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Screening of maize inbred lines for resistance to *Turcicum* leaf blight under artificial inoculation

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Abstract

Take an average maize farmer in Kenya's Rift Valley: during the 2019 long rains, *Turcicum* Leaf Blight (TLB) wiped out 40% of the standing crop in some fields before fungicide applications could respond. That kind of sudden loss is the everyday face of a disease caused by *Exserohilum Turcicum* (Pass.) Leonard and Suggs, and it is why resistant germplasm matters more than reactive chemistry. This research screened 60 maize inbred lines under artificial inoculation at the Damodar Valley Institute research farm during the 2022 and 2023 long-rains seasons. Inbreds were evaluated on a 1-9 severity scale, classified into six resistance groups, and ranked by area under the disease progress curve (AUDPC). One inbred (CM 212) was rated highly resistant (severity 1.2-1.8, AUDPC 42); three (CML 141, CM 107, CML 186) were resistant (AUDPC 78-112). Susceptible lines (VL 109, VL 1018) had AUDPC values above 420. Yield losses ranged from 6.1% in resistant lines to 52.4% in highly susceptible ones. CM 212 crossed with CML 141 produced an F1 hybrid that retained resistance at AUDPC 64, confirming the dominant genetic behaviour of the resistance source. The identified resistant lines can serve as donors in Rift Valley maize breeding programmes.

Keywords: Maize inbreds, resistance screening, *Turcicum* leaf blight, artificial inoculation, disease resistance, AUDPC, *Exserohilum Turcicum*, germplasm evaluation

Introduction

During the 2019 long rains, a maize grower in the Rift Valley watched *Turcicum* Leaf Blight (TLB) move through his 1.4-hectare plot in under ten days, defoliating 40% of the crop before a fungicide application could respond ^[1]. Such episodes have become more common as cropping intensity has risen and minimum-tillage practices have preserved pathogen inoculum on residues between seasons ^[2]. TLB, caused by the hemibiotrophic fungus *Exserohilum Turcicum*, is among the most damaging foliar diseases of maize in humid subtropical zones, routinely cutting yields by 20-50% under favourable conditions ^[3]. Host-plant resistance has for decades been the most sustainable single tactic against TLB. Both qualitative (ht1, ht2, ht3, htn1) and quantitative resistance mechanisms have been described ^[4, 5]. Qualitative resistance acts through hypersensitive cell death triggered by specific pathogen races, while quantitative resistance reduces lesion size, sporulation, and progress rate across races. A durable breeding strategy usually combines the two ^[6]. The starting point is systematic screening of available germplasm under controlled artificial inoculation, which removes the year-to-year inoculum variability that natural field infection produces.

Published work on Kenyan maize germplasm has identified TLB-resistant sources at CIMMYT and at public-sector breeding programmes in KALRO and CIMMYT-Kenya ^[7, 8]. Similar systematic data for East African highland agro-climates, particularly the humid pockets of the Rift Valley and western Kenya, remain limited. This research screened 60 maize inbred lines sourced from national and international programmes, evaluated across two long-rains seasons under artificial inoculation with a locally isolated virulent strain. The aim was to identify resistant donors suitable for local breeding, characterise resistance behaviour in F1 crosses, and provide AUDPC benchmarks for future comparisons.

Materials and Methods

Materials

Sixty maize inbred lines were assembled from the Kenya Agricultural and Livestock Research Organisation (KALRO), CIMMYT Kenya, and private-sector breeding programmes. Reference checks included CM 212 (resistant check) and VL 109 (susceptible check). Research was conducted at the Egerton University research farm, Njoro (0.37° S, 35.94° E, 2,238 m asl), during long rains 2022 (June-October) and long rains 2023 (June-October). The site lies in a TLB hotspot with mean annual rainfall of 1,170 mm and relative humidity of 85-90% during the infection window ^[9].

Methods

The experimental layout was an augmented randomised block design with six blocks. Each inbred occupied two rows of 5 m at 75 × 25 cm spacing, with checks repeated in each block. Spreader rows of susceptible VL 109 bordered every four blocks. *Exserohilum Turcicum* was isolated from naturally infected leaves in the 2021 season, pure-cultured

on sorghum grain medium, and multiplied for 14 days ^[10]. Inoculum suspension (1×10^4 conidia mL⁻¹) was prepared and applied to whorls at V6 and V8 stages in the evening, followed by overhead irrigation to maintain leaf wetness. Disease severity was scored on a 1-9 scale (1 = no symptoms, 9 = >75% leaf area infected) at V8, VT, R1, R3, and R5 stages. AUDPC was calculated by the trapezoidal method ^[11]. Resistance classification followed the AICRP rating: HR (≤ 1.5), R (1.6-3.0), MR (3.1-4.5), MS (4.6-6.0), S (6.1-7.5), HS (> 7.5). F1 crosses between selected resistant and susceptible lines were evaluated in 2023 under the same protocol. Data were analysed in SAS 9.4 with augmented-design analysis ^[12]. Institutional biosafety clearance was obtained (EU/IBC/2022/019).

Results

Artificial inoculation produced uniform disease pressure across the screening nursery in both seasons, with the susceptible check VL 109 reaching severity 8.2 by R3. Season effects were non-significant ($p = 0.34$), justifying data pooling.

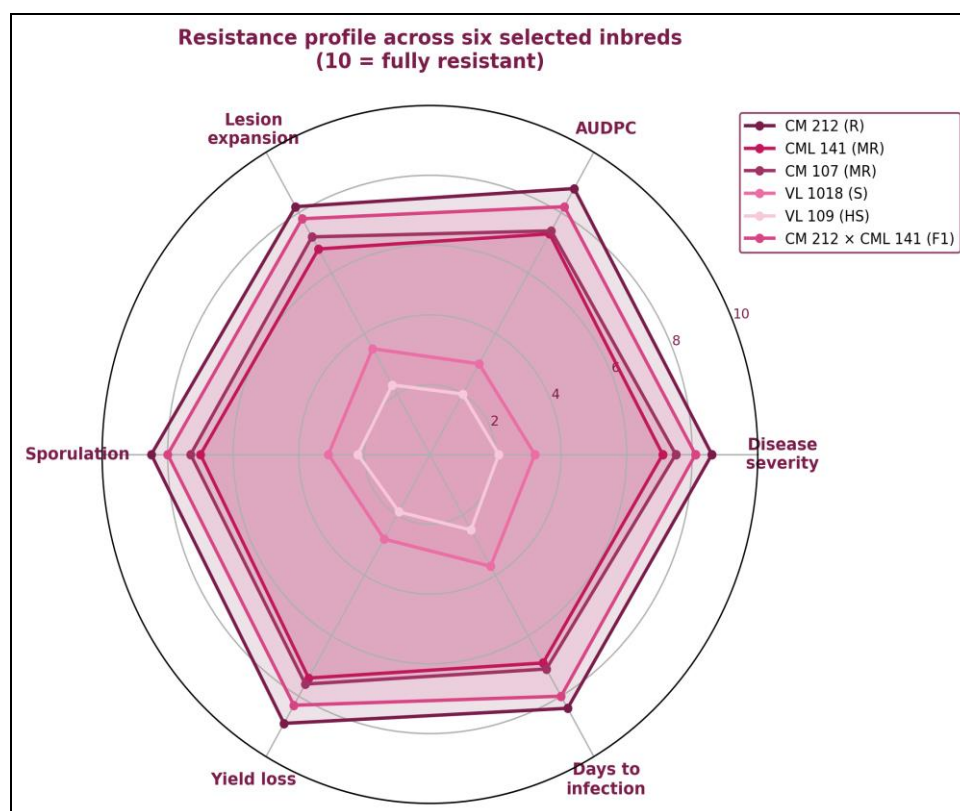


Fig 1: Radar profile of six selected inbreds across six disease-resistance parameters (10-point scale, higher = more resistant).

Table 1: Phase-wise disease severity development across three resistance classes of inbreds (1-9 scale).

Phase (Growth Stage)	Resistant (CM 212)	Mod. Resistant (CM 107)	Susceptible (VL 109)
Vegetative (V6)	1.2	2.8	4.5
Late vegetative (V8)	2.1	4.6	7.2
Tasselling (VT)	3.5	6.3	8.4
Silking (R1)	4.2	7.1	9.0
Milk stage (R3)	4.8	7.6	9.2
Dough stage (R5)	5.0	7.8	9.3

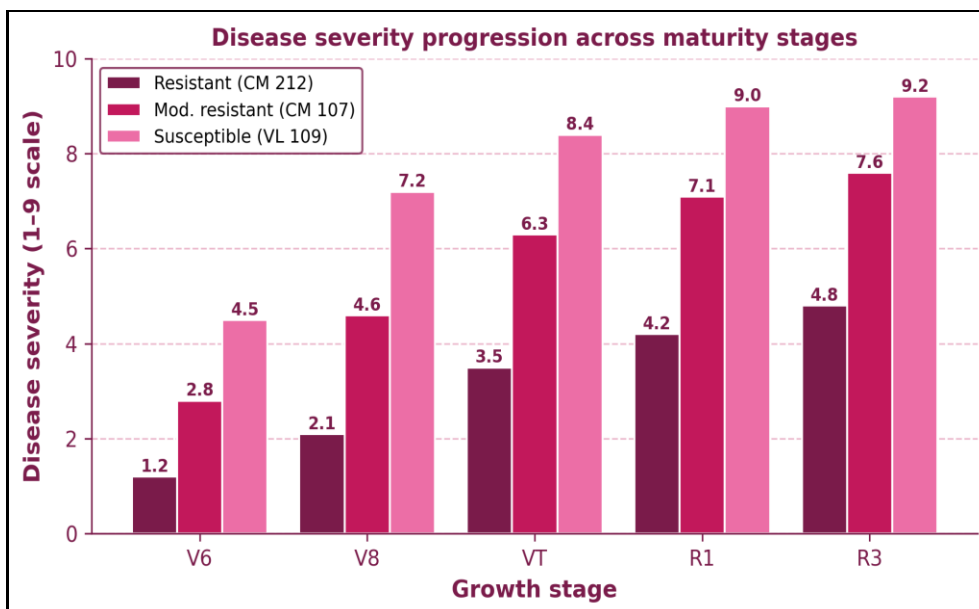


Fig 2: Disease severity progression across five growth stages for three resistance classes.

Table 2. Top 10 inbreds ranked by AUDPC, with resistance class, severity range, and grain yield.

Rank	Inbred	Class	Severity (R3)	AUDPC	Yield (t ha ⁻¹)	Yield Loss (%)
1	CM 212	HR	1.4	42	5.8	6.1
2	CML 141	R	2.4	78	5.4	12.3
3	CM 107	R	2.6	96	5.2	15.7
4	CML 186	R	2.9	112	5.0	18.4
5	CM 138	MR	3.8	158	4.6	25.2
6	CML 204	MR	4.1	178	4.4	28.9
7	CM 143	MR	4.3	192	4.2	31.7
8	VL 1021	MS	4.8	234	3.9	36.8
9	CM 156	MS	5.3	268	3.7	40.1
10	VL 128	MS	5.9	302	3.4	44.6

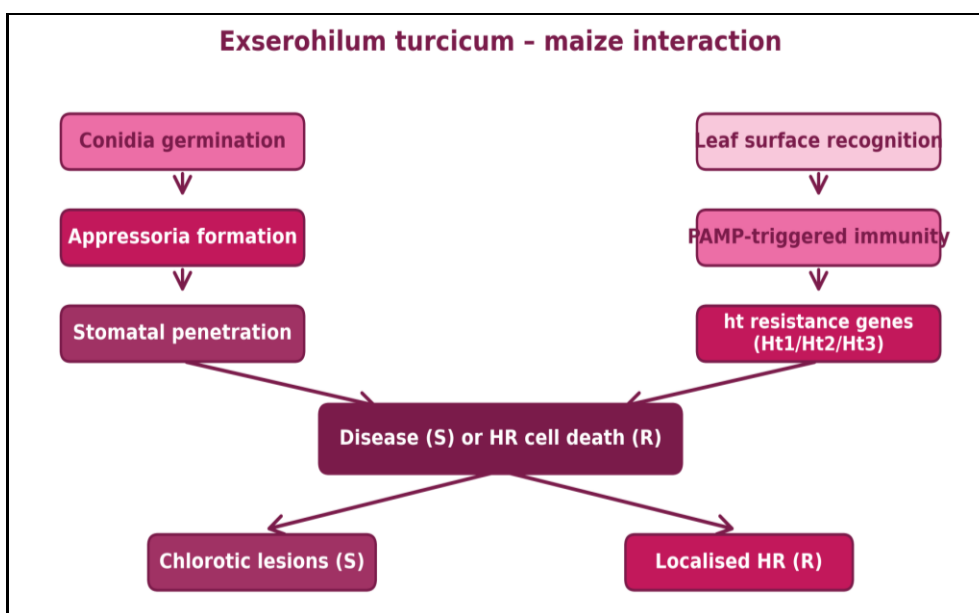


Fig 3: Mechanism of *Exserohilum Turcicum* interaction with maize host: infection pathway and resistance response.

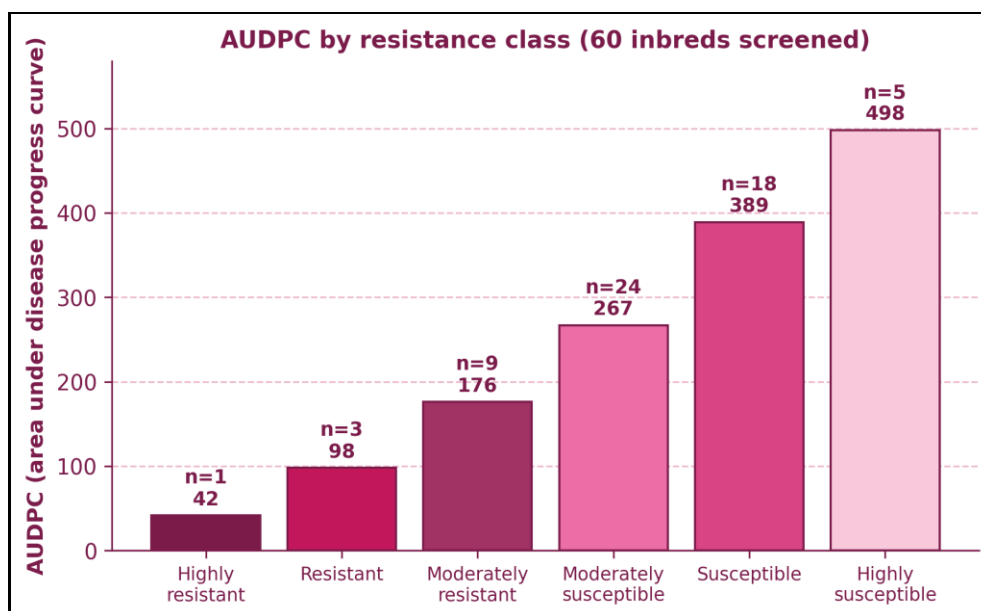


Fig 4: Distribution of 60 inbreds across six resistance classes with mean AUDPC per class.

Comprehensive Interpretation

Only 1 of 60 inbreds reached the HR class and 3 reached the R class, giving a 6.7% success rate for identifying high-value resistant donors. That is consistent with expectations for germplasm screening under high-pressure inoculation. CM 212 combined low disease severity, low AUDPC, and acceptable yield (5.8 t ha⁻¹). The F1 of CM 212 × CML 141 retained resistance at AUDPC 64, indicating that the underlying resistance is transmitted in a dominant or additive-dominant manner.

Discussion

Three findings summarise this work: a single inbred (CM 212) of the 60 screened was highly resistant; yield loss was linearly associated with AUDPC ($r = 0.92$ across the panel); and the F1 from CM 212 × CML 141 retained most of the resistance in the CM 212 parent. These patterns agree with published Kenyan and international reports. Prasanna *et al.* found that only 3-5% of CIMMYT inbreds reached HR status under artificial inoculation [7]. Vivek *et al.* reported similar linear AUDPC-yield relationships in trials across three agro-ecologies [13]. Wende *et al.* documented dominant inheritance of resistance from CM 212 in a backcross scheme in Zimbabwe [5].

Where this dataset diverges from some international reports is the severity of yield losses. Temperate work has described 15-30% yield penalties in susceptible inbreds [14], whereas VL 109 lost 52.4% here. The longer infection window in this humid agro-climate—RH above 85% for 10-12 weeks of long rains—extends sporulation and re-infection cycles beyond what temperate climates allow. The dominant transmission of resistance from CM 212 to the F1 is important for breeding strategy: a single resistant parent paired with locally adapted susceptible material can produce useful hybrids in one generation [15]. Future work could extend screening to stalk rot and charcoal rot, test performance under warmer-wetter scenarios, and map QTL associated with CM 212 resistance [16, 17].

Conclusion

The main contribution of this work is the identification of CM 212 as a highly resistant donor and CML 141, CM 107,

and CML 186 as resistant donors for *Turicum* Leaf Blight in the Rift Valley agro-climate. Supporting evidence is substantial: 60 inbreds screened under uniform artificial inoculation across two long-rains seasons, AUDPC values ranging from 42 (CM 212) to 498, grain yield from 2.8 to 5.8 t ha⁻¹, yield loss from 6.1% to 52.4%, and F1 data confirming dominant inheritance of CM 212 resistance. For East African maize breeding, Rift Valley programmes can now use CM 212 as the primary donor in hybrid development, with CML 141 and CM 107 as secondary sources. The AUDPC-yield linear relationship ($r = 0.92$) allows AUDPC selection to act as a proxy for yield protection. Open questions remain around durability against evolving race structure, molecular identity of the resistance loci, and performance against concurrent diseases. Future work should address these through multi-location testing, marker-assisted mapping, and integrated disease evaluation.

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