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THE NORTHERN MAIZE LEAF BLIGHT DISEASE IN TANZANIA: REASONS AND REISSUANCE METHODS

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A B S T R A C T

The northern leaf blight disease occurrence, intensity, distribution and resurgence in Tanzania have signalled attention to maize breeders. A clear understanding of its disease genetics, mode of gene actions and heritability plays significant roles in the successful designing breeding strategies and control. Gene actions and heritability provides information on choosing the appropriate breeding methods to be employed. Therefore, this review aimed at bridging the disease genetics information and the practical application of breeding methods to be employed by breeders in fields. Information gathered here are important tools to breeders for selection of the best breeding methods that are used to impart resistance to northern leaf blight disease in maize.

Keywords: Gene actions, *Helminthosporium turcicum,* maize resistance, resurgence.

INTRODUCTION

This review highlights the northern leaf blight (NLB) disease. The NLB disease has increased its importance in Tanzania. Therefore, the major focus of the study was on causative agent, transmission, epidemiology, the resurgence of NLB disease and disease symptoms. There is also discussion on sources of resistance to NLB and disease mode of inheritance. Current efforts to control the disease, genetics of northern leaf blight disease resistance, gene action estimation from diallel cross and generation mean analysis. Roles of heritability, heterosis, maternal effects in NLB disease resistance and breeding strategies for resistance are also explained.

Maize production constraints in Tanzania: Maize production in Tanzania is carried out by small-scale farmers who account for up to 85% of the total maize produced in the country (Bisanda *et al.,* 1998). Despite maize research programme efforts in breeding for high yielding cultivars, the average yield in the country is still low. According to research, the average yield under farmers condition is still resting at 1.2 t ha^{-1} (Aquino *et al*., 2001; Makurira *et al.,* 2007). The low yield is attributed to socio-economical, biotic and abiotic constraints (Katinila *et al*., 1998; Pixley *et al.,* 2006).

With the reduction of price subsidy, the prices of farm inputs increased beyond small farmers reach (Mwakalobo and Kashuliza, 1999). Prices of agricultural inputs like seed are 30 times what it used to be in the 1990's while the price for maize increased only three times during the same period. The situation has forced less than 35% of farmers to use purchased seeds (Morris, 2001). In addition, due to lack of seeds availability in the country, farmers are forced to use recycled seeds which further complicates the situation (Doss *et al.,* 2003). These factors have to lead to reduced maize yield which has resulted in food shortages and frequent hunger (Katinila *et al.,* 1998).

The abiotic factors include low-N, low-K and drought while biotic factors include stalk borer (*Busseola fusca*) and African armyworms (*Spodoptera exempta*). The common diseases are Leaf rust (*Puccinia maydis*), Brown spot (*Physoderma maydis*), Northern leaf blight (*Helminthosporium turcicum),* Phaeosphaeria leaf spot (*Phaeosphaeria maydis),* Tassel smut (*Sphacelotheca reiliana*), Gibberella stalk rot (*Gibberella zeae),* Fusarium

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ear rot *(Fusarium moniliforme)* and Fusarium stalk rot (*Fusarium moniliforme)* (Bisanda *et al*., 1998; Nkonya *et al.,* 1998*).* Among the biotic factors, northern leaf blight (*Exserohilum turcicum)* is one of the major constraints to maize production in Tanzania.

Northern corn leaf blight (NLB) disease in Tanzania: Northern leaf blight is one of the major diseases affecting cereal production (Pixley *et al.,* 2006). The disease can be found in all 21 maize growing regions, including the marginal areas, which were previously considered unfavourable for disease development. To date, most of the improved maize varieties grown in Tanzania are susceptible to NLB disease (Kanampiu *et al.,* 2003). There is a need for breeding for NLB disease resistant varieties which will curb the current outbreaks. In order to deal with this problem, an effective breeding strategy is needed. The use of available sources of resistance from some of the inbred lines, landraces and exotic materials may kick-start the introgression of resistance genes into the currently released and new cultivars.

Causative agent and transmission of northern leaf blight disease: The disease is caused by the fungus *Exserohilum turcicum* (Pass.) K. J. Leonard & Suggs [anamorph]. It is one of the major diseases of maize, sorghum and pearl millet. The primary hosts are maize (*Zea mays*) and sorghum (*Sorghum bicolor*) while the secondary host is pearl millet (*Pennisetum glaucum*). Densely populated cultivars facilitate movements of spores from one plant to another and thus increases disease severity (Adipala *et al.,* 1995). On the maize plant, the disease starts at the lower leaves and then spreads to other parts of the plant (Elliott and Jenkins, 1946). Wind and rainfall splash spreads spores from disease to healthy plants (Amusa *et al.,* 2005; Boland *et al.,* 2004). The disease also survives on wild hosts of the graminae family and attack maize in the next season. It survives from one season to another in the form of conidia on crop residues which acts as the source of inoculums to the new cropping season (CPC, 2001; Esele, 1995).

Disease epidemiology: The severity of the disease occurs when conditions are favourable. High humidity associated with low temperature and cloudy weather is conducive conditions for disease development on the host plant (Singh *et al.,* 2004). Heavy dew on the growing plant has also been cited as one of the factors leading to NLB disease severity (Dingerdissen *et al*., 1996). Conidia germination on leaves is high when the temperature ranges from 18 to 27°C (Levy and Cohen, 1983). Levy (1989) mentions that high relative humidity and presence of susceptible hosts are other factors that influence the disease epidemiology. Ceballos *et al.* (1991) reports that, disease severity is high for early maturing maize varieties than late maturing varieties. This implies that, late maturing cultivars are relatively more resistant than early maturing maize varieties.

The resurgence of NLB disease: Past decades witnessed breeders containing the NLB disease in maize production. Previous studies showed the concentration of the disease to high humidity and low temperature areas of the world (Raymundo and Hooker, 1981). Recently, the disease has resurged and is affecting all maize growing regions in the world (CIMMYT, 2002; Mwangi, 1998). Researchers have been associating the resurgence of NLB disease with many factors.

Planting susceptible cultivars coupled with the extensive use of fungicides to control the disease is one reason towards this scourge. Small-holder farmers are forced to use cheap and susceptible genotypes due to their low purchasing power, at the same time commercial farmers may use susceptible materials and intensify spraying regimes (Adipala *et al.,* 1993; Amusa *et al*., 2005; Kaliba *et al.,* 1998; Pataky *et al.,* 1998). Many resistant cultivars were developed to confer gene- for- gene, monogenic and race specific type of resistance (Robinson, 1987; Robinson, 2004). This type of resistance can easily be broken down and succumb to new emerging races (Campaña and Pataky, 2005). Susceptible genotypes act as the source of inoculums builds up which in turn could result in disease resurgence.

Transhumance and tendency of farmers to exchange recycled seeds among themselves are other factors contributing to NLB severity and occurrence. A survey conducted in southern Tanzania by Nathaniels and Mwijage (2000) reported seed exchange among farmers in Nachingwea district as one of the sources of planting materials. A similar observation has been found in Zambia where 40 % of seed exchange among farmers exists (Gwanama and Nichterlein, 1995). Recent studies have shown that land scarcity is another source of disease resurgence, land scarcity forces farmers to practice intensive farming while compromising crop rotation, improved fallow and rotational woodlots (Kimaro *et al.,* 2008; Nyadzi *et al*., 2003). This tendency has resulted in the increased number of fungal spores sufficient to cause the disease outbreak (Okori, 2004).

The effects of trade liberalization on agricultural sector

could have contributed significantly to NLB resurgence. Trade liberalization has been associated with double effects on NLB disease occurrence. First, trade liberalization was accompanied with the reduction of agricultural input subsidy that resulted in the increased input prices and lower crop yields (Jean and Christina, 1991; Kaliba *et al.,* 1998; Mwakalobo and Kashuliza, 1999). Increase in input prices has forced farmers to resort to cheap and NLB disease susceptible cultivars which increase inoculums in the field. The second effect is agricultural policy regulations to attract investors in the agriculture sector. Governments were obliged to reduce the strict importation and crop inspection rules and regulations. The result was the introduction of inferior and NLB susceptible genotypes in agricultural fields which lead to the increased NLB fungal inoculums (Geisler, 1992).

The high amount of inoculums from different sources have the possibility of increasing the recombinant hybridization which results into pathogen new races development in the area (Robinson, 1987). There are reports of new NLB disease races around the world. The emergence of new races 0, 1, 23 and 23N in NLB has posed a constant threat to the efforts of controlling the disease in maize breeding programmes worldwide (Ferguson and Carson, 2007; Ogliari *et al.,* 2005). In East Africa, Mwangi (1998) observed the presence of races 0, 1, 2, 3, 12 and three unknown races.

Other studies have cited climatic changes as a contributor of NLB disease severity (Boland *et al*., 2004; Chakraborty *et al.,* 2000). According to Griefenhagen and Noland (2003), the world's temperature is escalating such that it's temperature will rise by 3^0C next century. The rise in temperature will favour diseases development including fungus sporulation which is expected to bring further NLB disease threats to the world. Pathogen environmental competence has been cited by researchers as one of the reasons of disease resurgence in crops (Godfray *et al.,* 1999; Robinson, 1987). Robinson (1987) further reported on a maize landrace which was higher resistant to disease in Malawi, the same variety was highly susceptible to the same disease in Kenya. This implies that the pathogen had high environment competence and increased pathogenicity in Kenya than in Malawi. The same scenario can be used to explain the susceptibility to NLB disease of genotypes being currently introduced in different countries without enough testing time in the target countries. The result is the build up of inoculums in maize growing areas.

Mutation can be another source of NLB resurgence in maize germplasm. Breeding advances in maize have resulted in more uniform genotypes. Genetic uniformity increases the chances of pathogen mutations, new race emergence and increased pathogenicity (Ogliari *et al.,* 2005; Smale and DeGroote, 2003). Pataky *et al.* (1998) reports on epidemics of NLB in Florida due to mutation of the pathogen on a super sweet hybrid maize cultivar. Mutation can also occur on plants themselves through altering the genetic structure and thus become more susceptible to diseases. Jenks *et al.* (1994) reports on the effect of reduced epicular wax structure in sorghum which exposed the genotype to *Exserohilum turcicum* attack.

Disease symptoms: The disease starts with small water-soaked spots that appear on leaves. The small water-soaked spot dry up and join in the elongated brown lesion which can reach the size of 20 x 400 mm (Mwangi, 1998). Some researchers have recorded lesion sizes of 2.5 x 150 mm (CIMMYT, 2004). However, Pataky *et al.* (1998) reported much lower lesions on partially resistant maize genotypes. The severity of the disease depends on weather conditions, plant growth stage (Figure 1), pathogenicity and genotype susceptibility (CIMMYT, 2004; Levy, 1989).

Figure 1. Early infestation (A) leads to loss of photosynthetic leaf area at reproductive phase (B).

Under severe conditions, the whole leaf can be covered by the disease and dry up. The accompanying NLB disease effects are reduced photosynthetic area, plant lodging, secondary infection, stalk rot, plant death and subsequent lower yields per unit area (CIMMYT, 2004).

Sources of resistance to NLB disease: Researchers have been using resistant materials to control the NLB disease effects in maize. Sharma and Payak (1990) used CM104 and CM105 NLB disease resistant inbred lines from CIMMYT to analyse the mechanisms of leaf blight disease resistance in maize. In Uganda, researchers used cultivars Babungo 3, EV8342-SR, Mo 17 and H99 as sources of resistance to NLB disease and recorded useful results (Lipps *et al.,* 1997; Ojulong *et al.,* 1996). According to Freymark *et al.* (1993) and Pratt *et al.* (1997), Mo17 provides polygenic NLB resistance to maize plants. Other researchers reported CML 202 as the source of horizontal resistance to NLB in tropical Africa maize (Schechert *et al.,* 1999). In India, Inbred lines CM104 and CM105 from CIMMYT confer durable resistance to NLB disease (Levy, 1989; Sharma and Payak, 1990). According to Singh *et al.* (2004) early maturing, CM 145 and medium maturing lines from CIMMYT, CM 104, confer resistance to northern leaf blight disease. It was further noted that, population 31 from CIMMYT was a reliable source of the gene pool for NLB resistance (Singh *et al.,* 2004). In practice, durable resistance can be achieved by population improvement through recurrent selection (Campaña and Pataky, 2005; Ceballos *et al.,* 1991; Ogliari *et al.,* 2005). According to Ogliari *et al.,* 1999) L30R and L40 maize inbred lines are some sources of monogenic resistance to NLB disease.

Disease control: Various ways are used to contain the disease. These are cultural, chemical and biological controls. Biological control includes the use of natural enemies and resistant cultivars.

Cultural control methods aim at reducing the number of inoculums in the field. Methods like the destruction of crop residues so as to remove the infected plants and breaking the pathogen breeding cyle, crop rotation, weeding and intercropping have shown some levels of controlling the disease. According to Sharma and Duveiller (2004), optimal fertilizer rates, moisture management and timely planting increase resistance and the yield on maize production. Other researchers have found similar results (Reuveni and Reuveni, 1998). However, cultural control measures are sometimes limited due to land availability, labour shortages and farmers purchasing power.

Northern leaf blight disease can be controlled by using a number of fungicides. Fungicides differ in the ability of controlling the disease. The commonly used fungicides include Zinc ethylenebis (zineb), Dithane (mancozeb) M-45, O-Ethyl-S.S-diphenyl dithiophosphate (Edifenphos), Difolatan (Captafol), and benzenedicarbonitrile (chlorothalonil). The use of fungicides has managed to control the disease to a remarkable point. However, they are not sustainable, are expensive and pose environmental hazards (Chakraborty *et al.,* 2000; Matthews *et al*., 2003; Reuveni and Reuveni, 1998; Shelepchikov *et al.,* 2008).

Various biological control agents have been tested to combat the disease. The most promising is the use of *Bacillus subtilis* inoculums (Reis *et al.,* 1994). Biological controls have the advantages that, they are environmentally friendly, do not require industry processes and do not develop resistance to pathogens (Bacon *et al.,* 2001). However, they have slow and unpredictable actions; they require specialized skills for rearing and ways of releasing them to the field. Furthermore, biological agents can multiply excessively and turn to be pathogens of other crops (Jutsum *et al.,* 1988). Thus, breeding for resistance remains the reliable method.

Breeders and farmers have reported diseases as one of the major factors that limit crops production and employ some measures to reduce the effect. Previous breeding studies have significantly contributed to disease controls (Brewster *et al.,* 1992; Sharma and Payak, 1990; Welz *et al.,* 1999). In maize, breeding for NLB disease resistance started much earlier than 1961 (Ceballos *et al.,* 1991). Although it seems to start earlier, more efforts are still needed as new challenges arise. Following the difficulty in controlling NLB due to high input prices, new races and unreliable biological control, more breeding for resistance is highly demanded (CPC, 2001; Mwakalobo and Kashuliza, 1999). The use of maize resistant to NLB is the cheapest and more reliable approach towards combating the disease (Hughes and Hooker, 1971; Welz and Geiger, 2000). However, integration more than one control methods such as cultural, chemical and biological bring good and sustainable results than applying the single approach in isolation. Thus, integrated pest control is highly appreciated.

Genetics of northern leaf blight disease resistance: Resistance to NLB disease in maize is located on chromosomes 3, 5, and 8 (Welz and Geiger, 2000; Wisser *et al.,* 2008). Brewster *et al*. (1992) studied Mo17 maize line and found that, NLB disease resistance was linked to chromosome 3, the short arm of chromosome 4, and the long arm of chromosome 6. Northern leaf blight disease resistance is controlled by six dominant Ht1, Ht2, Ht3, HtN, NN and HtM and one recessive ht4 genes (Ferguson and Carson, 2004; Ferguson and Carson, 2007; Pratt, 2006; Singh *et al.,* 2004; Wisser *et al.,* 2006). All these provide qualitative inheritance in the form of dominance or partial dominance. According to Pataky *et al.* (1998) HtN gene confers partial resistance to NLB disease. Other researchers have reported on the durable resistance to NLB conferred by major genes. Ogliari *et al.* (2005) reports on dominant HtP genes inducing resistance to NLB pathogen and recessive rt genes inducing resistance to specific NLB pathogen races.

Several modes of gene actions are involved in controlling the inheritance of NLB disease in maize. Additive, dominance, and epistatic gene action are involved in controlling the disease (Ogliari *et al.,* 2005). However, additive gene action was found to be more important than others (Hughes and Hooker, 1971; Ogliari *et al.,* 2005). Maternal effects are less important for the traits associated with the inheritance of NLB disease resistance. Sigulas *et al.* (1988) found non-significant maternal effects on 16 maize genotypes. Other researchers have reported non-significant cytoplasmic and maternal effects on the inheritance of NLB disease in maize genotypes (Welz and Geiger, 2000).

Gene action estimation from diallel cross: Gene action can be estimated by using various mating designs. Mating designs are methods used to produce progenies in breeding programmes (Dabholkar, 1992). They enable breeders to estimate genetic variances and combining abilities. Estimation of combining abilities enables the prediction of progenies performance based on the performance of parents. General combining ability measures the averages of all line crosses to a common progenitor while specific combining ability estimates the specific performance of combinations between lines (Griffing, 1956). There are various mating designs depending on the objectives (Stuber, 1980). The common mating designs include: top cross, poly cross, biparental progeny, diallel and partial diallel, North Carolina I, II and III and line x tester mating designs. A diallel mating design can be used to estimate the GCA, SCA and other genetic effects from all possible combinations. By using diallel cross it is possible to evaluate parents, F_1 hybrids, reciprocals and maternal effects (Gupta and Kageyama, 1994; Stuber, 1980). In addition, diallel mating designs are suitable for cross pollinated crops like maize by which GCA and SCA and their interaction with the environment are taken care of (Griffing, 1956; Hayman, 1954). According to Griffing (1956) estimation of genetic variances is made in terms of the combining ability by which effects are considered in terms of GCA and SCA i.e. $v_{ij}=g_i+g_j+s_{ij}$ if reciprocals are excluded, and $v_{ij}=g_i+g_j+si_j+r_{ij}$ if reciprocals are considered. Where, g_i and s_{ij} are GCA and SCA , r_{ij} is the reciprocal effect involving the i^{th} and j^{th} parents respectively. The analysis conducted at one site can be modelled as:

$y_{ijkl}=\mu+ r_1 + b_{lk} + g_i +g_j+ s_{ij}+ e_{ijkl}$

where y_{ijk1} = yield (or any other trait) of the cross between lines i and j in block k; μ = overall mean; r_1 = replication effect, $\Sigma_1 r_1 =0$; b_{lk} = effect of the k^{th} block in the 1^{th} replication, $\Sigma_k b_{1k} =0$; g_i = the GCA of parent i, $\Sigma_i g_i =0$; g_j = the GCA of parent j, $\Sigma_j g_j =0$; s_{ij} = SCA of the cross between parents i and j, $\Sigma_i s_{ij} =\Sigma_k s_{ij} =0$;

e_{ijkl} =random error (assumed as normally and independently distributed i.e. $\mu=0$ and $\sigma^2=1$). The $g_i +g_j+ s_{ij}$ is the genotypic contribution for cross i x j.

A relatively larger GCA/SCA variance ratio demonstrates the importance of additive genetic effects and the lower ratio indicates a predominance of dominance and/or epistatic gene effects (Christie and Shattuck, 1992). The Significant contribution of GCA and SCA is then interpreted for breeding purpose application. If GCA is significant, it means additive gene effect is important and thus selection could improve the germplasm. If SCA is significant then, dominance gene effect is important and thus hybrid vigour could be achieved in crosses among inbred lines. If GCA and SCA are both significant, GCA/SCA ration is used for interpretation. In this case, if the ratio = 1, then both are important and if the ratio >1 then additive gene action is more important than dominance gene effects.

Depending to whether the selfed parents and or the reciprocals and F1's are included in analysis, it can be further divided into a subdivision. Griffing (1956) suggested four possible experimental methods:

parents, one set of F1's and reciprocal included, parent and one set of F1's are included but not the reciprocals,

one set of F1's and reciprocals are included but not the parents and, one set of F1's but neither parents nor reciprocals are included.

Depending on the type of parents used for crosses, fixed or random models are used for analysis. If parents are the genotypes under consideration, this is referred to the fixed model (model I), whereas the random model (model II) is applied if the parents are a random sample from the reference population (Griffing 1956).

Gene action estimation from generation mean analysis: Generation mean analysis (GMA) is another method used in gene action estimation. It utilizes six population means P_1, P_2, F_1, F_2, BCP_1 and BCP_2 to estimate genetic effects (Carson, 1995). The method is efficient in partitioning epistasis and non-allelic gene effects (Hettiarachchi *et al.,* 2009). Thus, it is used to study populations which have distinct wide contrasting traits like disease resistance because it analyses one trait at a time (Frank and Hallauer, 1997).

Generation mean analysis has been employed in various crops and traits to estimate genetic effects in contrasting characteristics. In maize, GMA has been used to generate useful information. For example, it has been used for twin cobs study ((Frank and Hallauer, 1997)) and inheritance of NLB disease (Campaña and Pataky, 2005; Carson, 2001). Several studies have shown that, NLB disease inheritance is mainly controlled by additive gene action while dominance and epistasis contributions are normally non-significant (Carson, 1995; Jenkins and Robert, 1952). However, other studies observed the significant contribution of additive, dominance and non-allelic gene interaction in controlling NLB disease resistance in maize (Lingam *et al.,* 1989). Generation mean analysis can be employed to estimate epistasis and non–allelic gene interaction in inbred breeding materials. Generation mean analysis is a powerful tool for separation of additive, dominance, epistatic additive x additive, epistatic additive x dominance and epistatic dominance x dominance effects which cannot be obtained in diallel cross studies. In addition, previous screening studies showed a wide reaction range on NLB disease resistance among parents which satisfied one of the requirements of GMA studies applications.

The role of heritability in NLB disease resistance: Heritability information is used by breeders in designing appropriate breeding strategies. According to Stanfield (1991) heritability value (<0.2) is classified as low, medium (0.2–0.5) and high (>0. 5). High narrow sense heritability is the indication of additive gene action involvement for controlling particular traits especially under weak dominance effects (Jawaharlal *et al.,* 2011). In breeding for NLB disease resistance, many reports show medium to high heritability. For example, Hughes and Hooker (1971) and (Chaudhar and Mani (2010) reports the heritability range of 35 – 85%. This range implies selection strategies like recurrent selection could be used to improve maize populations under those maize populations as suggested by Ceballos *et al.,* (1991). However, heritability estimates can be influenced by parent materials and environment interactions. Thus, NLB heritability is more accurate and reliable when based on specific crosses and the target test environments where the new varieties will be deployed.

The role of heterosis in NLB disease resistance: Heterosis is an important trait used by breeders to evaluate the performance of offspring in relation to their parents. It estimates the enhanced performance of hybrids compared to their parents. Heterosis can be positive or negative. The interpretation of heterosis depends on the nature of trait under study. For example, a positive heterosis is preferred in yield studies because it shows an inclination towards high yield (Duvick, 2011). On the other hand, a negative heterosis is preferred in disease resistance like NLB. A negative heterosis in disease resistance shows that, breeding materials leaned towards resistance direction while a positive heterosis would imply skewness towards susceptibility trend. Breeding strategies like recurrent selection accumulate gene frequencies among genotypes and are likely to fit for populations with high heterosis.

The role of maternal effects in NLB disease resistance: In plants, maternal effects occur due to cytoplasmic and nuclear gene interactions of female parents. If they are highly significant they could dictate which breeding materials to be used as a female parent. Maternal effects are responsible for the distortion of gene effects estimation by inflating genetic variances. Traits mainly controlled additively are likely to be influenced by the presence of maternal effects and thus reduced selection response could occur. The majority of investigators report absence, low or non-significant contribution of maternal effects on the inheritance of NLB disease resistance (Sigulas *et al.,* 1988; Welz and Geiger, 2000). Although many researchers have indicated non-significant maternal effects contribute to

the inheritance of NLB disease in maize, further investigation in different genetic backgrounds may be justified. The reason could be that, maternal effects have been found to contribute significant effects on the inheritance of leaf blight (*Exserohilum turcicum)* in sorghum (Durga *et al.,* 2008). Maize and sorghum are all cereals and the disease causative agent is the same.

CONCLUSION

A clear understanding of the Northern leaf blight disease genetics, transmission mechanism, epidemiology, symptoms, gene actions and heritability are the major factors towards finding resistance sources and instruments for the effective curbing of the menace. Breeders use information from gene actions and heritability to estimate the number of genes that confers resistance and selection of suitable breeding strategies. Information gathered here is vital and important tools to be incorporated in maize breeding programs for increased knowledge and control approaches.

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