Phyton-
International Journal of
Experimental Botany

Tech Science Press

DOI: 10.32604/phyton.2022.020721

REVIEW

Distribution, Etiology, Molecular Genetics and Management Perspectives of Northern Corn Leaf Blight of Maize (*Zea mays* L.)

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Received: 08 December 2021 Accepted: 23 March 2022

ABSTRACT

Maize is cultivated extensively throughout the world and has the highest production among cereals. However, Northern corn leaf blight (NCLB) disease caused by *Exherohilum turcicum*, is the most devastating limiting factor of maize production. The disease causes immense losses to corn yield if it develops prior or during the tasseling and silking stages of crop development. It has a worldwide distribution and its development is favoured by cool to moderate temperatures with high relative humidity. The prevalence of the disease has increased in recent years and new races of the pathogen have been reported worldwide. The fungus *E. turcicum* is highly variable in nature. Though different management strategies have proved effective to reduce economic losses from NCLB, the development of varieties with resistance to *E. turcicum* is the most efficient and inexpensive way for disease management. Qualitative resistance for NCLB governed by Ht genes is a race-specific resistance which leads to a higher level of resistance. However, some Ht genes can easily become ineffective under the high pressure of virulent strains of the pathogen. Hence, it is imperative to understand and examine the consistency of the genomic locations of quantitative trait loci for resistance to NCLB in diverse maize populations. The breeding approaches for pyramiding resistant genes against *E. turcicum* in maize can impart NCLB resistance under high disease pressure environments. Furthermore, the genome editing approaches like CRISPR-cas9 and RNAi can also prove vital for developing NCLB resistant maize cultivars. As such this review delivers emphasis on the importance and current status of the disease, racial spectrum of the pathogen, genetic nature and breeding approaches for resistance and management strategies of the disease in a sustainable manner.

KEYWORDS

Northern corn leaf blight; etiology; *Exherohilum turcicum*; pathogenic variability; disease resistance; management strategies

1 Introduction

Northern corn leaf blight (NCLB) caused by the fungus *Exherohilum turcicum* (synonym *Setosphaeria turcica*), is a destructive foliar disease of maize, sorghum, and related grass species [1]. The disease is widely distributed and economically the most important foliar disease of maize [2,3]. The disease has a worldwide distribution predominantly in areas with 75%–90% relative humidity and 22°C–25°C temperature during the cropping season [4,5]. NCLB causes enormous damage to the maize crops, and grain yield losses range from 24% to 91% [6,7], depending on the growth stage of the crop at which infection occurs, the severity of the outbreak, the resistance of the host plant and the virulence of the pathogen. The disease is more destructive if it appears prior to silk emergence. Disease development during the early growth stages results in the premature death of leaves. Hence the loss of photosynthetic area affects grain yield as well as fodder quality; this is of particular significance under temperate climatic conditions since fodder is fed to cattle during the lean season [8,9]. Different races of *S. turcica* have been identified throughout the world such as the races 0, 1, 2, 3, 12, 13 23, N, 1N, 2N, 3N, 13N, 23N and 123N based on their virulence against various resistant genes (*Ht1*, *Ht2*, *Ht3*, *HtM*, *Htn1*, *ht4*, *HtP*, *HtNB*) in maize [10,11]. Host plant tolerance relies on the efficacy of resistance against all virulent pathogen races in the region. The fungus *S. turcica* is considered to be extremely variable in cultural features, pathogenicity and genetic traits. Hence, the lack/loss of significant durable resistance in the maize genotypes is due to the presence of variability and continuous change in the racial spectrum of the pathogen [12]. Genetic diversity and pathogenicity of the pathogen are important factors in the resistance of the host plant. Hence, identifying the heterogeneity of pathogen isolates is an important step in the creation of a programme for disease management for a specific area and development of multi-racial disease-resistant cultivars. Deployment of resistant cultivars is by far the most successful and cost-effective way to manage the NCLB. Resistance to NCLB in maize can be obtained by breeding with qualitative and quantitative resistance, either independently or in combination. The Ht (*Helminthosporium turcicum*) genes are recognised for conferring qualitative resistance controlled by a single gene (and mostly dominant) which contributes to a higher level of resistance. Quantitative resistance is regulated by many genes and shows a significant reduction in NCLB disease severity, particularly in areas where race population of *S. turcica* is very high. Quantitative trait loci (QTL) or linkage mapping is an important approach to study polygenic and complex forms of disease tolerance. QTL for NCLB resistance has been established in many populations [13–16]. The present review lays out the rationale for NCLB disease development, variability and population structure of causal pathogen, genetics of resistance, the progress of gene identification against NCLB, and management strategies.

2 Distribution and Importance of NCLB

NCLB, also known as *turcicum* leaf blight (TLB) is among the most prevalent diseases of maize disseminated worldwide, particularly in regions with high humidity and moderate temperature [17–19]. The disease was first reported in 1876 from Italy by Passerine. The disease is distributed in the continents of Asia, Europe, North America, Africa, Oceania and South America (Fig. 1; CABI, 2019). Presently NCLB is a potential threat to maize cultivation in Europe, Australia, North-Eastern United States,

Sub-Saharan Africa, and in areas of North Korea, India, and China [20,21–23]. Butler [24] first reported the NCLB in India on sorghum, and Mitra [25] reported it from Punjab on sorghum and maize. The disease is most prevalent in all the major maize growing regions of India during the rainy (*Kharif*) as well as winter (Rabi) season [26]. Almora, Bajaura, Mandya, Dharward, Imphal and Kashmir are the hot spots for NCLB in India. This disease occurs sporadically in most temperate, humid maize-grown areas and is of particular concern in the tropical highlands, where conditions favour disease development [27]. The disease was also found to be the major restraint of maize production under the temperate climatic conditions of Ahangar et al. [28].

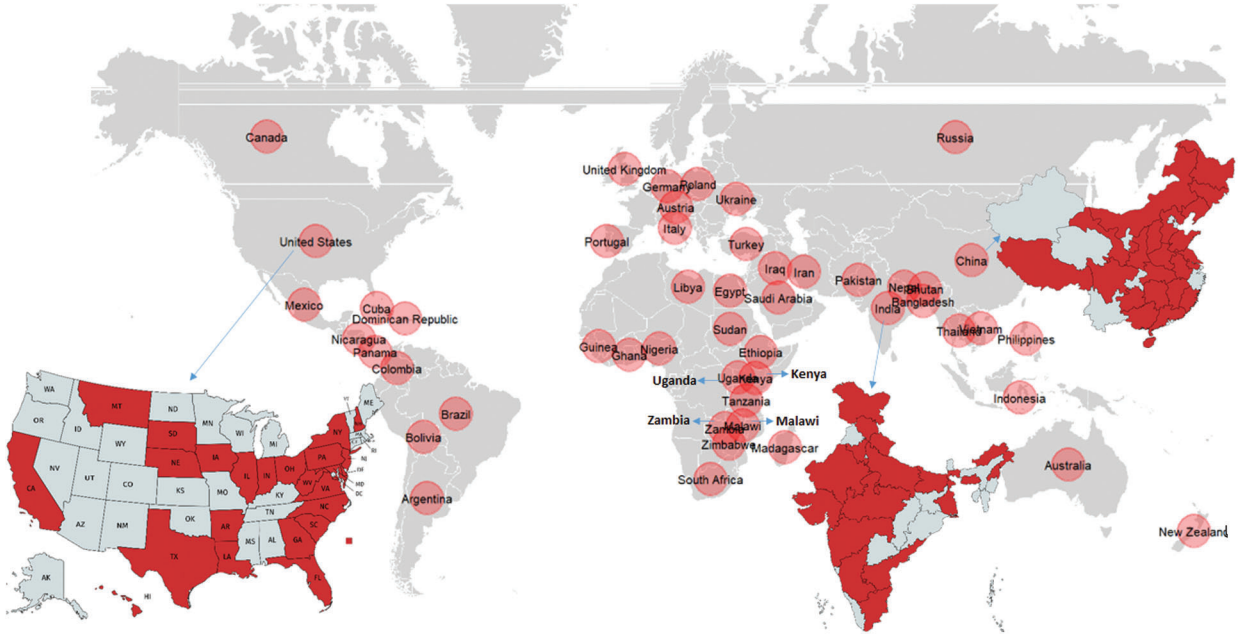

Figure 1: Worldwide distribution of NCLB along with a major focus of the distribution in USA, India and China (Data source: https://www.cabdirect.org/cabdirect/abstract/20046500257)

In maize, NCLB is a widespread foliar disease mainly found in temperate and tropical environments that cause yield reductions of up to 70% [29]. The disease also induces qualitative changes in the seed, such as reduced sugar content and germination potential, in addition to predisposition of infected plants to stalk rot [30,31]. The degree of yield losses due to NCLB depends on the growth stage of the crop at which infection occurs, the severity of the outbreak, the resistance of the host plant and the virulence of the pathogen. If the disease occurs before silking, a 40% yield decrease can occur [32], but if infection deferred until 6–8 weeks after silking, yield losses are minimal [33]. However, yield losses reach up to 50% when the disease occurs severely at 2–3 weeks after pollination [17]. The disease can substantially reduce the grain yield of maize over a wide range from 28% to 91% [7,32]. Average losses of 60% have been reported in Kenya, Uganda, Ethiopia, South Africa and Zambia [34]. Maize crops in the temperate belt of Kashmir are ravaged by this destructive disease with losses in the range of 27.6%–90.7% of total grain yield, particularly if the disease develops prior to silk emergence [35].

3 Disease Development

3.1 Taxonomy

NCLB/TLB is caused by *Exherohilum turcicum* [Pass.] Leonard and Suggs, synonym *Setosphaeria turcica* (Luttrell). The fungus is heterothallic ascomycete that belongs to the subclass

Loculoascomycetidae, order Pleosporales. Phylogenetic studies based on different loci indicated that *Exserohilum* belongs to the family Pleosporaceae, order Pleosporales [36]. The pathogen is a polycyclic, facultative parasite of maize. Leonard and Suggs have proposed the nomenclature of the organism as *Exherohilum turcicum* (Pass.) K. J. Leonard and E. G. Suggs, as an imperfect stage and teleomorphic phase was described in 1957 as *Trichometasphaeria turcica* by Luttrell and later modified to *Setosphaeriaturcica* (Luttrell) Leonard and Suggs. Normally, the causal agent of NCLB is defined by its imperfect stage *Exherohilum turcicum* in which a conidial hilum is strongly protuberant (Fig. 2e). The use of the name *Exserohilum* over *Setosphaeria* was claimed according to the Article 57.2 of the International Code of Nomenclature for algae, fungi and plants [37]. 38 taxa in *Exserohilum* have been listed by MycoBank which are distinguished on the basis of morphological features [38]. Hernández-Restrepo et al. [39] described 11 *Exserohilum* phylogenetic species based on nine nuclear loci, viz., ITS, LSU, act, tub2, cam, gapdh, his, tef1 and rpb2, as well as phenotypic data. *Setosphaeria* differs from *Trichometasphaeria* by the production of non-clypeateascomata which can be erumpent or superficial and produce larger ascospores [40]. Eight *Setosphaeria* species have been described by mating of compatible isolates [40,41]. The sexual stage of the fungus, *Setosphaeria turcica* rarely occurs under natural conditions [42]. The fungus exists with three distinct mating forms present in nature [43]. Mating of *S. turcica* is attained by inoculating compatible strains onto culture media with sterilized trashes of natural substrates such as maize leaf or wheat straw. Bunkoed et al. [44,45] first conducted the study on sexual reproduction of *S. turcica* in Thailand. Pseudothecia were found on highly infected corn leaves from natural fields. Conidiophores are simple, cylindrical, olive brown, shaped individually or in groups of two to four from stomata in necrotic leaf lesions (Fig. 2c). Single conidium is formed terminally on the conidiophores (Fig. 2d) which then resumes growth to one side of the conidial attachment and eventually produce another conidium at the new tip [40,41,46]. The vegetative hyphae remain mostly immersed, septate, branched, olivaceous brown, smooth about 3–7.5 µm wide. The conidia of the fungi are olivaceous-grey, elongated and spindle-shaped often less curved on one side (Fig. 2f) compared with the conidia of *Helminthosporium maydis*, which are more curved. The average size of conidia is about 20–150 µ with one to nine septa. A conspicuous spore feature of the conidia that distinguishes it from other more common species attacking maize is the protruding hilum [17,45].

3.2 Symptoms

About 14 days after infection, the disease symptoms appear as small, oval, greyish green and water-soaked spots which grow into elongated, spindle-shaped necrotic lesions [38]. NCLB lesions are elongated elliptical greyish, measuring up to 12 mm wide and 2.5–15 cm long which run parallel to leaf margin [46,47] (Figs. 2a and 2b). On mature lesions, distinct dark grey areas develop associated with fungal spores [48]. Spore formation causes the appearance of lesions to olive, dark, grey or black in colour [49]. NCLB is essentially a leaf disease and symptoms usually appear at any stage of the crop on the lower leaves spreading upwards [50,51]. The disease spots first appear on lower leaves and the number of spots increases and spreads up with the development of plant, leading to a complete blighting of the foliage. Symptoms range from cigar-shaped lesions on the lower leaves to the total loss of the foliage (Fig. 2b), thereby reducing the amount of leaf area required for photosynthesis [47]. Lesions of NCLB vary in shape and size depending on the race of *E. turcicum* and tolerance level of the genotype. Lesionfeatures vary among maize genotypes based on their resistance and interaction with different races of the pathogen. Race 1 develops oval to circular, tan lesions on leaves about 1.2 to 2.5 cm in size while Race 2 develops about 0.5 to 2.5 cm long oblong, brown spots. Race 3 and Race 4 causes narrow, long, bordered grey lesions on leaves and Race 0 develops only small flecks or spots. *E. turcicum* develops appressoria and penetrates the leaf surface directly. In incompatible reactions, the pathogen penetrates into xylem vessels and causes leaf chlorosis while as in compatible interactions it strongly colonizes the mesophyll and results on leaf necrosis and development of typical NCLB symptoms.

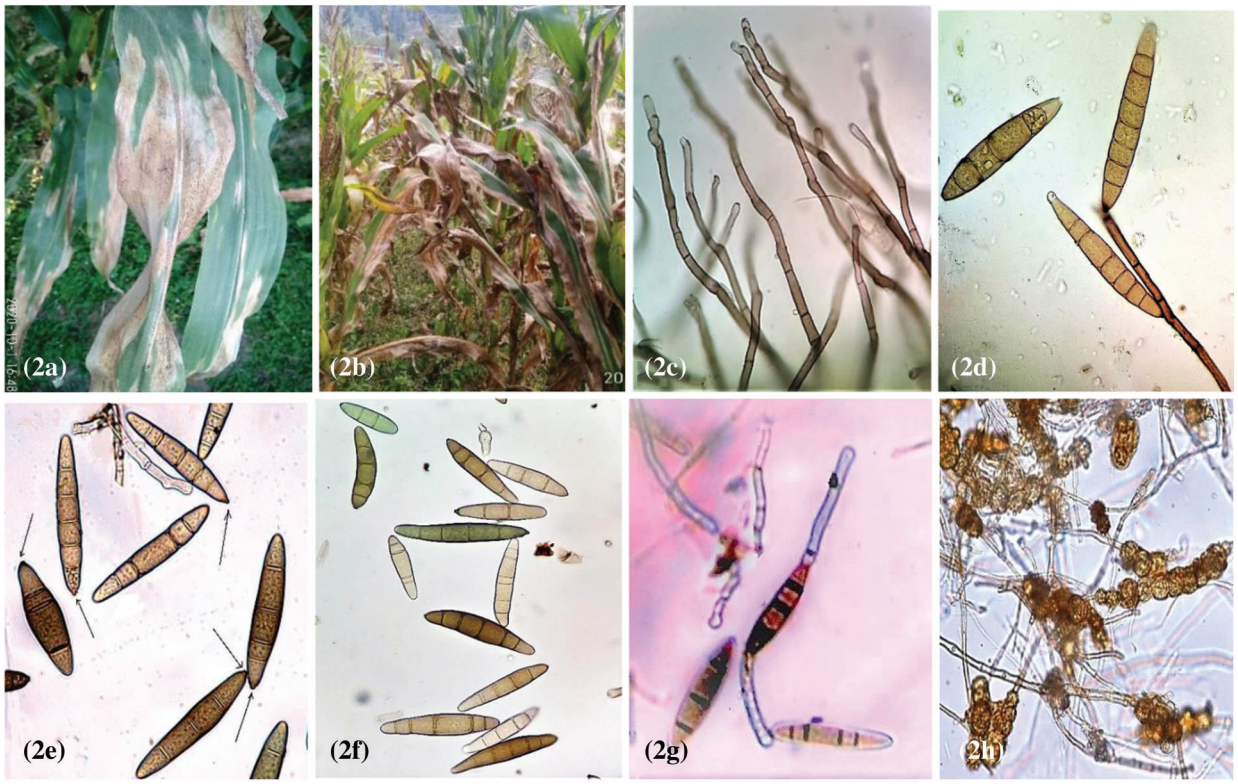

Figure 2: Symptoms and etiology of *S. turcica*. Elongated elliptical greyish NCLB lesions (2a), blighting and death of leaves (2b), cylindrical conidiophores (2c), conidial formation on conidiophore (2d), conidia with protruding hilum (2e), elongated to spindle-shaped conidia (2f), bipolar germination of conidium (2g) and dormant conidiophores (2h)

3.3 Etiology

This fungus has saprophytic survival which over-winters as dormant mycelia, conidia and chlamydospores (Fig. 2h) on maize residues left on the soil surface. Mycelia and conidia of this fungus from infected crop residues, in or on the soil, serve as the primary inoculum for the next crop [52,53]. The secondary inoculum is caused by disease lesions on leaves on which the fungus develops conidia that are spread over long distances by wind and rain [54,55]. Seed-borne nature of the fungus has been also reported which remains viable in the seed for 28 months [56]. The NCLB is favoured by mild temperatures (between 15°C to 25°C), high relative humidity (90%–100%), extended periods of leaf wetness (rain or dew at least 4 h) and frequent light showers [17,57–59]. Germination of *E. turcicum* conidia is bipolar (Fig. 2g) and develops infection 3–6 h after inoculation. Germ tubes grow at an angle and generate simple or forked terminal appressoria from which penetration pegs grow [60]. Penetration occurs directly through leaf cuticle and epidermis and occurs rarely through stomata [61]. The pathogen produces a range of secondary metabolites and toxins to allow penetration and colonization. Two pathogenicity related genes of the *S. turcica* genome encode xylanase enzymes that destroy the arabinoxylan in the plant cell wall responsible for its integrity thus leading to pathogen infiltration [62]. Infection pegs expand into or between, the epidermal cells of the dorsal or ventral sides of the leaf [2,63]. Penetration usually occurs 12–18 h after inoculation [50,64]. After penetration, the fungus develops a vesicle-like structure within the epidermal cells, giving rise to secondary hyphae that appear intracellular in the mesophyll tissue in different directions [63,65]. The hyphae begin to progress within the

chlorenchyma tissue, culminating in cell death and lesion development. The cells later become devoid of all cytoplasm, separate and disorganized [60]. The hyphae grow from the xylem to the underlying healthy tissues, infiltrate the normal bundle sheath and grow quickly in neighbouring mesophyll cells resulting in the enlargement of the lesions. Inside the tissue, the hyphae secrete a HT toxin called Monocerin [66] which comprises low molecular weight, water-soluble compounds that inhibit chlorophyll synthesis [67]. Mycelial threads aggregate into pseudoparenchymatous masses in sub-stomatal chambers. Conidiophores produced by these thick masses arise through the stomata and grow conidia extensively [63,64]. The incidence and severity of NCLB vary from site to site and year over year depending upon the virulence of the pathogen, response of the plants and prevailing environmental conditions [5]. Disease lesions on maize leaves develops at a faster rate during the night than day period. Thus, day lengths shorter than 12 h increases disease development. NCLB is commonly considered to be sporadic in frequency, depending on the environmental factors and the disease tolerance of the plant [68,69]. In general, the increase in the prevalence of the disease might be attributed to mono-cropping practices, high humidity, morning fogs, extended dew periods, minimum tillage and the use of uniform and highly susceptible varieties [69–72].

4 Variability and Population Structure of *E. turcicum*

Genetic diversity and pathogenicity of the microorganism are important factors for the host resistance and production of effective disease control strategies. Novel races of the pathogen are being frequently developed and the pathogen is being shifted to new regions. The fungus *E. turcicum* is considered to be extremely diverse in terms of cultural traits, pathogenicity and genetic composition. Molecular diversity of *E. turcicum* isolates varies considerably from region to region [73]. Higher molecular variability has been observed among-populations of *E. turcicum* from different hosts compared to the populations from different locations [74]. Genotypic diversity and gametic phase equilibrium in *S. turcica* populations develop more in tropical regions than populations from temperate regions. Higher sexual recombination rates have also been observed in tropical climates, whereas populations in temperate areas tend to be more clonal. *S. turcica* populations are highly adaptable in both temperate and tropical climates, as an extensive migration was also found within agro-ecological zones [73]. Isolates of *E. turcicum*, from different locations, varies in parasitic fitness in terms of the effectiveness of invasion, sporulation and lesion size, as well as in colour, type of mycelium, growth rate and sporulation in culture [75–78]. Highly virulent isolates exhibit more infection on different differentials. In a study by Muiru et al. [79], *E. turcicum* isolates from Germany, Kenya and Austria showed a varied response on the differentials indicating a high virulence complexity and variability of the pathogen. The aggressiveness of the various *E. turcicum* isolates differs in terms of lesion density, area under the disease progress curve (AUDPC), lesion size, length of incubation period and rate of lesion expansion [73,80]. Assefa [81] indicated significant differences among *E. turcicum* isolates in their virulence and the mean virulence rating was significantly correlated with spore length and rate of germination. Wathaneeyawech et al. [22] found substantial variability among 478 isolates of *E. turcicum* collected from Thailand. Significant morphological variability was also detected among the *E. turcicum* isolates from Argentina and Brazil for all measured variables viz., length, width and number of septa [53]. Substantial variations in morphology [28,82,83,84], pathogenicity [77,79,85], and genetic diversity [86,87] have been observed among the *E. turcicum* isolates from different agro-ecological regions. Knox-Davies et al. [88] reported ample evidence of heterokaryons and their perpetuation by conidia, and proposed that high variability in the fungus population might be related to heterokaryosis. Bunkoed et al. [44] first examined the sexual stage of *S. turcica* and proposed that sexual reproduction had induced genetic variation in this pathogen. In addition, virulence may be improved and new physiological races might have been produced by sexual hybridization. Two mating types MAT-1 and MAT-2 of *S. turcica* have been reported. These mating types are regulated by a single locus having two highly dissimilar alleles. In tropical environments, sexual

hybridization is responsible for greater adaptation ability due to the existence of equal fractions of MAT-1 and MAT-2 [73]. Li et al. [89] reported three mating types, 'Aa', 'a', and 'A', among which 'a' was the dominant type in China's Heilongjiang Province.

The prevalence of NCLB has increased in recent years and new races of the pathogen have been reported worldwide. NCLB intimidation to maize production is mainly due to the presence of *S. turcicum* races and the potential of the pathogen for the development of new races. Vidal-Villarejo et al. [90] reported that new pathogen lineages of *S. turcica* are not generated by race-specific virulence. High mutation rates of the pathogen may be the frequent origin of new races. Identification of races of the pathogen present in an area and the recognition of their spatial distribution are important steps in the generation of resistant cultivars in different pathogen systems where major genes regulate resistance. *S. turcica* races are described based on their phenotypic reaction whenever inoculated into a series of different maize lines [91]. The disease has spread around the world with a variety of distinct races such as Races 0, 1, 2, 3, 12, 13 23, N, 1N, 2N, 3N, 13N, 23N and 123N [11,79]. An increased number of *S. turcica* races identified from different regions of the world contributed to the quick loss in tolerance of many hybrids containing Ht genes [92]. Race designations are based on resistance genes and their corresponding virulence matches. For example, *S. turcica* Race 0 is avirulent for all the *Ht* genes, while Race 1 is only effective (virulent) for the *Ht1* gene; Race 23N is virulent in response to *Ht2*, *Ht3*, and *Htn1* genes; Races 3 and 4 are virulent in response to *Ht2*, *Ht3*, *Htn* genes, and Race 12 is virulent against *Ht1* and *Ht2* genes [Table 1]. Races 0 and 1 are more prevalent, whereas Races 2N, 23, and 23N are rarely found [93]. The Race 123N with the highest virulence complexity tends to infect all of the cultivars with matching *Ht* genes [94]. The race distribution of *S. turcica* is considerably variable in different regions of the world. Fourteen races of *S. turcica* have been reported from diverse geographic locations of China with a dominancy of Race 0 and 1 [11]. A total of 12 races have been found in samples from Germany, Kenya and Austria, with Race 2 appearing more frequently [79]. From Turkey eight diverse races of *S. turcica* were found, namely 0, 1, 2, 123, N, 3N, 12N, 1N, among which Race 0 and Race 1 were the most common [95]. Similarly, 17 races of *S. turcica* were identified in Canada with Race 0, 1M, 1N and 1MN being the most prevalent [96]. Race 0, 1 and 23N of *S. turcica* are found in Argentina and Brazil with Race 0 in abundance at a frequency of 83% and 65%, respectively. Therefore, Race 0 has the highest abundance throughout the world [97]. The physiological race of *S. turcica* present in the Indian subcontinent has been determined to be Race 2 [98]. Studies have also shown that *S. turcica* migration is likely over long distances, which could shift virulence to new regions [99]. Selection pressure, sexual recombination within the pathogen, and the capability to migrate long distances may create more virulent populations and contribute to spatial and temporal race population shifts. Distribution of *S. turcica* races on larger geographical regions of the world impresses upon the monitoring of pathogen diversity on large scales and over time to fully understand factors influencing the evolution of pathogen races.

Table 1: Gene-by-gene interaction between the pathogen and host plant

Race	Ht gene reaction				
	Ht0	*Ht1*	*Ht2*	*Ht3*	*HtN*
0	P	N	N	N	N
1	P	P	N	N	N
2	P	N	P	N	N
3	P	N	N	P	N
N	P	N	N	N	P

(Continued)

Table 1 (continued)

Race	Ht gene reaction				
	Ht0	*Ht1*	*Ht2*	*Ht3*	*HtN*
12	P	P	P	N	N
2N	P	N	P	N	P
23	P	N	P	P	N
23N	P	N	P	P	P
123N	P	P	P	P	P

Notes: N = Incompatible reaction between *Avirulence*(*Avr*) gene and *Ht* gene, infection do not occur (= host resistance).
P = Compatible reaction between the *Avr* and *Ht* genes (= host susceptibility).

5 Genetic Nature and Breeding Approaches for NCLB Resistance: Qualitative and Quantitative Genes

NCLB resistance is either qualitative which is usually race-specific and inherited from a single gene (monogenic) although quantitative resistance is race nonspecific and polygenic [100,101]. Monogenic or race-specific resistance for NCLB is controlled by *Ht1*, *Ht2*, *Ht3* and *Htn* genes. Gene *Ht1* (Ht for *Helminthosporium turcicum*) confers a chlorotic lesion type and was the first single gene resistance, identified by Hooker [102] from the inbred line, GE339 and popcorn cv 'Ladyfinger'. In maize lines with *Htn* gene, lesion formation is delayed in such way that plants in the field remain free from lesion until shortly after pollination while the expression of *Ht1*, *Ht2* and *Ht3* resistant genes occur as chlorotic lesions with minimum sporulation [91]. Symptoms produced in resistance responses by the different *Ht* genes are considerably variable. *Ht1* shows necrotic lesions with chlorosis, maize lines with *Ht2* resistance gene exhibits chlorosis and small lesions, *Ht3* shows chlorotic spots while as in *Htn1* resistance reaction there is no lesion development. *E. turcicum* forms appressoria and penetrates the leaf surface directly in both compatible as well as in incompatible reaction types. However, the pathogen penetration in xylem vessels and colonization to mesophyll restrict in resistant interactions [103]. Six dominant genes (*Ht2*, *Ht3*, *Ht1*, *Htn1*, *Htm1*, and *HtNN*) and two recessive genes (*ht4* and *rt*) have been identified to provide resistance to the various races of *S. turcica* [13,59,104–106]. *HtP* and *Ht1* are found on chromosome 2L (Bin 2.08) and mapped 10 cM from one another [58]. *Htn1* and *Ht2* were found on chromosome 8L [107,108] and *rt* is positioned on chromosome 3L (bin 3.06) [59]. *Htn1* encodes for a wall-associated receptor-like kinase which functions as core element of innate immune response by detecting pathogens or host-derived elicitors [109].

The specific region of chromosome 8 (bin 8.05–8.06) of the maize genome harbours a locus responsible for a substantial level of NCLB tolerance in maize germplasm. This is because in various biparental populations, several NCLB QTL and two major gene loci, *Ht2* and *Htn1* have been mapped to bin 8.05-8.06 [108,110,111]. The introgression of *Ht3* gene from *Tripsacumfloridanum* was carried out in maize [112] and mapped on bin 7.04 [113]. The *ht4* gene which provides race-specific resistance was found near the centromere on the chromosome 1 [114]. *HtP* has been mapped on chromosome 2 on bin 2.08. The gene *HtNB* which confers non-lesion resistance is located on chromosome 8.07 bin, flanked by *MAC216826-4* and *umc2218* at distances of 3.3 and 3.4 cM, respectively. Bins 1.05/7 and 9.05 were found to possess population-specific genes for resistance to *S. turcica* [13]. Among the up-regulated maize transcripts, one coiled coil (CC) forming, nucleotide-binding site (NB), leucine-rich repeats (LRR) [CC-NB-LRR] factors are found to be encoded by a gene *GRMZM2G005347*. The new plant resistance gene was designated as *St* referring to *S. turcica*. Comparative genomics showed that the CC-NB-LRR encoding *St*genes in maize were found on chromosome 2, and chromosome 5 in sorghum [115].

Recently, three NCLB resistance candidate genes viz., *CDPK21*, *HEX9* and *MKKK18* were identified with annotation functions of sugar signalling, calcium signalling, and MAPK signalling pathways [116].

Quantitative resistance also called as horizontal or non-race specific resistance shows a substantial reduction in the incidence of NCLB disease and is controlled by several genes (polygenic). QTL is a chromosomal region that is associated with a quantitative trait. QTLs usually include genes that regulate the quantitative trait of a plant like yield and NCLB resistance. The mapping of the QTL and followed by its introgression into cultivars is regarded as QTLian breeding. The QTL or marker-trait associations (MTAs) can be identified through QTL mapping and genome-wide association studies (GWAS) approaches, respectively [117]. The former differs from the latter in that it uses constructed mapping populations (GWAS is based on diverse natural accessions called association panels). Numerous QTLs for NCLB resistance associated traits have been identified through QTL mapping and GWAS approaches (Table 2). Quantitative NCLB resistance is characterised by fewer, often smaller, lesions and a lengthy incubation period [114,118]. The extended incubation duration is usually expressed in young plants and is closely associated with the disease severity in adult plants of the region [119]. QTL which confers lesions length, width, and area have been mapped on chromosomes 1, 3, 5, and 8 [120], while the QTL conferring incubation period and area under the disease progression curve (AUDPC) have often been located on chromosome 8L [121,122]. Numerous QTLs for NCLB resistance were identified from different populations and distributed throughout the genome [14,112,122–126]. Four QTLs associated with NCLB resistance and a candidate gene, *GRMZM2G024612* were also identified by association mapping [123]. One QTL at chromosome 1 (*qNLB1.06*) and another QTL at chromosome 8 (*qNLB8.06*) were found to be closely linked and functionally related to *Ht2* [2,127]. The QTL *qNCLB5.04* was found to be located at chromosome 5 and associated with NCLB disease scores and the width of the lesion [125]. Wang et al. [128] identified a new QTL, *qNCLB7.02* for resistance to NCLB in maize. Recently, Rashid et al. [16] found 21 significant SNPs across three panels, some of them found to be co-located with major genes like *Ht2*, *Ht3* and *Htn1* and previously reported QTL for NCLB. Another study [129], by using nested near-isogenic line library revealed the role of *liguleless1* for resistance to NCLB. The mutants for *liguleless1* were found to be susceptible to NLB as a lack of ligule in maize resulted into highly erect leaves. However, this fact still needs confirmation as contrasting results were obtained for the correlation of leaf angle with NCLB resistance in different populations [129]. Galiano-Carneiro et al. [94] identified 17 QTL for NCLB resistance governing 3.57%–30.98% of the phenotypic variation. Moderate to high genomic prediction accuracies were observed between 0.58 and 0.83 based on population and continent. Recently, Ranganatha et al. [130] evaluated CML153 (susceptible) and SKV50 (resistant) based 344 $F_{2:3}$ population for NLCB resistance and identified two major QTLs namely *qNCLB-8-2* (phenotypic variation of 16.34%) and *qNCLB-5* (phenotypic variation of 10.24%). Nevertheless, numerous QTLs have been identified in different populations by different research groups, but integrating the data of all the QTLs is an efficient approach to identify the consensus or stable regions harbouring multiple QTLs. Such studies were carried out using the meta-QTL analysis approach which declares regions (within certain confidence intervals) possessing two or more QTLs as meta-QTL. This helps in pinpointing the multi-allelic QTLs for NCLB and even multiple disease resistance [131]. Martins et al. [132] tested and advocated the use of this approach for identification of multiple disease resistance QTLs in maize. Genomic selection is a promising approach for simultaneous detection of favourable alleles in training sets and prediction of genotypic performance on basis of genome estimated breeding values [133]. Technow et al. [134] carried out the genomic prediction for NCLB resistance using the flint and dent training sets. The study revealed the prediction accuracies of 0.706 (dent) and 0.690 (flint). This indicates the potential use of genomic selection for NCLB resistance. Furthermore, the prediction accuracies can be further improved by the use of large diverse training sets. The detection of more than one QTL supports the theory that quantitative genes control resistance to *S. turcica*. Shared genetic

regions were also identified conferring resistance to *E. turcicum* in both maize and sorghum. Several promising candidate genes have been identified with known roles in resistance to leaf blight including genes related to R-gene mediated resistance [135]. Novel identification/evaluation of NCLB-resistant quantitative trait loci and genes has been done which could improve the maize varieties (Table 2).

Table 2: Summary of the genetic mapping studies for NCLB resistance using different mapping populations in various genetic backgrounds

S. No.	Chromosome No.	Bin/Position	Marker used	Maize lines	Mapping population	Trait observed	Reference
1	2, 5 and 8	–	SNPs	CML 153 and SKV 50	$F_{2:3}$	Percent disease index	[130,136]
2	1, 2, 3, 4, 8, 9	24.83, 36.35, 97.46, 46.38, 1.60, 23.05, 26,65, 34.12, 32.88	SNPs	B73, B97, and CML322	333 RILs	Disease severity	[126]
3	1, 8, 5, 6, 2	1.06, 8.05, 5.05, 6.05, 2.01	SNPs	BC_5F_4	NILs	AUDPC and leaf angle	[129]
4	7, 9, 1, 2, 4, 5, 8, 10	1.07, 1.08, 2.02, 2.04, 4.03, 5.04, 6.01, 8.08, 9.04, 10.04,	SNPs	(T1, T2, T5), (A1, A2, A10, A3, A4, A5, A11)	742 F_1	PDI	[94]
5	6, 7, 8, 1, 10	7.04, 7.02, 8.06, 8.03, 1.06	SNPs	CIMMYT Tropical inbred lines	Association mapping population (376, 224, 324)	PDI	[16]
6	1, 4, 6, 10	62217921, 62241862, 45334706, 45346069, 57, 920, 894	SNPs	800 converted lines	Sorghum conversion panel (800)	AUDPC	[135]
7	1, 2, 3, 4, 5, 8, 9, 10	1.03, 1.05, 2.05, 4.05, 5.04, 8.03, 9.03	SNPs	NC304, NC344, Ki3, NC262, Oh7B, H100	8 BC3F4:5 population (1,611 lines)	AUDPC	[137]
8	2, 3, 4, 5, 8, 9	2.05, 3.04, 4.05, 5.04, 8.03, 9.03, 9.04	SNPs	NC304, NC344, Ki3, NC262 H100	12 $F_{2:3}$ families	Diseased leaf area	[15]
9	1, 4, 5, 6, 7, 8, 9, 10	1.01, 4.04, 7.02, 8.03, 9.03, 10.04	SNPs	Qi319, Ye478	314 RILs	Disease score and lesion size	[128]
10	4	4.01/4.05, 4.08/ 4.10	SSR	CM 212, CM 338	$F_{2:3}$ families	PDI, AUDPC	[138]
11	1, 3, 5, 7, 9	1.03, 3.08, 5.04, 7.05, 9.03	SNPs	K22, BY815	207 RILs	Disease score	[125]

(Continued)

Table 2 (continued)

S. No.	Chromosome No.	Bin/Position	Marker used	Maize lines	Mapping population	Trait observed	Reference
12	3, 4, 6, 7, 8, 10	103166745, 103769943, 186590896, 160053330, 33447828, 91684720, 92335869, 37657703, 91956279	SNPs	Inbred lines	999 mapping panel	AUDPC	[124]
13	8	8.06-8.08	SSRs	Bramadi, 183/ Zi330, Huangzao 4, P111, B73	291 and 356: F_2 population	Disease severity	[139]
14	1–10	2.08, 3.05, 5.03, 5.05, 6.05, 7.02, 9.03	SNPs	Maize inbred lines	Association mapping panel (1487)	Disease score	[112]
15	1, 2, 3, 4, 5, 6, 7, 8, 9, 10	12136678, 183754852, 289465566, 1210644, 160834095, 177670891, 190880589, 116055358, 125153323, 151397247,	SNPs	RILs	Nested association mapping population (5,000)	Incubation period and disease severity	[123]
16	1	1.02, 1.06	RFLP, SSR	Tx303, B73	82 $TBBC_3$ introgression lines	AUDPC, Disease severity	[2]
17	1, 2, 3, 4, 6, 8, 9	1.06, 2.00–2.01, 2.02, 3.05, 3.09, 4.07–4.08, 4.08, 6.05, 6.07, 8.05. 8.07, 8.08, 9.02,9.04	SSR	B73, Mo17	302, RILs	Incubation period, weighted mean disease	[140]
18	1, 2, 6, 8	1.02, 1.05–1.06, 2.02–2.03, 6.05, 8.02, 8.05	SNPs, SSR	Ki14, B73	RILS	AUDPC	[141]
19	1, 2, 3, 4, 5, 6, 8, 9	1.06–08, 2.06. 3.01, 3.03, 4.03, 4.06, 5.03, 5.04, 6.05–07, 8.02–06, 9.02	RFLP, SSR	D32, D145	220 F_3 families	Disease severity	[110]

6 Genomics and Proteomics of *S. turcica*

Developments in high-throughput and cheaper sequencing platforms resulted in the sequencing of a number of fungal genomes (www.fungalgenomes.org). Fungi typically have small genome size, ranging from 20 to 50 Mbp and low volume of long repeats unlike eukaryotes. The complete genome of *S. turcica* was assembled in 2011 using Roche (454), Sanger Fosmids, and shredded consensus from Illumina assembled data (www.jgi.doe.gov). The size of its genome is 43 Mbp and comprised 11,702 predicted gene models. Later, the race 23N of *S. turcica* strain Et28A was sequenced again using IlluminaHiSeq and PacBio Sequel technologies, and assembled to approximately 43,480,261 bp on 30 scaffolds [142]. In total, 13,183 protein-coding genes were predicted, 13,142 of them were well annotated. This *S. turcica* genome resource is important for understanding the genetics behind pathogen evolution and infection mechanisms. The culturing of *S. turcica* on artificial media is feasible and the fungus is amenable to genetic alteration using *Agrobacterium tumefaciens*-mediated transformation [62]. Considering the availability of genome size and ease of genetic alteration, genomics should prove vital for thorough understanding of the *S. turcicum*.

Proteomic analysis is an effective approach for investigating the gene products to better understand the gene expression of resistant genes or mode of action of plant against pathogens. Leaf proteins were isolated from control and *S. turcica* infected leaves (inoculated for 72 h) and tested for differentially expressing proteins using two-dimensional electrophoresis and mass spectrometry-based recognition. A total of 137 proteins displayed more than 2-fold variations in abundance, including 50 up-regulated proteins and 87 down-regulated proteins [113]. About 48 protein spots were successfully identified by MS analysis, including 10 unique up-regulated, 20 down-regulated protein spots. These proteins were further grouped into nine functional classes considered to be involved in several functions, including energy metabolism (46%), protein destination and storage (12%) and disease protection (18%). The expression of photosynthesis-related proteins and metabolism-related proteins were found to be decreased by inoculation with *S. turcica*. The findings showed that the dynamic regulatory network functioned through the relationship between the A619 Ht2 and *S. turcica* resistant lines. The resistance mechanisms of A619 Ht2 consisted primarily via the direct release of defensive proteins and the regulation of primary metabolism, mainly photosynthesis and carbohydrate metabolism.

7 Genome Editing for NCLB Resistance

The first reference genome sequence for maize was published (B73 RefGen-v1) during year 2009 based on the sequencing of fosmids and bacterial artificial chromosomes [143]. Since release of B73 reference genome sequence, it had been widely used in functional genomics of maize. Plant diseases are amongst the major factors for yield losses in maize that necessitates the adoption of economically feasible novel genome editing technologies for production of genetically engineered disease resistant maize. A loss/gain of function mutation in these genes via genome editing methods such as CRISPR-Cas9 (Clustered Regularly Interspaced Short Palindromic Repeats-Cas9), could shed light on the function of these genes and their role in disease resistance [144]. Techniques for the cloning of quantitative disease-resistance genes and subsequent analysis have been explained by Yang et al. [145]. Recently two genes namely *ZmCCT10* and *ZmAuxRp1* were cloned for disease-resistance in maize. RNAi based silencing of *ZmAuxRP1* results in stalk rot resistant plants, while plants which shows overexpression of *ZmAuxRP1* gene becomes more vulnerable to *Gibberella* stalk rot. This gene controls resistance by influencing the synthesis of indole-3-acetic acid and benzoxazinoid acid [146]. Major QTL, *qRfg1* and *qRfg2* that provides resistance to *Gibberella* stalk rot [147] has been found to be the part of *ZmCCT10*. Altered histone modification in the cis regulatory region of *ZmCCT10* is the main cause for imparting the disease resistance in maize [148].

The use of RNAi to hinder specific genes in the pathogen relies on the availability of the information about the pathogenesis pathways to be targeted. In such instances, a double-stranded RNA molecule, complements a particular pathogen gene, is expressed in the host and transmitted to the pathogen during infection and causes silencing of specific genes. Micro RNAs (miRNAs) are regulators for gene expression involved with certain biotic stress reactions. Wu et al. [149] showed that *miR811* and *miR829* confer high tolerance to NCLB. Genome editing facilitates the functional study of genes and the integration of novel traits into major crop plants. Site-specific endonuclease-based systems allow site-specific modifications of the genome by producing double-stranded DNA breaks in genes of interest with a low chance of off-target results. Subsequently, cellular DNA repair machine homologous recombination and non-homologous end-joining pathways repair the cut end. By understanding the molecular pathways involved in disease resistance, breeders will be able to develop crop varieties with durable resistance to plant pathogens through adoption of RNAi and CRISPR-Cas9.

8 Management of NCLB: Need for an Integrated Approach

A plant's inherent resistance to infection by pathogen could most likely be a safe, inexpensive and eco-friendly disease control strategy. However, to develop disease-resistant cultivars, it is necessary to consider the structure of the population and the evolutionary potential in pathogens. In environments where there is a continuous change in the racial spectrum of the pathogen and the population is highly diverse, exploiting quantitative resistances are recommended. This can be achieved by using cultivar mixture and production of complex hybrids, like three-way and double-cross hybrids, with inbred lines varying in tolerance level [150–153]. The qualitative resistance is typically recommended in locations where the pathogen diversity is low. Marker assisted backcrossing can result in the pyramiding of *Ht* genes in maize. Planting hybrids with good NCLB resistance is an economical, effective and sustainable method of avoiding yield losses in maize. Great efforts have been made worldwide to develop, identify and utilize germplasm with TLB resistance. Extensive evaluation of maize germplasm revealed new and durable resistance sources against NCLB [36,61,98,154–157]. Although the primary approach to manage NCLB is to use plant resistant genotypes, in some situations, farmers may consider the application of fungicide a useful approach, where environmental factors are favourable for NCLB. Selecting the proper timing for application is essential in determining the efficacy of fungicides and economic benefit. The efficiency of different fungicide applications to manage NCLB has been studied extensively [158,159,160–162]. The disease management by use of fungicides seems to be cost-effective when used on NCLB-susceptible maize varieties, and when applied during tasseling or flowering [163]. The best growth stages for trifloxystrobin + epoxiconazole fungicide applications to reduce NCLB were between V10 and V14, showing coincidence with the disease onset [164]. Demethylation inhibitor fungicides (DMI), had shown greater efficiency for controlling NCLB [165]. Among the DMI fungicides, propiconazole is the most effective in reducing the severity of the disease. The quinine oxidation inhibitor (QoI) fungicides known as strobilurin induce favourable physiological activities of plants like improved stalk strength and sustained green leaf tissue by delayed leaf senescence [164]. The fungicides prothioconazole + trifloxystrobin exhibited the highest chemical control efficiency for NCLB [166]. Though the chemical application has been proved very effective in the management of NCLB, excessive chemical usage has hazardous effects on human health and the environment [167]. Hence, minimum dosage of fungicides in combination with other cultural practices and moderate levels of host plant resistance is the best approach for the control of NCLB.

9 Conclusion and Future Perspectives

NCLB is the most important re-emerging foliar disease of maize, limiting maize production. The disease has a worldwide distribution and its development is favoured by cool to moderate temperature with high relative humidity. Disease development usually originate from mycelia and conidia of the causal fungus from infected crop residues left in farm fields. Early detection of the disease development is of great

importance in the management of NCLB. The prevalence of the disease has increased in recent years and new races of the pathogen have been reported worldwide. The fungus *E. turcicum* is highly variable in nature. Identification of races of the pathogen present in the area and ability to understand their geographical distribution are important steps for the development of disease resistant cultivars. Distribution of *S. turcica* races on larger geographical regions of the world impresses upon the monitoring of pathogen diversity on large scales and over time to fully understand factors influencing the evolution of pathogen races. Though chemical measures are available for the control of the TLB. They are difficult to sustain and have not been adopted particularly in marginal farming systems under high altitude rainfed conditions. Most efficient and cost-effective ways to manage the disease is to develop varieties with resistance against *E. turcicum. Ht* genes confer race specific qualitative resistance against NCLB inherited by single gene; however, the resistance tends to break down under the pressure of high virulent races of *S. turcica*. Hence, pyramiding of multiple *Ht* genes will play a crucial role for the development of durable resistance against a number of pathogen races. Quantitative resistance to NCLB is favoured in high disease pressure environments. The discovery, validation and introgression of NCLB resistant genomic regions would surely prove vital to achieve improved genetic gains for grain yield. The utilization of genome editing technologies like CRIPSR-Cas9 will help in the development of resistant cultivars that would be relatively easy to release unlike transgenics. Furthermore, omics approaches such as proteomics and metabolomics would pave the way for a better understanding of the molecular mechanism of NCLB and defence response of the host plant.

Author's Contribution: Study conception and design: M.A.A; S.H.W; M.C; Literature collection: Z.A.D; J.R; F.M; M.B; Draft manuscript preparation: M.C; S.K.A; S.A.W; K.A.D; A.E.S; Review, editing: C.B; O.K; M.A.H; S.H.W; M.A.A; All authors have read and approved the final version of the manuscript.

Funding Statement: The authors received no specific funding for this study.

Conflicts of Interest: The authors declare that they have no conflicts of interest to report regarding the present study.

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