Journal of Maize Research and Development (2015) 1(1):71-85

ISSN: 2467-9305 (Online)/ 2467-9291 (Print)
DOI: 10.5281/zenodo.34286

A review on threat of gray leaf spot disease of maize in Asia

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ARTICLE INFO

Article history:
4th September, 2015 Revised:
10th October, 2015 Accepted:
8th November, 2015

Keywords:
Cercospora,
epidemiology,
inheritance, resistance

ABSTRACT

Biotic and biotic constraints are yield limiting factors in maize producing regions. Among these gray leaf spot is a yield limiting foliar disease of maize in high land regions of Asia. This review is done from related different national and international journals, thesis, books, research papers etc. The objectives of this review are to become familiar with genetics and inheritance, epidemiology, symptoms and disease management strategies etc. High relative humidity, temperature, minimum tillage and maize monoculture are important factors responsible for disease development. The sibling species of *Cercospora zeae-maydis* (Tehon and Daniels, 1925) Group I and Group II and *Cercospora sorghai var. maydis* (Chupp, 1954) are associated with this disease. Pathogens colonize in maize debris. Conidia are the source of inoculums for disease spread. Severe blighting of leaves reduces sugars, stalk lodging and causes premature death of plants resulting in yield losses of up to 100%. Disease management through cultural practices is provisional. The use of fungicides for emergencies is effective however; their prohibitive cost and detrimental effects on the environment are negative consequences. The inheritance of tolerance is quantitative with small additive effects. The introgression of resistant genes among the crosses of resistant germplasm enhances the resistance. The crosses of resistant and susceptible germplasm possess greater stability than the crosses of susceptible and resistant germplasm. The development of gray leaf spot tolerant populations through tolerance breeding principle is an economical and sustainable approach to manage the disease.

INTRODUCTION

Pests and disease are destroying about one fifth of all crop production around the world and at least 10% of the global food production is lost through plant disease alone (FAO, 2000) mainly in West Africa and South Asia. Maize (*Zea mays* L.) crop suffers from various biotic and abiotic constraints resulting in considerable yield loss. Among these, gray leaf spot is one of the most destructive and yield-limiting foliar disease in the world (Tehon and Daniels, 1925). The disease has been getting agricultural importance in tropical, subtropical and temperate maize growing areas worldwide in the last 30 years (Pingali and Pandey, 2002).

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Historical perspective

Gray leaf spot (GLS) is caused by a fungus namely *Cercospora zeae-maydi*s (Tehon and Daniels, 1925). It was first observed in Illinois, USA in 1925 (Tehon and Daniels,1925). The pathogen first identified from a sample collected by Tehon and Daniels and confirmed by Chupp in 1953.There were very few records of this disease during the 1940s (Roane,1950). The disease was endemic in proportion and occasional outbreak during the period of the 1970s (Latterell and Rossi, 1983). The disease was recognized as destructive and yield limiting when increased incidence occurred in North Carolina (Leonard,1973). The severity and distribution of the pathogen has been increasing and the disease has become the most destructive throughout the maize growing regions of USA (Stromberg and Donahue, 1986; Ward *et al.,* 1999). This disease also occurred in South America (Chupp, 1953). GLS was first observed at Grey town in 1988-1989, Cedera in 1992 (Gevers *et al.,* 1994) and Kwazulu Natal in 1988 (Ward and Nowell,1997).

It was reported that GLS reached epidemic level in Natal South Africa during 1991-1992 (Gevers *et al.*,1994) and the first report was made in 1990 (Nutter and Jenco, 1992). It was reported that GLS was observed in Southern, Central and West Africa in the late 1980s and early 1990s (Ward and Nowell, 1997). Since then the pathogen became pandemic and spread rapidly in other provinces of South Africa as well as other countries in Sub Saharan Africa (Ward *et al.,* 1999).

Similarly it was recognized as a yield-limiting disease of maize in Asia, particularly in temperate regions of China (Ward *et al.,* 1999), India, South East Asia and the Philippines (Coates and White, 1994). Kim (2006 and 2008 personal communication) reported that GLS occurred in the northern part of China. Similarly GLS was first time observed in Nepal in 2006. It became epidemic in the Dhungkhark VDC of Kavre Planchowk district and Kaleswor, Gotikhel VDCs of Lalitpur district of Nepal (Dhami, 2006 unpublished; Manandhar, 2007). After few years; this disease was reported from hilly districts of eastern, western and mid western hills of Nepal. Similarly this disease was found epidemic in Bhutan (Katwal, 2008). The incidence and severity of this disease is increasing in others parts of Asia.

Importance of Maize

Maize is the important staple cereal and principle food of inhabitants of high land regions of Asia particularly in Nepal, Bhutan, China and India. It is mostly grown in upland rain fed conditions during summer season and low land in winter season. Relay and intercropping with finger millet, legumes, and potatoes and in rotation with wheat and barley are common practice in different parts of hilly regions. In case of Nepal, more than two thirds of maize production was directly consumed at farm level in high land areas of Nepal (Paudyal *et al.,* 2001). In the lowland areas less than 50% of the maize production is used for human food and a significant part goes to the market. Small and poor farmers use maize green cobs and early harvesting to combat hunger. It is a source of green fodder and dry stover to feed the cattle. The dry stalks are a source of domestic energy (fuel wood) as well as a means to prevent soil erosion. In accessible areas, it is becoming an industrial as well as a commercial crop. However in high land regions it is used as human food rather than other purpose. Since past few years, GLS disease became the major problem to reduce grain yield in these regions. It was estimated that a 50-100% yield loss was observed in Bhutan (Katwal, 2008)

and >80% was estimated in Nepal (NARC, CIMMYT and DOA, 2007 unpublished).The maize growing environments, production systems and socioeconomic characteristics of Nepal, Bhutan and other high land regions of Asia are relatively similar. In a view of these circumstances maize is the most important crop interms of food feed, fodder etc. It is urgent need to increase maize production for sustainable livelihood in these regions and is ranked as a high research priority problem of maize production by national and international maize research organizations.

Epidemiology and factors associated with disease development

The GLS epidemic primarily depends upon three factors that interact with time and space. (1) The initial amount of inoculums (2) rate of reproduction of the pathogen within season and (3) proportion of healthy tissues remaining to be infected. High relative humidity, temperature (Latteral and Rossi, 1983; Stromberg, 2000) wide adoption of minimum and conservation tillage and maize monoculture (Payne and Waldron, 1983; Ward *et al.,* 1999) are equally important for GLS pathogen development. Relative humidity of 95% is optimal for germ tube elongation and formation of appressoria (Thorson and Martinson, 1993). Paul et al.(2005) observed that, sporulation is high at 100% relative humidity and 25^{O}C-30^{O}C temperature but the number of lesions and lesion expansion were not significantly different with >25^{O}C temperature. GLS was slow to develop when the mean daily temperature dropped below 20^{O}C (Ward, 1996; Nowell, 1997). Generally disease severity increases during mid to late summer due to favorable conditions for lesions expansion (Paul *et al.,* 2005). Ward and Nowell (1998) reported that incidence and severity of disease is usually associated with the amount and distribution pattern of rainfall. Early rains favor the development of primary lesions. Disease severity occurred more on warm humid, prolonged overcast, misty and cloudy days (Rupe *et al.,* 1982; Stromberg, 2000). In temperate regions maize monoculture, growing susceptible, local cultivars (de Nazareno *et al.,* 1993; Manadhar, 2007), plowing by locally fabricated ploughs and other biophysical factors favor pathogen development. Residues in neighboring fields may serve as a potential source of inoculums (de Nazareno *et al.,* 1993). The practice of stacking maize stalks in the field, feeding maize stovers to animals, use as animal bedding and use of und decomposed compost may spread the pathogen inoculums.

Similarly, stalk mulching, partially harvested maize stovers left standing in relay and intercropping fields may stabilize the pathogen. Blowing wind in the dry season may facilitate the dissemination of the pathogen up to 80-160 km each year (Ward *et al.,* 1999). The deficiency of mineral nutrients may have a potential role in GLS epidemics (Smith, 1989; Ward, 1996).

Disease cycle

Cercospora zeae-maydis (Tehon and Daniels,1925) is a polycyclic facultative pathogen (Chupp, 1953; Stromberg and Donahue, 1986). The fungi over winters as mycelium and stromata in infected maize residues left over the soil surface (Payne and Waldron, 1983). After harvesting maize fungus colonize on residues and produces conidia and disease cycle starts in spring (Payne and Waldron, 1983; Stromberg, 2000). The conidia disseminated to new corn plants by wind and splashing rain drops (Lipps, 1998). These new born conidia provide primary inoculums to infect newly planted maize fields (Latterell and Rossi, 1983; Payne and Waldron, 1983). The spores (conidia) infect the lower leaves through the stomata

and colonize leaf tissues. Conidia are produced from two to four weeks after initial leaf infection. Sporulation may be delayed in genotypes with moderate levels of resistance (Beckman and Payne, 1983). The fungus can remain dormant during the dry part of summer and then become active when conditions are favorable (Stromberg, 2000). The latent period of the pathogen is longer and can take as long as 14-28 days after infection for lesions to sporulate (Beckman and Payne, 1982; Stromberg, 1986). In about two weeks, these lesions will generate new spores and produce appresoria over stomata before penetrating the host tissue. Secondary cycles of disease are initiated by conidia produced within the lesions. Prior to grain filling very few infection cycles occur because of the long latent period (Beckman and Payne, 1982). Under favorable climatic conditions, disease progress can be rapid during the grain filling stage of crops.

Disease symptoms

The expression of symptoms depends on the genetic background of the genotype (Kim *et al.*, 1989). Resistant genotypes express the fleck type of lesions due to resistance gene (Latterell and Rossi, 1983). Moderately resistant genotypes exhibit chlorotic lesions (Roane *et al.*,1974) and the susceptible genotypes display necrotic spots (Latterell and Rossi, 1983). Early symptoms of GLS can be confused with symptoms of other foliar diseases particularly with southern leaf blight and northern leaf blight (Stomberg, 1986). GLS has two distinct features. Lesions occure as gray to tan in color and are distinctly rectangular in shape (5-70 mm long by 2-4 mm wide), and tan spot running parallel to leaf veins (Laterell and Rossi, 1983; Ayers *et al.*,1984; Stromberg, 1986).

The fungus generally produces spores on the lower side of leaves and the spore bearing structures may appear as small black specks. Early symptoms of infection include pinpoint lesions surrounded by yellow haloes. The early lesions are transparent when the leaf is held against the light while mature lesions are completely opaque (Latterell and Rossi¸1983; Smith and White, 1987). Leaf veins restrict pathogen growth and lesion width, but lesion width may vary with the distance between veins and proximity to other lesions. The lesions merge and kill entire leaves during favorable weather condition. The severe blighting of leaves and leaf sheaths are followed by stalk rotting and severe lodging (Stromberg, 1986), and premature death of leaves (Latterall and Rossi, 1983; Stromberg and Donahue, 1986; Stromberg, 2000). If the incidence and severity of disease is high during anthesis, the affected plants are fully dried but the ears have green husks, fresh silks, barren or partially filled ears and shrunken kernels (Manadhar, 2007).

Effects of GLS on crop

GLS reduces the grain yield and quality of silage maize. The yield loss has been estimated and quantified by researchers in Iowa, Virginia, Africa, China, Nepal and Bhutan. Researchers reported that, grain yield loss was found high when disease severity occurs during vegetative and tasseling/silking to grin filling stage and low grain yield loss was found after grain filling stage. Other several factors may contribute to this response, including yield potential of the cultivars, growth stage of crops and the ability of leaf blighting to predispose the variety to stalk rots. Documented yield losses of maize attributed to GLS vary from 11 to 69% (Ward *et al.*, 1999). Most of the researchers estimated that losses as high as 100% occurred when the pathogen attacked before the flowering stage (Stromberg and Donahue, 1986, and Lipps *et al.*, 1996). The blighting of leaves and stalk rotting caused the premature

death of leaves which reduced the amount of sugar and resulted in significant yield loss. Early blighting of the leaves above the ear leaf has led to yield losses of more than 50%. Blighting and premature death of the upper eight or nine leaves which contribute 75-90% of the sugar for grain filling resulted in a high level of yield reduction (Allison *et al.,* 1996).

Rupe *et al.* (1982) found that symptoms appearing before anthesis irrespective of planting date caused greater yield loss. Nutter and Jenco (1992) observed that disease severity at late dough stage resulted in a variation in yield of up to 90%. Late planted maize has greater GLS severity and a higher reduction in yield than earlier planted maize (Lipps, 1995; Manandhar, 2007).

Species of Cercospora

The genus *Cercospora* is a member of deuteromycetes and belongs to one of the largest groups of plant pathogenic fungi (Goodwin *et al.,* 2001). Initially *Cercospora zeae-maydis* was considered to be the sole causal agent of gray leaf spot. Recently it was accepted that three genetically distinct species of *Cercospora* are associated with this disease. Among the two sibling species of *Cercospora zeae-maydis;* Group I *Cercospora zeae-maydis* and Group II *Cercospora zeina* (*Crous et al.,* 2006) and *Cercospora sorghai var. maydis* were associated with this pathogen. The two sibling species (Group I and Group II) are genetically distinct but morphologically similar (Carson *et al.,* 1997; Wang *et al.,* 1998) and uniform internally with a genetic similarity of approximately 93 to 94% (Wang *et al.,* 1998). The genus *Cercospora sorghai var. maydis* is saprophytic and found in maize tissues (Carson and Goodman, 2006). It is associated with GLS lesions however, its pathogenicity is not confirmed (Chupp, 1953).The internal transcribed sequence of the *C. zeina* isolate was more similar to that of an isolate of *C. sorghi* var. maydis than to that of *C. zeae-maydis*. Although Group I can be distinguished from Group II by its faster growth rate of conidia (8-12mm per week) when compared to that of *C. zenia* (4-5 mm per week) in artificial media. Group I has the ability to produce cercosporin, longer conidiophores and broadly fusiform conidia, whitish to grayish mycelia, irregular edge and visible quantities of reddish toxin (cercosporin) where as Group II contains mycelia whitish to grayish in color with olive green mycelia, irregular edges on top and no visible reddish toxin. Although two isolates have some differences in morphology and the production of cercosporin. They produce exactly the similar symptoms in maize. Group I is prevalent and predominates over *C. zeina* throughout the maize growing areas of the eastern and midwest regions in the USA, Latin America, China, India, Nepal and Bhutan. Group II species are confined to Africa and the Eastern US. Meisel *et al.* (2009) found that, Group II (*Cercospora zenia*) is the causal agent of GLS in Southern Africa. Similarly Crous *et al.* (2006) reported that, the Group II pathotype is prevalent and predominant in East Africa but the origin of the pathogen is unknown. It is generally accepted that *C. zeina* originated in Africa but spread from sorghum (indigenous host) to maize. *C. zeina* has higher genetic variability in Africa compared to the USA (Dunkle and Levy, 2000). They also argued that the GLS pathogen was introduced to the USA from Africa.

Inheritance of gray leaf spot

Different gene actions are involved for early season and late season resistance to gray leaf spot (Bubeck *et al.,* 1993, Coates and White, 1998). The expression of resistance is affected by the genetic background of a susceptible parent (Kim *et al.,* 1989) and microclimatic

conditions (Payne and Waldron, 1983). Several genetic studies (Verma, 2001; Menkir and Ayodele, 2005; Donahue *et al.,* 1991) reported that the resistance to GLS was quantitatively inherited with a preponderance of additive gene action and possible minor dominant and epistatic gene effects which, contributed to the resistance. Manh (1977) reported that, additive genetic effects accounted for 82 to 96% of the total variation in GLS resistance among generations, although dominance and epitasis provided some contribution. In diallel cross analysis of GLS resistance Gevers and Lake (1994) found that additive and non additive genetic effects were important in GLS resistance. South African researchers found high frequency of quantitative resistance to GLS present within commercial hybrids (Nowell, 1977). In addition to quantitative resistance, a qualitative resistance to GLS was observed in maize genotype in South Africa (Gevers *et al.,* 1994) and it was observed that non-additive genetic effect plays a significant role in resistance mechanism. He reported that crosses between resistant and the most susceptible inbreeds resulted in resistant hybrids due to the predominantly additive nature of gene actions and major dominant effects of some genes. Quantitative resistance to GLS has been found to impact on lesion size, latent period and sporulations (Ayers *et al.,* 1994). Host resistance is regulated by a small number of quantitative loci with five or more genes involved which are inherited additively (Ayers *et al.,* 1985; Thompson *et al.,*1987; Bubeck *et al.,*1993; Saghai-Maroof *et al.,* 1996). Clements et al. (2000) found that five quantitative trait loci (QTLs) were significantly associated with GLS resistance. Four of them were associated with ear height relative to plant height. Li-yu *et al.* (2007) reported that a total of 57 QTLs for GLS resistance were found and located in each chromosome. They were primarily found in chromosome 1, 2, 4, 5 and 8. Ward and Nowell (1988) reported that QTL 1 and 2 had additive effects for GLS resistance, 4 had a dominant/recessive component and 8 had a recessive effect. Chromosome 8 was included in both parental lines for higher GLS resistance in hybrids. Saghai-Maroof *et al.* (1996) observed that the QTLs located in three chromosomes (1, 4 and 8) had large effects on GLS resistance and were consistent. QTLs with smaller effects were found in chromosome 2 and 5. Chromosome-1 QTL had the largest effect. However, the findings regarding the chromosome 5 might have been false. Chromosome 4 belonged to the susceptible parent and all 3 chromosome (1,4 and 8) were from the resistant parent. The use of inbred strains that were highly resistant to GLS produced highly resistant crosses (Ivanovic *et al.,*1982; Gevers and Lake, 1994) and the intermediate GLS resistant inbreed strains produced highly susceptible hybrids (Huff et al., 1988).Whereas Coates and White (1994) reported that several inbreeds line identified as resistant to GLS did not produce resistant hybrids in crosses with a susceptible tester line. The introgression of resistance genes through the crosses of resistant with resistant germplasm enhanced the high level of resistance which was useful to develop resistant inbred strains (Menkir and Ayodele, 2005). They observed that the GLS score was significantly higher among the crosses of susceptible with resistant germplasm as compared to the F_1s of resistant with susceptible germplasm. They also reported that the F1s of resistant with susceptible germplasm were more durable. However the yield difference was not significant. The cytoplasm genes contributed significantly to the variation in GLS scores among hybrids, hence from the crosses between susceptible and resistant lines, the resistant line (VA14) could be used as a female parent to enhance the level of resistance (Menkir and Ayodele, 2005).

Disease Management

Cultural practices reduce the pathogen inoculums but some losses from disease are inevitable in areas where the disease is endemic and of epidemic proportion. However these practices are recommended as immediate actions to minimize yield loss.

Crop rotation and cropping pattern

Maize is the only host crop this fungus is known to attack. Rotation of the non host crop for two years can reduce the disease inoculums effectively where the management of conservation tillage and field sanitation is equally important (Lipps, 1998; Wolf, 2002). However there is no alternative crop to replace the maize for crop rotations in hills. The possible crops for rotation are soybean and potato. Mixed cropping of soybean with maize, relay and intercropping of finger millet are widely used practices. Mixed or inter cropping hinders air circulation inside the crop field which helps to increase relative humidity and favors disease development. Cultivation of wheat in maize cultivated field is not recommended, because *Gibrella zeae*, is one of the most common causes of corn stalk rot. Wolf (2002) pointed out that the incidence and severity of head scab in wheat may be due to ear rot of maize.

Tillage practices

There is a positive correlation between tillage practices and disease epidemics. Conventional tillage incorporates the surface residues in to the soil. The burial of infested debris facilitates rotting and deprives the fungus of a food base. However conventional tillage may be effective only in regions where external inoculums are minimal (Payne *et al.*, 1987). Zero and minimum tillage favor the disease development because of old maize residues left over the soil surface in the field.

Residues and Weed management

The infected residue of a previous crop left over the soil surface is the principal source of inoculums. There was a strong positive correlation between the amount of infected maize residue and disease inoculums (Asea *et al.,* 2002; de Nazareno *et al.,* 1993a). They reported that disease intensity was higher in a high residue treated plot than a non treated plot. The collection of stovers which are stacked in the field and near the home stead, is a common practice. This practice may help to keep the field clean and reduce disease inoculums. However it is not always practiced for the following reasons, maize stalks are mulched and dried stovers are used for animal bedding. The use of un decomposed compost also harbors and disseminates the disease inoculums. Weed management practices increase air flow within the crop canopy, reduce relative humidity and help limit the time period favorable for pathogen infection (Wolf, 2002).

Maintain the plant density

High plant density creates high relative humidity and a microclimate which favors disease development (Beckman and Payne,1983; Payne and Waldron,1983; Ayers *et al.,* 1985) where

as de Nazareno *et al*.(1993a and 1993b) argued that high plant density has less disease incidence because of less air flow to disseminate secondary inoculums.

Adjustment in time of planting

Most of the researchers reported that, late planted maize was more affected than early planted maize because disease development was slow due to unfavorable environmental condition early in the season (Payne and Waldron, 1983). They also suggested planting early maturing cultivars earlier in the season to minimize the yield loss. The late planted maize tended to develop more severe GLS, because the plants experienced initial infection at earlier stages and there was a greater opportunity for multiple cycles of infection before the plants

reached their physiological maturity (Stromberg and Donahue, 1986; Bhatia *et al.,* 2002). Early maturing cultivars escape from disease because plants face first cycle of infection at physiological maturity stage. Assured irrigation is crucial for timely planting but in these regions planting maize primarily depends upon monsoon rain.

Balanced use of fertilizers

Application of chemical fertilizers significantly affected GLS progress (Okorai *et al.,* 2004). They reported that GLS epidemic was significantly higher in non fertilized plots than fertilized plots. They also observed that a single application of nitrogen increased the predisposition of plants to GLS but a combined application of nitrogen and phosphorus at a recommended level significantly reduced the predisposition effect of a high nitrogen level. The unbalanced use of nutrients caused host nutrient deficiency and losses of resistance status predisposed the plants to GLS (Smith, 1989; ward, 1996). Maize growers in hills and remote areas do not have access to fertilizers because of high cost and less developed infrastructures. The use of farm yard manure and compost is a common and widely adopted practice in these areas. These organic manures are useful for improving the soil‟s physical properties but they do not supply the required amount of nutrients to the maize plants.

Use of fungicides

Fungicides are only recommended for an emergency on susceptible hybrids and previously infected crop fields. Tilt (active ingredient propiconazole) and Quadris (azoxystrobin) are effective to manage GLS. The use of fungicides to control GLS in maize seed production is cost effective but it is not directly applicable to grain production (Shaner *et al.,* 1999). Smith (1988) found that the Benzimidazole group of fungicides has commonly used in many crops and in some cases pathogens have developed resistance rapidly. The use of fungicide is beyond the access of resource constrained farmers and moreover increases the production cost, hazardous to human health and has a negative impacts to environment.

Tolerance crop breeding principle

The development of a host that is resistant to biotic and abiotic factors is cost effective and environmentally sound. The two terminologies are frequently used in resistant ie. horizontal resistance and vertical resistance (Vanderplank, 1978). Horizontal resistance (tolerance) remains effective while being extensively used in agriculture for long periods in an environment conducive to disease. This tolerance crop breeding principle is the use of quantitatively inherited genes to breed new crops to combat biotic stress (Kim *et al.,* 2009). This principle depends on the number of genes and gene action involved (Kim, 2000). This principle does not aim at absolute (100%) controls but attempts to attain partial control (95%). The concept of tolerance is similar to "partial resistance", "general resistance", "horizontal resistance", "durable resistance", and "mature plant resistance" (Kim 1994a, 1996b). It is partial and race- non specific in phenotype, oligogenic or polygenic in inheritance and is conditioned by additive or partially dominant genes (Gevers and Lake, 1994) and allows the survival and development of the pathogen. The tolerant host is attacked by the pathogen in the same manner as the susceptible genotypes, but there is little or no loss in biomass production or yield (Kim, 1996b; Singh, 2005). It provides the space for host flexibility and host adjustment in a changing environment. This is useful to producers particularly for those who are subsistence farmers of underdeveloped countries (Kim, 1996b; Ward *et al.,* 1999).

Resistance is synonymous with complete resistance, true resistance and vertical resistance with hypersensitive response. With this principle host plants provide the negligible space for pathogen development. In the case of resistance, the reproduction rate of pathogen „r" is 0 or close to 0, but in the case of tolerance „r" is never 0. Because „r" is smaller than 1 (100%) but greater than 0, this principle aims at absolute (100%) control, complete or a high resistance until resistance genes work, but can be lost through an associated and matching change in the virulence genes in the pathogen (Vanderplank, 1978). The complete control (100%) by a single gene always creates selection pressure that may invite about the mutation of pest (Kim, 2000).

Future Strategies

Increase crop diversity and broadening the base of germplasm

Maize is not a native crop in Asia thus the genetic base is narrow particularly in temperate regions. The maintenance of adequate genetic diversity in crop plants is a prerequisite for plant breeding (Goodman, 1999). The crop plants gradually become vulnerable to disease and pests because of an elimination of host diversity within a very homogeneous host population (Strange and Scott, 2005). The genetic vulnerability in locally used breeding materials and commercial hybrids would enhance the disease severity (Givers and Lake, 1994). The genetic mixtures possess greater stability of performance and their inherited resistance to disease is more effective and more durable (Wolf, 1993). Exotic germplasm is a potential source of new alleles for introgression into adapted germplasm to increase the variability (Goodman, 1985). The use of a resistance source for conversion and incorporation would be better from the sources in the same heterotic group (Kim, 2000). These are a useful source of alleles for resistant to disease, insect pests and for broadening the genetic base of temperate germplasm (Goodman 1999). Eberhart *et al.* (1995) proposed the use of elite exotic germplasm with high yield potential and resistance to disease and insects as a good strategy for integrating genetic

diversity into maize breeding populations. It is crucial to cross the CIMMYT and IITA maize germplasm with locally adapted and introduced germplasm.

Selection and development of tolerant cultivars

The development of locally adapted tolerant cultivars enhances the durability of resistance (Nowell, 1997). The breeder should practice selecting the tolerant genotypes from adapted germplasm based on yield potential and stand ability under disease pressure. Plants with mild symptoms of the disease and good yield at maturity will have the highest tolerance (Kim, 2000). The incorporation of new genotypes, either local or exotic, in the evaluation of a breeding program increases the availability of genes for resistance that were not previously available. For example; in Nepal, NARC and CIMMYT scientists found Deuti, Manakamana-3 and Ganesh-1 to be relatively tolerant with GLS (NARC, CIMMYT and DOA, 2007 unpublished).

Similarly Ashom I and Ashom II varieties were found tolerant in Bhutan (Katwal, 2008). These improved open pollinated varieties should be crossed with GLS resistant materials either locally developed or introduced. As the inheritance of GLS resistance is mainly quantitative in nature, the frequency of resistant alleles in a population can be increased by population improvement techniques. Recurrent selection can be an effective method to incorporate and accumulate the resistant genes in elite breeding materials if several genes with additive gene action are involved. This method of selection increases the

frequency of favorable alleles for the trait under selection (Goodman, 1999) and maintains the genetic variability of the population through the recombination of genes between cycles of selection and permits continued selection.

The International Maize and Wheat Improvement Centre (CIMMYT), Institute of International Tropical Agriculture (IITA) and International Corn Foundation (ICF) have been given the high priority problem of managing GLS disease in these regions. CIMMYT, IITA and ICF are providing financial, technical and germplasm support. CIMMYT has been conducting collaborative GLS disease research activities in Nepal and Bhutan (Manandhar and Katwal personal communication). The gray leaf spot screening nursery has been completed in disease hot spot area in Nepal. Based on their GLS disease response and overall agronomic performances, resistant population ZM627, ZM401, ZM525, and 07SADVI have been identified. Similarly some tolerant inbreeds and CIMMYT hybrids having good yield potential were identified. OPvs, synthetics, hybrids and inbreeds will continuously testing in artificial inoculated as well as natural GLS hot spot area to evaluate their tolerance level.

CONCLUSION

GLS is still causing enormous yield losses in tropical, subtropical and temperate regions. It has threatened the sustainable food production and livelihood of the communities in Asia. This condition will become worse in the developing countries where maize is the staple food. Thus it is becoming the major concern of plant breeders and pathologists. The effort and research focused on this disease has been mainly concentrated in the USA and Africa. There is not a sufficient source of information about this disease in Asia. Very limited work has been done in the molecular aspect in China. In collaboration with CIMMYT/Mexico and ICF/Korea, scientists of NARC/Nepal and Bhutan have initiated preliminary research. Thus the concerned national and international organizations primarily CIMMYT and IITA should concentrate their efforts in under developed countries otherwise this disease may become the

primary cause of grain yield loss in these regions. Chemical recommendation for disease management is only the acceptable for emergency situations. Chemicals should not be applied in breeding nurseries. The breeding of crops through the tolerance principle is effective for resource constrained farmers. This is the durable and economical means for disease management. This principle is equally important for an eco-friendly environment.

ACKNOWLEDGMENTS

The authors would like to express their sincere thanks to Agriculture Botany Division, Agricultural Research Council Singh Durbar, International maize and Wheat Improvement Centre, Mexico, International Corn Foundation and International Agricultural Research Institute of Kyungpook National University of Korea for their kind support and cooperation. The authors also would like to express their gratitude to S.P. Khatiwada chief of Agriculture Botany Division, NARC.

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