Similarly, [a] can be large while [σ^2_A] is still nonsignificant (statistically zero), particularly when the effects of individual genes are very small (Kearsey and Pooni 1996). The relationship between [d] and [σ^2_D] is also similar except that the significance and magnitude of the estimates of [σ^2_D] are affected by ambidirectional dominance and not gene dispersion. Significance of both [a] and [σ^2_A] in all the five crosses over two seasons suggested the substantial contribution of [a] gene effects in the inheritance of FSR disease resistance. The presence of nonsignificant dominance genetic effect [d] with significant dominance genetic variance [σ^2_D] indicated the ambidirectional dominance of genes controlling resistance to FSR disease in both post rainy and *summer* seasons in the cross VL1218 × CM212. While, in the cross VL1043 × CM212, [d] was nonsignificant and [σ^2_D] was significant only in post rainy season. Nonsignificant estimates of both [d] and [σ^2_D] in summer season of the cross VL1043 × CM212 suggested the absence of dominance. Further, significant negative [d] and significant [σ^2_D] indicated the directional dominance for decreasing alleles in the three crosses *viz.,* VL108867 x CM202, VL121096 x CM212 and VL1218 x CM202 over two seasons except for the cross VL1218 x CM202 in summer season where significant [d] and nonsignificant [σ^2_D] indicated low dominance in the inheritance of resistance. Thus, the magnitude and direction of [a] and [d], and magnitudes of [σ^2_A] and [σ^2_D] varied with the genetic background of the crosses. Our results indicating the role of dispersed genes with predominantly additive effects and variance. This study augurs well with those reported by Roy (2000) and Archana et al. (2019). The predominance of additive effects and variance in this study for the FSR resistance draw adequate support from theoretical expectations of greater σ^2_A than σ^2_D (Moll and Stubber 1974; Hallauer 1985; Dudley 1997; Bernardo 2010, 2014). This is because, loci that exhibit dominance as well as epistasis also contribute to σ^2_A indicating that any segregating locus with either no dominance or partial dominance or complete dominance or overdominance contribute to σ^2_A (Bernardo 2010, 2014). Hallauer and Miranda (1988) and Bernardo (1996) reported that estimates of σ^2_A are about 67% and 200% greater than σ^2_D, respectively for grain yield in maize. Considering that dispersion of genes also reduces σ^2_A (Hanson 1959), intermating in early (F_2/F_3) segregating generations not only help achieve near complete association of genes but also increases the frequency of genes that contribute to σ^2_A. Increase in σ^2_A as a result of intermating is attributed to autoconversion (self-conversion) of nonadditive genetic variances including epistasis to σ^2_A. This conversion occurs because heterozygotes get fixed into homozygotes (Goodnight 1998; Acquaah 2012). For estimation of the number of effective factors, differences between parents and variation in F_2 and backcrosses are needed. The FSR resistance was under the control of more groups of effective factors. This result is in accordance with that reported by Archana et al. (2019) for FSR resistance in maize. Allard (1960) noted that major genes are believed to have a complement of modifiers and number of genes estimated is not necessarily the actual number. Each effective unit may be considered as a block of closely linked genes which segregate as a unit. Potence ratio in the F_1 and F_2 generation revealed the preponderance of over dominance but in negative direction in the genetic control of resistance to FSR. In all the five crosses, one or two cycles of intermating followed by simple selection for resistant inbreds can be made as [h] + [l] < [d] + [i] and high narrow-sense heritability was also evidenced by many group of genes controlling the resistance reaction. Reciprocal recurrent selection will also be more rewarding in obtaining FSR resistant maize inbred lines considering the importance of both additive and non-additive genetic effects in the inheritance of resistance.

Authors' contribution

Conceptualization of research (HCL, BMSB); Designing of the experiments (HCL, AMR, NM); Contribution of experimental materials (HCL); Execution of field/lab experiments and data collection (BMSB, HCL, NM); Analysis of data and interpretation

(BMSB and HCL); Preparation of the manuscript (BMSB and HCL).

Declaration

The authors declare no conflict of interest.

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