

TURCICUM LEAF BLIGHT OF MAIZE AND BREEDING FOR ITS RESISTANCE

BY

S. A. K. M. KAISER

Department of Plant Pathology, Faculty of Agriculture,
Bidhan Chandra Krishi Viswavidyalaya,
Kalyani, West Bengal.

Key Words : Maize, Turcicum, Leaf blight, Breeding

Turcicum leaf blight of maize caused by *Exserohilum turcicum* (= *Helminthosporium turcicum*) acts as a major constraint on maize production in Indian agriculture. The disease may cause considerable damage to the standing crop by reducing yield and grain value under favourable climatic conditions. Virulent strains of *E. turcicum* on some released materials (hybrid Ganga 5, Deccan, Pio x 104, Pio x 114 and Kisan) resulted a major epidemic during 1974 in many parts of India. Symptoms of the disease appear in the form of lesions (3 to 18 x 1 to 4.5 cm), elliptical in shape and greyish green to tan in colour. The disease is favoured by cool temperature (17° to 25°C), high rainfall and high humidity, and it may appear at any stage of plant growth. Crop debris usually serves as the source of primary inoculum. Epidemiology of the disease is determined by (1) the source(s) of primary inoculum, (2) spread of the inoculum on the host surface and (3) the condition(s) favourable for the inoculum to germinate, cause infection and its secondary spread.

Resistance to *E. turcicum* in maize is chemical in nature and is caused by the production of phytoalexin (hydroximic acid - DIMBOA) isolated from monogenic resistance source (Ht gene). Two types of resistance to *E. turcicum* are known in maize : (1) quantitative (polygenic) and (2) qualitative (monogenic). The quantitative form of resistance is efficient against all races of the pathogen and is easily inherited, while monogenic form is race specific. Considerable variation is observed in the population of *E. turcicum* which exists in nature atleast in the form of three different races (race 1, race 2 & race 3). Resistance or susceptibility to these races is conferred by the involvement of different genes (Ht, Ht₂ & Ht₃) in a certain cultivar. Race 1 is known to be avirulent to genes Ht, Ht₂ & Ht₃, race 2 is virulent to gene Ht but avirulent to genes Ht₂ & Ht₃, while race 3 is virulent to genes Ht₂ & Ht₃ but avirulent to the Ht₂ gene. Since the known races of this pathogen are virulent on specific genes for resistance, the most effective way to control them would be to combine the Ht & Ht₂ or Ht & Ht₃ genes in a breeding programme. The continued use of monogenic source would only increase the selection pressure. Therefore, the best alternative for all breeding programmes, with farmers as the final target, would be the incorporation of both polygenic and monogenic resistance in the same cultivar because due to the interaction of both types of resistance together in the same plant a higher level of resistance may be obtained than with either alone. This may be effectively accomplished by the population improvement method through recurrent selection.

INTRODUCTION

Maize (*Zea mays* Linn.) is attacked by 16 diseases of major importance under Indian condition as delineated by Renfro and Ullstrup (1976). Out of these Turcicum or Northern leaf blight (*Exserohilum turcicum* (Pass.) Leonard & Suggs, synonym of *Helminthosporium turcicum* Pass., asexual stages of *Setosphaeria turcica* (Luttrell) Leonard & Suggs; and *Trichometasphaeria turcica* (Pass.) Luttrell respectively) has gained its importance in recent years as it acts as a major constraint on maize production in Indian agriculture. The disease is widely prevalent over different parts of India including Himalayan and Sub Himalayan regions, Peninsular India and Bihar. It appears on the crop grown both in the *kharif* season (summer/monsoon) and *rabi* season (winter/spring) depending upon the physical factors influencing growth and spread of the pathogen, and it may attack all types of corn. Some places in India, namely Dharwar (Karnataka), Karimnagar (Andhra Pradesh), Dholi (Bihar), Kolhapur (Maharashtra), Bajaura (Himachal Pradesh) and Kalimpong (West Bengal) have been declared as "hot spots" of this disease due to its regular occurrence and high incidence. Loss in grain yield due to this disease is found to vary from 27.6 to 90.7% (Payak and Sharma, 1978; Rai, 1987) depending upon the intensity of infection. Appearance of virulent strains of *E. turcicum* on some released materials like hybrid Ganga 5, Deccan, Pio x 104, and Kisan resulted to a major epidemic during 1974 in many parts of India (Sharma and Payak, 1981). Progressive susceptibility to *E. turcicum* has been also recorded during the recent years on some released materials like Kisan, Tarun, Vikram, A-de-Cuba, Vijay, Ageti-76, Diara, Hunius and Safeda under Kalimpong condition (Kaiser, 1986). Hence, considering in terms of geographical distribution and potentiality to cause significant yield reduction the importance of the disease is of great significance.

Disease symptoms

The disease is potentially important in areas where temperatures drop at night while humidity is high (Ullstrup, 1955, 1960; Robert, 1964; Leon, 1987). Normally, the lesions start developing in the lower leaves, but if the infection occurs late, most of them may develop in the upper leaves. Lesions produced are generally large (3 to 18 cm long and 1 to 4.5 cm wide), elliptical in shape and grayish green to tan in colour. In high humid condition, lesions may be covered with masses of dark green conidia of the fungus. In case of severe infection, the complete foliage may be killed. The ears and kernels are not generally affected but some lesions may appear in the outer husks.

The disease is favoured by cool temperature (17° to 25°C), high rainfall, high plant population (Popov, 1968; Markov, 1969; Das and Chattopadhyay, 1984a). Moreover the intensity of infection depends upon the stages of plant growth and

the resistance of the genotype to this disease. Plants attacked at the seedling stage suffer badly and the loss in yield may even go upto 100% (Jugenheimer, 1975 ; Das and Chattopadhyay, 1984b ; Rai, 1987).

Mode of survival, epidemiology and infection

Crop debris usually serves as the source of primary inoculum. The pathogen is not carried through seed as the cobs are not affected.

Epidemiology of the disease is determined by the following factors : (1) the source(s) of primary inoculum, (2) spread of the primary inoculum on the host surface and (3) condition(s) necessary for the inoculum to germinate, cause infection and its secondary spread (Popov, 1968 ; Markov, 1969 ; Leon, 1987).

Dispersal of the fungus from both near or distant sources are necessary in the epidemiology of the disease. In moist temperate conditions, the fungus overwinters as mycelium, and conidia are produced on the infested maize debris left on the ground. In such conditions, conidia may also develop into thick walled resting spores (chlamydospores) which remain viable for a long period of time. However, during warm and humid conditions, conidia are produced on the maize debris. These conidia acting as primary inoculum are spread when splashed by rain or carried by wind from distant places and deposited on the surface of maize leaves. Once deposited, the conidia germinate when free water, mainly in the form of dew, is available on the leaf surface for about 6 to 16 hrs at a temperature of 18° to 24°C. On germination, conidia penetrates through stomata by means of germ tubes invading the parenchyma cells. Lesions develop in about 7 to 10 days after infection. Secondary spread of the fungus is accomplished by rain water/irrigation and wind which disseminate the conidia produced on the lesions.

Mechanism of resistance

Resistance to *E. turcicum* in maize is chemical in nature and caused by the production of *phytoalexins* following host-pathogen interaction. Isolation of *phytoalexins* from maize with monogenic resistance (Ht gene) has been well studied (Lim *et al.* 1968, 1970 ; Payak *et al.*, 1974). The *phytoalexin* isolated is an inhibitory substance known as hydroximic acid (DIMBOA). It appears to be defensive in function and it can significantly inhibits the germination of conidia of *E. turcicum* at a concentration of 1 to 10 ppm. Percentage of infected leaf area has been also found to be significantly higher in genotypes lacking DIMBOA as compared to those where it is present. Multigenic lines are able to produce significantly more inhibitory substances than monogenic lines. The incorporation of Ht gene into multigenic lines may dilute their ability to produce inhibitory substances.

RESISTANCE BREEDING

Control of *Turcicum* leaf blight of maize may be effectively accomplished by using host resistance which has been quite successful in India and other countries. Before launching a breeding programme breeders and pathologists together should consider several factors including races of the pathogen and the types of genes involved in the genotype to be effectively used in the breeding strategies. Moreover, appropriate breeding method should be followed as per requirement to develop a desirable type of cultivar (hybrid/composite).

Races of the pathogen and host resistance

Resistance to *E. turcicum* in maize are mainly of two types : (1) quantitative and (2) qualitative. The quantitative form of resistance was identified long ago (Jenkins and Robert, 1952) and also reported by various workers (Jugenheimer, 1975 ; Ramamurthy *et al.*, 1980 ; Das, *et al.*, 1985). This resistance is recognised mainly by the reduction in lesion's size and number but with little effect in decreasing the amount of sporulation on the lesion surface. This resistance has been identified to have a polygenic inheritance which is easily inherited but not expressed in the seedling stage. It is efficient against all known races of the pathogen, and is non-race specific. Because of the various genes involved in this resistance it is also called horizontal in its inheritance.

The qualitative form of resistance is conferred by the Ht single dominant gene, and it is expressed as either chlorotic or necrotic lesions in both seedling and developed plants when they come in contact with an avirulent or virulent race, respectively. This is also known as monogenic resistance or chlorotic lesion type of resistance and it is mostly associated with the suppression of sporulation. The gene involved in it was identified in the corn inbred GE 440 and popcorn variety Ladyfinger, and has been effectively utilised in many countries (Hooker, 1961,1963; Hooker *et al.*, 1964, 1965).

Another type of resistance which mostly reduces the number of lesions in adult plants has been identified as HtN which differs from the quantitative type in that it is inherited as a single dominant gene. It was identified in South Africa in Pepitilla race of maize. But this type of resistance is unstable and it breaks under suitable environments conducive for the growth and spread of the pathogen (Leon, 1987).

A new race 2 of the pathogen identified in Hawaii and Indiana in 1972 and 1979 respectively, was found to be virulent to plants carrying Ht gene, but avirulent to the inbred NN 14B carrying the Ht₂ gene. The most recent biotype reported from South Carolina, USA is race 3. It is avirulent on plants known to carry the Ht gene but virulent on inbred NN 14B (Leon, 1987). Considering the facts as

presented here the following virulence formulae may be developed in the host-pathogen relationship.

Race	Virulence formula (for effective/non-effective genes)	Avirulent to	Virulent to
Race 1	Ht Ht ₂ Ht ₃ /0	Ht Ht ₂ Ht ₃	—
Race 2	Ht ₂ Ht ₃ /Ht	Ht ₂ Ht ₃	Ht
Race 3	Ht/Ht ₂ Ht ₃	Ht	Ht ₂ Ht ₃

Breeding strategies

From the evidences produced here it may be understood that a considerable variation exists in the population of *E. turcicum*. If breeders continue incorporating and selecting for a specific gene against a particular race they may further enhance the increase of other races. Since the known races of the pathogen are virulent on specific genes for resistance, the most effective way to control them would be to combine the Ht and Ht₂, or Ht and Ht₃ genes in a breeding programme. The continued use of monogenic source would only increase the selection pressure for the development of new races of *E. turcicum*.

Both monogenic and polygenic resistances have some advantages and disadvantages in them. One of the advantages in monogenic resistance is that it decrease the amount of sporulation thereby reducing the secondary spread of the disease. But this does not always hold true in case of large amount of inoculum coming from the neighbouring fields. Moreover, in such conditions cultivars with monogenic resistance have been found to yield less as compared to that in cultivars with polygenic resistance. This lower yield obtained in the cultivars with monogenic resistance may be attributed to the amount of leaf area which becomes chlorotic due to resistant reaction thus reducing the photosynthetic capabilities. The polygenic resistance would be the better means to decrease the effects of the disease where spread of *E. turcicum* within a certain field is considered to be insignificant.

One of the main reasons for the continuing use of monogenic resistance particularly in case of hybrid development is that it is easy to incorporate the single dominant genes in the parents. One of the main objections for use of polygenic resistance is the difficulty to transfer multiple genes for resistance without affecting desirable agronomic characters in the susceptible parent. This may require for a backcrossing programme to recover the desirable characters of the susceptible parent. But it is well known to any breeder with a certain degree of experience that incorporation

of monogenic resistance from a source is not an easy job, either because in such type of cross with the resistance donor, many undesirable characters may come up thus increasing the amount of time and labour in the selection programme.

According to Leon (1987) in the improvement of any maize variety, or line, the breeders should try to incorporate and select for higher level of polygenic resistance in the existing programme. Once the adequate level of polygenic resistance is achieved, monogenic resistance gene may be incorporated to decrease the possibilities of damage to a minimum. Selection for polygenic resistance is quite impossible in a material with monogenic resistance already incorporated. Therefore, the best alternative for all breeding programmes, with farmers as the final target, would be the incorporation of both polygenic and monogenic resistance in the same cultivar because due to the interaction of these two types of resistance together in the same plant a higher level of resistance is obtained than with either alone. This level of resistance is also directly proportional to the amount of lines or families with monogenic resistant genes used in the formation of hybrid or synthetic variety.

Classification of genotypes for resistance to E. turcicum

In a polygenic type of resistance wide range in the level of resistance to *E. turcicum* is expressed by the different genotypes (Jugenheimer, 1975; Ramamurthy *et al*, 1980; Kaiser, 1986; Rai, 1987). Based on their level of resistance the different lines are classified into different categories on a suitable disease rating scale. The scale as practiced in the All India Coordinated Maize Improvement Project (AICMIP) is described here for example (Payak and Sharma, 1982).

Score	Category
1	Resistant (R)
2	Moderately Resistant (MR)
3	Moderately Susceptible (MS)
4	Susceptible (S)
5	Highly Susceptible (HS)

In a breeding programme reaction of hybrids to this disease may be observed by crossing (single cross) the inbred parents in all possible combinations as follows: (1) $R \times R$, (2) $R \times MR$, (3) $R \times S$, (4) $MR \times MS$, (5) $MR \times S$ and (6) $S \times S$. In such type of cross combinations resistance may be partially dominant over susceptibility, or in certain combinations genetic interactions may be observed. It has been observed that in case of several F_2 $R \times S$ combinations certain R inbred lines are capable of throwing high frequency of R plants in F_2 as compared to others (Rai, 1987).

Breeding technique

The first step in breeding for Turcicum leaf blight (TLB) resistance will be to standardise the screening technique. The standard technique followed by Pioneer Seed Company (PSC), Bangalore is cited here for an example (Rai, 1987).

In a block where the TLB screening is required for inbred lines and segregating material, a spreader range is planted to a susceptible hybrid for every alternate range of test material. In addition, every 11th row is planted to the same susceptible hybrid which is generally referred as indicator row. The spreaders and the indicator rows are planted along with test material. At keen-high stage whorl inoculation is done on spreader ranges by using the dried powdered diseased leaves as inoculum. In a dry season, sprinklers are used to increase the atmospheric humidity. Disease symptoms may develop on the inoculated plants in about 15 days after inoculation. At the time of flowering disease will be very severe on the spreaders and the disease will start spreading to the test material. This technique is suitable for any large breeding programme and may be accepted with/without modification.

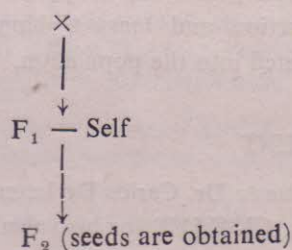
BREEDING METHODS

Any breeding programme for the development of TLB resistance will require a suitable breeding method. Three type of breeding methods are followed by PSC in Bangalore to develop TLB resistant maize material.

Pedigree Method

Susceptible line
(good combiner, good
per se yielder)

Resistant line
(resistant to TLB)



F_2 seeds are planted in TLB nursery. In F_2 population, some of the $R \times S$ crosses may show high percentage of resistant plants while the other crosses may show very low percentage of R plants. In crosses where the percentage of resistance becomes high, $R F_2$ plants are selfed and advanced like in the normal pedigree method, while in other crosses MR and R plants are sib-mated for one generation.

Modified Gamatic Selection Method

This method is generally followed to trap favourable genes from a promising TLB S population, and is consisted of several steps.

Step 1—R inbred lines from the bulk pollen of S population are pollinated.

Step 2—Progeny of population $\times$ inbred cross is raised. Desirable plants are then selected under disease free condition and the selected plants are sib-mated. Only the pollinated plants are finally selected and harvested.

Step 3—Progeny from the harvest of previous season is grown. Low epiphytotic condition is developed. MR and R plants are selected and sib-mated, and only pollinated plants are selected and harvested.

Step 4—Progeny is raised from the selected ears. Moderate epiphytotic is developed. Selected plants are selfed and only the selfed plants are selected for harvest.

Step 5—Progeny from the above harvested seed is raised and allowed to self under TLB condition to develop inbred line.

Population Improvement Method

The objective of this method is to incorporate TLB resistance to a promising population along with improvement of population for other trials. Both monogenic and polygenic type of resistance are incorporated through bulk pollen from selected resistant lines during mass sibbing. In the next generation, plants show both monogenic as well as polygenic type of resistance. Resistance plants are again sib-mated, and the resulting population is expected to show three types of resistance viz., 1) Monogenic, 2) Polygenic and 3) combination of Monogenic and Polygenic. Selection and mass sibbing are continued till acceptable level of resistance is incorporated into the population.

ACKNOWLEDGEMENT

Grateful thanks are due to Dr. Carlos De Leon, Scientist from International Maize Research Programme, CIMMYT for his valuable suggestions and informations in the subject.

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(Accepted for publication 7th December 1987)