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Inheritance of Resistance to Downy Mildew Disease in Maize

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ABSTRACT

This study was carried out in the disease nursery at Agricultural Research Station of Sakha during 2014 and 2015 maize growing seasons. Theinheritance of resistance to sorghum downy mildew disease in maize was investigated. The obtained results indicated that, the additive gene effect, additive by additive and additive by dominance gene effects in two tested maize single crosses (SC) were significant and generally were in the resistance direction (have negative sign). While thedominance and dominance bydominance gene effects were notsignificant in two tested maize genotypes. Therefore, recurrent or mass selection scheme using the advanced generations of two tested crosses (SC 10 and SC120) would be appropriate to accumulate genes for resistance to this disease. As well as, the degree ofdominance (patience ratio) for disease in the twotested crosses were in partial dominance and in direction of resistant parent (less than one and has negative sign). Moreover, the estimates of heritability in broad sense were high, while it was low in narrow sense and in the genetic advance in the tested maize crosses. It seems likely that the environmental variation play a serious role in development of this disease. Moreover, the cytoplasmicconstitution of the female parent are interfere in conditioning theinheritance of resistance to sorghum downy mildew disease in maizeespecially in case of the single cross 120.

Keywords: maize, Sorghum, downy mildew, inheritance resistance.

INTRODUCTION

Sorghum downy mildew (SDM)caused by *Peronosclerospora sorghi* Weston and Uppal is a serious disease of maize ,grain and forage sorghum *P. sorghi* perennially infects *Sorghum spp. and Zea mays L.* throughout the world and is characterized by distinct systemic and localized phases .The disease is initiated by hyphae from germinating soil borne oospores or airborne conidia which infect seedlings .Soil borne oospores are the major source of inoculum and yield loss is directly related to oospores initiated infection (Frederiksen,1980).Downy mildew caused heavy losses in grain yield up to 80% depending upon type of cultivar and severity epidemics (Williams,1984; Graig *et al*., 1989;Anahousur and Laxman, 1991 and Sadoma,2003). This disease became a prominent disease of maize globally during the years when rapid spread of sorghum as grain and forage crop. SDM is prevalent in the peninsular India, in the states of Karnataka,Tamil Nadu and Andhra Pradesh causing yield losses of 30% and higher (Payak, 1975 and Krishnappa *et al*., 1995) The SDM infects its host soon after the seedling emergence until about one month after planting, causing yield losses of 50 – 100% for susceptible cultivars (Jampatong *et al.,* 2013). It was recognized that the genetic basis of SDM resistance must be understood for efficient breeding programs. So it exists enough scope to improve productivity of maize through breeding for high yielding single cross maize hybrids with resistance to downy mildew disease. However, utilization of host plant resistance seems to be the most effective and economical way for controlling sorghum downy mildew (SDM) in maize (Premalatha *et al.*, 2012). It can be concluded that , host –plant resistance is an effective and economical means of controlling downy mildew (DM) diseases in maize (*Zea mays* L.) Singbaraudom and Reufro 1982). Host resistance is more effective in the SDM management and resistance has been reported to be under polygenic control necessitating identification of resistant quantitative –trait Loci QLs (Lohithaswa *et al*., 2015)

Therefore, the presented work aimed to study the inheritance of resistance to downy mildew disease in maize, and to choice the scheme that will breeder using it for development of resistant maize genotype.

MATERIALS AND METHODS

This study was carried out in the disease nursery at Agricultural Research Station of Sakha during 2014 and 2015 growing seasons and its prepared to studying sorghum downy mildew (SDM) disease reaction. The field layout was prepared as follows:

Preparation of field downy mildew disease nursery

The highly susceptible Sudan grass (*Sorghum sudanens*) piper Black variety was sown in every fourth row throughout the field at least four weeks prior to the expected date of planting of tested maize genotypes. Three rows of the surrounding border were also planted with the same Sudan grass variety. Sudan grass rows act as infector plants to help spreading the asexual spores (conidia) to the tested materials and to serve as indicator for the uniform distribution of the disease inoculums throughout the field. After establishing of the infector rows, and after the appearance of abundant sporulation of the pathogen by producing downy growth on the leaf surfaces (4 weeks), the tested rows of tested maize genotypes were planted. After emergence, seedlings were challenged by conidia needed for infection blown by wind from the infector rows. Sudan grass was cut monthly about 20- 30 cm above soil level. This is to increase spores production needed for infection around the tested materials.

Field trails:

Tow commercial single crosses (SC) of maize were developed as follows:

1 – SC 10 as obtained by crossing between line 7 (moderate resistance) x line 63 (resistance).
2 – SC 120 as obtained by crossing between line34 (susceptible) x line 63 (resistance).

Pedigree of the parental lines used in this experiment are shown in Table (1).Percentages of downy mildew disease were estimated using the formula according to Chaube and Punder (2005).

$$\text{Infection(\%)} = \frac{\text{No. of infected plants}}{\text{Total no. of plants (healthy + infected plants)}} \times 100$$

In 2014 growing season, three parental lines were grown and crossed to obtain the F1 seeds. Seeds of the F1 were planted in 2015 growing season and at flowering were selfed and crossed to their parents to obtain F2, and two backcrosses i.e. BC1 and BC2 seeds; respectively.

Table 1. Pedigree of the parental lines and the code number for each genotype

Genotypes	Origin
Inbred line 7	Developed from American early variety by Maize Section, A.R.E.
Inbred line 34	Developed in maize program in Sakha agriculture research station.
Inbred line 63	Developed from tep.5 variety by Maize Section, A.R.E.

Seeds of six population i.e. P1, P2, F1, F2, BC1, and BC2 of each single cross were grown in separated experiments. Each experiment was arranged in a randomized complete block design with four replications. Each replicate included three rows from each parent (P1 and P2) and F1.and four rows for each backcrosse (BC1 and BC2), and seven rows for F2 plants (segregating generation). The rows were 5m long and 70cm width. Each row included 20 hills, 25 cm distance. This experiment was carried out under field disease nursery and late sowing date (25 July). All agricultural practices were applied as recommended. The disease was estimated after 35 days from sowing on individual plant for each generation according the scale which illustrated by EL-Zeir and Tolba (1999) as follows:

0=no infection, 1= local infection and 2= systemic infection.

Statistical and genetical analysis:

Statistical and genetic analysis for downy mildew disease resistance were calculated according to Gamble (1962), and Mather and Jinks (1977).

The mean, variance and standard deviation were calculated for parents (P1 & P2), F1, backcrosses (BC1 & BC2) and F2 populations of each cross (SC10 and 120).

Type of gene effects, nature of dominance, heritability in broad and narrow senses, and expected genetic advance upon selection were estimated as following:

Type of gene effects:

The following formulae given by Gamble (1962) were used to estimate the type of gene effects:

Additive gene effect (a) =$\bar{B}C1-\bar{B}C2$

Dominance gene effect(d)=$\bar{F}1+2\bar{B}C1+2\bar{B}C2-½\bar{P}1-½\bar{P}2-4\bar{F}2$

Additive × Additive (aa) = $2\bar{B}C1+2\bar{B}C2-4\bar{F}2$

Additive × Dominance(ad)=$½\bar{P}2+\bar{B}C1-½\bar{P}1-\bar{B}C2$

Dominance × Dominance(dd)=$\bar{P}1+\bar{P}2+2\bar{F}1+4\bar{F}2-4\bar{B}C1-4\bar{B}C2$

The variance (V) formulae were as following:

The variance of additive gene effect (Va) =$V\bar{B}C1+V\bar{B}C2$

The variance of dominance geneeffect (Vd) = $V\bar{F}1+16V\bar{F}2+4V\bar{B}C1+4V\bar{B}C2+¼V\bar{P}1+¼V\bar{P}2$

The variance of additive × additive (Vaa) = $4V\bar{B}C1+4V\bar{B}C2+16V\bar{F}2$

The variance of additive × dominance(Vad) = $¼V\bar{P}2+V\bar{B}C1+¼V\bar{P}1+V\bar{B}C2$

The variance of dominance × dominance(Vdd) = $V\bar{P}1+V\bar{P}2+V\bar{F}1+16\bar{F}2+16\bar{B}C1+16\bar{B}C2$

Where:

$\bar{P}1$, $\bar{P}2$, $\bar{F}1$, $\bar{F}2$, $B\bar{C}1$ and $B\bar{C}2$ is the means of P1, P2, F1, F2, BC1 and BC2, respectively.

Nature of dominance:

To study the nature or degree of dominance {the potency ratio (p)} was calculated according Mather and Jinks (1977) as following:

$$\text{Potency ratio (p)} = \frac{\bar{F}1 - ½(\bar{P}1 + \bar{P}2)}{½(\bar{P}2 - \bar{P}2)}$$

Complete dominance is assumed when (P) equals 1, while, partial dominance is presumed when (P) equals less than 1, and over-dominance is existed when scores more than 1. The positive and negative signs indicate the direction of dominance to either parents.

Heritability estimates:

Heritability was estimated in both broad and narrow senses according Mather and Jinks (1977) as follows:

Heritability in broad sense (HBS) for F2 generation =

$$\frac{½VA + ¼VD}{½VA + ¼D + VE} \times 100$$

Heritability in narrow sense (HNS) for F2 generation=

$$\frac{½VA}{½VA + ¼V + VE} \times 100$$

Where:

VE (Variance of environmental) = VP1+VP2+VF1/3

VA = Variance of additive gene effect

VD = Variance of dominance gene effect

Expected genetic advance upon selection:

The expected genetic advance (Gs) upon selection was calculated using methods developed by Mather and Jinks (1977) as follows:

$Gs = K.\ HNS.\ \sigma F2$

Where:

Gs= the expected genetic advance, K = 2.06

HNS= Heritability in narrow sense, and $\sigma f2$= F2 standard deviation.

RESULTS

Inheritance of resistance to downy mildew disease in maize plantsPwas studied. Mean performance of parents and both of it, cross populations disease reaction in the two tested crosses was represented in Table (2).

Table 2. mean performance of parents (P1,P2), F1,F2and back crosses (BC1,BC2) populations disease infection for the two tested crosses

Population	Disease infection%	
	SC 10	SC 120
P1	8.33e	8.33f
P2	18.33c	36.76c
F1	10.03d	29.66d
F2	46.87b	49.43b
BC1	6.25f	20.00e
BC2	51.25a	67.50a
CV	0.747	2.397
L.S.D at 5%	0.325	1.605

Data in Table (2) gives means performance of parents (P1 and P2)), first and second generations (F1and F2) and back-crosses generations (BC1 and BC2) in single crosses 10 (resistance hybrid) and 120 (susceptible hybrid) maize cultivars, for disease infections %.

The means values for single cross 10 indicated that P1 (line 63) as a male parent have the lowest infection percentage as compared with P2 (line 7) the values were 8.33 and 18.33% in P1and P2, respectively. While the first generation was intermediate (10.03%) between them. The F2 (segregation generation) was recorded high infection percent (46.87%). The back-crosses BC1 was recorded the lowest infection % (6.25%) comparing with BC2 which have the highest infection % (51.25%).

The means values for single cross 120 indicated that , P1 (line63) as male parent was recorded low infection % (8.33%) as compared with P2(line 34) which was recorded 36.76 infection percent, while the F1 generation

was intermediate value(29.66%) between two tested parents (P1 and P2), the F2 generation was recorded high infection percentage value (49.43%). On the other hand, the BC1was recorded low infection % (20.00) comparing with BC2 generation which recorded the highest infection percentage (67.50) by the tested disease.

Type of gene actions:

Table (3) gives the estimates of type of gene actions i.e. additive (a), dominance (d) and epitasis (aa, ad and dd) for the infection percent of downy mildew disease in SC10 and SC120 maize cultivars. The presented data in Table (3) reported that, the additive gene effect in two tested maize cultivars was significant and generally was in the resistant direction (has negative sign), the values were -1.99 and -2.01, the values of additive by additive(aa) were also highly significant and were also in the resistant direction, these values were -2.70 and -2.54, the values of additive by dominance(ad) were also significant and in resistant direction ,it recorded -2.08 and -2.12 in SC10 and 120 maize cultivars , respectively.On the other hand, estimates of dominance gene effect was not significant and in resistant direction in case of sc10 (has negative sign), while it was in susceptible direction in case of SC 120(has positive sign), the values were -1.81 and +1.65, moreover, the dominance by dominance (dd) was also not significant and in resistant direction in case of SC10(has negative sign) and in susceptible direction in case of SC120 (has positive sign), the values were – 0.73 and + 0.85 inSC10 and 120 maize cultivars , respectively. The above mentioned results indicated that, the additive gene effect was play quite important role in inheritance of resistance to downy mildew disease in maize.

Nature and degree of dominance (patience ratio):

Data presented in Table (3) showed that, the potency ration values for degree of dominance were less than one and have negative sign (-0.6)in sc10 and positive sign (+0.4) in SC120 cultivars, indicating that partial dominance was observed in direction of the resistant parent in case of SC10 and in direction of susceptible parent in case of SC120.

Heritability % in broad and narrow sense:

Data presented in Table (3) illustrated that, the heritability estimates in broad sense were very high in two tested single crosses i.e. SC10 and SC120, the values were 85.0 and 80.2%, respectively. On the other hand, the heritability estimates in narrow sense were slightly low, the values were 21.0 and 18.0 % in SC10 and SC120 maize cultivars, respectively.

Genetic advance%:

Data presented in Table (3) found that the genetic advance upon selection values in SC10 and SC120 were recorded suitable values i.e. 29.41 and 25.58 % respectively.

Table 3. Estimates of type of gene action, degree of dominance, heritability% (in broad and narrow senses), and genetic advance % for infection% by downy mildew disease in maize single crosses 10 and 120 cultivars.

Genetic advance (%) upon selection	Heritability%		Degree of dominance	Type of gene action					Downy mildew infection%	Maize hybrids
	Broad sense%	Narrow sense%		dd	ad	aa	Dominance (d)	Additive (a)		
29.41	85.0	21.0	-0.6	-0.73	-2.08*	-2.70**	-1.81	- 1.99*	10.03	SC 10
25.58	80.2	18.0	+0.4	+0.85	-2.12*	-2.54**	+1.65	-2.01*	29.66	SC 120

***and** significant at 0.05 and 0.01 levels of probability, respectively.**

Data presented in Table (4) showed the role of cytoplasmic constitution of the female parent in determining the inheritance of resistance to sorghum downy mildew disease in maize, the obtained data indicated that, the female cytoplasmic was very importance especially in case of SC 120 maize cv.,since, the susceptibility character was transferred from female parent (Line 34) to the F1 hybrid (SC120).

Table 4. The cytoplasmic constitution of the female parent in determining the inheritance of resistance to sorghum downy mildew disease in maize

F1 hybrid	Male parent		Female parent
SC10 (R)	Line 63 (R)	X	Line 7(MR)
SC 120 (S)	Line 63 (R)	X	Line 34(S)

DISCUSSION

Breeding for disease resistance is regarded as one of the most economic and important means in the integrated control of the downy mildew disease of maizeby incorporating genetic resistance available from desirable sources.However, studying inheritance of resistance to downy mildew disease in maize was quite importance for choice of the scheme which will breeder using it for development of resistant maize genotypes. The obtained results in this study indicated that, the additive gene effect , additive by additive and additive by dominance gene effects in two tested maize single crosses were significant and generally were in the resistance direction (have negative sign). While the dominance and dominance by dominance gene effects were not significant in two tested maize cultivars. This result explained that, the additive gene effect was effective and play quite important role in inheritance of resistance to downy mildew disease in maize. Therefore, either recurrent or mass selection scheme using the advanced generations of two tested crosses (sc10 and 120) would be appropriate to accumulate genes for resistance to tested disease. These results were in accordance with those reported by Motawei (2011) who illustrated that, the additive type of gene effect was predominant and of higher magnitude than non-additive gene effect for inheritance of resistance to downy mildew disease of maize. Similar results were also obtained by EL-Zeir and Tolba (1999), Raswandi *et al.* (2014) and Maruthi and Jhansi Rani (2015), they found that, the additive gene effect play an important role in conditioning resistance to the downy mildew disease in maize, therefore, resistance to downy mildew in tested maize inbred lines is governed by additive gene action; hence, selection procedures can be used to improve the level of downy mildew resistance in tested new inbred lines. The breedingprogram will

consider development of conventional hybrids, such as single crosses, three way crosses, and top crosses. The presented results also indicated that, the average degree of dominance (potency ratio) for infection by downy mildew disease in two tested crosses were in partial dominance and in direction of resistant parent (less than one and has negative sign). Moreover, the estimates of heritability in broad sense were high, while the heritability estimates in narrow sense and estimates of genetic advance were low in tested maize crosses. It seems likely that the environmental variation play a serious role in development of infection. These results are agreement with EL-Zeir and Tolba (1999); and Motawei (2011) ,they reported that, heritability of downy mildew disease in broad since estimates was high ,it ranged from 74 to 94 % , while it was low in narrow since estimates. The genetic advance value was also low. It is means that the environmental deviation plays a serious role in the inheritance of resistance to downy mildew disease in maize. Moreover, the obtained results also indicated that, the cytoplasmic constitution of the female parent play an important role in determining the inheritance of resistance to sorghum downy mildew disease in maize, especially in case of SC 120 maize cv.

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توريث المقاومة لمرض البياض الزغبى في الذرة الشامية

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تم تنفيذ هذه الدراسة بحقل العدوى بمحطة البحوث الزراعية بسخا خلال موسمى 2014 و 2015 م. حيث تم دراسة توريث المقاومة ضد مرض البياض الزغبى في الذرة الشامية. وقد دلت النتائج المتحصل عليهاعلى الأتى: أن الفعل الجيني المضيف وكذلك الفعل الجيني لمضيف مع المضيف , وأيضاالفعل الجيني المضيف مع الفعل الجيني السيادي كانوا ذومعنوية وفى إتجاه الأب المقاوم للاصابه (ذات إشاره سالبه). بينما كان الفعل الجيني السيادي وأيضا الفعل الجيني السيادي مع السيادي كان غيرمعنوي, في كل من صنفي الذرة المختبره. وبناءاعلى هذه النتيجة فإن الإنتخاب الإجمالى أوالمتكررفي الأجيال المتقدمه للهجينين المختبرين (هجين فردى 10 وهجين فردى120) ممكن أن يؤدى إلى تجميع جينات المقاومه لهذا المرض. إن درجة السيادة للصفة المرضيه في صنفي الذرة المختبرين , كانت سيادة جزئيه وفى إتجاه الأب المقاوم للاصابه (قيمتها أقل من الواحد الصحيح و القيمة تحمل اشارة سالبة). علاوة على ذلك فإن نسبة التوريث في معناها الواسع كانت مرتفعه, في حين كانت منخفضة في معناها الضيق وفى مقدار التحسين الوراثي بالانتخاب في صنفي الذره تحت الإختبار .هذه النتيجة تدل على أن الظروف البيئيه تلعب دورا هاما وخطيرفي تطور هذا المرض. كما دلت النتائج أيضاعلى أن السيتوبلام الأموى يلعب دورا هاما في وراثة صفة المقاومه لمرض البياض الزغبى في الذره الشاميه وخاصة في حالةالهجين الفردي 120.