Recent advances in the population biology and management of maize foliar fungal pathogens *Exserohilum turcicum*, *Cercospora zeina* and *Bipolaris maydis* in Africa

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Maize is the most widely cultivated and major security crop in sub-Saharan Africa. Three foliar diseases threaten maize production on the continent, namely northern leaf blight, gray leaf spot, and southern corn leaf blight. These are caused by the fungi *Exserohilum turcicum*, *Cercospora zeina*, and *Bipolaris maydis*, respectively. Yield losses of more than 10% can occur if these pathogens are diagnosed inaccurately or managed ineffectively. Here, we review recent advances in understanding the population biology and management of the three pathogens, which are present in Africa and thrive under similar environmental conditions during a single growing season. To effectively manage these pathogens, there is an increasing adoption of breeding for resistance at the small-scale level combined with cultural practices. Fungicide usage in African cropping systems is limited due to high costs and avoidance of chemical control. Currently, there is limited knowledge available on the population biology and genetics of these pathogens in Africa. The evolutionary potential of these pathogens to overcome host resistance has not been fully established. There is a need to conduct large-scale sampling of isolates to study their diversity and trace their migration patterns across the continent.

1 Introduction

Food demand driven by exponential human population growth over the past fifty years has shifted cropping systems from farms with high genotypic diversity to genetically uniform crops (termed monocultures) (Zhan et al., 2014). More recently, there has been an increased adoption of conservation agriculture (Rodenburg et al., 2020; Jat et al., 2021; Reicosky, 2021). These two factors have led to favorable conditions for crop pathogens that persist in the soil, including some foliar pathogens, to cause severe global disease outbreaks (Dill-Macky and Jones, 2000; Bateman et al., 2007; Simón et al., 2011; Bebber, 2015).
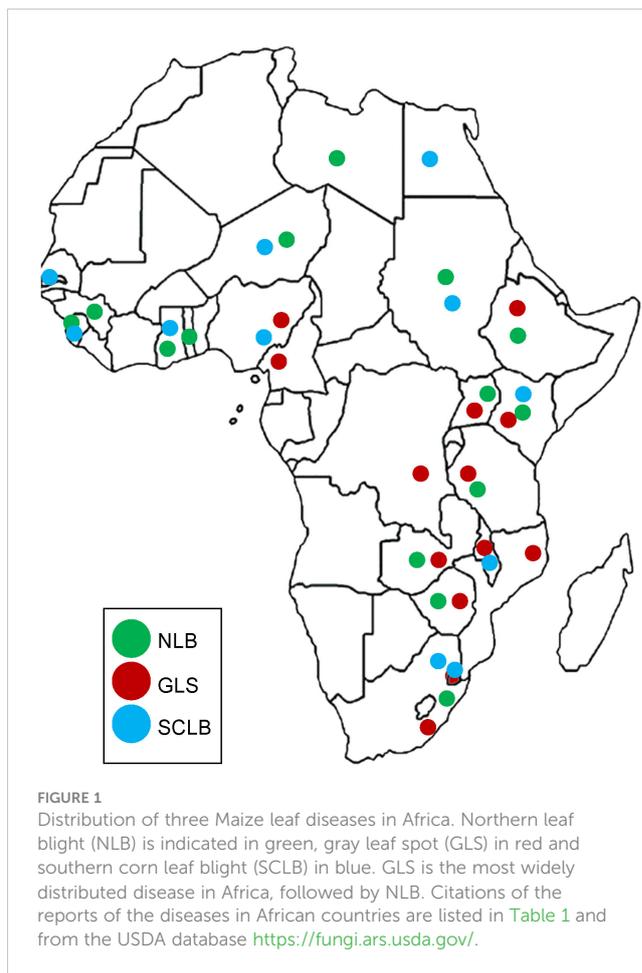
Maize production in the 2021/2022 production season was calculated at one billion tons (FAOSTAT, 2024). Yield has increased at a rate of 3.2% per year between 1972 and 2021 (Knoema, 2023). This is more than the 2.4% yield increase required per year to meet the expected global production demand by 2050 (Ray et al., 2013). Despite the cultural and food security importance of maize in many countries in sub-Saharan Africa, only 7.5% of the global maize crop is grown on the continent (FAOSTAT, 2024).

A worldwide survey indicated that biotic factors were responsible for 23% of maize yield losses annually (Savary et al., 2019). The three foliar fungal diseases - northern (corn) leaf blight (NLB), gray leaf spot (GLS), and southern corn leaf blight (SCLB), contributed to more than 4% of these estimated yield losses (Savary et al., 2019). Sub-Saharan Africa yield losses for NLB were estimated at more than 1% (Figure 1). Unfortunately, survey data was not gathered for the other two diseases. However, GLS is widespread on the African continent (Nsibo et al., 2021), and SCLB has been reported from Egypt, Gambia, Ghana, Kenya, Malawi, Nigeria, Sierra Leone, South Africa, Sudan, and Eswatini (Rong and Baxter, 2006; Aregbesola et al., 2020) (Figure 1). Here, we review recent advances in the population biology and management of the causal pathogens of NLB, GLS, and SCLB, with a focus on Africa.

2 Global distribution and causal agents of NLB, GLS, and SCLB

2.1 Northern leaf blight

Northern leaf blight also known as Northern corn leaf blight (NCLB) or Turcicum leaf blight (TLB), has persisted for decades as a major foliar disease in maize producing regions of the World (Drechsler, 1923; Savary et al., 2019). Following its first discovery in Parma Italy, NLB was only well documented in the United States of America (USA) in 1878 and only emerged as an outbreak in 1889 (Drechsler, 1923). The disease has since appeared in the Americas, and Asia (Shi et al., 2017; Bashir et al., 2018; Mueller et al., 2020; Navarro et al., 2021). In Africa, NLB was first reported in Uganda in 1924 and has since been reported in several sub-Saharan African countries (Figure 1; Table 1) (Emechebe, 1975; Adipala et al., 1995; Abebe and Singburaudom, 2006; Haasbroek et al., 2014; Nieuwoudt et al., 2018; Berger et al., 2020).

FIGURE 1
Distribution of three Maize leaf diseases in Africa. Northern leaf blight (NLB) is indicated in green, gray leaf spot (GLS) in red and southern corn leaf blight (SCLB) in blue. GLS is the most widely distributed disease in Africa, followed by NLB. Citations of the reports of the diseases in African countries are listed in Table 1 and from the USDA database https://fungi.ars.usda.gov/.

The causal pathogen of NLB is *Exserohilum turcicum* (Pass.) K.J. Leonard & Suggs, which is the asexual form of this hemibiotrophic Dothideomycete (Leonard and Suggs, 1974). Researchers have defined physiological races of *E. turcicum* based on six maize qualitative resistance genes namely; *Ht1, Ht2, Ht3, Htn1, Htn2,* and *Htm1*, that *E. turcicum* is able to overcome (Jordan et al., 1983; Jindal et al., 2019; Navarro et al., 2021; Muñoz-Zavala et al., 2023). The race groups are determined based on the screening of a differential set of maize lines, each with a different resistance gene (Leonard et al., 1989). Routine screening of *E. turcicum* races is carried out using maize differential genotypes in some maize producing countries (Weems and Bradley, 2018; Jindal et al., 2019; Turgay et al., 2020; Navarro et al., 2021; Muñoz-Zavala et al., 2023). However, maize resistance responses in these germplasm sets are highly variable and dependent on growth room/glasshouse conditions, and genetic background effects (Weems and Bradley, 2018; Jindal et al., 2019). To date, there are 29 described race groups based on the combinations of maize resistance genes that can be overcome, with race 0 defined as being able to overcome all known resistance genes (Table 2 – see references within). Race 0 has been described from countries in all the continents except South America (Table 2). Twelve of the race combinations have been reported from African countries (Table 2), although screening has only been done on *E. turcicum* isolates from South Africa, Kenya, Uganda, and Zambia (Craven and Fourie, 2011).

TABLE 1 The epidemiological characteristics of NLB, GLS and SCLB causal pathogens.

	NLB	GLS	SCLB
The first report	1878, New Jersey, USA (Drechsler, 1923)	1925, Illinois, USA (Tehon and Daniels, 1925)	1923, USA (Drechsler, 1925; Robert, 1953)
The first report in Africa	1924, Uganda (Emechebe, 1975)	1988, KwaZulu-Natal South Africa (Ward et al., 1997)	1974, Mpumalanga, South Africa (Rong and Baxter, 2006)
Causal pathogen (s) in Africa	*Exserohilum turcicum* (Pass.) K.J. Leonard & Suggs	*Cercospora zeina* (Crous & U. Braun)	*Bipolaris maydis* (Y. Nisik. & C. Miyake) Shoemaker
Old teleomorph name for genera	*Setosphaeria* (Alcorn, 1988)	Unknown	*Cochliobolus* (Alcorn, 1988)
Pathogen lifestyle	Hemibiotroph (Ohm et al., 2012)	Necrotroph (Benson et al., 2015)	Necrotroph (Ohm et al., 2012)
Lesion structure	Long oblong, cigar-shaped, tan or grayish (White, 1999)	Rectangular, tan to grey lesions delimited within veins (Latterell and Rossi, 1983)	Spindle-shaped (Race T) and rectangular parallel sided lesions (Race O) with chlorotic borders (Jeffers, 2004)
Lesion length	2.5 to 30 cm (White, 1999)	0.5 to 7 cm (Latterell and Rossi, 1983)	2 to 3 cm (Jeffers, 2004)
Asexual structures	Pale to olivaceous brown straight or slightly curved conidia with a hilum, 5-10 septa (Alcorn, 1988).	70-180 × 2-3 µm hyaline, 6-10 septa (Crous et al., 2006)	Fusoid, straight or curved conidia with one germ tube from each end and protuberant conidial hilum, 2-8 septa (Alcorn, 1988)
Sexual structure	Pseudothecia reported *in-vitro* (Abadi et al., 1993)	Unknown	Ascospores reported when intercrossed with other *Helminthosporium* spp. (Nelson, 1960).
Optimal growth conditions	18-27°C and high humidity	Temperature 22-30°C, relative humidity is > 90%	20-32°C and high humidity
Toxin production in culture	Monocerin (Robeson and Strobel, 1982)	Cercosporin (*C. zeae-maydis*) (Wang et al., 1998). Unknown toxin for *C. zeina*	Race T produces toxins I, II, III and IV (Karr et al., 1974)

Research on maize resistance genes to *E. turcicum* has revealed that the *Htn1* gene encodes ZmWAK-RLK1, a wall-associated receptor-like kinase (Hurni et al., 2015). Further research provided evidence that maize *Ht2* and *Ht3* genes encoded the same ZmWAK-RLK which corresponded to a different allele of ZmWAK-RLK1 (Yang et al., 2021). This is consistent with previous work that *Htn1*, *Ht2*, and *Ht3* map to chromosome 8 (Yang et al., 2021). This brings into question the validity of using *Ht* genes only

to allocate *E. turcicum* race classes when screening with differential maize panels. The authors propose that responses may be confounded by "modifier genes" as a result of (i) different genetic backgrounds, and (ii) variation in the size of the introgression surrounding an *Ht* gene in each differential maize line (Yang et al., 2021). This may explain why some isolates of *E. turcicum* gave different responses and were thus classified as race 2 or race 3 on the "differential" maize lines carrying *Ht*2 and *Ht*3 genes. In addition, it is well known amongst maize pathologists that responses to the pathogen are very sensitive to environmental conditions, which confounds reproducibility in *E. turcicum* race screening using differential panels (Weems and Bradley, 2018).

2.2 Gray leaf spot

Globally, GLS is the second most economically important foliar disease of maize after NLB, and is the most important foliar disease in the USA and Canada (Mueller et al., 2016, Mueller et al., 2020). Gray leaf spot disease was first reported in 1925 (Tehon and Daniels, 1925), and only became economically important in the late 1970s in the USA (Latterell and Rossi, 1983) and has since been reported in the Americas (Zhu et al., 2002; Juliatti et al., 2009; Neves et al., 2015; Mueller et al., 2020) and Asia (Manandhar et al., 2011; Liu and Xu, 2013). In Africa, GLS was first reported in 1988 in South Africa (Ward et al., 1997) and has since been reported in sub-Saharan Africa (Figure 1; Table 1).

Cercospora zeae-maydis Tehon & E.Y Daniels (Tehon and Daniels, 1925), and *Cercospora zeina* Crous & U. Braun (Crous and Braun, 2003) cause GLS. *Cercospora zeae-maydis* is predominant in the Americas and Asia, whereas *C. zeina* is found in Africa, Brazil, some parts of Asia, and the Eastern corn belt of the USA (Wang et al., 1998; Goodwin et al., 2001; Okori et al., 2003; Meisel et al., 2009; Neves et al., 2015; Duan et al., 2022). Although other *Cercospora* spp. have been associated with GLS, namely *Cercospora* spp. CPC 12062, a single isolate from South Africa (Crous et al., 2006) and *Cercospora sorghi* var. *maydis* Ellis & Everh. reported in Kenya (Kinyua et al., 2010) and Brazil (Neves et al., 2015), their role in pathogenicity has not been determined. The rest of this review will, therefore, focus on *C. zeina*, which is the predominant pathogen in Africa.

2.3 Southern corn leaf blight

Southern corn leaf blight, also known as Maydis leaf blight, was first reported in the USA in 1923 (Drechsler, 1925) and became a serious concern in the 1970s (Smith et al., 1970). Since then, SCLB reports have emerged from Western Europe, Asia and Africa (Munjal and Kapoor, 1960; Ullstrup, 1972; Fisher et al., 1976; Gregory et al., 1979; Carson et al., 2004; Singh and Srivastava, 2012; Manzar et al., 2022). The first report of SCLB in Africa was an outbreak in 1974 in South Africa, which resulted in the withdrawal of Texas male-sterile cytoplasm (T-cms) maize germplasm from the country's breeding programs (Levings, 1990; Rong and Baxter,

TABLE 2 The physiological races of *Exserohilum turcicum* and *Bipolaris maydis* and their global distribution.

Disease	Race	Resistance in host that is over-come by the Race	Distribution by continent and country					Authors references
			Africa	Asia	Europe	North America	South America	
Northern leaf blight	0	Virulent to all known R genes	Kenya, South Africa, Uganda, Zambia	China	Germany	Canada, USA		Sun et al., 2005; Dong et al., 2008; Zhao et al., 2008; Muiru et al., 2010; Craven and Fourie, 2011; Ramathani et al., 2011; Weems and Bradley, 2018; Jindal et al., 2019
	1	Ht1	Kenya	China, Israel	Austria, Germany, Hungary	Canada, USA	Brazil	Abadi et al., 1989; Sun et al., 2005; Ferguson and Carson, 2007; Dong et al., 2008; Muiru et al., 2010; Ramathani et al., 2011; Weems and Bradley, 2018; Jindal et al., 2019
	2	Ht2	Kenya, Uganda	China	Austria, Germany	Canada, USA	Brazil	Jordan et al., 1983; Sun et al., 2005; Zhao et al., 2008; Muiru et al., 2010; Ramathani et al., 2011; Weems and Bradley, 2018; Jindal et al., 2019
	3	Ht3	Kenya	China	France, Italy	Canada, USA		Sun et al., 2005; Dong et al., 2008; Zhao et al., 2008; Muiru et al., 2010; Ramathani et al., 2011; Jindal et al., 2019
	12	Ht1, Ht2	Kenya	China	Germany	Canada, USA		Sun et al., 2005; Dong et al., 2008; Zhao et al., 2008; Muiru et al., 2010; Weems and Bradley, 2018; Jindal et al., 2019
	13	Ht1, Ht3	Kenya, Zambia	China	Germany	Canada, USA	Mexico	Dong et al., 2008; Zhao et al., 2008; Muiru et al., 2010; Zhu et al., 2011; Weems and Bradley, 2018
	23	Ht2, Ht3	Kenya, Zambia	China	Germany	Canada, USA	Mexico	Sun et al., 2005; Ferguson and Carson, 2007; Dong et al., 2008; Muiru et al., 2010; Zhu et al., 2011
	123	Ht1, Ht2, Ht3	Kenya	China		USA		Sun et al., 2005; Zhao et al., 2008; Muiru et al., 2010; Weems and Bradley, 2018
	N	Htn1	Kenya, Uganda	China		Canada		Sun et al., 2005; Dong et al., 2008; Muiru et al., 2010; Weems and Bradley, 2018; Jindal et al., 2019
	1N	Ht1, Htn1		China		Canada, USA	Brazil	Gianasi et al., 1996; Dong et al., 2008; Weems and Bradley, 2018; Jindal et al., 2019
	2N	Htn2, Htn1		China		USA	Mexico	Windes and Pederson, 1991; Welz et al., 1993; Dong et al., 2008
	3N	Ht3, Htn1	Kenya	China	France, Italy	Canada		Windes and Pederson, 1991; Welz et al., 1993; Dong et al., 2008
	12N	Ht1, Ht2, Htn1		China		Canada	Brazil	Gianasi et al., 1996; Dong et al., 2008; Jindal et al., 2019
	13N	Ht1, Ht3, Htn1	Kenya, South Africa	China		Canada		Sun et al., 2005; Muiru et al., 2010; Human et al., 2020
	23N	Ht2, Ht3, Htn1	Kenya, Uganda, South Africa, Zambia	China	Austria, Germany	USA	Mexico	Sun et al., 2005; Ferguson and Carson, 2007; Dong et al., 2008; Zhao et al., 2008; Muiru et al., 2010; Human et al., 2020
	123N			China			Brazil	

(Continued)

TABLE 2 Continued

Disease	Race	Resistance in host that is over-come by the Race	Distribution by continent and country					Authors references
			Africa	Asia	Europe	North America	South America	
		Ht1, Ht2, Ht3, Htn1						Gianasi et al., 1996; Dong et al., 2008; Ma et al., 2020
	M	Htm1				Canada, USA		Weems and Bradley, 2018; Jindal et al., 2019
	1M	Ht1, Htm1				Canada, USA		Weems and Bradley, 2018; Jindal et al., 2019
	2M	Ht2, Htm1				Canada		Zhu et al., 2011; Jindal et al., 2019
	13M	Ht1, Ht3, Htm1				Canada		Inderbitzin et al., 2010; Jindal et al., 2019
	3M	Ht3, Htm1				Canada		Hulme, 2009; Jindal et al., 2019
	23M	Ht2, Ht3, Htm1				USA		Weems and Bradley, 2018; Jindal et al., 2019
	MN	Htm1, Htn1				USA		Weems and Bradley, 2018
	1MN	Ht1, Htm1, Htn1				Canada, USA		Weems and Bradley, 2018; Jindal et al., 2019
	2MN	Ht2, Htm1, Htn1				USA		Weems and Bradley, 2018
	12MN	Ht1, Ht2, Htm1, Htn1				Canada, USA		Zhu et al., 2011; Weems and Bradley, 2018; Jindal et al., 2019
	13MN	Ht1, Ht3, Htm1, Htn1				Canada		Jindal et al., 2019
	23MN	Ht2, Ht3, Htm1, Htn1				USA		Weems and Bradley, 2018; Jindal et al., 2019
	123MN	Ht1, Ht2, Ht3, Htm1, Htn1				Canada		Jindal et al., 2019
Southern corn leaf blight	0	Virulent for all	All maize producing countries	All maize producing countries	All maize producing countries	All maize producing countries	All maize producing countries	Smith et al., 1970; Mwangi, 1998; Balint-Kurti et al., 2007; Wang et al., 2010; Pavan and Shete, 2021
	T	cms-T	South Africa			USA		Leonard, 1977a, b; Rong and Baxter, 2006; Wang et al., 2010
	C	cms-C		China				Wei et al., 1988; Gao et al., 2000
	S	cms-S		China				Wang et al., 2010; Zhao et al., 2012

2006) (Table 1). Since then, no reports on SCLB have emerged from the country and on the rest of the continent until two decades later in Kenya (Mwangi, 1998) and recently on seeds in Nigeria (Biemond et al., 2013), thus indicating that SCLB can be a seedborne disease. These reports indicate that SCLB is present in Africa (Figure 1), currently at levels where its severity and occurrence are still insignificant. However there is the potential of SCLB becoming a severe and serious phytosanitary threat to maize production in Africa.

Bipolaris maydis (Y. Nisik. & C. Miyake) Shoemaker is the causative pathogen of SCLB (Smith et al., 1970). Previously known as *Cochliobolus heterostrophus*, *B. maydis* has been adopted as the most widely accepted species name (Rossman et al., 2013). Four physiological races of *B. maydis* (races O, T, C, and S) are known globally (Smith et al., 1970; Wei et al., 1988; Levings, 1993; Manamgoda et al., 2014), while races C and S only exist in China (Wei et al., 1988; Wang et al., 2010; Zhao et al., 2012; Bruns, 2017) (Table 2).

Overall, the USA was the first to report NLB, GLS, and SCLB in the early 20th-century (Drechsler, 1925; Tehon and Daniels, 1925). This could have been a result of the USA' system of Land Grant Universities with farmer extension services, vigilant crop disease diagnosis activities, and adoption of hybrid maize breeding (Duvick, 2001). At that time, maize production expanded and the planting of monocultures increased, creating a high risk of disease if susceptible genotypes were planted (Dodd, 2000; Duvick, 2001).

Gray leaf spot differs from NLB and SCLB, with no known physiological races among its populations. Physiological races of *E. turcicum* and *B. maydis* follow the "gene for gene" hypothesis. Races are classified based on a pathogen's ability to overcome resistance genes in maize inbred lines, often by loss of an avirulence gene recognized by a specific maize resistance gene (Leonard et al., 1989). However, the GLS-maize pathosystem has no known virulence genes or major resistance genes, so there are no known physiological races.

3 Disease epidemiology and economic impact of NLB, GLS and SCLB on maize

Disease epidemiology entails an understanding of the dynamics of disease development and proliferation in space and time (Milgroom and Peever, 2003). Several biotic, abiotic, and edaphic factors contribute to plant diseases (Milgroom and Peever, 2003). The knowledge on the above mentioned predisposing factors and epidemiological parameters, such as infection efficiency, latent period, and spore production in disease development, is therefore crucial in deciding the nature of control strategies to adopt (de Vallavieille-Pope et al., 2000; Milgroom and Peever, 2003). This section reviews the epidemiology of NLB, GLS, and SCLB, the factors that favor their development, and their economic impact.

3.1 Northern leaf blight

Upon infection of maize leaves, *E. turcicum* causes greyish lesions that start as chlorotic flecks and later mature into elliptical or cigar-shaped lesions from 2.5 to 30 cm in length (White, 1999) (Figures 2, 3). Disease establishment occurs within 6–18 h post-infection, and mature lesions develop within two weeks of host-pathogen interaction under favorable environmental conditions (Levy and Cohen, 1983; Bentolila et al., 1991; White, 1999; Kotze et al., 2019) (Table 1; Figure 2). The pathogen invades the host through the epidermis and blocks the vascular tissues (Kotze et al., 2019). This causes plant lodging and a reduction in photosynthetic leaf area, leading to 30%–91% yield losses in cases of severe infections during silking and grain filling (Tilahun et al., 2012; Nwanosike et al., 2015; Jindal et al., 2019; Berger et al., 2020). The NLB disease is a splash- and wind-borne polycyclic disease that spreads via conidia from infected debris left in the fields (Figure 2), and from secondary infections over long distances across fields (Schwartz and David, 2005).

3.2 Gray leaf spot

Cercospora zeina invades the host leaf tissues intracellularly resulting in irregular chlorotic lesions that after 14 days post infection, mature into grey to tan linear rectangular lesions that run parallel with leaf veins (Latterell and Rossi, 1983; Ward et al., 1999) (Figures 2, 3). Extensive disease development results in the coalescence of the lesions, blighting, necrosis of the leaf tissue, reduced photosynthetic area and plant lodging (Paul and Munkvold, 2005; Lennon et al., 2016). The calculations made based on spore size (40 - 165 μm × 4 - 9 μm), wind speed (varies per location) and the height of vertical mixing of the atmosphere above the crop, estimate flight distances of spores to range between 0.1 - 40 km as wind speed increases from 1 to 10 m/s (Ward et al., 1999). The spores have also been reported to spread to a distance of 80 - 160 km annually, making it a fast-spreading disease (Manandhar et al., 2011). Yield losses due to GLS have been estimated to be 20–80% (Latterell and Rossi, 1983; Ward et al., 1999; Manandhar et al., 2011).

3.3 Southern corn leaf blight

Irrespective of race, *B. maydis* infections in maize generally take between 12 to 18 h for fungal penetration, and 2 to 3 days to form mature lesions (Singh and Srivastava, 2012) (Figure 2; Table 2). The race O causes small diamond-shaped lesions that elongate into rectangular lesions limited within veins to a length of 20-30 mm that later coalesce, resulting in the entire leaf blighting (Jeffers, 2004; Singh and Srivastava, 2012). The race T causes oval-shaped yellow to brown lesions that are larger than race O (Jeffers, 2004; Singh and Srivastava, 2012), and produces a T-cms-specific polyketide toxin (T toxin) that is specific to T-cms maize genotypes (Condon et al., 2018). This race, whose origin is still a mystery, was implicated in a serious epidemic in the USA in 1970 (Bruns, 2017). The SCLB disease thrives in hot and humid agroecosystems (Warren, 1975) (Table 1). Yield losses of 10–40% due to SCLB infections have been reported, depending on the physiological race, environment and the maize hybrid grown (Fisher et al., 2012; Bruns, 2017).

All three fungal pathogens infect the same plant parts (leaves). They have similar growth requirements of moderate temperatures between 20°C and 30°C with relative humidity above 90% favoring disease establishment. Yield losses from individual pathogens can be 10-80%. Therefore, there is a need to determine the impact of combined infections by measuring the percentage of co-occurrence of these three diseases on a plant, field, and larger spatial scale to model their combined potential yield losses. This will facilitate the development of management strategies that target both single and co-infections.

4 Diagnosis of NLB, GLS, SCLB and identification of their causal pathogens

Crop disease diagnosis and the identification of the causal organism up to species level are becoming more critical. This is because more disease epidemics are emerging globally as a result of increased anthropogenic activities, such as global trade and expansion of pathogen ranges due to climate change (Hulme, 2009; Elad and Pertot, 2014; Prakash et al., 2014; Chaloner et al.,
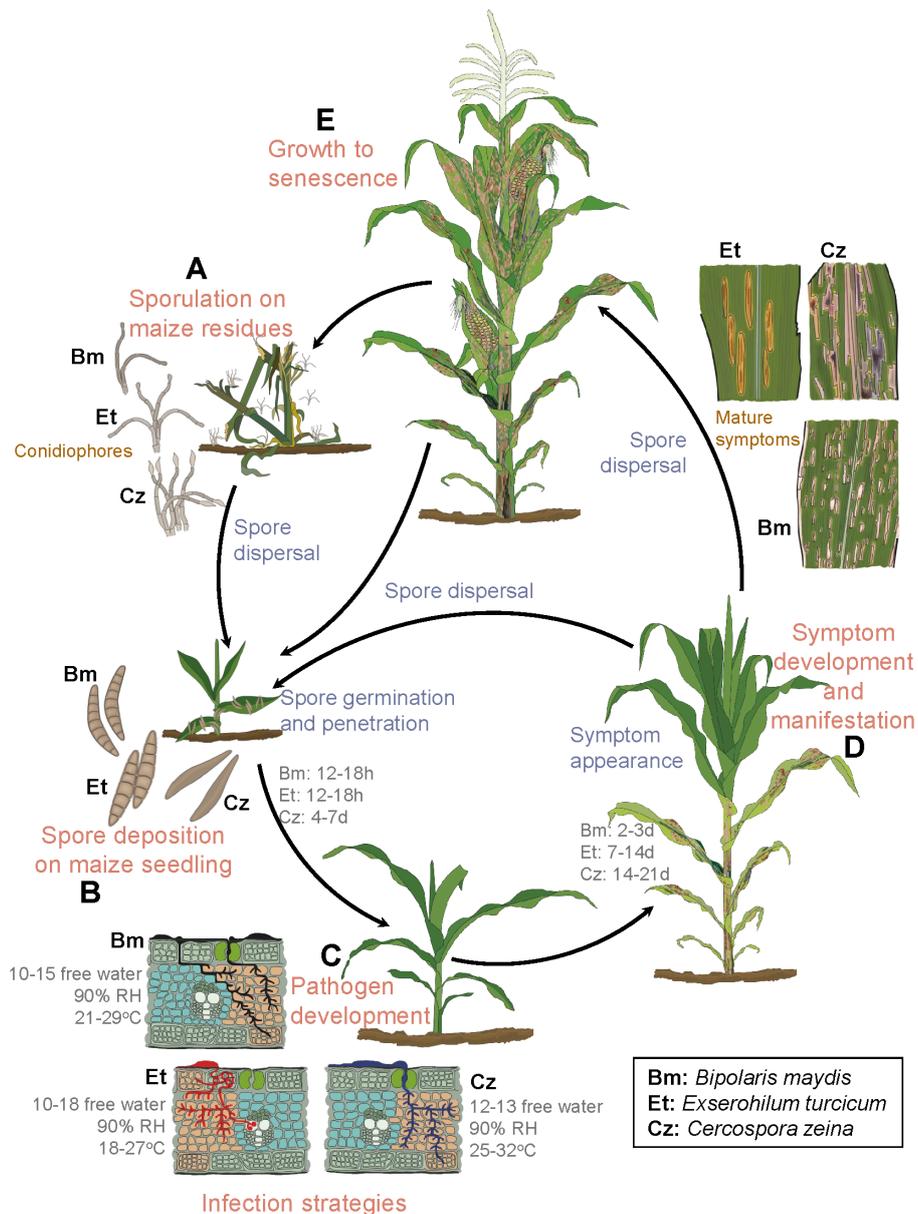
FIGURE 2

Asexual life cycles of *Exserohilum turcicum*, *Cercospora zeina*, and *Bipolaris maydis*. **(A)** Primary inoculum overwinters on maize debris as conidiophores until the next growing season, when they are dispersed in the form of conidia. **(B)** Under favorable conditions, conidia are dispersed and land on young maize plants. **(C)** Conidia germinate, penetrate plant cells, and later develop into small chlorotic spots. **(D, E)** Mature lesions develop from the lower leaves to younger leaves. These later give rise to conidia (secondary inoculum), which disperse to the younger plants, and the cycle repeats. Et, *E. turcicum* (Leonard, 1977b; Levy and Cohen, 1983; Bentolila et al., 1991; White, 1999; Kotze et al., 2019); Cz, *C. zeina* (Beckman and Payne, 1982; Latterell and Rossi, 1983; Ward et al., 1999; Wisser et al., 2011) and Bm, *B. maydis* (Jeffers, 2004; Wisser et al., 2011; Singh and Srivastava, 2012). Citations refer to sources for details of each pathogen's disease cycle. **(C)** leaf cross sections were adapted from Supplementary Figure S1 of Wisser et al. (2011). The unit for free water is hours. RH = relative humidity.

2021). Failure to accurately diagnose diseases and correctly detect the causal pathogens leads to inadequate or delayed implementation of control measures, thus causing a reduction in crop yield and quality (Miller et al., 2009). Similar to other plant diseases, NLB, GLS, and SCLB and their corresponding causal pathogens, have been diagnosed based on symptoms, morphological characteristics, and molecular phylogenetics.

4.1 Field diagnosis of NLB, GLS, and SCLB

Traditionally, plant disease diagnosis is performed through conventional visual field inspection of infected plant tissues (symptoms) using experienced technical human resources (Bock et al., 2010). Two standard scales (1–5 and 1–9, where 1 = resistant and 5 or 9 = susceptible) are being used to rate the severity of NLB
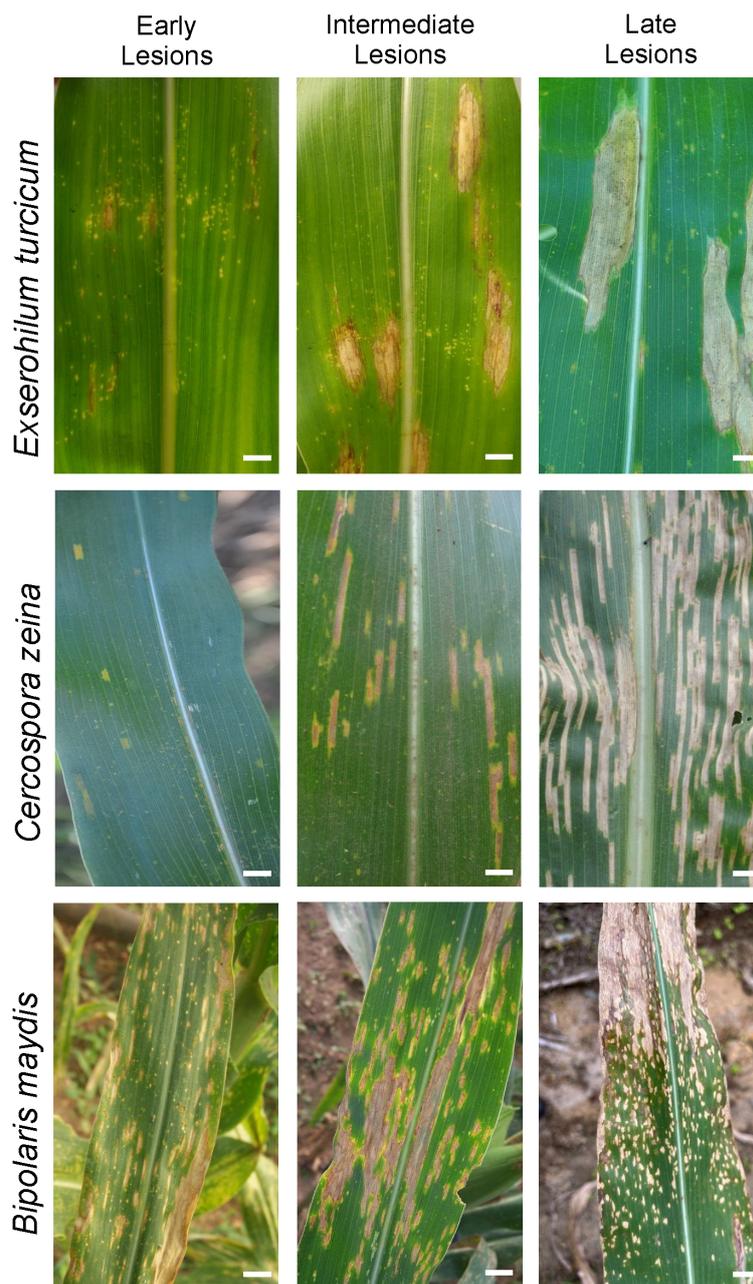
FIGURE 3

Symptomatic differences in NLB, GLS, and SCLB in maize. Each pathogen causes distinct disease symptoms during the intermediate and late stages of the infection cycle. Symptoms are prone to misidentification at early stages. All three diseases exhibit chlorotic spots in their early stages, making them difficult to diagnose. In the intermediate to late stages, each disease assumes its distinct lesion shape (i.e., cigar-shaped lesions for NLB, fine rectangular lesions for GLS, and rectangular lesions with irregular margins for SCLB symptoms, especially at the late stage). SCLB and GLS are not as clearly distinct as NLB. Scale bars = 2 cm.

(Abebe et al., 2008; Asea et al., 2009; Vivek et al., 2010; Kumar et al., 2011), GLS (Bubeck et al., 1993; Munkvold et al., 2001; Danson et al., 2008; Chung et al., 2011; Berger et al., 2014; Benson et al., 2015), and SCLB (Zwonitzer et al., 2009; Chung et al., 2010, Chung et al., 2011; Singh and Srivastava, 2012) (Table 2). Some plant pathologists have preferred using the 1 to 9 scale in the field and later converted it to a scale of 1 to 5 using the following formula: 0.5 * (disease score (1 to 9 scale) + 1) = disease score (1 to 5 scale) (Vivek et al., 2010). These scales are used to assess disease severity over time which can be expressed as area under disease progress curve (AUDPC) or disease index (Ma et al., 2022). While this traditional method has been refined over time, it is plagued by the inherent subjectiveness of disease estimates and is time-consuming (Nutter et al., 1993; Bock et al., 2008; Poland and Nelson, 2011). Sometimes, morphological traits are misleading because of the similarities between disease symptoms. For instance, race O lesions of SCLB are sometimes mistaken for GLS, whereas the initial symptoms of NLB, GLS, and SCLB (chlorotic spots) can potentially lead to misidentification (Figure 3).

Digital imaging techniques based on standard RGB images or hyperspectral images captured manually with cameras, mobile phones, or captured automatically with drones or by satellites hold great promise for crop disease diagnostics (Mohanty et al., 2016; DeChant et al., 2017). These methods involve training computer models with datasets of disease images which have been pre-classified by plant pathologists. The models are then tested on new sets of disease images to evaluate the accuracy of diagnosis. Examples of machine learning methods that have been used for plant disease diagnosis are spectral angle mapper (SAM), partial least squares regression (PLSR), support vector machines (SVMs), and convolutional neural networks (CNNs) (Xie et al., 2012; Stewart and McDonald, 2014; Mutka and Bart, 2015; Pauli et al., 2016; MuLaosmanovic et al., 2020).

This is a very active area of research that also being applied to maize foliar diseases such as NLB and GLS with accuracies of greater than 90%, but it is still in its infancy since most models are being trained on images with only one disease symptom type (Zhang, 2013; Xu et al., 2015; Mohanty et al., 2016; Qi et al., 2016; Singh et al., 2016; DeChant et al., 2017; Zhang et al., 2018; Craze et al., 2022; Pan et al., 2022). Attempts are underway to diagnose individual foliar diseases with more field-realistic images with multiple disease symptom types; for example a neural network model was developed to identify GLS symptoms on maize leaves which had mixed symptoms of NLB, common rust, and white spot disease (Craze et al., 2022).

These high-throughput diagnostic methods are a foundation for understanding pathogen ecology, epidemiology, and biology. Their integration into management programs for several plant diseases has the potential to foster a more targeted approach for the prevention of epidemics.

4.2 Morphological and physiological diagnosis and detection

For years, pathogen identification has relied on conventional techniques such as culturing, re-inoculation, microscopy, and biochemical assays (Sharma and Sharma, 2016). Morphological methods, which depend on visible signs of post-fungal infections, such as symptoms and fungal propagules, can be used to distinguish between *E. turcicum* and *B. maydis*, based on a hilum. *Bipolaris maydis* has a subtle hilum (Alcorn, 1988), while *E. turcicum* has a truncated, prominent hilum with a bubble (Leonard, 1974) (Figure 4). Cercosporoid fungi, however, are mainly distinguished based on conidia, hila, and pigmentation of their asexual structures (Crous and Braun, 2003; Crous et al., 2006; Nsibo et al., 2021). *Cercospora zeina* conidia are characterized by their septate, hyaline, thin walls, smooth apex, and thick darkened and refractive hila (Figure 4). These characteristics are similar to those of *C. zeae-maydis*. However, they differ in conidia shape, conidiophore length, and growth rate (Crous et al., 2006). Furthermore, *C. zeae-maydis* produces a photoactive phytotoxin, cercosporin, *in vitro*, whereas *C. zeina* does not (Goodwin et al., 2001; Crous et al., 2006; Swart et al., 2017).

Disease diagnosis of NLB, GLS, and SCLB based on morphological differences of the fungal morphology is possible.

However, this often requires isolation and culturing of the fungal pathogen, which is time-consuming. This makes these approaches inadequate for accurate and timely species-level identification (McCartney et al., 2003; Pryce et al., 2003).

4.3 Molecular identification

More advanced methods of identification, such as PCR-based amplification of nucleic acids and sequencing, are increasingly being employed for *E. turcicum*, *C. zeina*, and *B. maydis*. These methods can be more sensitive, are highly specific, faster, and require limited prior knowledge of the pathogen or expertise in plant pathology (McCartney et al., 2003; Ward et al., 2004). PCR amplification followed by sequencing of a fragment of the nuclear ribosomal DNAs (rDNAs), particularly the internal transcribed spacer (ITS), nested between conserved sequences of the 18S, 5.8S, and 28S rRNA gene regions, has been extensively employed for fungal species identification. The ITS marker is used as a universal barcode and is applicable for *E. turcicum* (Goh et al., 1998; Weikert-Oliveira et al., 2002; Ramathani et al., 2011; Haasbroek et al., 2014; Hernández-Restrepo et al., 2018), *C. zeina* (Dunkle and Levy, 2000; Crous et al., 2006; Meisel et al., 2009; Korsman et al., 2012; Liu and Xu, 2013; Bakhshi et al., 2015; Neves et al., 2015) and *B. maydis* (Goh et al., 1998; Emami and Hack, 2002; Manamgoda et al., 2012; Gogoi et al., 2014) (Table 3). The ITS region has multiple (identical) copies in the genome and it's PCR products are small (less than 1 Kb) which allow for easy PCR amplification, even in dilute or partially degraded DNA (White et al., 1973; Gardes et al., 1991; Lee and Taylor, 1992; Schoch et al., 2012).

Other available DNA targets for pathogen identification include regions of the translation elongation factor 1-α, calmodulin, β-tubulin, glyceraldehyde-3-phosphate dehydrogenase and mating type genes (Carbone and Kohn, 1999; James et al., 2006; Walker et al., 2012). Most of these gene regions have been employed for the identification of *E. turcicum* (Ramathani et al., 2011; Haasbroek et al., 2014; Hernández-Restrepo et al., 2018), *C. zeina* (Meisel et al., 2009; Bakhshi et al., 2015; Muller et al., 2016; Nsibo et al., 2019, Nsibo et al., 2021), and *B. maydis* (Leonard, 1974; Turgeon et al., 1995; Manamgoda et al., 2012; Tan et al., 2016) in Africa and around the world (Table 3).

Various species-specific PCR diagnostic tools that do not require sequencing have been developed (Table 4). For *E. turcicum*, mating-type genes are currently the only available species-specific diagnostic method. The amplification of PCR products of 608 bp and 393 bp using either a *MAT1-1*F or *MAT1-2*F primer together with MAT_CommonR primer indicates the presence of *MAT1-1* or *MAT1-2*, respectively (Henegariu et al., 1997; Haasbroek et al., 2014) (Table 4).

A species-specific PCR diagnostic that can differentiate three maize *Cercospora* species (*C. zeina*, *C. zeae-maydis*, and *Cercospora* sp.) was based on the histone *H3* gene region (Crous et al. (2006). A multiplex PCR was used where universal primers CylH3F and CylH3R amplify a 389-bp fragment common to all three species. This universal primer pair is multiplexed with species-specific primers
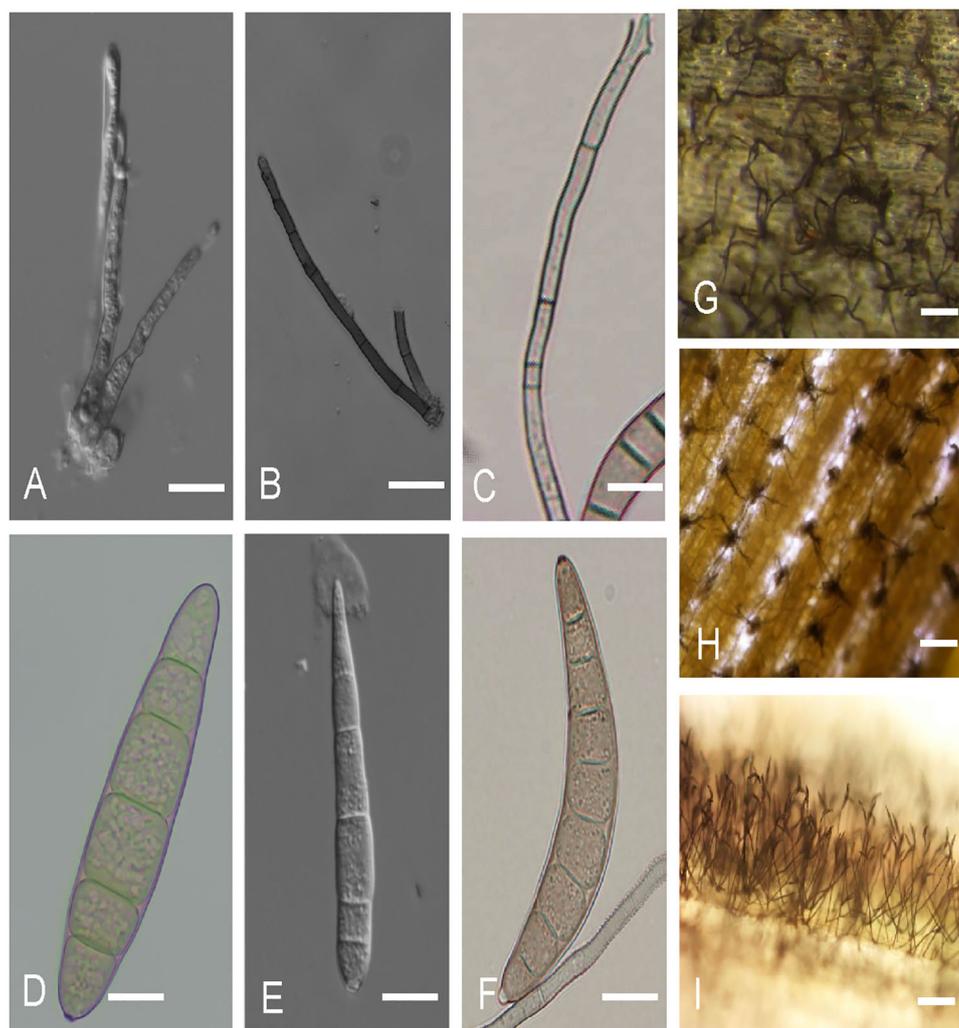
FIGURE 4
Asexual structures of *Exserohilum turcicum*, *Cercospora zeina*, and *Bipolaris maydis* in maize. **(A–C)** illustrates the conidiophores for **(A)** *E. turcicum*; **(B)** *C. zeina*; **(C)** *B. maydis*. **(D–F)** illustrate conidia of **(D)** *E. turcicum*; **(E)** *C. zeina*; **(F)** *B. maydis*. **(G–I)** Conidiophores of **(G)** *E. turcicum*, **(H)** *C. zeina* and **(I)** *B. maydis* on the surfaces of maize leaves Scale bars: **(A–F)** = 10 µm, **(G–I)** = 100 µm. **(C, F, I)** photos provided by Ms. Anu Elizabeth Ajayi, International Institute of Tropical Agriculture (IITA), Nigeria.

CzeaeHIST, CzeinaHIST, or CmaizeHIST in three separate PCR reactions for each unknown sample. Each reaction produces the common 389-bp fragment, and one of the three reactions will give a species-diagnostic 284-bp fragment (Crous et al., 2006) (Table 4). Limitations of this approach is the need to do three PCR reactions per sample, and the requirement for highly optimized PCR conditions to ensure only the correct species-specific primer binds to the target.

A cytochrome P450 reductase (*cpr1*) has been used to distinguish *C. zeina* and *C. zeae-maydis* from other maize pathogens. The CPR1_F and CPR1-R primers amplify a 164bp product from *C. zeina* and *C. zeae-maydis* but not from other maize pathogens (Korsman et al., 2012). Furthermore, the assay can also be used to differentiate *C. zeina* and *C. zeae-maydis* based on melting temperature differences between the products that can be measured after a real-time PCR reaction (Korsman et al., 2012).

Rapid identification of *C. zeina* or *C. zeae-maydis* is routinely carried out using primers in the cercosporin biosynthesis *CTB*7 gene region, since different sizes are produced for *C. zeina* compared to *C. zeae-maydis* (Swart et al., 2017; Nsibo et al., 2019, Nsibo et al., 2021). *Cercospora zeina* mating type markers amplify fragments that differentiate *C. zeina* MAT1-1 from MAT1-2 strains, with no amplification from species like *C. zeae-maydis* (Muller et al., 2016; Nsibo et al., 2019, Nsibo et al., 2021) (Table 4). For *B. maydis*, a multiplex mating-type PCR assay was optimized using primers MAT113, MAT123, and MATcon5 to amplify 702-bp (*MAT1-1*) and 547-bp (*MAT1-2*) fragments unique to *B. maydis* (Gafur et al., 1997) (Table 4).

Recently, high throughput diagnostics are being employed for the early detection of crop diseases. For example, nano-material-enabled sensors including carbon-based, metal- and metal oxide-based nanomaterials, are currently being used in the early detection of plant diseases based on the changes in the physiology of plants (Li et al., 2021). In addition, the RNA programmable nuclease of CRISPR/Cas is a nucleic acid detection tool that is currently being

TABLE 3 The universal molecular bar codes used in the identification of the causal pathogens of NLB, GLS and SCLB.

Locus	Definition	Primer Name	Oligonucleotide (5'→3')	Specificity	GenBank numbers			Reference
					E. turcicum	C. zeina	B. maydis	
ITS	Internal transcribed spacer region	ITS1	TCCGTAGGTGAACCTGCGG	Universal	NR_163537	DQ185081	NR_138224	Berbee et al., 1999; White, 1999; Meisel et al., 2009; Haasbroek et al., 2014; Tang et al., 2015
		ITS4	TCCTCCGCTTATTGATATGC	Universal				
TEFIα	Translation elongation factor 1 alpha	EF1-728F	CATCGAGAAGTTCGAGAAGG	Universal	LT896674	DQ185093	KM093793	Carbone and Kohn, 1999; Crous et al., 2006; Meisel et al., 2009; Neves et al., 2015
		EF1-986R	TACTTGAAGGAACCCTTACC	Universal				
CAL	Calmodulin	CAL-228F	GAGTTCAAGGAGGCCTTCTCCC	Universal	LT852468	DQ185117	HQ699077	Carbone and Kohn, 1999; Crous et al., 2006; Neves et al., 2015
		CAL-737R	CATCTTTCTGGCCATCATGG	Universal				
ACT	Actin	ACT-512F	ATGTGCAAGGCCGGTTTCGC	Universal	LT837686	DQ185105	AY748989	Carbone and Kohn, 1999; Crous et al., 2006
		ACT-783R	TACGAGTCCTTCTGGCCCAT	Universal				
TUB	β-tubulin	Bt1a	TTCCCCCGTCTCCACTTCTTCATG	Universal	LT899336	–	KX835024	Glass and Donaldson, 1995; Hernández-Restrepo et al., 2018
		Bt1b	GACGAGATCGTTCATGTTGAACTC	Universal				

These gene regions require PCR amplification, sequencing and phylogenetic analysis for species identification.

employed for crop disease diagnosis (Zhang et al., 2020; Wheatley et al., 2021; Liang et al., 2024). These methods are yet to be optimized for the detection of *E. turcicum*, *B. maydis* and *C. zeina*.

5 Genomic information for *Exserohilum turcicum, Cercospora zeina* and *Bipolaris maydis*

The development of molecular diagnostic tools and population genomics studies (see later) for these fungi will be increasingly supported in the future by genomics data, especially genome sequences. The first genome sequences that were available were developed using short-read Illumina sequencing, namely for USA strains of *E. turcicum* and *B. maydis* (Condon et al., 2013) and an African strain of *C. zeina* (CMW25467) from Zambia (Wingfield et al., 2017) (Table 5). Subsequently, these genome sequences were improved, for example, by including RNAseq data for better annotation, and using long read sequencing such as PacBio.

Details of the reference genome sequences for *E. turcicum*, *C. zeina, B. maydis* are presented in Table 5. It should be noted that some genome sequences are available from GenBank (https://www.ncbi.nlm.nih.gov/), whereas others are on the Mycocosm site at the Joint Genome Institute (JGI) (https://genome.jgi.doe.gov/portal/). Currently, the reference sequence for the NLB pathogen *S. turcica* (*E. turcicum*) Et28A race 23N and another USA strain NY001 race 1 are based on Illumina data (Table 5) (Condon et al., 2013). These assemblies resulted in genome sizes of 43 Mb and 38.4 Mb, respectively. However, due to short read sequencing it is likely the repetitive parts of these genomes are not fully assembled. The only genomics data set for an African isolate of *E. turcicum* is an *in planta* RNAseq time course for strain 2 (race 23N) and strain 103 (race 1) from South Africa (Human et al., 2020).

The reference genome for *C. zeina* is strain CMW25467 from Zambia in Africa (Wingfield et al., 2022). This high-quality genome sequence was determined using PacBio, resulting in 22 scaffolds (Table 5). In addition, Illumina genome sequences for 30 isolates of *C. zeina* from five countries in Africa were used for a population genomics study (Welgemoed et al., 2023). Recently, PacBio genomes for *C. heterostrophus* (*B. maydis*) race T strain C4 and race O strain C5 from the USA were reported (Haridas et al., 2023). These assemblies were 37.8 and 36.5 Mb in size in 70 and 53 scaffolds, respectively. Interestingly, the T-toxin biosynthetic cluster in race T was situated as dispersed genes within large stretches of repetitive DNA (Haridas et al., 2023). Overall, the number of predicted protein coding genes in these maize foliar pathogens were in a similar range of 11702 – 12547 (Table 5).

Genomic sequence information for these maize fungal pathogens opens several new avenues for disease control, as well as a deeper understanding of maize-pathogen interactions. Protein-coding gene catalogues of *E. turcicum*, *C. zeina*, and *B. maydis* can be searched for effector genes, known to be important for pathogenicity. Machine learning tools such as Effector P are used for this (Sperschneider and Dodds, 2022), as was done for *Bipolaris* spp. and *E. turcicum* (Condon et al., 2013; Human et al., 2020).

TABLE 4 The species-specific molecular bar codes used in the identification of the causal pathogens of NLB, GLS and SCLB.

Locus	Definition	Primer name	Oligonucleotide (5'→3')	Species	Amplicon size (bp)	Date published
CPR*	Cytochrome P450 reductase	CPR1_1F	TCCACTCTCGCTCAATTCG	*C. zeina*	164	2012
		CPR1_1R	GCCTTCATCGCCATATGTTC			
		CPR1_1F	TCCACTCTCGCTCAATTCG	*C. zeae-maydis*	164	
		CPR1_1R	GCCTTCATCGCCATATGTTC			
CTB7	Cercosporin toxin biosynthesis 7	CTB7-F	AAGAGTGCTTGTGAATGG	*C. zeina*	618	2017
		CTB7-R	GATGCGGGTGAAGTAGAAA			
		CTB7-F	AAGAGTGCTTGTGAATGG	*C. zeae-maydis*	925	
		CTB7-R	GATGCGGGTGAAGTAGAAA			
HIST	Histone H3	CylH3F	AGG TCC ACT GGT GGC AAG	*Cercospora* sp.	389	2004 and 2006
		CylH3R	AGC TGG ATG TCC TTG GAC TG			
		CzeinaHIST	TCGAGTGGCCCTCACCGT	*C. zeina*	284	
		CzeaeHIST	TCGACTCGTCTTTCACTTG	*C. zeae-maydis*	284	
		CmaizeHIST	TCGAGTCACTTCGACTTCC	*Cercospora* sp.	284	
MAT	Mating types	Cz*MAT1-1*F	TCACCCTTTCACCGTACCCA	*C. zeina*	631	2016
		Cz*MAT1-1*R	CACCTGCCATCCCATCATCTC			
		Cz*MAT1-2*F	CGATGTCACGGAGGACCTGA		409	
		Cz*MAT1-2*R	GTGGAGGTCGAGACGGTAGA			
		*MAT1-1*F	CTCGTCCTTGGAGAAGAATATC	*E. turcicum*	608	2014
		*MAT1-2*F	GCTCCTGGACCAAATAATACA		393	
		MAT_CommonR	AATGCGGACACGGAATAC			
		MAT113	AGGTAGTTTGAGGTGAGGGCAGATGATG	*B. maydis*	702	1997
		MATcon5	TCTTTGTTTTCCTGTGACTGCCTGTTG			
		MAT123	CTGGGCTGATTGGGGGCTTGATAC		547	
		MATcon5	TCTTTGTTTTCCTGTGACTGCCTGTTG			

*Primers can distinguish between *C. zeina* and *C. zeae-maydis* based on differences in their quantitative PCR (qPCR) melting peaks (Korsman et al., 2012).

TABLE 5   Reference genome sequences.

Disease	Northern (corn) leaf blight	Northern (corn) leaf blight	Gray leaf spot	Southern corn leaf blight	Southern corn leaf blight
Old teleomorph name	*Setosphaeria turcica* Et28A v2.0 (race 23N)	*Setosphaeria turcica* NY001 v2.0 (race 1)	Unknown	*Cochliobolus heterostrophus* race T strain C4	*Cochliobolus heterostrophus* race O strain C5
Current species names	*Exserohilum turcicum*	*Exserohilum turcicum*	*Cercospora zeina* CMW 25467	*Bipolaris maydis* ATCC 48331	*Bipolaris maydis* ATCC 48332
Genome size (Mb)	43	38.4	41.7	37.79	36.5
Genome scaffold count	407	489	22	70	53
Scaffold N50 (length of scaffolds)	2.14 Mb	0.23 Mb	4 Mb	2.06	2.12 Mb
Scaffold L50 (# contigs in N50)	8	45	5	7	6
Repeat coverage (%)	13%	nd	AT rich component ~33%	13%	12%
Predicted genes	11702	12547	11570	11324	11808
Sequencing technology	Sanger, 454, Illumina	Illumina	PacBio	PacBio	PacBio
JGI version for this data	v2.0	v2.0	not available at JGI	v6.0	v4.0
JGI weblink	https://mycocosm.jgi.doe.gov/Settu3/Settu3.info.html	https://mycocosm.jgi.doe.gov/Settur3/Settur3.info.html	not available at JGI	https://mycocosm.jgi.doe.gov/CocheC5_4m/CocheC5_4m.info.html	https://mycocosm.jgi.doe.gov/CocheC5_4m/CocheC5_4m.info.html
GenBank Accession #	GCA_000359705.1 (v1.0)	not available at Genbank	MVDW02	GCF_000354255.1 (v1.0)	GCA_000338975.1 (v1.0)
Provenance of strain	USA	New York State, USA	Mkushi, Zambia	USA	USA
Reference	Condon et al., 2013 (v1.0)	Condon et al., 2013 (v1.0)	Wingfield et al., 2022	Haridas et al., 2023	Haridas et al., 2023

Fungal effector genes interact with host proteins either directly or indirectly, and effector discovery is the first step in identifying host targets, eventually leading to effector-based breeding (Vleeshouwers and Oliver, 2014).

Gene catalogues for these maize pathogens can also prove useful for developing novel approaches for disease control, such as RNAi-based fungicides. In a recent study in *C. zeina*, a phylogenomic approach was used to first determine that this pathogen had the machinery for RNAi (Marais et al., 2024). This entailed comparing the protein-coding gene catalogue of 99 *Dothideomycetes* fungal genomes, and then drawing phylogenetic trees for orthogroups of Dicer-like, RNA-dependent RNA Polymerase and Argonaute. RNAi targets were identified in the *C. zeina* gene catalogue, allowing design of gene-specific dsRNAs. In a proof of concept, the dsRNA treatment disrupted the metabolic activity of the fungus *in vitro*, and reduced GLS disease when applied to inoculated maize leaves (Marais et al., 2024).

6 Management of NLB, GLS, and SCLB

Effective disease management strategies should aim to interfere with the most vulnerable stages of the pathogen life cycle to reduce the rate of disease development (Ward and Nowell, 1998; Shah and Dillard, 2010; Reddy et al., 2013). Cultural practices, chemical usage, and host genetic resistance are extensively employed in managing NLB, GLS, and SCLB.

6.1 Cultural practices for the control of NLB, GLS, and SCLB

Similar management strategies, including the use of tillage practices, rotation with non-host crops, and manipulation of environmental factors, are being used against NLB, GLS, and SCLB to reduce the amount of initial inoculum of the causal pathogens in the field (Ward and Nowell, 1998; Hooda et al., 2017). Deep tillage ensures the burial and destruction of pathogen inoculum in the soil (Payne and Waldron, 1983; Huff et al., 1988). Rotations for at least two years with non-host crops reduce fungal inoculum, especially in seasons with low disease incidence (Sharma and Payak, 1990; Ward and Nowell, 1998). In addition, manipulation of favorable environmental conditions (temperature, relative humidity, and leaf wetness) for pathogen development, especially early in the growing season, is crucial for hindering early season disease development (Ward and Nowell, 1998). Although

these cultural practices are useful in managing these diseases and may be effective in low-risk areas, they are less effective when the disease is well-established (Ward et al., 1997; Lipps et al., 1998; Ward and Nowell, 1998).

6.2 Chemical control of NLB, GLS and SCLB

Broad-spectrum fungicides, specifically demethylation inhibitor (DMI), quinone outside inhibitor (QoI), and succinate dehydrogenase inhibitor (SDHI), are effective against NLB, GLS, and SCLB, especially in susceptible hybrids (Reddy et al., 2013; Weems and Bradley, 2017; Dai et al., 2018; Neves and Bradley, 2019; Sun et al., 2023). Furthermore, Iturin A2, a *Bacillus subtilis* compound, was developed into a fungicide effective against *B. maydis* and other fungi (Gong et al., 2006; Kim et al., 2010; Ye et al., 2012). Iturin A2 could potentially treat other maize pathogens like *E. turcicum* and *C. zeina* and should be tested. Despite fungicide effectiveness, resistance has developed in other cereal pathogens like *Zymoseptoria tritici*, *Pyrenophora teres* f. *teres*, and *Magnaporthe oryzae* (Bohnert et al., 2018; Ellwood et al., 2019; Garnault et al., 2019). A few fungicide sensitivity studies have been conducted on *E. turcicum* and *B. maydis* to DMI, QoI, and SDHI fungicides. To date, all have revealed high sensitivities to fungicides, with no resistance buildup yet (Chapara et al., 2012; Weems and Bradley, 2017; Yuli et al., 2017; Hou et al., 2018). However, Africa lacks baseline sensitivity studies on *E. turcicum*, *C. zeina*, and *B. maydis*, despite increasing fungicide demands and use in large-scale field plantings. These studies are needed before fungicide resistance monitoring in maize foliar pathogens is initiated in Africa. Chemical control is also too expensive for many smallholder farmers. Therefore, affordable and long-lasting strategies such as host resistance through breeding need to be integrated and utilized.

6.3 Breeding for resistance against NLB, GLS and SCLB

Host plant resistance is the most economical, eco-friendly, and adjustable approach for maize disease management. Nelson et al. (2018) pointed out that effective resistance depends on the effect and strength of resistance genes in the host. Major genes provide complete or near-complete resistance, while quantitative resistance involves multiple minor genes with small additive effects (St. Clair, 2010).

6.4 Qualitative breeding for resistance

Resistance to *E. turcicum* in maize is both qualitative and quantitative and can be used either separately or in combination with qualitative resistance following a gene-to-gene model (Welz and Geiger, 2000; Ogliari et al., 2005) (Table 2). Qualitative

resistance is mediated by *Helminthosporium turcicum* (*Ht*) resistance genes (Welz and Geiger, 2000). The four well-known *Ht* genes include *Ht1*, *Ht2*, *Ht3*, and *Htn1*, where the functions of *Ht1, Ht2* and *Ht3* have yet to be characterized (Van Staden et al., 2001; Yin et al., 2003; Ogliari et al., 2005). The *Htn1* gene is highly conserved in *E. turcicum* hosts, particularly maize, sorghum, rice, and foxtail millet (*Setaria italica*), and encodes a wall-associated receptor-like kinase that confers resistance against race 12 (Hurni et al. (2015). Other resistance genes include *HtP* against races 123x and 23rx (Ogliari et al., 2005) and the recessive genes *ht4* and *rt* that confer resistance to a wide range of *E. turcicum* races (Ogliari et al., 2005).

For GLS, a qualitative resistance locus is yet to be characterized. To date, GLS resistance is qualitatively inherited. Few major resistance quantitative trait loci including *Qgls8* derived from teosinte (Zhang et al., 2017), and *gRgls1* and *qRgls2* from maize (Zhang et al., 2012) have been precisely defined.

Qualitative genes can confer resistance to *B. maydis* races. For instance, *rhm* gene mainly protects maize against race O and, to a lesser extent, race T strains (Zaitlin et al., 1993; Chang and Peterson, 1995). Chang and Peterson (1995) proposed a two-gene model in which two homozygous recessive genes, *rhm1* and *rhm2* which, in combination, increased host resistance to *B. maydis*. This was confirmed by the reduced lesion size as compared to the control experiment (Chang and Peterson, 1995) or the effect of an individual gene (Simmons et al., 2001). Qualitative resistance is effective against race O, while the best defense against race T is to avoid T-cms maize germplasm in breeding programs (Leonard, 1977a). Resistance to C and S races is so far unknown (Rong and Baxter, 2006).

6.5 Quantitative breeding for resistance

Quantitative disease resistance is known to reduce disease severity and incidence, rather than completely eliminate the disease (Young, 1996; Poland et al., 2009). In recent years, QTL mapping studies have characterized several traits of crops, including resistance to several plant pathogens (Bernardo, 2008; Xu and Crouch, 2008).

Quantitative trait loci for resistance against NLB span the entire maize genome and have been identified in several mapping populations (Welz and Geiger, 2000; Wisser et al., 2006; Chen et al., 2016; Wang et al., 2018; Wende et al., 2018; Rashid et al., 2020). Using techniques such as genome-wide nested association mapping, QTLs with several potential candidate genes have been characterized and confirmed to confer resistance against NLB (Poland et al., 2011; Rashid et al., 2020). Although many QTLs are known to confer resistance to a broad spectrum of *E. turcicum* races, some QTLs are known to confer race-specific resistance to NLB (Chung et al., 2010, Chung et al., 2011).

Hot spots of QTLs conferring resistance to GLS span discrete regions of chromosomes 1, 2, 3, 4, 5, and 7 (Lehmensiek et al., 2001; Berger et al., 2014). Most notably, a candidate gene encoding a

maize caffeoyl-CoA O-methyltransferase that confers quantitative resistance to GLS and SCLB has been cloned, implicating lignin and the phenylpropanoid pathway in maize defense against foliar diseases (Yang et al., 2017).

Many of these QTLs are derived from bi-parental crosses between susceptible and resistant genotypes tested under different disease pressures, germplasm backgrounds, and environmental conditions (Clements et al., 2000; Lehmensiek et al., 2001; Balint-Kurti et al., 2008; Berger et al., 2014). A majority of the QTLs are environment-specific; however, many QTLs expressed in several environments have also been characterized (Berger et al., 2014). These can be introgressed into maize genotypes grown in different environments. Molecular breeding to develop GLS resistant maize for small-holder farmers in Africa has been reported (Kibe et al., 2020). Recently, advanced populations of maize developed by CIMMYT were used in a combination with linkage and association mapping with genome wide SNP markers to identify QTLs for GLS and NLB resistance in East Africa (Omondi et al., 2023).

Using recombinant inbred lines (RILs), Carson et al. (2004) identified 11 QTLs spanning chromosomes 1, 2, 3, 4, 7, and 10, which are associated with SCLB resistance. Additional SCLB resistance QTLs have been characterized from different maize genotypes at different maturity stages (Balint-Kurti and Carson, 2006; Zwonitzer et al., 2009; Negeri et al., 2011).

Thus, qualitative, and quantitative resistance breeding are crucial for managing NLB, GLS, and SCLB. Maize geneticists have made great progress in identifying the genomic loci associated with resistance to one or more of these three diseases. To identify these loci tools such as the nested association mapping (NAM) panel, and the availability of genome wide SNP markers (Benson et al., 2015) have been employed. Importantly, some loci appear to confer multiple disease resistance, and recent advances have validated some QTL in independent maize populations (Lopez-Zuniga et al., 2019). A limitation is that most of these studies have been carried out in the USA and Europe often with inbred lines adapted to these temperate climates, with disease scoring carried out against local populations of each pathogen (Technow et al., 2013). Molecular marker-assisted breeding tools can now be employed to introgress these resistance alleles into germplasm adapted to maize-producing regions in Africa to determine whether crop protection is conferred against pathogen populations on the continent.

7 Recent advances in population genetics of the causal pathogens of NLB, GLS, and SCLB

Pathogen survival is based on its ability to adapt to constant environmental changes through evolution (McDonald, 1997). Therefore, management strategies to counteract these fast-changing lifestyles must be guided by understanding the genetics of populations and their evolution in response to changing environments rather than a focus on individual "model" pathogen strains (McDonald, 1997).

7.1 Population genetics of *Exserohilum turcicum*

Microsatellite markers have replaced earlier techniques such as Random Amplified Polymorphic DNA (RAPD) and Amplified Fragment Length Polymorphism (AFLP) markers to study the global population structure of *E. turcicum*. Reports from Asia, Europe, the Americas, and Africa show that *E. turcicum* is genetically and genotypically diverse, with higher diversity in Asia and Africa (Borchardt et al., 1998b; Ferguson and Carson, 2004; Dong et al., 2008; Haasbroek et al., 2014; Tang et al., 2015; Nieuwoudt et al., 2018). European *E. turcicum* populations are characterized by low genetic diversity (Borchardt et al., 1998a; Turgay et al., 2021) and are partially differentiated due to the Alps (Borchardt et al., 1998a). African geographic boundaries, like mountains and the large lakes of the Rift Valley, may affect *E. turcicum* population structure, although this has not yet been investigated. Sexual recombination is a major evolutionary factor in *E. turcicum's* global population structure, even in Europe, where sexual occurrences are rare, based on the frequency distribution of mating types and a lack of observed sexual structures in nature or in the laboratory (Fan et al., 2007; Turgay et al., 2021). Mating-type genes were found to be equally distributed and frequent in several countries where mating-type studies have been conducted, except in Europe, indicating sexual recombination (Haasbroek et al., 2014; Human et al., 2016; Weems and Bradley, 2018). Even though *S. turcica*, the sexual stage of *E. turcicum* is very rare in nature, with the only existing report being from Thailand (Bunkoed et al., 2014), it has been induced under laboratory conditions using Sach's medium with barley culm (Moghaddam and Pataky, 1994; Fan et al., 2007).

Population genetic analysis allows for the study of physiological race distribution, potential race re-emergence, and the identification of alternative hosts. However, limited knowledge exists on the population genetic diversity and race diversity of *E. turcicum* in Africa, with the exception of populations in Kenya and South Africa. Therefore, understanding the population structure of *E. turcicum* in maize-growing African countries is needed.

7.2 Population genetics of *Cercospora zeina*

Prior to the classification of the GLS causal pathogens into two distinct species (Crous et al., 2006), all studies conducted on GLS referred to the disease as being caused by *C. zeae-maydis* (Latterell and Rossi, 1983; Lipps, 1998; Ward et al., 1999; Okori et al., 2003). As more studies based on taxonomy, molecular and phylogenetic tools emerged, it became evident that there were two sibling species, *C. zeina* (formerly known as *C. zeae-maydis* type II) and *C. zeae-maydis*. Initial molecular studies of *Cercospora* isolated from maize in Africa indicated the presence *C. zeae-maydis* type II (*C. zeina*) using AFLP and RFLP markers with genetic relatedness to Type II, and not Type 1 isolates in the Americas (Wang et al., 1998; Dunkle and Levy, 2000; Okori et al., 2003; Liu and Xu, 2013; Muller et al., 2016). Subsequent surveys in sub-Saharan Africa confirmed that *C. zeina* was the only causal pathogen in Africa since to date, no isolates of *C. zeae-maydis*

have been found in a collection of more than 1000 isolates from East and Southern Africa (Nsibo et al., 2021).

Populations studies have shown that *C. zeina* is a highly diverse pathogen in Africa with a partially defined population structure within and among countries (Okori et al., 2003, Okori et al., 2015; Muller et al., 2016; Nsibo et al., 2019, Nsibo et al., 2021). Given that maize is non-native to Africa, the dominance of *C. zeina* on the continent is attributed to more than one introduction, followed by several sexual recombination events, intra-continent gene flow, migration, and local adaptations (Nsibo et al., 2021). This work was further refined by genome sequencing of 30 isolates of *C. zeina* from two countries in East Africa (Kenya, Uganda) and three countries in Southern Africa (Zambia, Zimbabwe and South Africa) (Welgemoed et al., 2023). This showed population differentiation but no major differences in diversity indices between regions, indicating two possible introductions over the approximately 500-year time period since maize entered the continent (Welgemoed et al., 2023). The study of *C. zeina* populations from other continents and the search for alternate hosts is underway to explore alternative hypotheses, including the hypothesis that *C. zeina* in Africa was derived from a host jump from an unidentified grass species onto maize (Dunkle and Levy, 2000; Crous et al., 2006).

Although sexual structures of *C. zeina* have not been observed under field and laboratory conditions, cryptic sexual recombination has been suggested based on the presence of mating-type genes with equal distribution and frequency, in addition to the low levels of linkage disequilibrium among some populations (Groenewald et al., 2006; Muller et al., 2016; Nsibo et al., 2021). Possible explanations for the failure of laboratory experiments to induce or discover the sexual stage of *C. zeina* may include the absence of environmental parameters that the pathogen encounters in nature to trigger sexual recombination. It could also be a failure to systematically monitor the development of an ascocarp in this presumably asexual pathogen (Dyer et al., 1992) or fertility decline in the pathogen (Dyer and Paoletti, 2005).

There is clear evidence that *C. zeina* is a well-established pathogen in Africa with the potential to threaten food production on the continent if not monitored to determine its diversity and migration patterns and deploy more effective management strategies.

7.3 Population genetics of *Bipolaris maydis*

There is limited information regarding the genetic diversity of *B. maydis*. RAPD markers have been used to understand the genetic structure of *B. maydis* populations, especially in India, where most reports have emerged. In India, *B. maydis* has been reported to be highly diverse, with little to no population differentiation (Karimi, 2003; Jahani et al., 2011; Gogoi et al., 2014), suggesting that gene flow plays a major evolutionary role in the population structure of the pathogen. Furthermore, the physiological race O is the most predominant race in India (Gogoi et al., 2014; Pal et al., 2015), with high genetic variability among isolates of the same race (Gafur et al., 2002; Pal et al., 2015). Sexual recombination is another major evolutionary factor driving observed genetic diversity (Gafur et al., 1997, Gafur et al., 2002). The availability of the *B. maydis* genome (Condon et al., 2013) offers a unique opportunity to

develop more robust molecular markers, such as microsatellite and single-nucleotide polymorphism (SNP) markers, that can be exploited to enable comprehensive studies of the pathogen from all the countries where the disease exists.

Bipolaris maydis is a potential threat to maize production in Africa although it has only been reported in Kenya (Mwangi, 1998) and South Africa (Rong and Baxter, 2006). The risk of its spreading to other countries is heightened by the fact that *B. maydis* is both an air- and seed-borne pathogen (Aylor and Lukens, 1974; Manoj and Agarwal, 1998; Biemond et al., 2013). Due to increasing anthropogenic activities and global trade, unreported incidences of the pathogen in the rest of Africa are possible. Therefore, countries where SCLB has not yet been reported must be vigilant through the establishment of phytosanitary regulations and bodies that test and ensure the movement of healthy seeds across geographical boundaries. Methods such as roguing, seed dressing, and proper storage to minimize contamination have been suggested as alternative ways to ensure seed health (Biemond et al., 2013).

8 Breeding for multiple disease resistance against NLB, GLS and SCLB

Qualitative and quantitative disease resistance strategies, either individually or in combination (McDonald and Linde, 2002), are important for the management of NLB, GLS, and SCLB. These strategies are based on the development of advanced maize genetic populations and screening for disease resistance across multiple environments (see examples in next section). Multi-environment field testing aims to expose the maize populations with the "diversity" of pathogen genotypes (i.e. races). This, however, can now be done more systematically by supplementing the pathogen diversity by artificial inoculation if pathogen population genetics and race typing data are available.

The co-occurrence of maize foliar diseases such as NLB, GLS and SCLB in some maize production regions of Africa presents an additional challenge for maize breeders. As described above, many resistance QTL are available for each disease, however each locus may have a small effect and thus breeders need to introgress several QTL for durable resistance (Nelson et al., 2018). Researchers have, therefore, searched for multiple disease resistance loci in maize and other plants (Wiesner-Hanks and Nelson, 2016).

A multiple disease resistance QTL associated with resistance to NLB, GLS and SCLB was identified by association mapping with a panel of 253 genetically diverse maize genotypes that had been scored for each disease in the field (Wisser et al., 2011). Moderate resistance to all three diseases was found to be associated with alleles of a maize glutathione S-transferase (GST) gene (Dean et al., 2005; Wisser et al., 2011). This maize GST may play a general defense role against all three diseases through detoxification of fungal secondary metabolites.

In another study, quantitative resistance to both GLS and SCLB was associated with alleles of the *ZmCCoAOMT2 gene*, which encodes a caffeoyl-CoA *O*-methyltransferase (Yang et al., 2017). This enzyme is involved in the phenylpropanoid pathway and lignin production, thus potentially contributing to defense barriers against the invading fungal pathogens. In another study, the search for

robust QTL for resistance to NLB and SCLB was carried out using the maize Nested Associated Mapping (NAM) panel, high density markers, and field testing in the USA and China. Some of the identified QTLs conferred resistance to both NLB and SCLB, and one of the candidate genes was *ZmCCoAOMT2*, providing validation of the previous finding (Li et al., 2018).

Backcross populations developed between four multiple disease resistant and two susceptible maize lines were used to identify several QTLs associated with resistance to NLB, GLS, and/or SCLB (Lopez-Zuniga et al., 2019). Several of these QTL conferred resistance to two of the diseases, and six to all three (NLB, GLS and SCLB) (Lopez-Zuniga et al., 2019). Further work validated these QTL by developing populations in more uniform genetic backgrounds, and two QTL were confirmed to be associated with resistance to all three diseases (Martins et al., 2019).

A take home message from these studies is that quantitative resistance to each of these three foliar diseases is generally conferred by many QTL in a single maize genotype, each with minor but additive effects (Li et al., 2018). In addition, QTL conferring resistance to more than one disease are rare and would be limited to common responses to the different pathogens (Martins et al., 2019).

Pyramiding qualitative resistance genes has been successful in other cereal pathosystems, such as wheat against the Ug99 races of stem rust (Zhang et al., 2019). However, combining QTLs for Ug99 resistance has been proposed as the most durable strategy for resistance (Singh et al., 2006). Pyramiding QTL would be the most durable strategy in maize against NLB, GLS, and SCLB when integrated with other management strategies.

Recent findings about both qualitative resistance genes like the Ht genes and multiple disease resistance QTLs described above have potential to benefit maize disease resistance breeders in Africa. However, several factors need to be fulfilled namely (i) access to germplasm and research capacity, (ii) efficacy of resistance loci in local environments, and (iv) ongoing research into pathogen population dynamics across the continent (Nsibo et al., 2021). Understandably, tightened phytosanitary regulations limits the ease with which maize germplasm can be shipped around the world. Fortunately, many African countries have historical access to maize germplasm through national breeding programmes, seed companies or NGOs such as the CGIAR institutes (Berger et al., 2020; Kibe et al., 2020). There are ongoing successes in releasing stress-tolerant maize varieties to farmers in Africa, which provides a good foundation to build on (Worku et al., 2020).

This highlights the importance of regional and international networks to address the threat of these three maize foliar diseases in Africa. Regional collaboration is important since fungal pathogens do not respect country borders. A successful example is the ongoing collaboration between universities in South Africa and Kenya which started with surveillance of the GLS pathogen *C. zeina* (Nsibo et al., 2021). Subsequently, CIMMYT came on board to expand the project to maize disease resistance breeding (Omondi et al., 2023). The collaboration had a strong component of capacity building with maize foliar disease workshops and postgraduate exchanges and training. The network is now supporting new maize disease reports in other countries of southern Africa. International linkages are key, for example collaboration with a

German University has brought in whole genome-based population genomics expertise to the *C. zeina* project (Welgemoed et al., 2023), and funding from the British Society for Plant Pathology has facilitated expansion of the project to *E. turcicum*.

9 Conclusion

This review summarizes recent advances in NLB, GLS, and SCLB disease resistance breeding, as well as the ecology and population genetics of their causal pathogens in Africa. All three diseases exist on the continent and threaten its maize production and food security. These diseases are polycyclic in nature and can infect maize under overlapping environmental conditions within a single growing season. With the increasing adoption of conservation agriculture and monocropping, foliar diseases are likely to escalate to all maize-producing countries owing to the accumulation of inoculum and shared dispersal mechanisms. Several management strategies at the commercial level, particularly cultural practices, fungicide usage, and breeding for resistance, are being increasingly adopted and used in Africa. However, since most farming is on a small scale, fungicide usage is not widespread due to its cost implications and its aftereffects on soil and human health. As such, there is an increasing adoption of breeding for resistance at a small-scale level, used in combination with cultural practices. Notably, limited knowledge is available on the population biology and genetics of *E. turcicum*, *C. zeina*, and *B. maydis* in Africa; thus, the evolutionary potential of these pathogens to overcome resistance has not been fully established. Therefore, there is a need to conduct large-scale sampling of isolates across the continent to study their diversity and trace their migration patterns across the continent.

Author contributions

DN: Data curation, Formal analysis, Investigation, Methodology, Software, Validation, Visualization, Writing – original draft, Writing – review & editing. IB: Conceptualization, Data curation, Formal analysis, Resources, Software, Supervision, Validation, Writing – review & editing. DB: Funding acquisition, Investigation, Methodology, Project administration, Resources, Supervision, Validation, Visualization, Writing – review & editing.

Funding

Acknowledgments

The authors acknowledge Glenda Brits from the Department of Education Innovation at the University of Pretoria for illustrating Figures 1 and 2.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

The author(s) declared that they were an editorial board member of Frontiers, at the time of submission. This had no impact on the peer review process and the final decision.

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