Forage Res., **47** (1) : pp. 11-18 (2021) **http://forageresearch.in**

GENETICS OF RESISTANCE AGAINST *HELMINTHOSPORIUM* IN MAIZE (*ZEA MAYS* L.): AN OVERVIEW

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(Received : 9 May 2021; Accepted : 25 June 2021)

SUMMARY

Maize (*Zea Mays*) plant is affected by number of foliar diseases among of them, Southern Corn Leaf Blight (SCLB) or *Maydis* Leaf Blight (MLB) incited by the *Helminthosporium maydis* anamorph: *Bipolaris maydis*, telomorph: *Cochliobolus heterostrophus* is one of the most devastating disease of maize in India as well as in the world. Three different races namely race O, T and C of *helminthosporium* are found responsible for causing this disease. *Bipolaris* has the potential to reduce the yield significantly up to 70% depending upon the susceptibility, race of pathogen and environmental conditions.Genetic architecture of maize possess the diverse source of resistance against *Helminthosporium* which are qualitative, quantitative and multiple disease resistance types in nature but often represents quantitative nature of resistance. A single gene, *rhm*, imparts a qualitative-like resistance against race O which inherits recessively with additive gene action predominantly. It is implied at the seedling stage but quantitative resistance is needed after silking stage. An additional two resistance genes have been identified in the inbred NC250 that are not allelic to *rhm* and this resistance is maintained in mature plants. Plenty of attempts have been made to identify the resistance source and to dissect the quantitative trait loci (QTL) responsible for resistance or multiple disease resistance to SLB across different maize populations. QTLs responsible for multiple disease resistance were identified of elucidated with the help of meta analysis of available studies.

Key words : Maize, *Helminthosporium maydis,* genetic architecture, QTL mapping, *rhm* allele, qualitative and quantitative resistance gene and meta analysis

Maize (*Zea Mays* L.) belongs to family poaceae and tribe maydeae and considered as foremost principal cereal crop which is being cultivated for food, feed and fodder. In India, maize is grown for dual purpose *i.e.* for grain as well as fodder. Maize also provides raw material for various agro based and other industries (Arya *et. al*., 2015). It is cultivated worldwide under warm and humid conditions. Being C_4 plant, maize represents highest potential of carbohydrate productivity per day (Dayal *et. al*., 2014). Queen of cereals (Maize) is attacked by more than 65 pathogens including fungi, bacteria and viruses. Few of them are causing serious reduction in yield (Rahul and Singh, 2002). Southern Corn Leaf Blight (SCLB), Northern Corn Leaf Blight (NCLB), Grey Leaf Spot (GLS) and different type of rust are few major fungal diseases associated with maize. Southern Corn Leaf Blight (SCLB) which is also known as *Maydis* Leaf Blight (MLB) is significant foliar disease which is incited by fungus *Helminthosporium maydis* or *Cochliobolus heterostrophus* (or *Bipolaris maydis* (Nisik.) Shoemaker), which is a necrotrophic ascomycete, forming tan elliptical lesions, which occurs after anthesis (White, 1999). This disease prevails under wide range of environment from warm humid temperate to tropical and temperature ranging between 20-30^{0}C. Temperature affects the spore production (Warren, 1975). It is affecting the crop almost all maize growing regions but under favorable conditions such as hot and humid tropical and temperate conditions, has the potential to reduce the yield up to 65 to 70 % in hot and humid tropical and temperate areas of the world (Wang *et* al., 2001, Ali *et* al., 2011a). MLB can cause considerable grain yield loss depending upon the race, susceptibility of the variety and environment (Thompson and Bergquest, 1984). MLB is caused by three different races namely O, T and C of the same pathogen. During the year 1970, in southern part of United States, MLB caused the serious epidemic, which seriously devastated whole crop leading to the complete failure of crop due to race T, while in entire United State, yield reduction was estimated by 15 %. While the 50% yield loss that southern cms-T hybrids suffered during 1970-71 is

the exception, even hybrids with moderate genetic resistance to SLB have been shown to suffer yield losses of 0.7-0.8% for every 1% increase in affected leaf area between 0 and 25% (Hooker, 1972; Byrnes and Pataky 1989). Extent of yield loss was up to 40 % or more when crop inoculated with race 'O'in inoculated yield loss trials.

Disease symptoms and cycle

Symptoms developed by pathogen first appear on lower leaves and proceed on entire plant. The symptoms when infected with strain "O" appears as adolescent, tiny and diamond shaped lesions and elongates upon maturation. Extension of lesions is limited by adjacent veins resulting into rectangular shapeof lesion. Lesions may coalesce which gives burning effect to large areas of the leaves. Symptomatic lesions which are produced by race T are different in shape and size. They are oval in shape and larger in size than those produced by the O strain. (Fig.1). Race 'T' strain severely damage the maize cultivars having t-male sterility. *Bipolaris maydis* represents polycyclic disease cycle. It liberates both asexual conidia and sexual ascospores. Asexual cycle occurs in nature and is of primary concern.

Race O and T can be distinguished with the help of pathogenicity test and study of physiological/ morphological characters on culture media (Leonard, 1977; Warren *et al*., 1977).

Cytology of disease

T-toxin, which is produced by race T, is a long chain of polyketides which precisely binds to URF13 which is encoded by *T-urf13* gene found in mitochondrial genome of cms-T maize (Wise *et al.,* 1987). This URF3 is a polypeptide of 13 kDa and found in mitochondria (inner membrane) and acts as a ligand gated channel (Levings, 1990; Levings and Siedow, 1992). This membrane starts to leak and lose function once T-toxin binds to URF13.This leads to death of and pathogen starts to colonize in maize tissue. It represents the first evidence of role of mitochondria in causing plant disease. It also clearly set forth the significance of mitochondrial solidarity in control of eukaryotic cell death. Conidia are capable to germinate followed by penetration in the epidermal cells and stomata of host of both susceptible and resistant hybrids. Chloroplast destructs followed by collapse of cell wall when hyphae enter into the chlorenchymatic tissue of the susceptible plants. (Hesseltine *et al.,* 1971). Resistant factor(s) in maize against *Helminthosprium Maydis* and *Exserohilum turcicum*is seems to be present in the chlorenchyma and in xylem respectively (Hilu and Hooker, 1965; Hesseltine *et al*., 1971).

Genetics and inheritance pattern of disease resistance

Resistance in maize for SCLB is classified into two major classes *i.e.* Qualitative resistance (major gene resistance) which shows major effects and provides resistance which is race-specific. Quantitative resistance (Minor gene resistance) has multigenic basis, shows minor effects and generally provides intermediate level of resistance which is non race specific. Qualitative resistance which shows hypersensitive response (HR), is quickly breakdown

Fig. 1. Symptoms of *Maydis* leaf blight.

in the field, however few exceptions are available (Steffenson, 1992) but quantitative resistance governed by quantitative genes tends to be more durable (Parlevliet 2002). Qualitative and quantitative resistance is basically potent against biotrophic and necrotrophic pathogens respectively. Another type of disease resistance where more than one disease is controlled by single gene is called multiple diseases resistance (MDR) but this phenomenon is yet to be explored well. Most of the maize hybrids showed moderate level of quantitative resistance to MLB, which was governed by several genes with partial dominance in nature (Pate and Harvey, 1954). The basic concept underlying the phenomenon of resistance is R-genes (Jones and Dangl, 2006). Interaction between resistant gene and pathogens is similar to key-lock approach. If pathogen has the virulence genes, it is capable to infect the host irrespectiveits genetic architecture of the host plant. Most of R genes codesfor the nucleotide binding site (NBS) and a leucine-rich-repeat (LRR) region. Almost all plant species are found with the abundance of NBS-LRR types of genes (Meyers *et al*, 2003) and maize genome is available with 109 NBS-encoding genes (Cheng *et al*, 2012). The host defence reaction is supposed to suppress by the suppressor which is encoded by *Rhm* alleles (Keen 1990). On the other hand, a host function is encoded by *Rhm* allele which is identified by the pathogen. Host devoid of *rhm* alleles abscond the recognition by the pathogen. (Chang and Peterson,1995). Different types of sterile cytoplasm are found in maize and specificity of available races of pathogen to available sterile cytoplasm is different which is given in table 1. Resistance against race T is mainly governed by cytoplasmic factors and determined by nuclear factors which inherit quantitatively and shows dominance and additive effects (Hooker 1978; Johnson 1976). Resistance against race O is governed by nuclear genes only.

Qualitative and quantitative resistance against different races is further discussed as given below:

A) Qualitative resistance

It often refers to an "all-or nothing" disease response that is conditioned by the availability or non availability of a single major gene. Although a single gene, *rhm*(resistance to *Helminthosporium maydis*), imparts a qualitative-like resistance to SLB Race O, it is inherited recessively. It is still yet to be confirmed whether *rhm1* involves in resistance against race T, but its effectiveness lies at seedling stage only and it is linked with few alteration at gene expression or protein levels (Simmons *et al*. 2001).To describe the resistance for race O, different models have been proposed like; Pate and Harvey, 1954, proposed multiple gene model; Craig and Fajemisin, 1969, given two linked recessive gene model and Thompson and Bergquist in 1984 proposed two independent recessive genes with complementary effects model. The one v/s two gene controversy reemerged when transposon tagging was used to map the location of *rhm* and an unusually high mutation frequency could be accounted for only by the presence of two recessive genes, *rhm1* and *rhm2* (Chang and Peterson, 1995). At present time, resistance to race O is considered to be governed by *rhm* gene which was discovered by Smith and Hooker, 1973 in Nigerian material. Since then, it is being used as significant source of resistance against race O and has been transferred to many modern maize cultivars. The location of *rhm* gene was elucidated on chromosome 6 (short arm) and probe UMC85 and agrP144 were showing very closely/tightly association with *rhm* locus (Zaitlin *et al*., 1993). Inheritance pattern of adult plant resistance is not understandable completely, but it is supposed to be governed by two complementary recessive genes which show independent segregation from *rhm* gene. It is shown that *rhm1* gene provides adequate resistance which is qualitative in nature, during early stages of growth, but at the time or after silking, quantitative resistance is needed (Craig and Daniel-Kalio, 1968). *rhm* gene represent the first document attempt of breeders in

TABLE 1
Specificity of three races of SCLB for different sterile cytoplasm in maize

Race	Toxin produced	Susceptible Host	Remark	Reference
Race - T	T-toxin	Maize cultivars possessing Texas male sterile cytoplasm (T-cms)	Plants have gene *T-urf 13*, which encodes for T-toxin	(Wei *et al*. 1988)
Race - C	C-toxin	Maize cultivars having cytoplasm male sterility (C-cms)	Available only in China.	(Hooker *et al*. 1970; Smith, 1975)
Race - O	O-toxin	All maize cultivars having normal cytoplasm (N).	Affects all types of maize plants which are devoid of resistant gene	

USA to use resistant to *B. maydis* from tropical maize germplasm. Global transcript profiling revealed no consistent difference in expression of 8,000 to 13,000 genes between *rhm* mutants and wild-type infected plants 24 hours post-infection (Simmons *et al*., 2001). This recessive mode of inheritance implies lack of functional protein, and thus Avr recognition is not likely the mode of action of this gene. Furthermore, juvenile *rhm* plants exposed to SLB exhibit a chlorotic flecking reaction, but post-anthes is, *rhm* is only partially effective at deterring disease symptoms. During 1950s and 1960s, maize inbreds in which sterility was incorporated from Texas cytoplasm (cms-T) was predominantly used for seed production of hybrids. In other crops, like wheat and rice, qualitative resistance is enormously exploited. Few major gene showing resistance, namely, *Ht, Rp and rhm* genes used in resistant breeding against northern leaf blight (Welz and Geiger, 2000), common rust (Ramakrishna *et al*., 2002) and southern corn leaf blight (Smith and Hooker, 1973).

B) Quantitative resistance

Resistance against race O of *Bipolaris maydis* is elucidated as quantitative in nature which shows additive gene action predominantly. It also displays significant dominance effects in some populations (Lim, 1975; Lim And Hooker, 1976; Thompson and Bergquist, 1984; Burnette and White, 1985; Holly and Goodman, 1989). Major gene which can represent the complete immunity or resistant is yet to be discovered, that is why maize breeders are bound to rely on polygenic, quantitative resistance to SLB.

Classical studies of inheritance

The first study that described quantitative inheritance to SLB was conducted by Pate and Harvey (1954). Crosses between susceptible and resistant inbreds yielded progeny that were always intermediate in resistance, suggesting partial dominance of this trait. Lim and Hooker (1976) studied general and specific combining ability among four double cross parents in a diallel. General, but not specific, combining ability was highly significant, indicating that a parent's genetic contribution to resistance was consistent across all combinations, and independent of its other partner in a cross. Thus, resistance against MLB was displaying additive gene action predominantly. In an evaluation of tropical inbreds and their progeny, Holley and Goodman (1989) identified both additive and recessive forms of gene action. Welz and Geiger, (2000) also reported additive gene action along with declining trend of disease severity in a study related with high heritability and negative value of selection differential. Pate and Harvey (1954) also studied the genetics of resistance in inbred lines and partially dominance was observed. Numbers of genes involved were not confirmed but they suggested involvement of small number of genes based on the ease with which they were able to transfer the resistance by backcrossing. Resistance to race T and O was linked with additive genetic effects and is displaying partially dominant nature (Lim, 1975). He has also observedtight association between resistance and heterosis in single crosses. Lim and Hooker (1976) also studied the genetics of resistance in double cross hybrids and showed the predominance of additive gene effects. It was observed that major part of total variation in resistance is contributed by the additive genetic effect (Sheih and Lu, 1993). Shah *et al*. (2006) observed differences for SCLB which were highly significant ($p<0.01$) in two maize populations while Rahman *et al.* (2005) observed significant differences among the maize varieties for resistance to MLB. Inbreds with stay-green character had high photosynthetic activities, high protein and lipid content and showing resistant to MLB (Choi *et al*., 1994) Maturity is also considered as an important factor in resistance for SCLB. Late maturing genotypes were showing more resistance to disease than early maturing genotypes (Ceballos *et al*., 1991). If weather conditions are congenial and infection occurs prior to silking, it can cause the damage at significant level.It is elucidated that different genetic system are involved in additive and recessive type of gene action. Resistance against MLB supposed to be governed by few genes which act in a positive epistatic fashion with the resistant gene (Holley and goodman, 1989). Lim and Hooker (1989) studied some quantitative source of resistance available in corn belt germplam by using different combinations of normal and cms-T cytoplasm in conjunction with race O and/ or race T of pathogen. There research demonstrated that resistant gene were partially dominant and that no cytoplasmic-genomic interaction was associated with resistance. The efficiency of S_1 recurrent selection for yield and attributing traits under inoculums of MLB was evaluated. Based on frequency distribution curve, most of the S_1 lines were representing moderate resistance indicating the efficiency of S_1 line recurrent selection to improve resistance against MLB because

S_1 lines were supposed to have high concentration of proteins, lignins, phenolics and callose which is adding in resistance to SCLB (Durrishahwar *et al,* 2008). In resistant/moderate resistant lines, grain yield is not affected by the pressure of inoculums (Shivankar and Shivankar, 2000).

Locating and mapping of QTLS

Since resistance against SCLB governed quantitatively, QTL mapping is being used to locate different genetic loci. Number of attempts have been made to dissect quantitative trait loci (QTL-chromosomal region affecting the expression of a quantitative trait) responsible for determining resistance to SLB in different segregating populations (Poland *et al.,* 2011; Carson *et al*., 2004; Balint-Kurti *et al.*, 2005, 2006 and 2008; Zwonitzer *et al*., 2009; Negeri *et al.*, 2011; Kump *et al*., 2011; Liu *et al*., 2011 and many more.). However, genes underlying the response were difficult to identify due to lack in the precision of the positional estimates of the resultant loci. Furthermore, germplasm studied to elucidate the quantitative loci offers the limited opportunity to examine only two alleles per locus that is why usefulness of results are very confined because significant number of alleles yet to be studied. The genes underlying a quantitative trait are more difficult to map for several reasons, most of which stem from the segregation of multiple loci for the trait, the small effects these individual genes have and the interactions both within the organism's genome and between its genome and environment (Lynch and Walsh, 1998; Mackay, 2001). Some QTLs gives the same temporal and spatial expression. Due to the interaction between genotype and environment there is possibility to detect the different QTL under different circumstances (Yan *et al*., 2003).

Source of resistance to SCLB in maize

SCLB is considered as one of the most destructive disease and can cause the significant yield reduction in maize crop. But, at the same time, great potential lies for disease resistance. Identification of the resistant source/gene is extremely important to achieve the greater success in maize breeding programme. Inbred are convenient to usein maize breeding because they can be genotyped, sometime phenotyped. Further, they can be studied in diversified environment because of their genetic uniformity, stability and their vigour (Mubeen et al., 2017). Various successful attempts to identify the resistance source against MLB have been made in different populations. Some of the identified resistant source are RSSSCC6, RBS10C6 (Aziz *et al.,* 1992); P138 and D Huang 212 CN 165, 313, C8605-2, Chang 7-2, Qi 318, Qi 319, Shen 137, Dan 9046, Zhongzi 01, Shen 136, 8065 (Wang et al., 2004); V 334, V 336, V 341, V 345, V 373, V 383, V 398, V 400, V 407, V 418, V QL2, VQL 17, CM 145, CM 153 (Chandrashekara et al., 2014); NRL-4, EV-1097, SP-3, NCML-73, NC-2703, NRL-6 and Local-Y (Mubin et al., 2017) and V53, V 178, V 190, V 336, V 340, V 341, V 345, V 348, CM 104 and CM 145 (Srivastava et al., 2017). In spite of these identified sources, many more examples are available which shows the resistance for SLB.

Multiple disease resistance

Qualitative and quantitative resistance is major types of available resistance in maize. Other than this, multiple diseases resistance (MDR) is also available in maize. In case of MDR, more than one disease is controlled by a single gene(Wisser *et al*, 2005; Chung *et al,* 2010) but this phenomenon has yet to be explored well. A QTL responsible for resistance to several diseases *i.e.* NCLB, GLS and SCLB in RIL population were identified (Zwonitzer*et al*., 2009). To solve this puzzle, the phenomenon of MDR for these three diseases was elucidated with the help of meta-analysis at genome level.The meta-analysis of QTLs can be described as refinement of QTLs position on the consensus map and can be used as an approach to dissect consensus QTLs (Gordon *et al*., 2004). Approx 389 QTLs were identified for these three major diseases which were showing random distribution on all available chromosomes (10 chromosomes) out of which 63 QTLs were showing multiple disease resistance *i.e*. they were controlling more than one disease (Ali *et al*., 2013). Each resistant QTL/gene is located on specific region of a chromosome. For resistance against NCLB, chromosome 3 harbors the maximum number of QTLs while chromosome 10 harbors the least number of QTLs. For SCLB resistance, chromosome 3 and 1 possess maximum QTLs. Among the three diseases, minimum QTLs were identified for grey leaf spot (GLS) and maximum of those identified QTLs were located on chromosome 4 and minimum on chromosome 6. More than 15 QTLs were observed for each disease on chromosome number 4 that is why this chromosome is found to be

more consistent for all three diseases. In case of actual or real QTLs, total 44 real QTLs were identified in maize genome based on 4 QTL as standard which were randomly distributed on different chromosomes. Maximum number of real QTLs was harbored by first three chromosomes and minimum real QTLs were possessed by chromosome 6 and 10 (Ali *et al*., 2013). Underlying gene of resistance *i.e*. *Ht1* and *Ht2* also confirmed within the real region of real QTL. Number of pathogens from different taxonomic groups attacks on maize plants. Genes, which governs multiple disease resistance are supposed to be under positive selection pressure (Wisser *et al*., 2011). Meta-analysis revealed several QTL with very low Confidence interval (CI) in meta analysis. Further it can be suggested that several QTLs showing lower CI can be reduced significantly through meta-analysis (Chung *et al*., 2010). Maize genome is available with the abundance of the meta QTLs and more than 60 MDR QTLs for NCLB, SCLB and GLS were observed (Ali *et al*., 2013).

CONCLUSION

MLB is the one of the prominent problem which can drastically reduce maize yield. Inheritance pattern of *Maydis* leaf blight is both qualitative and quantitative in nature with additive and sometimes dominant gene action. Quantitative resistance is conditioned by small effects of many genes. For efficient utilization of available genetic variation for SLB resistance, the number of genes involved, their combinations and interactions with environment must be elucidated. Durability of the resistance depends on the evolutionary capacity of pathogen and population structure. Cytoplasmic and genetic diversity must be maintained to cop up with the early breakdown of resistant. It is a need of present scenario, where maize crop suffers due to various foliar diseases, to breed the maize cultivars displaying the resistance against multiple diseases. Though, it is very difficult but, introgression breeding programme should be aimed to incorporate QTLs for multiple disease resistance to avoid the crop from universal vulnerability. It would be better if focus should be placed on S_1 recurrent selection during population improvement.

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