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REVIEW

Turcicum leaf blight—sustainable management of a re-emerging maize disease

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Abstract *Turcicum* leaf blight of maize caused by the fungus *Setosphaeria turcica* is a serious foliar disease of maize distributed widely throughout the world and causing significant yield losses. The disease is more prevalent in humid weather with temperature between 20–28 °C and causes small cigar-shaped lesions to complete destruction of the foliage. Though there are several management practices available, identification and deployment of host plant resistance is a pragmatic approach to control the disease. However, qualitative resistance is unstable and breaks down easily by emergence of new races of the pathogen in maize necessitating the development of durable TLB resistant cultivars. Application of modern molecular tools and availability of high-density molecular marker data are expected to accelerate efforts to develop resistant hybrids. This review provides a focuses on current status, and future research needs especially biological control and sustainable integrated management strategies of TLB.

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Introduction

Turcicum leaf blight (TLB) also called as northern corn leaf blight (NCLB) caused by the ascomycete fungus, *Setosphaeria turcica* [(Luttrell) Leonard and Suggs.)] with its conidial state *Exserohilum turcicum* [(Pass.) (Leonard and Suggs.)], is of worldwide importance [87, 82, 14, 92]. The disease was reported for the first time from New Jersey, USA in 1878. The symptom of the disease in maize is a severe defoliation especially during grain-filling period [92]. The intensity of the disease is severe in mid-altitude tropical regions due to high humidity coupled with low temperature and cloudy weather. The first serious outbreak of TLB was occurred in Connecticut, USA during 1889. In India too, severe losses of 25–90% in grain yield due to TLB epiphytotics have been reported [13, 44, 32]. Lately there has been an increase in the TLB disease incidence in the maize producing regions. Vivek et al. [101] reported that the incidence and severity of TLB had increased, especially in Southern Africa, in the past 3–5 years. The major reasons hypothesized for increased incidence were, viz, breaking down of qualitative resistance, which is not stable, introduction of temperate susceptible germplasm into tropical environments, thus giving rise to increased TLB severity. The management practices like crop rotation, seed treatment and application of fungicides have been recommended [51, 88] to reduce the losses due to TLB. However, the host plant resistance (HPR) has been considered as most appropriate and economical strategy due to several advantages like environmental friendly,

convenient to adapt at farmers' level. In fact, HPR plays a significant role in integrated disease management approach as an important component. Thus, identifying resistant genes and genotypes to this disease and combining them with yield traits is a priority of maize breeding program [90]. The resistance breeding has been emerging as an important area of research in recent years due to recent surge in increased losses due to disease incidence. It is important, therefore, to identify more diverse sources of resistance to TLB, as the increase in the disease severity has the potential of threatening maize grain productivity with a negative influence on food protection. Sibiya et al. [92]. The review outlines the basis for revised management approaches by way of integrating use of resistance germplasm/genotypes with adjustments in cultural management and/or fungicidal control practices to make maize production both more profitable and more sustainable globally.

Distribution and economic impact

It is serious problem in the Northeastern United States, in sub-Saharan Africa, and in areas of China, Latin America, and India [1, 21]. In India, it was reported for first time by Butler during 1907 from Bihar. Later it was reported from many parts of the country, viz., Lalmardi, Srinagar [48], Punjab [67], Himachal Pradesh [13], Sikkim, Meghalaya, Tripura and Assam [88] and Kashmir valley [81]. The endemic areas (hot spots) of the disease in India have been identified, which are, Arabhavi, Nagenahalli in Karnataka, Kolhapur in Maharashtra, Karimanagar in Andhra Pradesh, Dholi in Bihar and Almora in Uttarakhand [55]. It is observed in most maize growing areas that have high humidity coupled with moderate temperatures.

The disease affects maize from seedling stage to till harvest. If the disease epidemics start at an early stage, it causes premature death of blighted leaves as a result losing their nutritive value as fodder, causes alterations of the seeds, resulting in reduced total sugar content, lowered germinative capacity, restricted starch formation, chaffy kernels, and reduced vigor and yields, and the heavily infected plants are predisposed to stalk rots [81]. Yield losses due to TLB can be large, but vary depending upon environmental conditions and geographic location. The losses were found to be directly proportional to disease intensity [13, 20]. However, yield losses under experimental conditions of artificially created disease epiphytotics were estimated to the extent of 66% in susceptible variety Basi and 56.0% in CM 202 [80]. Maize yield decreases up to 0.6 t ha^{-1} when its severity reaches more than 75%, [92].

Disease symptoms

TLB starts as long, elliptical, gray-green lesions measuring 3–15 cm in length. As the lesions mature, they become tan with targeted, darkish zones of fungal sporulation. Because the disorder develops further, the lesions may just coalesce, forming enormous blighted areas, and entire leaves may emerge as blighted [100]. The disease mainly harms the lamina, sometimes infects sheath and bract also, but does not harm fructose directly [89]. The infection occurs from the lower lamina, gradually extends to the upsides [112]. The disease injures or kills the leaf tissues, as a result reducing the area of green chlorophyll which manufactures food for the plant. If significant leaf area is blighted, the vigor and yields are reduced. If plenty of the green area is killed, starch formation is restrained and the kernels are chaffy and are not appropriate for fodder because of the lowered nutrition value. Pant et al. [76] studied the effect of disease on photosynthesis and determined that infected leaves showed a significant reduction in CO_2 fixation with increase in the severity of the leaf blight. The leaf temperature and transpiration rate extended with increase in severity of the disease in direct proportion. When the weather is damp and rainy, spore production causes the lesions to appear dark gray, olive or black; gray black mold layer will occur on the disease spots. The gray black mold layer is the pathogen's conidiophores and conidia [53].

The pathogen

Setosphaeria turcica is a hemibiotrophic ascomycete fungus, living on live plant tissue before causing necrosis, drawing nutrition from dead tissue [60]. Nine species (Table 1, [23]) are included in the genus *Setosphaeria* (anamorphic stage *Exserohilum*) [93, 23]. The type species for the group is *S. turcica* [57].

The pathogen survives in temperatures ranging between 17–28 °C with moderate to high humidity but can tolerate harsher conditions also [106]; like many ascomycete plant pathogens, *S. turcica* is notion to spend nearly all of its existence as a haploid organism simplest becoming diploid for a brief stage during sexual recombination earlier than undergoing meiosis to produce haploid ascospores [86]. Population genetic reviews on *S. turcica* established on RAPDs and mating variety assessments have, however, verified high genotypic diversity and even distribution of the two mating types on maize grown beneath tropical conditions compared to *S. turcica* populations accrued in temperate areas [7, 86]. The cause of this high genetic diversity is unknown, but presence of a sexual stage has

Table 1 Characteristics of species of the genus *Setosphaeria*

Species	Lifestyle	Host	Location
S. turcica	Hemibiotrophic plant pathogen, heterothallic	Maize, sorghum	Widespread worldwide
S. rostrata	Plant pathogen, opportunistic human pathogen, heterothallic	Grasses, bananas, humans	Worldwide
S. prolata	Plant pathogen, heterothallic	Maize, wheat, other cereals seeds and leaves	Ethiopia, Guatemala, Jamaica, Kenya, USA
S. holmii	Plant pathogen, heterothallic	*Cymbopogon*, wheat *Dactyloctenium*, rice	Egypt, Kenya, Nigeria, USA
S. pedicellata	Plant pathogen, heterothallic	Wheat roots, rice, maize, *Echinochloa*	Egypt, India, Pakistan, USA
S. monoceras	Isolated from plants, heterothallic	*Echinochloa* (leaves), rice	Australia, Cuba, India, Israel, Japan, Turkey, Russia, USA
S. glycinea	Heterothallic	Soybean	–
S. khartoumensis	Isolated from grain in culture, homothallic	Sorghum	Sudan
S. minor	Isolated from plants, heterothallic	*Dactyloctenium*	Australia

been suggested. Established on DNA sequence information derived from 28S rDNA sequences, *Setosphaeria* species are placed within the *Pleosporaceae* family. The centre of origin for *S. turcica* has been suggested to be both Mesoamerica and East Africa through a coevolution with either maize or sorghum [7]. The pathogen allows penetration and colonization with the production of a range of secondary metabolites and toxins. The *S. turcica* genome includes two genes encoding xylanase enzymes, which degrade arabinoxylan in the plant cell wall causing loss of integrity and aiding penetration [19]. Chung et al. [15] traced the time direction of infection and early colonization of maize leaves using light microscopy and observed the process of conidium germination, appressorium formation and penetration by way of the plant cuticle and epidermal cell wall. After this, hyphae grow toward and enter the vasculature, the place they proceed to grow and ramify inside vascular bundles without causing seen symptoms for seven days following inoculation. Defense responses like callose deposition and the accumulation of auto fluorescent phenolic compounds are induced around the site of infection. A HT-toxin (*Helminthosporium turcicum)* isolated from *S. turcica* [111] has been recognized as a lipophilic phytotoxin referred to as Monocerin. Monocerin-treated maize and sorghum plants inhibited chlorophyll synthesis, reduced root growth and lesions and necrosis on susceptible genotypes [4]. Furthermore, leaves were treated with Monocerin broaden necrosis that spreads throughout the vascular system, suggesting that Monocerin HT-toxin is involved not only in penetration, but also in later degrees of infection [17].

Epidemiology

Understanding the epidemiology of TLB on maize enables us to devise rational disease management strategies that take into account the pathogen's life strategies. TLB varies in incidence and severity from year to year and from one locality to another depending largely on genetic makeup of the plants and prevailing environmental conditions [29]. The disease is favored by mild temperatures ranging from 20–28 °C, relative humidity from 90 to 100%, and low luminosity [69, 95]. The infection of *S. turcica* on maize occurs from seedling to harvesting; nevertheless, maximum severity used to be seen from tasselling and 6–8 weeks after silking resulted in heavy loss [13]. Levy and Cohon [59] reported that the disease is more aggressive in young susceptible plants with an optimum temperature for infection and lesions at 28 °C. Tsen [98] studied the effect of sowing dates on the occurrence of disease and reported that optimum temperature of 20 to 26 °C and over 80% RH were congenial to disease development. He concluded that RH plays a greater role than the temperature for the severe outbreak of the disease. The TLB disease severity was maximum ranging from 71 to 84% during the crop sown in the months of September, October and November. Mallikarjuna et al. [63] reported a linear relationship between disease severities and meteorological factors namely temperatures and relative humidity. The disease is favored by low temperature (13–28 °C) and high relative humidity (89–93%). Losses due to TLB are more severe when the leaves above the ear are infected at, or slightly after, flowering. Humid and hot weather, late planting and an

abundance of prior maize residues expand the risk of disease infections [33, 29].

Inoculum sources and infection

Maize residues on the soil surface are the major source of inoculum for several fungal pathogens of maize. As shown in Fig. 1, pathogen overwinters as mycelium and chlamydospore in infected crop debris. The fungus spreads biotrophically during the initial infection process before switching to a necrotrophic lifestyle. At the onset of the next season, fungi in crop debris begin to sporulate according to higher temperatures and humidity. Spores (conidia) are then disseminated by means of wind and rain splash to freshly planted maize. Conidia can be carried over vast distances by wind and germinate in temperature ranging from 20 to 28 °C and during periods of extended leaf wetness (6 to 18 h) leading infection to maize tissue [34]. Secondary cycles of disease occur where conidia produced in disease lesions are disseminated within the crop and to other fields by rain splash and wind [29, 62].

Therefore, it is indicated that the maize stubbles and debris present in the upper layer of the field are considered as the potential sources of infection and may help in perpetuation of the disease.

Inoculum multiplication and inoculation technique

The mass multiplication of the pathogen was prepared on sterilized sorghum grain culture [46]. One hundred grams of sorghum grains placed in 500-ml conical flask was soaked in tap water for 24 h. The excess water was drained off. The material was sterilized twice at 24-h interval at 1.10 kg/cm^2 pressure for 1 h. The contents of the flasks were thoroughly shaken after sterilization to prevent clumping. The flasks were aseptically inoculated with pathogen culture and incubated at 27 ± 1 °C for 20 days, and the flasks were shaken every alternate day to avoid clumping. Within 3 weeks, the sorghum grains were covered with black mycelial growth and conidia of the fungus colonized sorghum grains. Such fully colonized sporulated sorghum grain culture was used for creating artificial epiphytotic conditions in the field following whorl drop method of inoculation [85]. The plants can also be inoculated at 3–4 leaf stage by applying two pinches (about 100 mg) of powder of diseased leaf obtained from actual lesion area. The powder containing 0.65 × 10^{4}conidia/g was applied in central whorls and then sprinkled with water. Finally, the whorls were covered with polythene to create humidity. The disease first manifested itself by 8–12 days after inoculation depending upon different genotypes. Disease appeared quickly in infector rows and became severe by the time of silking. Disease intensity was recorded using 1–5 rating scale on individual plant basis at silk drying stage [79]. The genotypes showing disease score between 0.1 and 2.0 was considered as resistant (R), 2.1–2.5 as moderately resistant (MR), 2.6–3.0 as moderately susceptible (MS), 3.1–4.0 as susceptible (S) and 4.1–5.0 as highly susceptible (HS) [79]. In order to reduce the subjectivity of disease severity visual estimates, diagrammatic scales have been developed for several pathosystems. In plant pathology, diagrammatic scales are a valuable tool for identifying genetic variations in disease resistance among plant genotypes. The first diagrammatic scale for assessing TLB was developed by Elliot and Jenkins [24]; nevertheless, their diagrams depict the entire

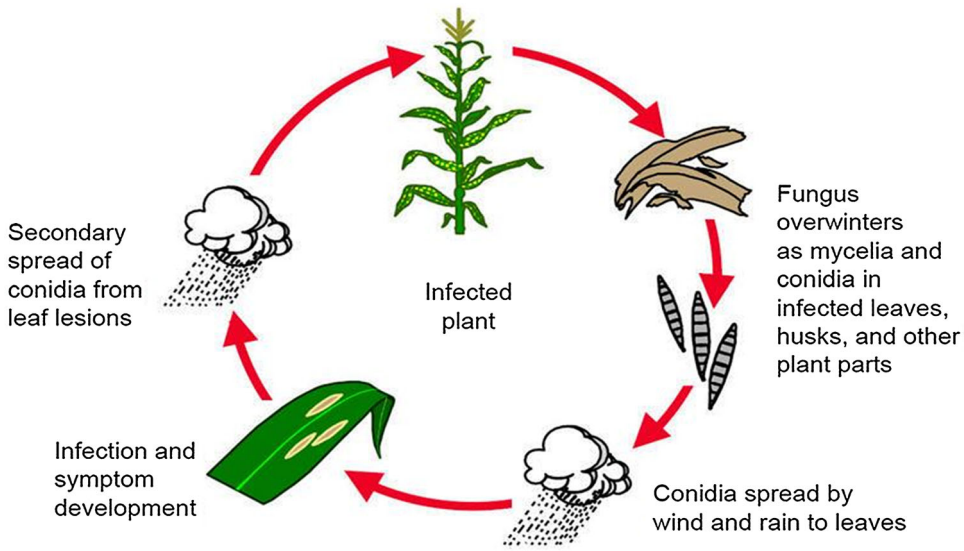

Fig. 1 Disease cycle of TLB (Courtesy-www.pioneer.com)

plant, which is able to, usually, restrict its use as a result of simultaneous prevalence of different leaf diseases. Another published diagrammatic scale, developed by Pataky [77], representing the severity levels on leaves with arithmetic increments, may confuse evaluators. Severity is undoubtedly the most sensitive criteria for screening maize genotypes based on differences in levels of resistance to TLB. Using the diagrammatic scale developed by Vieira et al. [100] was able to improve precision, accuracy and in the construction of disease progress curves (Fig. 2) of TLB.

Sources of resistance

TLB resistance sources across the globe have been identified by screening germplasm against TLB, and high levels of resistance have been reported in a number of inbred lines (Table 2). The review on resistant sources and their utilization in regular breeding program are available [103]. Progress of new maize cultivars with improved levels of disease resistance as well as greater abiotic stress tolerance might be extra sustainable and effective for increased maize yields as well as farmer's profitability, especially in the smallholder farming sector [72].

Genetics of resistance

The identification of physiological race is very important in the disease control and the study of maize and pathogen interaction [97, 112]. The physiological races are identified by the reaction of spot after inoculation. During the past two decades, an increasing number of novel races had been identified in China (0 and 1), in Mexico (23 N, 23 and 2 N), in Zambia (23, 23 N and 0) and in Uganda (0, 2, N, 23 N) [43, 66, 104, 11, 58, 68]. Extensive studies have been conducted to differentiate different races (Table 3) [56, 71, 25]. In northern China, Dong et al. [22] reported 13 different physiological races (0, 1, 3, 12, 13, 23, N, 1 N, 2 N, 12 N, 3 N, 23 N and 123 N) based on their infection types on the host differentials. Understanding the nature of resistance and its behavior is important to transfer and utilize the durable and stable resistance sources across different genetic backgrounds. Several studies have been conducted on nature of TLB resistance, and it has been found that resistance was partially dominant and is controlled by many genes, i.e., polygene [108, 5]. Polygenic resistance is expressed primarily as reducing the number of lesions with little decrease in the amount of sporulation compared to susceptible types.

In India, Pandurangegowda et al. [75] and Mallikarjuna et al. [63] studied the nature and type of gene action governing resistance to *E. turcicum* in six generations. They observed that all types of gene effects (additive, dominance and epistasis) were operating in various crosses. Dominance and dominance x dominance type interaction were present in the majority of crosses and inbred lines tested. In addition, they also found that additive and epistatic effects are important for disease resistance at anthesis stage. The lines/parents could be used for further evaluation for developing superior double cross/top cross hybrids. Inbred lines carrying resistant genes have been used in maize breeding program.

In order to identify the genomic regions responsible for resistance reaction, several efforts were made to map and locate the genomic regions. The efforts have led to

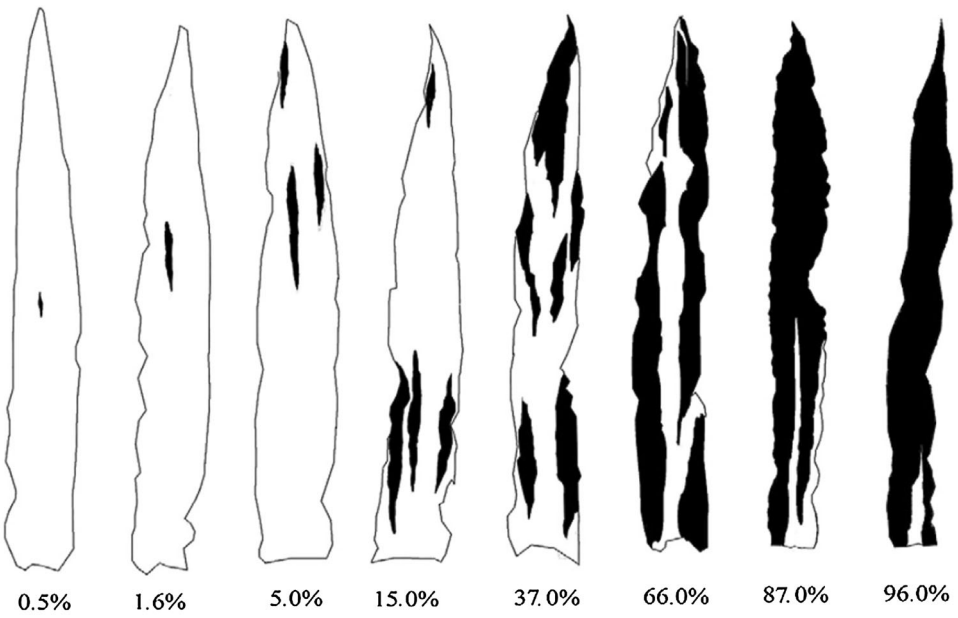

Fig. 2 Diagrammatic scale for the assessment of *Turcicum* leaf blight severity on maize leaves (Courtesy-[100])

Table 2 Maize inbred lines with reported resistance to TLB

Country of origin	Test site	Inbred line (s)	References
USA (Temperate)	USA	C.I. 23, K175, L97, Mo21A, NC34, T528, CI. 90A, CI.93A, Wh4971, H49, H52, H55, H60, C 103, CI. 28A, CI. 42A, CI. 64, IL545a, IL611a, IL676a, IL677a, IL685d, IL 731a, IL797a, AXH, H4110, H4460a, MAAW, MABI, R177, 4HC, 4JEOh S10 S_1 progenies, 69-1, Mo 17, H 99, OhS10 S_1- 90, RSSSCC6, W153R, Oh43, C103 C.I.66, C.H.586-12, H60, H95, Mo42, Ms75, Oh509A, W570, H102, H103, H110, H111, Pa392, Pa860, Pa877, Pa879, Pa891, SD40, SD41, SD42, SD44, SD46, SD47, SD48, SD101, ND256 and B115	[94, 31]
USA (subtropical)	USA Uganda Yugoslavia	Fla2AT115 (Florida), Fla2AT116 (Florida), Hi39 (Hawaii), H55 (Indiana), F (Kenya; a parent of hybrid H632), Narino 605	[61]
India	India, Kenya, Nigeria, Hawaii	CM-104, CM-105, CML 197, CML 202, CML 204, CM-118, KUI-1414A, NAI-147, B-37, PTR, Ade-C, Phill DMR-1, Phill DMR-8, CM-111, CM-501, CM-121, KDMI-12, CM-119, CM-136, CM-211, Hyd.sel.9, Hyd.sel.15, Hyd.sel.11, Indimyt 345, NAI- 147, SKV-6L, SKV6E, SKV-26, SKV-33, SKV-43, SKV-44, SKV-62 and inbred A (parent of H632), NAI-102, NAI-104, NAI-115, NAI-116, NAI-123, NAI-124, NAI-135, NAI-137, NAI-141,NAI-142, NAI-617, NAI-608, CML-124,CML-410, CM-114, CM-205 and SKV-50	[35, 85, 32, 73, 74, 75, 20, 64, 65]
Colombia	Kenya. Hawaii India	ICA127	–
Mexico (CIMMYT)	Germany	B93, B100, CML434, CML437, CML438, CML439, CML102, CML117, CML132, CML443, CML444, CML445, Eto -25, Eto-81B, Eto-182C	[30]
Ethiopia	Ethiopia	F7212	[10]
Kenya	Kenya. Nigeria. Zimbabwe	E12-3, E12-210, inbred A, S21 C3- 8, R11C2-64, H632 (inbred), H632F	[41]
Zimbabwe (CIMMYT)	Kenya. Zimbabwe	IntA-5-1, CML 199, CML 202, CML 204	–
Zimbabwe	Zimbabwe	M162 W	[52]
South Africa	South Africa,	B1138T	[34]
Thailand	Kenya, Nigeria	Narino 330	[52]
Philippines	Kenya, Nigeria	MIT 2, MIT 11	[52]
Australia	USA	D21, HB, 21H, 25	[40]
Switzerland	Switzerland	1511 C, 493B, R2038, Lanzarote	
Germany	Germany	D164, D 305, R2038IU 1414, SR52 F	[52]

Table 3 Interaction of differentials (Ht genes) with different races of TLB

Pathogen	Maize line						
Race designation	B73	Pa91	Pa91Ht1	Pa91Ht2	Pa91Ht3	B68	B68HtN
0	S	S	R	R	R	S	R
1	S	S	S	R	R	S	R
2	S	S	R	S	R	S	R
12	S	S	S	S	R	S	R
23	S	S	R	S	S	S	R
23 N	S	S	R	R	R	S	R
123 N	S	S	R	S	R	S	R

B73, Pa91, B68 = maize genotypes carrying or not carrying Ht or N genes. Race designation = able to cause virulence on corresponding line, i.e., maize line with gene Ht1 is susceptible (S) to race 1, but resistant (R) to races 0, 2, 3, or N or combinations thereof

identification of both qualitative (major gene) and quantitative (polygene) loci showing resistance to *E. turcicum* [103]. The study conducted by Hooker and others [37, 27, 39, 36, 38] has shown that six dominant genes (*Ht1, Ht2, Ht3, Htm1, Htn1* and *NN*) and one recessive gene (*ht4*) provide resistance to the various key races of *Setosphaeria turcica*. Plants homozygous for *Ht* gene have the highest level of resistance; however, it confers comparatively less protection to the host than in the case of polygenic resistance under conditions of heavy epiphytotics.

Quantitative trait loci and marker-assisted selection

The availability of the maize genome sequence offers new opportunities for a detailed understanding of the organization and architecture of disease resistance, fine mapping of QTL, validating candidate genes and identifying effective molecular markers to improve disease resistance. Molecular markers play greater role in identifying different genes responsible for desirable traits like disease resistance and allow to construction of high-density linkage maps [45]. Several QTLs for resistance to TLB have been mapped in maize by using different mapping populations, viz, $F_{2:3}$, RILs, NILs, NAM. (Table 4, adapted from [42]).

An avenue is open for pyramiding multiple genes by marker-assisted selection that may control different mechanisms for resistance [41, 12, 42]. To date, in the maize *S. turcica* pathosystem, clustering of major genes and QTL has only been observed at bin 8.05–8.06 [108]. Many efforts are still required to fine-tune these approaches to be readily usable by the maize breeders to develop broad stable sources of resistance against prevalent races by pyramiding qualitative and quantitative loci, because the stable expression allows resistance tests to be conducted without precise control of environmental conditions and is of great practical importance for the maize breeder.

Disease management

The disease is difficult to manage by means of both fungicides and crop rotation alone. Thus, an integrated approach is essential for effective management, especially under problematic conditions. Integration of resistant varieties, good cultural and use of recommended fungicides and biopesticides are necessary for the management of TLB.

Cultural practices

When considering reducing the severity of disease, different management strategies have to be initiated that are not only environmentally friendly but also cost effective [84]. TLB can be managed either through biological control, chemical protection or by introducing genetic resistance through plant breeding [50]. De Leon and Pandey [18] suggested crop rotation of 1 to 2 years or deep burying of infested maize residues before maize hybrids were planted reduces over wintering of the fungus and decreases disease pressure. Also, timely removal of over wintering infected crop residue will reduce the amount of available inoculums at the onset of the subsequent growing season. Patrick et al. [78] showed that the severity of TLB is less in fields when adequate potassium in the form of potassium chloride has been applied.

Biorationals

Disease control compounds which might be much less dangerous to the environment and non-target organisms than traditional fungicides had been targeted as biorational fungicides [110] also referred to as biopesticides. Different modes of action have been reported such as preventing spore germination, retarding sporulation and mycelial growth, and inducing systemic resistance [70, 110]. Biorationals are most effective when used preventively at short spray intervals; they may be used in fungicide rotations, thereby reducing conventional fungicide use and the development of fungicide resistance [96]. Selected biological control agents might add to the list of biorational pesticides and provide alternatives to traditional fungicides for TLB. Biological control for TLB has not received much attention due to pathogen variation thus preventing the development of suitable biological agents for control. However, potent control of *S. turcica* with BCAs has remained elusive. Use of plant products in disease management is a contemporary eco-friendly approach and gaining popularity considering that of its benefits over chemical compounds. These plant extracts are easily biodegradable, without any residue, non-phytotoxic and are easily absorbed by the plants and cost effective. The presence of naturally occurring substances in plants with antifungal properties had been stated and tested toward wide range of fungi infecting many commercially important crops [91]. However, studies on control of turcicum leaf blight of maize with the aid of plant extracts are meager. The ultimate goal of reducing fungicide use in maize production will be accomplished by using different biorational fungicides in rotations with traditional fungicides. Future studies are needed to explore their use as

Table 4 QTLs mapping studies on turcicum leaf blight

Parents	Resistance source	Population	QTL mapping	Trait	Flanking markers	References
B52 × Mo17	BS2	$F_{2:3}$	Interval mapping	Average percentage leaf tissue diseased	umc157, umc67	[26]
				Average Number of lesions/leaf	umc157, umc67	
D32 × D145	D32	F_3	Composite interval mapping	% diseased leaf area	csu61b, dup12 (dupssr12)	[105]
D32 × D145	D32	Recurrent selection	Selection mapping	–	bnlg615	[109]
B73 × T × 303	T × 303	NILs	–	IP, AUDPC, IP, lesion number, diseased leaf area	umc1754, umc2234	[14]
Ki14 × B73	Ki14, B97	RIL	Multiple interval mapping	AUDPC	PZA01041.2, bnlg1057	[113]
B73 × Mo17	B73	RIL	Composite interval mapping	AUDPC (AU06WMD)	bnlg1598, umc1396	[3]
B73 × CML52	CML52	HIFs	–	AUDPC	–	[16]
B73 × CML52	CML52	RIL	ICIM	3 diseased leaf area ratings	–	
NAM	CML103, CML247, CML52, CML69, Ki11, Ki3, M37 W, Mo17, Mo18 W, NC358, Tzi8	NAM	Joint linkage mapping	AUDPC	PZA02191.1, PZA00619.3	[83]
T × 303 × B73	T × 303	NILs	Association mapping	–	snp_01_0042, snp_01_0005	[42]

potential, economical phytochemical molecule against TLB of maize.

Chemical management

The use of fungicides is not always consistently profitable for maize. Profitable fungicide use in maize depends to a great extent on the grain yield potential, the disease susceptibility of the hybrid and the foliar disease severity throughout the growing season, the market price of maize and the price for fungicide application [6]. Demethylation inhibitor (DMI) fungicides, and particularly propiconazole, have shown the highest efficacy (Table 5) in controlling TLB both in vitro and in field conditions [8, 102, 28, 54, 49, 107, 99]. In addition, the quinine oxidation inhibitor (QoI) fungicides, commonly referred as strobilurin, had been proven to induce physiological benefits for plants, including improved stalk strength, longer preserved green leaf tissue and delayed plant senescence, either through a reduction in ethylene or in oxidative stress, an increase in photosynthetic capacity and translocation and regulation of the stomatal aperture and improved water-use efficiency [9, 6].

A quantity of such efforts opened new avenues for research; nevertheless, robust and comparatively cheap chemicals are yet to be developed for this disease. This is due to the existence and development of resistant strains of the pathogen posing serious problems in formulating effective control. Moreover, reliable forecasting systems are essential to determine the proper time of application of these chemicals for efficient control. Excessive use of chemicals also has a detrimental effect on the environment, farmers and consumer health [2]. Thus, there is a great demand for new methods to supplement the existing disease management strategies to achieve better TLB control.

Integrated disease management

Combination of two or more disease management options can offer improved disease control compared with a single control option approach. Adoption of the integrated disease management (IDM) technology is essential for economical

Table 5 Maize foliar fungicides and their efficacy against TLB

Mode of action	Fungicide	Company	Active ingredients	Chemical group	Efficacy on TLB
QoI strobilurins group 11	Aproach	DuPont	Picoxystrobin	Methoxyacrylates	Very good
	Headline AMP	BASF	Pyraclostrobin + metconazole	Methoxycarbamates and triazoles	Very good
	Headline EC and headline SC	BASF	Pyraclostrobin	Methoxycarbamates	Very good
DMI triazoles group 3	Tilt	Syngenta	Propiconazole	Triazoles	Good
Mixed modes of action	Quadris	Syngenta	Azoxystrobin	Methoxyacrylates	Good
	Quilt and quilt Xcel	Syngenta	Propiconazole and azoxystrobin	Triazoles and methoxyacrylates	Very good
	Stratego and YLD	Bayer	Prothioconazole and trifloxystrobin	Triazoles and oximinoacetates	Good
	Domark	Valent	Tetraconazole	Triazoles	No data
	Priaxor	BASF	Fluxapyroxad and pyraclostrobin		Unknown efficacy
	Folicur	Bayer	Tebuconazole	Triazoles	Very good

and effective management of TLB. Moderate levels of HPR can be combined with other cultural practices and/or application of minimum dosage of fungicide for control of TLB. The seed treatment with carboxin + thiram or benomyl + thiram followed by foliar application of mancozeb (0.25%) thrice at 10 days interval improved the seed emergence and seedling vigor and reduced the initial inoculum. This was found very effective integrated management approach against southern leaf blight of maize. Harlapur [32] studied the IDM of TLB by using carboxin power @2 g/kg for seed treatment followed by two sprays of mancozeb 0.25% as compared to control and observed significantly minimum percent disease incidence and maximum grain yield. The seed treatment with carboxin power (2 g/kg)/*Trichoderma harzianum* (6 g/kg) followed by three sprays of mancozeb (0.25%)/propiconazole (0.1%) at 30, 40 and 50 days after sowing will reduce disease severity. The success and sustainability of IDM strategy, especially with resource-poor farmers, greatly depends on their involvement in helping generate locally specific techniques and solutions suitable for their particular farming systems and integrating control components that are ecologically sound and readily available to them.

Conclusion and future strategies

TLB is one of the most devastating diseases causing up to 50% yield losses in maize. The disease develops under conditions of high humidity coupled with moderate temperatures and can be found in most regions where maize is grown. Epidemics of this disease are generally because of infested maize residues left in farm fields. Turcicum leaf blight can be controlled through conventional measures, but this has not been effective and has been difficult to sustain. We have highlighted different management strategies which are very effective in controlling the disease. The chemical fungicides used are mixtures of strobilurins and triazoles, pyraclostrobin, azoxystrobin + cyproconazole and picoxystrobin + cyproconazole reducing the disease severity and showing good yields. Biorational chemical molecule and probably some biological manipulate organisms keep some promise for managing this disease; however, more research is requisite before these procedures will be viable options. The disease is mainly controlled by resistant cultivars through qualitative and quantitative resistance. Nonetheless, qualitative resistance is unstable and breaks down easily with the aid of emergence of new races of the pathogen. As a consequence, identification of the *S. turcica* races present in an area and the understanding of their geographical distribution are vital in screening for resistance genes. The *Htn*1 gene confers quantitative and partial resistance against TLB with the aid of delaying lesion formation and introgressed into ultra-modern maize cultivars from the Mexican landrace Pepitilla in the Seventies. The strength of the *Htn*1 resistance relies on environmental conditions and maize genotype. A paradigm shift from thinking locally to regionally is necessary for cooperative evaluation of potential sources of resistance. It wills most effective be possible via using standardized disease screening protocols and methodologies, including common checks, to allow

consolidation and harmonization of knowledge. Moreover, progresses made within the "omics" area will revolutionize the chances for making improvements to pathogen identification and investigating host–pathogen interactions, epidemiology and growth of novel disease management practices. Therefore, there is a great demand for new methods to supplement the existing disease management strategies to achieve better TLB control. Ultimately, we have to rely on an IDM approach to mitigate the menace of TLB.

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