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*Full Length Research Paper*

# Genetic analysis of resistance to common smut in maize (*Zea mays L.*) using triple test cross

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**Understanding of gene action for resistance to maize common smut is essential for maize breeding programs, therefore, triple test cross analysis was employed to assess gene action controlling resistance to common smut in maize. Parental inbred lines and their progenies were evaluated using randomized complete block design with three replications in Karaj Field Station, Seed and Plant Improvement Institute, in 2007 and 2008 cropping seasons. Epistasis was observed for resistance to maize common smut. Partitioning of the total epistasis revealed that [i] type (additive × additive) and [I+J] types (additive × dominance and dominance × dominance) were highly significant. Additive ($L_{1i}$+$L_{2i}$) and dominance ($L_{1i}$ – $L_{2i}$) effects for resistance to maize common smut were also significant, over two growing seasons. Dominance ratio (H/D) $^{1/2}$ indicated that resistance to maize common smut was controlled by dominance effect. However, the direction of dominance ($r_{s,d}$) for this character in two growing seasons was not-significant which implies that dominant alleles were distributed in parents, therefore they did not express any directional dominance for this attribute. Since F′ was positive, therefore, dominant alleles increased disease severity of maize common smut. Cytoplasmic effects were also important for resistance to maize common smut. Combined analysis of variance showed that the effect of year and genotype × year interaction were highly significant. Generally, the additive, dominance and epistatic components were important in resistance to maize common smut. It is concluded that Genetic analysis of Resistance to Common Smut in Maize (*Zea mays* L.) using triple test cross recurrent selection procedure may be efficient in breeding for resistant to maize common smut, since it exploits additive and non-additive components of genetic variation for improvement resistance to maize common smut.**

**Key words:** *Zea mays,* triple test cross, common smut, gene action.

## INTRODUCTION

Common smut caused by *Ustilago maydis*, is one of the most serious diseases that occurs in maize growing areas throughout the world. Like many economically important plant-pathogen systems, breeding for genetic resistance in the host, is the most effective way to control common smut (Du Toit and Pataky, 1999). Employing an effective breeding procedure depends to a large extent on understanding of the genetic mechanisms controlling the characters to be improved (Sadat Noori and Sokhansany, 2004). Resistance to maize common smut and the nature of the host-pathogen interaction are still method of inoculation (Pataky et al., 1995). Hence, only limited molecular genetic information is available on inheritance of resistance to common smut in maize.

Various quantitative genetic approaches have been used for estimating the mode of gene action in controlling resistance to common smut disease in maize. Most of the genetic design used to analyze mode of gene action assume absence of non-allelic interactions, however, there are contrary evidences to this assumption (Ashfa et

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**Table 1.** Mean of squares of sums and differences.

| Difference | | | Sum | | |
|---|---|---|---|---|---|
| δ2e+2rδ2 m1 | n-1 | $\overline{L_{1i}} - \overline{L_{2i}}$ | δ2e+2rδm2 | n-1 | $\overline{L_{1i}} + \overline{L_{2i}}$ |
| δ2e | (n-)(r-) | Error | δ2e | (n-1)(r-) | Error |

**Table 2.** List of maize inbred lines used for triple test cross.

| Pedigree /Origin | Inbred lines | Parent/line designation |
|---|---|---|
| Unknown | $K_{1264/1}$ | $P_1$ |
| Hybrid 3114 (Petrisco company) | $K_{47/2\text{-}2\text{-}1\text{-}3\text{-}3\text{-}1\text{-}1\text{-}1}$ | $P_2$ |
| Derived from $MO_{17}$ | $K_{19}$ | $L_1$ |
| Derived from $K_{19}$ | $K_{19/1}$ | $L_2$ |
| Tlaltizapan – 8946 | $K_{3545/6}$ | $L_3$ |
| Tlaltizapan – 8946 | $K_{3545/7}$ | $L_4$ |
| Po- Tzu- Chia- I8946 | $K_{3544/1}$ | $L_5$ |
| Srinagar 8848 | $K_{3547/5}$ | $L_6$ |
| SYN- late | $K_{3615/1}$ | $L_7$ |
| SYN- late | $K_{3615/2}$ | $L_8$ |
| SYN-late | $K_{3651/1}$ | $L_9$ |
| SYN- late | $K_{3651/2}$ | $L_{10}$ |
| BSSS $C_5$ (Iowa stiff stalk synthetic) | $B_{73}$ | $L_{11}$ |
| $B_{73}$ back cross derived line [($A_{662}$×$B_{73}$)(3)] | $A_{679}$ | $L_{12}$ |

al., 2006). The triple test cross is one of the breeding poorly understood, partly because of the lack of a reliable designs that provides information about additive, dominance and epistatic gene effects (Jinks and Perkins, 1970; Pooni et al., 1980; Mather and Jinks, 1982). In the present study, triple test cross was used to assess additive, non-additive as well as espistatic gene effects in controlling resistance to common smut in maize.

## MATERIALS AND METHODS

### Plant materials and field design

The experimental materials for triple test cross analysis were produced following the procedure outline by Ketatta et al. (1976). $K_{1264/1}$ resistant and $K_{47/2\text{-}2\text{-}1\text{-}3\text{-}3\text{-}1\text{-}1\text{-}1}$ susceptible to common smut were crossed, as testers, with 12 randomly selected inbred lines as well as resulted $F_1$s (Table 2). The 51 genotypes including 12 inbred lines, three testers and 36 hybrids were evaluated using a RCB design with three replications under field conditions, in 2007 and 2008 cropping seasons.

### Inoculation method and disease assessment

Teliospores originally isolated from susceptible maize lines were surface disinfected using 0.5% copper sulfate solution, for 16 h on a horizontal shaker. The disinfected teliospores were spread on potato dextrose agar (PDA) in Petri dishes for five days at 23 - 25°C, to produce sporidia. The produced sporidia was diluted to a concentration of approximately $10^6$ spores.ml$^{-1}$ (Thakur et al., 1989). Ten uniform plants from each inbred line were selected and inoculated by tip injection method- using 3 ml inoculation. All of the infected plants were scored for rate of development of disease symptom on ear, after 3 - 4 weeks.

### Data analysis

Disease severity scores were transformed to a continuous scale from 0 to 7. Data were subjected to angle transformation prior to analysis of variance to normalize and improve homogeneity of variance. Analysis of variance for triple test cross data was performed following Singh and Chaudhary (1999). The genotype effect was partitioned into the effects of hybrids, parents, inbred lines, testers, $P_1$+ $P_2$ *vs* $F_1$, $P_1$ *vs* $P_2$, lines *vs* testers and hybrids *vs* parents. The presence of epistasis was estimated when the mean of squares for the ($\overline{L_{1i}} + \overline{L_{2i}} - 2\overline{L_{3i}}$) were significantly greater than error (Kearsey and Jinks, 1968; Singh and Chaudhary, 1999). Further epistatic effect was partitioned into [i] (additive × additive interactions), [j] (joint effect) and [l] (additive × dominance and dominance × dominance interactions) following Jinks and Perkins (1970) and Pooni et al. (1980). Additive and dominance components were also estimated from analysis of sums and differences assuming no epistasis. The expected mean of squares of sums and differences are shown in Table 1 (Jinks et al., 1969).

The expectation for $\delta^2_m$ and $\delta^2_{m1}$ in absence of epistasis and linkage is 1/4D and 1/4H, respectively (Jinks et al., 1969). Average degree of dominance was calculated as $(H/D)^{1/2}$, where (H) and (D) are the dominance and additive variance components, respectively. Direction effect of dominance was determined by correlation ($r_{s,d}$) between the sum ($L_{1i}$+ $L_{2i}$) and the genotypic differences ($L_{1i}$ - $L_{2i}$) for all the genotypes. Significant positive and negative correlations indicate a predominant direction towards decreasing and increasing values of the trait, respectively (Jinks et al., 1969; Khattak et al.,

**Table 3.** Mean of squares for genotypes.

| Source of variation | df | 2007 | 2008 |
|---|---|---|---|
| Replication | 2 | 621.36** | 2.01 ns |
| Genotypes | 50 | 849.09** | 365.48** |
| Hybrids | 35 | 887.42** | 387.87** |
| Parents | 14 | 808.457** | 328.23** |
| Lines | 11 | 773.853** | 304.83** |
| Testers | 2 | 1333.047** | 606.26** |
| $P_1+P_2V_s.F_1$ | 1 | 938.88** | 405.17** |
| $P_1V_s.P_2$ | 1727.20** | 807.36** | |
| Lines $V_s$. Testers | 1 | 139.92* | 29.52 ns |
| Hybrids $V_s$. Parents | 1 | 76.15 ns | 103.70** |
| Error | 100 | 46.86 | 19.63 |
| CV (%) | - | 17.92 | 16.52 |

***: Significant at the 5% and 1% levels of probability, respectively. ns: Non-significant.

**Table 4.** Analysis of variance for epistasis.

| Source of variation | df | 2007 | 2008 |
|---|---|---|---|
| Total epistasis | 12 | 5926.00** | 1529.85** |
| Epistasis ( i ) type | 1 | 17775.55** | 620.84** |
| Epistasis (I + j) type | 11 | 4848.78** | 1612.49** |
| Epistasis (i ) × Replication | 2 | 17.67 | 14.515 |
| Epistasis (I + j) ×Replication | 22 | 254.76 | 128.65 |
| Total epistasis × Replication | 24 | 235.00 | 116.22 |

***: Significant at the 5 and 1% levels of probability, respectively.

2001). Direction effect of dominance (F′) was determined as -1/4 F′= cov (sum. diff.). If F′ is negative it show that dominant alleles have decreasing effects, but if F′ is positive, it indicates dominant alleles have increasing effects. Combined analysis of variance was also performed for genotypes.

## RESULTS

Analysis of variance revealed significant differences among genotypes with respect to disease severity in 2007 and 2008 cropping seasons. Significant mean of squares were observed for lines and testers, crosses and parents *vs* crosses indicating that the parental lines used in present study responded differently to common smut in both growing seasons (Table 3). The significant mean of squares of $P_1$ *v*$_s$. $P_2$ and $P_1$ + $P_2$ *v*$_s$. $F_1$ for resistance to common smut showed existence of useful variations between testers ($L_1$ and $L_2$). High significant differences between $L_1$ and $L_2$ resulted into expression of high mean performance of their $F_1$ ($L_3$) as revealed by significant mean of squares due to $P_1$+ $P_2$ *v*$_s$. $F_1$. Since the two testers represented highly significant differences for resistance to common smut, therefore, they would provide precise estimate of epistasic variance, however, in the absence of epistasis we could estimate unbiased additive and dominance variance (Khattak et al., 2001, 2002; Virk and Jinks, 1997).

The analysis of variance for epistasis (Table 4) revealed significant overall epistasis for common smut in both growing seasons as was indicated by significant "$L_{1i}$+ $L_{2i}$- 2$L_{3i}$" variance. Furthermore, both additive and non-additive interactions that is, [i] type (additive × additive) and [I+J] types (additive × dominance and dominance × dominance) were also significant for resistance to common smut in 2007 and 2008 growing seasons.

Evaluation of response of hybrids to common smut infection (Table 5) indicated that $L_1 \times F_1$, $L_6 \times P_2$ and $L_4 \times F_1$ were the most susceptible hybrids with high disease severity, whereas, $L_2 \times P_1$, $L_6 \times P_1$ and $L_{10} \times F_1$ with low disease severity were determined as resistant hybrids in the growing season. In 2008, $L_3 \times P_2$, $L_6 \times P_2$ and $L_1 \times P_2$ were sensitive and $L_6 \times P_1$, $L_7 \times P_2$ and $L_4 \times P_1$ were resistance hybrids $L_6 \times P_1$ showed resistance to common smut in both growing seasons. In addition, cross of $L_6$, as resistance line, with, $P_1$ (resistant parent) produced resistant hybrid, however, when it was crossed with $P_2$ (susceptible parent), the resulted hybrid showed high susceptible behavior. Therefore, cytoplasmic effect may play a role in resistance to common smut as reported by

**Table 5.** Disease severity of maize common smut in 12 inbred lines and three testers in two growing seasons.

| | | $L_1$ | $L_2$ | $L_3$ | $L_4$ | $L_5$ | $L_6$ | $L_7$ | $L_8$ | $L_9$ | $L_{10}$ | $L_{11}$ | $L_{12}$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **2007** | $P_1$ | 33.33 | 5.68 | 19.36 | 16.03 | 35.76 | 5.53 | 40.50 | 21.83 | 19.76 | 8.76 | 25.00 | 12.83 |
| | $P_2$ | 38.40 | 29.98 | 58.20 | 33.76 | 38.90 | 64.66 | 17.66 | 32.60 | 12.23 | 36.26 | 29.93 | 19.53 |
| | $F_1$ | 75.50 | 38.70 | 21.66 | 61.00 | 33.90 | 22.53 | 36.50 | 49.16 | 31.66 | 4.20 | 56.43 | 30.40 |
| | | $L_1$ | $L_2$ | $L_3$ | $L_4$ | $L_5$ | $L_6$ | $L_7$ | $L_8$ | $L_9$ | $L_{10}$ | $L_{11}$ | $L_{12}$ |
| **2008** | $P_1$ | 36.93 | 15.66 | 16.03 | 10.33 | 10.73 | 6.50 | 24.03 | 15.26 | 16.60 | 8.76 | 25.00 | 12.83 |
| | $P_2$ | 45.16 | 29.60 | 49.86 | 28.46 | 29.23 | 45.26 | 8.56 | 34.66 | 25.66 | 36.26 | 29.93 | 19.53 |
| | $F_1$ | 35.03 | 40.70 | 19.00 | 34.06 | 18.96 | 18.73 | 29.63 | 33.83 | 21.26 | 4.20 | 56.43 | 30.40 |

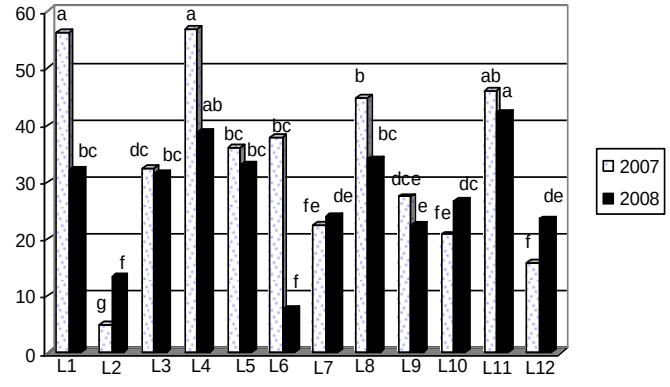


**Figure 1.** Disease severity for 12 lines in two 2007 and 2008 growing seasons. Bars followed by at least one letter in common, in each growing season, are not significantly different at the 5% level of probability.

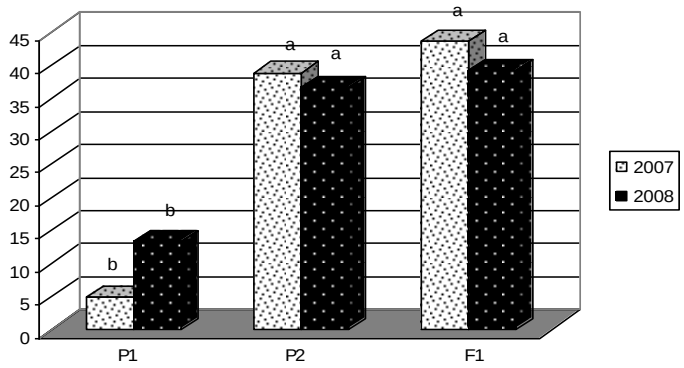


**Figure 2.** Disease severity for three testers in 2007 and 2008 growing seasons. Bars followed by similar letters, in each growing seasons, are not significantly different at the 5% level of probability.

Marton et al. (1985) and Choukan et al. (2008). Furthermore, this occurred in $F_1$, because $P_1$ was a resistant parent (male) and $P_2$ was a susceptible parent (female), therefore, $F_1$ was susceptible in two growing seasons (Figure 2).

In 2007, resistant hybrids resulted from cross between resistant $L_2$ and $P_1$. In $L_{10}\times F_1$ cross, probably $F_1$ (female) transferred resistance genes to progenies, therefore, its crosses with $L_{10}$ (resistant parent) developed into resistance hybrid (Figure 1). In 2008, cross between $L_4$ and $P_1$ was resistant due to resistant $P_1$ (male) parent. Although, $P_2$ (female) and $L_7$ (male) were susceptible and moderately resistant, respectively, their cross was resistant, this may be attributed to complementary gene effects between parental genes that resulted in resistant hybrid. Susceptible hybrids as $L_1 \times F_1$ and $L_4 \times F_1$ (in 2007) had susceptible parents, however, susceptible hybrids ($L_3 \times P_2$ and $L_7 \times P_2$) in 2008 resulted from susceptible and moderately susceptible parents.

Analysis of variance for sum ($L_{1i} + L_{2i}$) and differences ($L_{1i} - L_{2i}$) to assess direct test for additive and dominance component revealed significant effects of sums and differences in explaining the role of dominant gene effect in controlling resistance to common smut in maize in two growing seasons (Table 6). In 2007, dominance ratio $(H/D)^{1/2}$ was 1.3 implied over-dominance gene action in resistance to common smut in maize, however, this ratio, in 2008, was 1.0, suggesting presence of both additive and non-additive gene actions.

The direction of effect of dominance ($r_{s,d}$) for common smut resistance in maize in 2007 and 2008 was not significant which implied that the extent of gene effects of both increasing and decreasing alleles, dominant and recessive, respectively, were similar. Based on combined analysis of variance all the sources of variation were significant (Table 7).

## DISCUSSION

Genetic architecture of any crop species has a great bearing on success in its breeding procedures. It was proved that estimates of genetic parameters would be biased in presence of epistasis. It is imperative to get a clear picture by getting unbiased estimates of such parameters (Sofi et al., 2006). The triple test cross is one of the multiple mating designs that provide estimates of genetic architecture of polygenic traits. It is equally applicable in detecting epistatic bias in segregating and

**Table 6.** Mean of square of sums ($L_{1i}$+ $L_{2i}$) and differences ($L_{1i}$- $L_{2i}$) estimates of additive (D) and dominance (H) components, and dominance ratio $(H/D)^{1/2}$, narrow sense heritability ($h^2_n$) and correlation coefficient for sums and differences ($r_{s,d}$) from a triple test cross for resistance to common smut in maize in 2007 and 2008 growing seasons.

| Components | 2007 | 2008 |
|---|---|---|
| Sum ($L_{1i}$+ $L_{2i}$) | 805.14** | 614.98** |
| Difference ($L_{1i}$- $L_{2i}$) | 1394.90** | 563.79** |
| D | 489.78 | 375.48 |
| H | 853.78 | 654.47 |
| $(H/D)^{1/2}$ | 1.3 | 0.96 |
| $H^2_n$ | 0.34 | 0.48 |
| F' | 6251.88 | 3912.29 |
| $r_{s,d}$ | -0.13 | -0.15 |

***: Significant at the 5% and 1% levels of probability, respectively.

**Table 7.** Combined analysis of variance for maize common smut disease severity 2007 and 2008 growing seasons.

| Source of variation | df | SS | MS |
|---|---|---|---|
| Year (Y) | 1 | 1692.82 | 1694.82** |
| Error (a) | 4 | 625.39 | 156.34** |
| Genotype (G) | 50 | 49771.64 | 995.43** |
| G ×Y | 50 | 10957.39 | 219.14** |
| Error (b) | 200 | 6650.05 | 33.25 |
| CV (%) | - | - | 19.76 |

*,**: Significant at the 5 and 1% levels of probability, respectively.

non-segregating generations such as $F_2$, backcross and homozygous lines (Kearsey and Jink, 1968; Chahal and Jinks, 1978).

In this study, significant epistasis was observed for resistance to maize common smut and indicating presence of [i] and [I + J] type of epistasis for this attribute. The [i] type epistasis represents the fixable portion while [I + J] types show non-fixable portions of genetic variations (Saleem et al., 2009).

Ketata et al. (1976) proposed that standard hybridization and selection procedures could benefit from epistasis given it is of [i] type epistasis (additive × additive), however, [I + J] types of epistasis (additive × dominance and dominance × dominance) are not fixable by selection under self fertilization. Therefore, they would not be suitable for developing inbred lines. Subbaraman and Rengasamy (1989) reported that [I + J] types of non-allelic interactions could be useful in the development of hybrids.

The result of present study has revealed that the magnitude of maternal parent effect was more than that of paternal that may be interpreted as importance of cytoplasmic effects for resistance to maize common smut. Morton et al. (1985) and Choukan et al. (2008) also reported similar results. Therefore, it could be concluded that use of resistant genotypes as maternal parent may guarantee development of resistant hybrids.

The estimates of additive and dominance genetic components of resistance to maize common smut were biased to an unknown extent, because of the presence of epistasis. Epistasis was an important component of gene action in controlling resistance to maize common smut as revealed by the present study. Therefore, this component (epistasis) warrants its detection, estimation and consideration in determination of objectives, strategies and methodologies in maize breeding program for this trait. If the presence of epistasis is overlooked, as is the case in many reports- using designs that assume absence of epistasis, one would not only lose the information about the implication of epistasis, but would also obtain biased estimates of additive and dominance components of genetic variation which would led to inefficiency of breeding procedure (Zafar et al., 2008).

The dominance ratio $(H/D)^{1/2}$ in 2007 and 2008 growing seasons indicated over dominance and dominance gene action, respectively, implies that the dominance (H) was more important than additive (D) component in controlling resistance to maize common smut. Therefore, hybrid breeding is suggested as efficient approach in maize improvement for resistance to common smut. Positive F′ indicated that dominant alleles increased common smut disease severity; hence, parents with few dominant alleles are to be employed. Triple test cross employed in various crops have had varying results across years and locations due to mainly genotype × environment interactions, hence, a series of experiments are required for development of an efficient breeding procedures (Rangasamy, 1989; Tefera and Peat, 1997). In this study, combined analysis of variance revealed significant year and genotype × year interactions. Therefore, replicated experiments over years and locations are required for more precise estimation of gene effects.

Generally, recurrent selection procedure may be useful, since it will exploit both additive and non-additive components of variation. Such strategy would assist to increase the frequency of favorable alleles while maintaining genetic variation in breeding population (Doerksen et al., 2003; Sofi et al., 2006). Recurrent selection, infact, sets out favorable changes in the population performances and is designed as to use additive and non- additive components of variation.

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