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Genetic analysis of Fusarium stalk rot resistance in maize (*Zea mays* L.)

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Abstract
The present investigation was conducted to understand the genetics of resistance to Fusarium stalk rot (FSR) resistance in maize through six-generation mean analysis in the cross, CM202 × P12 during *kharif* 2017. CM202 was a resistant line to FSR disease while P12 was highly susceptible. The F_1 showed susceptible reaction to the disease. The scaling tests and joint scaling tests indicated the inadequacy of additive-dominance model and showed the presence of epistatic gene effects in the cross for Fusarium stalk rot resistance. Further study of genetic effects revealed the preponderance of additive and additive × additive gene effects in the expression of FSR and duplicate type of interaction was observed in the expression of disease. The potence ratio of more than one indicated that FSR could be under control of over dominance. The number of effective factor was found to be one with high heritability estimate in the inheritance of resistance to FSR which indicated the effectiveness of simple selection strategies in isolating superior inbreds.

Keywords: Generation means, additive-dominance model, duplicate gene interaction, simple selection

Introduction
Maize (*Zea mays* L.) has become a staple food in many parts of the world, with total production surpassing that of wheat or rice, serving as staple food, livestock feed, and industrial raw material (Troyer 2006) [20]. Approximately 65 pathogens infect maize (Rahul and Singh, 2002) [13] that include foliar diseases, ear rot, and stalk rots caused by fungi and bacteria. Globally, about nine per cent yield losses have been estimated in maize due to diseases (Oerke, 2005) [10]. Fusarium stalk rot (FSR) caused by *Fusarium moniliforme*, is one of the most devastating soil-borne diseases of maize, occurring in all continents of the world. In India, Fusarium stalk rot was first reported from Mount Abu, Rajasthan (Arya and Jain, 1964) [2]. The disease is common in Rajasthan, Uttar Pradesh, Bihar, Karnataka and Andhra Pradesh. Dry and warm (28-30º C) weather early in the season followed by wet weather two to three weeks after silking favours the disease incidence. Several disease management options have been recommended to reduce the impact of FSR including conventional tillage, crop rotation, foliar fungicide application and planting of resistant hybrids. Among these practices, planting of resistant cultivars can effectively reduce the rate of disease development and is widely recommended (Ward *et al.* 1997) [16]. To breed a genotype with high level of resistance to Fusarium stalk rot the knowledge of gene action involved in the expression of resistant reaction in the material being handled, is a very important pre-requisite. Earlier studies have indicated that resistance to stalk rot is quantitatively inherited and controlled by multiple genes with additive effects (Yang *et al.*, `2004) [21]. Various biometrical approaches have been developed to decipher the genetic architecture and mode of inheritance of Fusarium stalk rot. Generation mean analysis (Hayman 1958; Jinks and Jones 1958; Mather and Jinks 1971) [4, 5, 6] is one such approach, which elucidates information about nature and magnitude of different gene actions *viz.*, additive and dominance with an unambiguous test for epistasis. It also provides information about the type of epistasis *viz.*, additive × additive, additive × dominance and dominance × dominance operating in the inheritance of a character. Detection, estimation

and interpretation of non-allelic interactions from generation mean analysis is statistically reliable as it is based on first order statistics which are less confounded with each other when compared with higher order statistics based estimates. Thus, the objective of this research program was to use the generation mean analysis to study the inheritance of resistance to FSR in maize to initiate breeding program to develop resistant inbreds.

Material and Methods

The research work was conducted in the disease sick plots of Zonal agricultural research station (ZARS), V. C. Farm, Mandya. Two contrasting inbred parents (CM202 and P12) for FSR disease reaction were taken for study. The cross, CM202 (Resistant) × P12 (Highly susceptible) was generated during *Kharif* 2016 and F_1 was raised during *Rabi* 2016-17 along with the parents in a randomized complete block design with two replications. The F_1 was backcrossed to both parents and selfed simultaneously to produce F_2. Six generations *viz.*, P_1, P_2, F_1, F_2, B_1 and B_2 of the cross, CM202 × P12 were raised during summer 2017. Non–segregating generations (P_1, P_2 and F_1) consisted of 20 plants each, 250 plants in F_2 and 150 plants in backcross generations. Appropriate susceptible checks for FSR (Hema and Nithyashree) were sown after every 20th row to assure adequate disease load in the experimental plots. To ensure uniform disease incidence, artificial inoculation was done by following the procedure given by Indian Institute of Maize Research (IIMR), New Delhi. The pathogen *Fusarium moniliforme* was isolated by standard tissue isolation technique (IIMR, New Delhi). Inoculations were made with of 45-50 days old plants just after flowering stage, in the second internode above the soil level. Disease symptoms appeared in the inoculated plants about 20-25 days after inoculation. The disease intensity and severity was recorded following 1-9 rating scale as described by IIMR (New Delhi), according to discolouration in the second internode from soil to upper internodes on a scale from 1 (Highly Resistant) to 9 (Highly Susceptible) thereby providing for a total of nine classes where score1: slight discolouration of the inoculated internode, score 2: 50% discolouration of the inoculated internode, score 3: 51-75% discolouration of the inoculated internode, score 4: 51-75% of the inoculated internode is discoloured, score 5: Less than 50% discolouration of the adjacent internode, score 6: More than 50% discolouration of the adjacent internode, score 7: Discolouration of three internodes, score 8: Discolouration of four internodes and score 9: Discolouration of five or more internodes and premature death of plant.

Statistical analysis

To detect the presence or absence of epistasis, four scaling tests (A, B, C and D) of Mather (1949) [7] were used. The six parameter model (Jinks and Jones 1958; Mather and Jinks 1971) [5, 6] was used to estimate gene effects for the traits for which additive-dominance model was inadequate as indicated by joint scaling test of Cavalli (1952) [3]. The minimum number of effective factors differentiating the parents was worked out using the formula given by Wright (1968) [19] and the potence ratio (PR) which indicates the degree of dominance was computed from generation means as per Peter and Frey (1966) [12]. From the estimates of generation mean analysis heritability in narrow sense can be worked as per warner (1952) [17].

Results

Uniform disease score for Fusarium stalk rot was achieved through artificial inoculation. The results obtained through six generation mean analysis involving both segregating and non-segregating generations (P_1, P_2, F_1, F_2, B_1 and B_2) of the cross CM202 × P12.

The mean values along with standard error and variances of six generations (P_1, P_2, F_1, F_2, B_1 and B_2) of the maize in respect of Fusarium stalk rot are mentioned in Table 1 and briefly presented below. Wide range of mean disease score was observed among non-segregating populations and the disease score was maximum in susceptible parent, P12 (5.36) compared to resistant parent CM202 (2.7). While in the F_1 (CM202 × P12) recorded mean disease score of 5.37 compared to its parents. Among segregating generations, the disease score was 5.26 in F_2, 5.23 in B_1 and 4.46 in B_2. To test the adequacy of additive – dominance model A, B, C and D scaling tests were applied. The mean and variance of six generations *viz*., P_1, P_2, F_1, F_2, B_1 and B_2 of the cross CM202 × P12 in respect of Fusarium stalk rot disease score were subjected to scaling tests as per the method of Mather (1949) [7]. The results of scaling tests A, B, C and D in respect of disease score of six crosses are presented in table 2. The scaling tests A, B, C and D were significant in the cross, CM202 × P12, which was further confirmed by significance of additive and dominance components in joint scaling test (table 3). The mean and variance of six generations *viz*., P_1, P_2, F_1, F_2, B_1 and B_2 of the cross, CM202 × P12 for Fusarium stalk rot disease score were subjected to Joint scaling test. The results of joint scaling test for Fusarium stalk rot score is presented in Table 4.

After ascertaining the failure of additive-dominance model in explaining the inheritance of resistance to Fusarium stalk rot, the perfect fit six-parameter estimates of digenic interaction model were estimated following the methods of Jinks and Jones (1958) [5] and Mather and Jinks (1971) [6]. The gene effects estimated by using perfect fit model in respect of disease score are tabulated in Table 4. Opposite signs of [$\hat{h}$] and [$\hat{l}$] indicated the presence of duplicate type of epistasis in the inheritance of resistance to Fusarium stalk rot.

The number of effective factors was 1.19 and potence ratio of -1.01 and -1.85 was observed in F_1 and F_2 generations, respectively and narrow sense heritability of 61 per cent was recorded (Table 5).

Table 1: Estimates of means of generations with their standard error, variance and variance of mean for response to Fusarium stalk rot.

Generations /Populations (Sample size)			Fusarium stalk rot		
			Mean ± SE	Variance	Variance of mean
Parents	20	CM 202	2.79±0.43	0.18	0.02
	20	P12	5.36±0.85	0.70	0.05
F_1	30	CM 202 × P12	5.37±1.01	1.20	0.04
F_2	257	CM 202 × P12	5.26±2.24	5.01	0.02
B_1	157	CM 202 × (CM 202 × P12)	5.23±1.95	3.78	0.04
B_2	164	P12 × (CM 202 × P12)	4.46±1.79	3.20	0.03

Table 2: Estimates of scaling tests for Fusarium stalk rot scores of different generations

Cross	Scaling Test			
	A	B	C	D
CM 202 × P12	2.32**	-1.82**	2.17**	0.84*

* Significant at $P \leq 0.05$, ** Significant at $P \leq 0.01$, *** Significant at $P \leq 0.001$

Table 3: Estimates of components of generation means and test for adequacy of additive-dominance model for inheritance of Fusarium stalk rot

Disease	Cross	(m)	(d)	(h)	Chi square value
Fusarium stalk rot	CM 202 × P12	3.96	0.95	1.76	66.42**

* Significant at $P \leq 0.05$, ** Significant at $P \leq 0.01$, *** Significant at $P \leq 0.001$

Table 4: Estimates of components of generation means based on perfect fit solution (Joint Scaling Test) for Fusarium stalk rot

Cross	(m)	(d)	(h)	(i)	(j)	(l)	Type of epistasis
CM202 × P12	5.74**	-1.29**	-1.55	-1.67*	2.063**	1.174	Duplicate

* Significant at $P \leq 0.05$, ** Significant at $P \leq 0.01$, *** Significant at $P \leq 0.001$

Table 5: Number of effective factors in the genetic control of Fusarium stalk rot in F_2 generation, potence ratio (in F_1 and F_2) and heritability in narrow sense (h^2).

Disease	Number of effective factors in F_2	Potence ratio in F_1	Potence ratio in F_2	h^2
Fusarium stalk rot	CM202 × P12	CM202 × P12	CM202 × P12	CM202 × P12
	1.19	-1.01	-1.85	61%

Discussion

In the present study, to know the genetics of resistance to Fusarium stalk rot in maize, six generation mean analysis was carried out in the cross CM202 × P12. It was clear from the Table 1 that parents were highly diverse for disease reaction. Probably because of the diverse nature of the parents, even F_2 and backcross generations also exhibited a vast difference in their reaction to Fusarium stalk rot.

The adequacy of simple additive-dominance model was tested by A, B, C and D scaling tests of Mather (1949) [7] which confirmed the presence of epistatic gene effects in the cross CM202 × P12, thereby indicating the inadequacy of simple additive-dominance model to explain the inheritance of resistance to Fusarium stalk rot in maize. Hence the use of six parameter model for detection of epistatic gene interaction involved in the inheritance of this character was suggested. After knowing the inadequacy of additive-dominance model perfect fit solutions were applied to estimate the magnitude and direction of the digenic interaction effects for the cross.

The significant estimate of additive [$\hat{d}$] gene effect in negative direction was observed in the inheritance of resistance to Fusarium stalk rot. Among non-allelic interaction effects, significance of additive × additive [$\hat{i}$] and additive x dominance [$\hat{j}$] interaction effects was noticed in controlling the resistance to Fusarium stalk rot in maize. In the present investigation, generation mean analysis revealed that additive gene action was more preponderant which could be improved by progeny selection in early generations and also it is fixable in nature hence advanced generation of this cross can be utilized to derive superior inbreds with higher resistance. Opposite signs of [$\hat{h}$] and [$\hat{l}$] indicated the presence of duplicate type of epistasis in the inheritance of Fusarium stalk rot, which tends to reduce the trait expression and the genetic gain is faster with mild selection and less rapid with very intense selection (Roy, 2000) [15]. The studies conducted by Pe *et al*. (1993) [11] reported that resistance to stalk rot was quantitatively inherited and controlled by multiple genes with additive effects. The results obtained by Murali Krishna *et al*. (2013) [8] also revealed the role of epistatic effects *viz*., additive x additive, additive x dominance, dominance x dominance as well as dominance gene action in governing the resistance to post flowering stalk rot.

For estimation of the number of effective factors, differences between parents and variation in F_2 and backcrosses are needed. The Castle/Wright formula (Weber, 1950) [18] was used to estimate the number of effective factors in the cross CM202 × P12 for Fusarium stalk rot reaction. The Fusarium stalk rot resistance was under the control of one group of effective factors. This result is in accordance with that reported by Ranganatha (2014) [14] who reported that genetics of resistance to northern corn leaf blight was under the control of one group of effective factors and Carson and Hooker for Anthracnose leaf blight in maize (1980). Allard (1960) [1] 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 Fusarium stalk rot. In the cross CM202 × P12, simple selection for resistant inbreds can be made as [h] + [l] < [d] + [i] and high narrow-sense heritability (61 %) as also evidenced by one group of genes controlling the resistance reaction.

Conclusion

In the present investigation, generation mean analysis revealed that the cross with preponderance of additive 'd' gene action could be improved by progeny selection in early generations and also it is fixable in nature and hence superior inbreds with higher resistance could be derived. Duplicate epistasis indicated that population improvement programs need to be utilized for isolation of superior inbreds and used in the development of heterotic hybrids.

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