

THE MAIZE TRUST

FINAL REPORT FOR MYCOTOXIN PROJECTS

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Report due date	30 June 2026	Date range of activities reported	2015-2026
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1. Details of principal investigator

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2. Project title, duration and funding

Project title:	QTL mapping for resistance to maize ear rots	MTM project no.
Duration:	2015 - 2026	
Total MT funding received for project:		
All other funding received for this project (contributor and amount):	NRF (R300 000)	

3. Summary *(description of the project, capturing the main findings; maximum 250 words)*

Fusarium ear rot (FER) and Gibberella ear rot (GER), caused primarily by *Fusarium verticillioides* and *F. boothii*, respectively, are major constraints to maize production in sub-Saharan Africa. These pathogens reduce grain quality and yield while contaminating grain with harmful mycotoxins, including fumonisins (FUM), deoxynivalenol (DON), and zearalenone (ZEA). Resistance to these diseases is quantitative and strongly influenced by environmental conditions, complicating breeding efforts and highlighting the need to better understand the genetic and biochemical mechanisms underlying resistance. A doubled haploid (DH) maize population was developed from a resistant parent (CML390) and a susceptible parent (R2565Y), both adapted to African production systems. The doubled haploid (DH) population was evaluated over two seasons following artificial inoculation with the respective pathogens. Disease severity and mycotoxin accumulation data enabled classification of DH lines into resistant, intermediate, and susceptible groups and facilitated quantitative trait loci (QTL) mapping. Five putative QTL associated with resistance to disease and toxin accumulation (qGER1, qGER2,

qDON3, qDON4, and qFER5) were identified with qDON3 consistently supported across mapping approaches. To investigate biochemical mechanisms of resistance, untargeted metabolomics analyses were conducted on resistant (CML444) and susceptible (R2565Y) maize lines following FER and GER infection. Diterpenoids were strongly associated with reduced FUM accumulation, while piperidine carboxamide-like amines showed a negative association with ZEA accumulation, although their role in resistance remained inconclusive. Several lipid and fatty acid metabolites also displayed pathogen- and genotype-specific responses. This study provides the first public report of QTL associated with FER, GER, and mycotoxin resistance in South African-relevant maize germplasm. Integrating genetic and metabolomic analyses revealed key genomic regions and defence-related metabolites associated with resistance, advancing understanding of maize–Fusarium interactions and supporting future breeding efforts for improved disease and mycotoxin resistance.

4. Tasks, Milestones and Verification

Project goal:			
<i>List all tasks/activities (one per row) as mentioned in Section 4 of original proposal. This list should be comprehensive and include experimentation, publications, presentations, databases, manuals, human capacity development, etc.)</i> <i>Full details of scientific outputs can be given in Section 7.</i>			
Activity	Milestones	Means of verification	Reason(s) if not achieved
To develop and maintain a double haploid (DH) mapping population for QTL studies	The generation of DH mapping populations.	New seed source annually of DH mapping populations for disease screening trials.	N/A
To increase DNA marker resolution for QTL mapping	Identify and test additional SSR markers Perform DArT analyses on mapping population	An excel sheet with the newly identified SSR markers and their applicability in the mapping population An excel sheet with the SNP and silicoDArT information for the mapping population.	N/A
To phenotype the DH population for its response to <i>F. verticillioides</i> and	Conducting independent, plant response field trials to the major ear rot pathogens over two years.	A database of the plant response of the DH population to FER, GER and the associated mycotoxins.	N/A

<i>F. graminearum</i> and their mycotoxins		Statistically analysed data indicating significant differences between members of the population and correlations between disease severity and mycotoxin contamination.	
To map QTLs for resistance to FER, GER and associated mycotoxins using genotypic data (Obj 2) and phenotypic data (Obj 3)	Executing the mapping of QTL associated with FER, GER and/or the associated mycotoxins.	Physical map showing the distribution of mapped QTLs related to FER, GER and/or the associated mycotoxins for all 10 maize chromosomes.	N/A
To assess the metabolic response of ear rot resistant maize inbred lines to <i>F. verticillioides</i> and <i>F. graminearum</i> infection using untargeted metabolomics	Executing a field trial of different maize genotypes with differing responses to FER, GER and the associated mycotoxins. Analyses and identification of metabolites in maize genotypes when challenged with the pathogen. Identification and correlation of metabolites with mycotoxin resistance.	Collection and storage of maize material for analyses. Bioinformatic outputs that identifies the most significant metabolites. Additional bioinformatic and statistical analyses to accurately correlate metabolites to resistance to mycotoxins.	N/A

5. Results, Tables, Figures (provide a concise summary of results supported by tables and/or figures as appropriate)

1. To develop and maintain a double haploid (DH) mapping population for QTL studies

This objective was achieved. A double haploid population (n=200) has been maintained in collaboration with Corteva Agriscience over the past 3 maize-producing seasons (2022/3; 2023/4; 2024/5). The DH seed is stored at the Department of Plant Pathology, Stellenbosch University.

2. To increase DNA marker resolution for QTL mapping

This objective was completed. Briefly, a DH population (n=200) was subjected to genotyping by Diversity Array technology (DArT) and microsatellite analyses (SSRs) with published markers associated with resistance to FER, GER and associated mycotoxins. This produced an incredibly large genomic dataset for QTL mapping. From the DArT data, a total of 23 873 SNP and 12 509 SilicoDArT markers were generated, and the data was filtered based on parental informativeness, call rate and heterozygosity to determine an appropriate threshold for our dataset. A linkage map was successfully constructed spanning all 10 maize chromosomes consisting of 13 linkage groups. The map has sufficient coverage ([Table 1](#), Figs. [1](#) & [2](#)) when compared to the B73 reference genome, with exceptions on chromosomes 6 and 8, which have large gaps with inadequate coverage. The linkage map construction was performed under the supervision of Dr Renée Prins (Director, Cengen).

3. To phenotype the DH population for its response to *F. verticillioides* and *F. graminearum* and their mycotoxins

This objective was achieved. The DH population was evaluated for its response to FER and GER as well as associated mycotoxins, respectively, in independent, replicated field trials over two seasons (2022/3 and 2023/4). Disease severity for each disease, and the specific mycotoxins produced were determined. The results indicate the efficacy of artificial inoculation as inoculated and control groups differ significantly from each other ([Table 2](#)). Furthermore, the response of the population was skewed for both disease severity and mycotoxin production for FER but were normally distributed for GER and DON with moderate skewing for ZEA (Figs. [3](#), [4](#), [5](#) and [6](#)). Of the 135 DH lines evaluated for both diseases, 19 DH lines (14.1%) were resistant to both FER and GER in Y1 and Y2 with 10 DH lines (7.4%) susceptible to FER and GER in Y1 and Y2. Fourteen DH lines (10.4%) were resistant only to FER in both seasons whereas 32 DH lines (23.7%) were resistant to GER in both seasons. The remaining 108 DH lines (44.44%) were categorised as intermediately resistant ([Fig. 7](#))

4. To map QTLs for resistance to FER, GER and associated mycotoxins using genotypic data (Obj 2) and phenotypic data (Obj 3)

This objective was achieved. Genotypic data from DArT analysis was used to group, order, and space the DNA markers to generate the maize linkage groups. This was achieved in collaboration with Cengen. Putative QTLs were detected on chromosomes 2, 4, 5, 9 and 10 ([Table 3](#); [Fig. 2](#)). Due to differences in the skewedness of data, QTLs for GER, DON and FER were detected with moderate LOD scores. The biological significances of these QTLs were confirmed with additional analyses.

5. To assess the metabolic response of ear rot resistant maize inbred lines to *F. verticillioides* and *F. graminearum* infection using untargeted metabolomics

This objective was achieved. Inbred maize lines with known resistance (R2565Y and CML 444) to ear rots, and newly released CIMMYT inbred lines (n=10) with known response to ear rot, were evaluated for their metabolic response to ear rot pathogens by LC-MS/MS using an untargeted metabolomic approach. The maize-related metabolites were significantly associated with fumonisins and ZEA accumulation, most likely due to their mediation of defence responses in different maize genotypes. Di- and triterpenoids were highly significantly associated with fumonisins ([Fig. 8](#)), while isoleucine derivatives were significantly associated with ZEA ([Fig. 9](#)). From our results we noted an overlap of the qGER1 resistance peak with a triterpenoid glycosyltransferase gene interval on chromosome 2. This provides genetic and biochemical coherence, linking triterpenoid biosynthesis to ear rot defence.

Table 1. Summary of the linkage group characteristics including marker density, and physical coverage as compared to the B73 V5 maize reference genome.

Linkage group	Length (cM)	Physical length (Mbp) -B73	Marker density (markers/cM)	Coverage (%)	Number of markers	Portion of chromosome covered (Mbp)
1	286.5	301.35	0,20	88	47	27 - 298
2.1	117.9	243.67	0,14	15	17	204.9 - 241.7
2.2	38.9	243.67	0,10	68	4	24.4 - 191
3	217.1	233.3	0,19	90	42	26 - 236
4	295.3	246.3	0,15	98	44	3.9 - 246.3
5.1	206.8	223	0,20	82	41	1.2 - 184.3
5.2	4	223	0,75	0.37	3	219.9-220.8
6.1	76.5	175	0,20	8	15	164.3 - 178.3
6.2	42.2	175	0,07	-	3	Unk -10
7	265.4	184.54	0,08	98.77	22	2.26 -184.45
8.1	46.3	175.8	0,09	4.33	4	2.66 - 10.27
8.2	20.3	175.8	0,15	1.29	3	178.5 -180.8
8.3	33	175.8	0,30	-	10	Unk-166.5
9	227.6	150.17	0,18	100	41	10 -161
10	175.2	150.14	0,14	99.3	25	1-150
Total	2053	3076.54			274	

Genetic Length (cM): Length of each linkage group in centiMorgans; Physical Length (Mbp) - B73: Physical size of each chromosome in the B73 V5 reference genome; Marker Density (markers/cM): Number of markers per cM; Coverage (%): (Genetic length of our linkage group / Physical length of the corresponding B73 V5 chromosome in Mbp, converted to cM using average cM/Mb ratio) $\times$ 100 ; Number of Markers: Total markers mapped on each linkage group; Unk: Markers where the Mbp position could not be determined on the MaizeGDB platform.

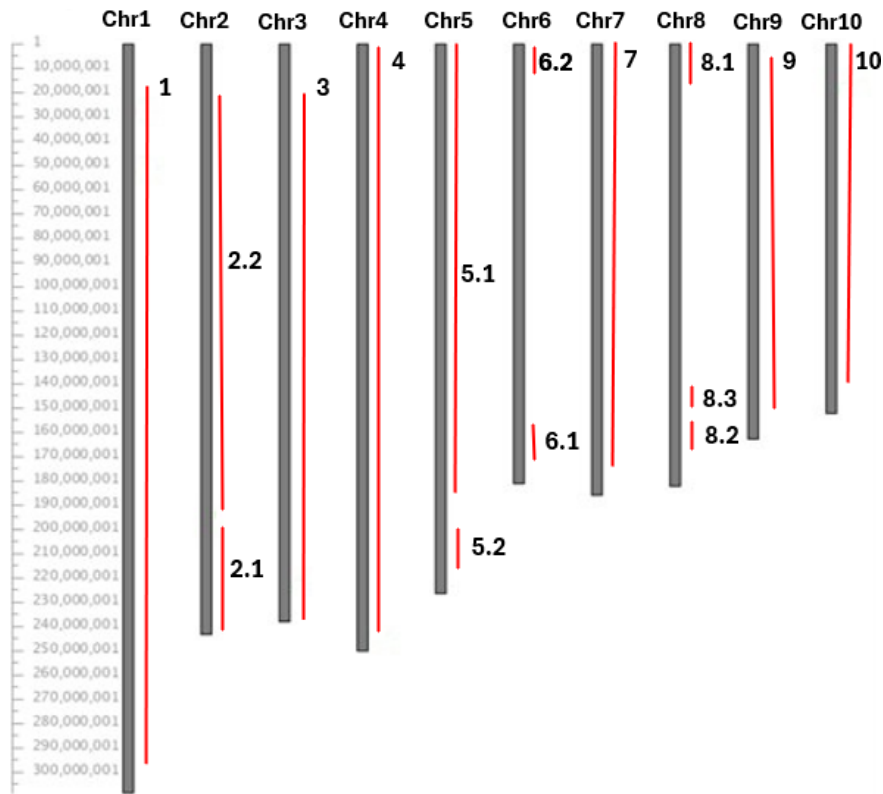


Figure 1. Alignment of our constructed linkage map (red) alongside with the B73 V5 maize reference genome (grey), illustrating marker distribution in mega base pairs (Mbp) and genome coverage across the 10 maize chromosomes. Coverage of the linkage map generated in this study is indicated in red. Source: Maize Genetics and Genomics Database (<https://www.maizegdb.org/>).

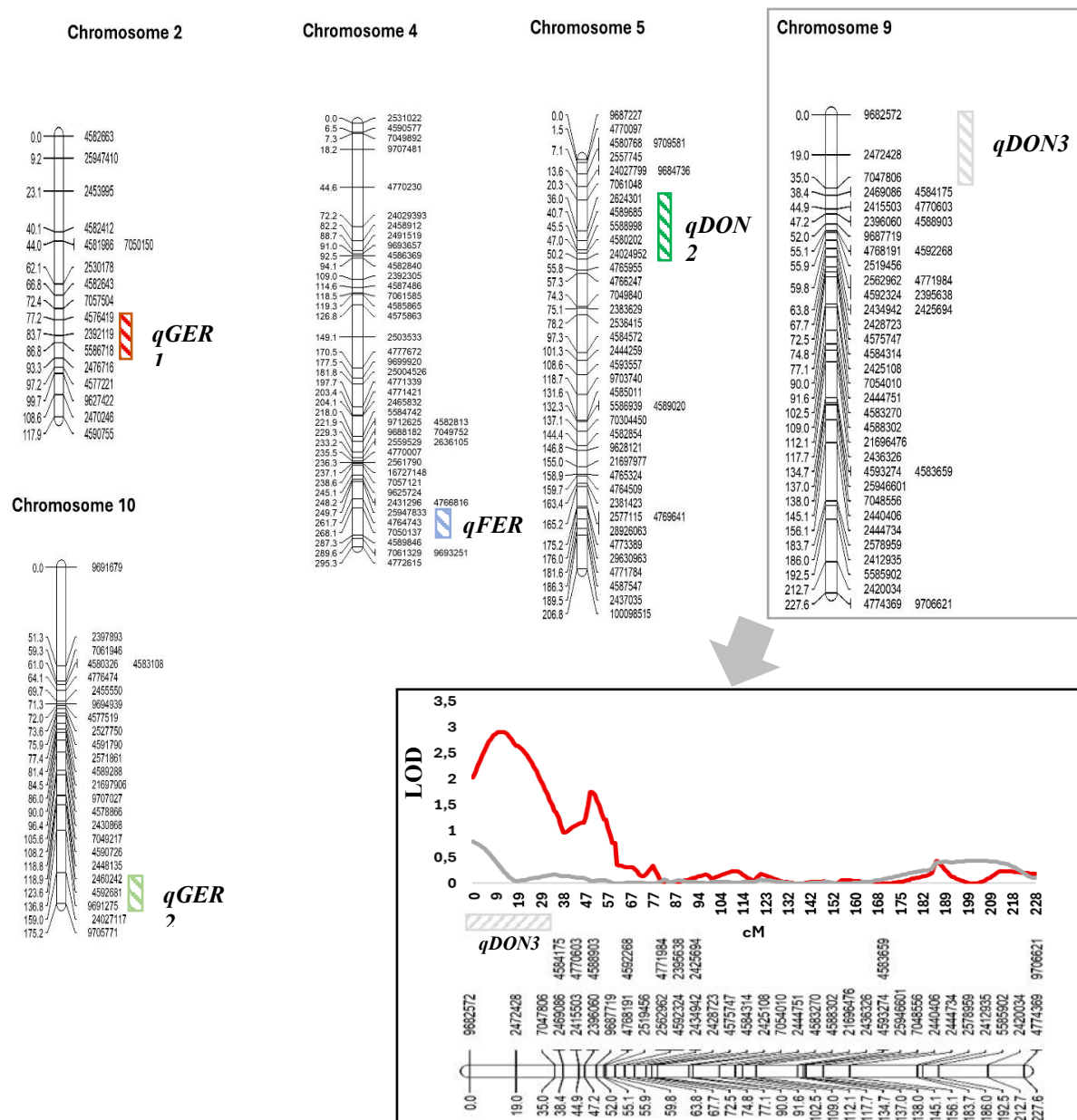


Figure 2. Linkage map segments for chromosomes 2, 4, 5, 9, and 10 showing marker distribution and positions of putative QTL associated with resistance to Fusarium ear rot (FER), Gibberella ear rot (GER), and deoxynivalenol (DON) accumulation. The QTL regions are highlighted, corresponding to *qGER1* (Chr 2), *qGER2* (Chr 10), *qDON3* (Chr 5), *qFER2* (Chr 4) and *qDON1* (Chr 9). Chromosomes displayed were selected based on the presence of significant or suggestive QTL. The sub-image (bottom right) shows the logarithm of the odds graph in relation to the marker positions with the marker distances in centimorgans (cM) on the x-axis and the logarithm of the odds score (LOD) on the y-axis.

Table 2. Summary of means and significance level of data gathered for infection indicators (Gibberella ear rot severity, deoxynivalenol and zearalenone) comparing *Fusarium boothii* inoculated and uninoculated samples of our maize doubled haploid population in 2022-2023 (Year 1) and 2023-2024 (Year 2).

	Mean (I)	Mean (C)
GER	13.27 A	2.0 B
DON	29.29 A	4.39 B
ZEA	10.34 A	2.47 B

GER = Gibberella ear rot severity %; DON = Deoxynivalenol (mg kg⁻¹); ZEA = Zearalenone (mg kg⁻¹); Y1 = Year 1; Y2 = Year 2; I = Inoculated; C= Uninoculated controls; means followed by the same alphabetical letter in a row are not significantly different according to Fisher's LSD at a 95% confidence level (P>0.05).

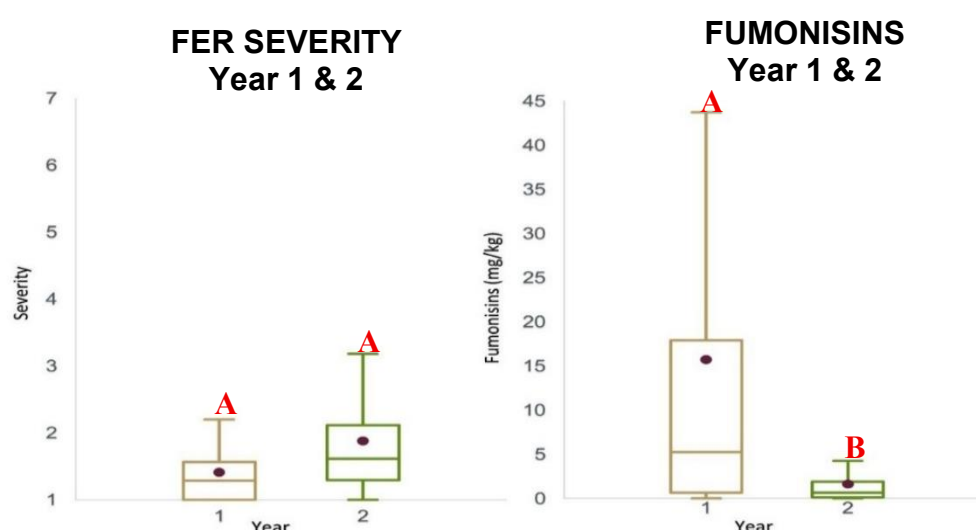


Figure 3. Box and whisker plots summarising the data of infection indicators for year 1 and 2 **Left:** Fusarium ear rot severity (FER) in year 1 and 2 in the absence of outlier effects **Right:** Fumonisin contamination in year 1 and 2 in the absence of outlier effects (mg kg⁻¹), means followed by the same alphabetical letter are not significantly different according to Fisher's LSD at a 95% confidence level (P>0.05).

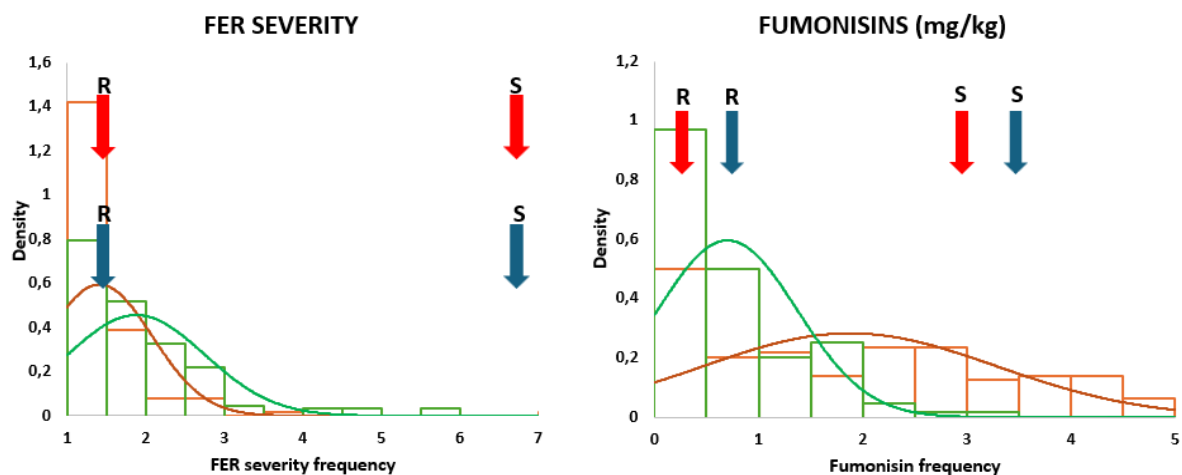


Figure 4. Frequency distribution graphs for Fusarium ear rot infection indicator data recorded following artificial inoculation of a doubled haploid population **Left:** Distribution frequency for Fusarium ear rot severity (FER) in year 1 (Green bars) and year 2 (Orange bars) **Right:** Distribution frequency for total fumonisins (FB1+FB2+FB3) in year 1 and 2. Curves show the normality distribution of the bars with the matching colour. Red arrows indicate where the FER/ fumonisin response of the maize parental lines (Resistant/R = CML444; Susceptible/S = R2565Y) were situated in year 2 versus year 1 (Blue arrows).

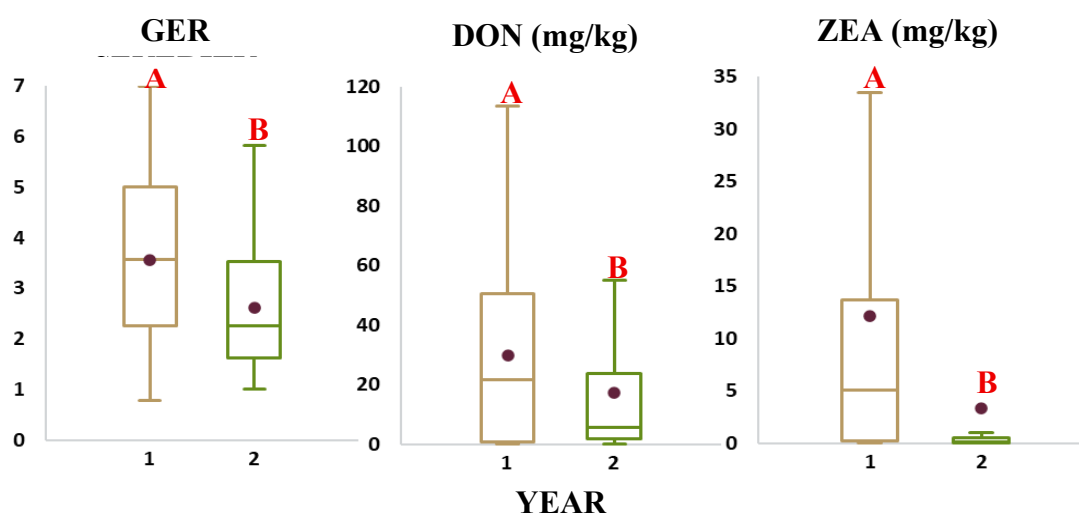


Figure 5. Box and whisker plots summarising the raw untransformed data of Gibberella ear rot (GER) infection indicators for year 1 and 2 following artificial inoculation **Left:** Gibberella ear rot severity (GER) occurrence in year 1 and 2 in the absence of outlier effects **Middle and right:** Deoxynivalenol (DON; mg kg⁻¹) and zearalenone (ZEA; mg kg⁻¹) contamination in year 1 and 2 in the absence of outlier effects. Means followed by the same alphabetical letter are not significantly different according to Fisher's LSD at a 95% confidence level ($P > 0.05$).

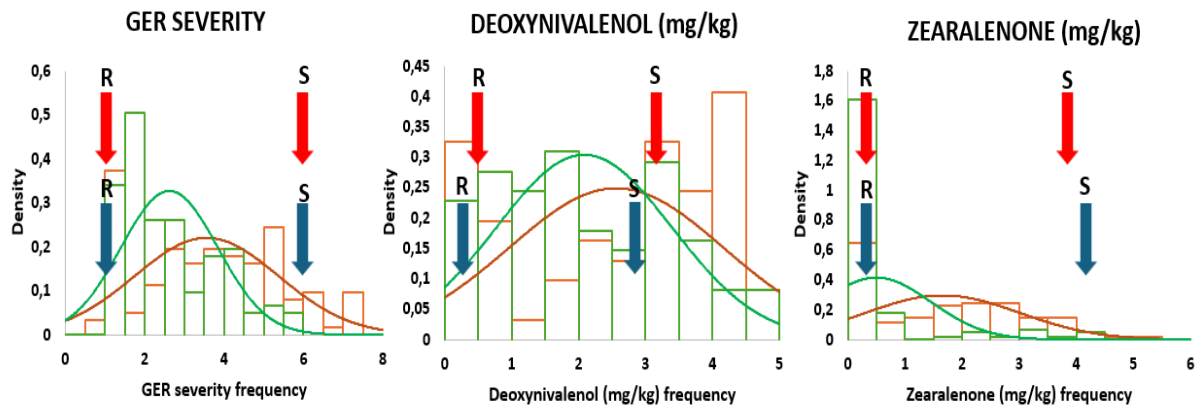


Figure 6. Frequency distribution graphs for *Gibberella* ear rot (GER) severity, deoxynivalenol and zearalenone **Left:** Distribution frequency for GER severity in year 1 (Green bars) and year 2 (Orange bars) **Middle:** Distribution frequency for deoxynivalenol in year 1 and 2 **Right:** Distribution frequency for zearalenone disease in year 1 and 2. Curves show the normality distribution of the bars with the matching colour. Red arrows indicate where the FER/ fumonisin response of the maize parental lines (Resistant/R = CML444; Susceptible/S = R2565Y) were situated in year 2 versus year 1 (Blue arrows).

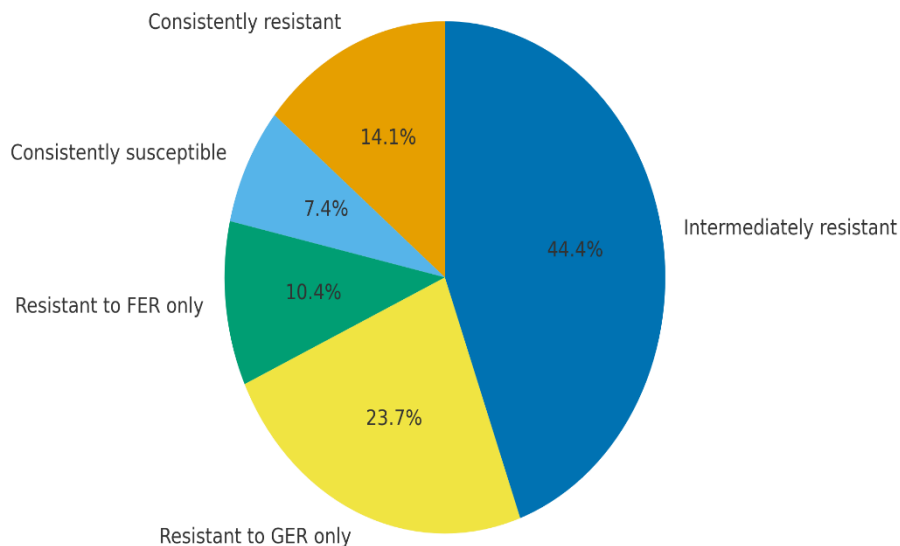


Figure 7. Pie chart showing the response categorisation of the phenotyped doubled haploid (DH) maize population (n= 135 lines) to *Fusarium* ear rot (FER) and *Gibberella* ear rot (GER) following inoculation of maize ears with *Fusarium verticillioides* and *Fusarium boothii*. Nineteen DH lines (14.1%) was resistant to FER and GER in both inoculation seasons (Consistently resistant). Ten DH lines (7.4%) was susceptible to FER and GER in both seasons (Consistently susceptible). Fourteen (10.4%) and 32 DH lines (23.70%) were resistant only to FER and GER, respectively. The remaining 60 DH lines (44.4%) were categorised as intermediately resistant.

Table 3. Summary of detected quantitative trait loci for *Fusarium* resistance-related traits in maize using the interval mapping, including genomic position, logarithm of the odds score (genome wide) and multiple QTL mapping) effect size and parental origin.

Trait	QTL ID	Chr	QTL interval	QTL peak marker	Position (cM)	LOD (GW*)	R ²	Additive	Origin
GERY1	<i>qGER1</i>	2	4576419-25586718	2392119	83.68	2.0 (3.0)	9.1	0.562370	CML390
GERY2	<i>qGER2</i> [#]	10	2460242-9691275	4592681	123.55	2.0 (3.0)	9.6	-0.375303	R2565Y
DONY1	<i>qDON3</i> [#]	9	9682572-7047806	2472428	19.0	2.6 (2.6)	11.8	11.2800	CML390
DONY2	<i>qDON4</i>	5.1	2624301-24024952	4589685	45.50	2.2 (2.6)	9.1	-8.65550	R2565Y
FERY2	<i>qFER5</i>	4	25947833-7050137	4764743	268.10	2.0 (2.7)	8.0	0.260317	CML390 Y

Y1 = Year 1, Y2= Year 2, DON = Deoxynivalenol, ZEA = Zearalenone, Chr = Chromosome, LOD = Logarithm of the odds, R² = Variance, Additive calculated as (Mean of Parent A -- Mean of Parent B)/2, *Genome-wide (GW) LOD significance threshold determined through permutation. [#]Multiple QTL analysis only validated the QTL detected for GERY2 (*qGER2*) and DONY1 (*qDON3*).

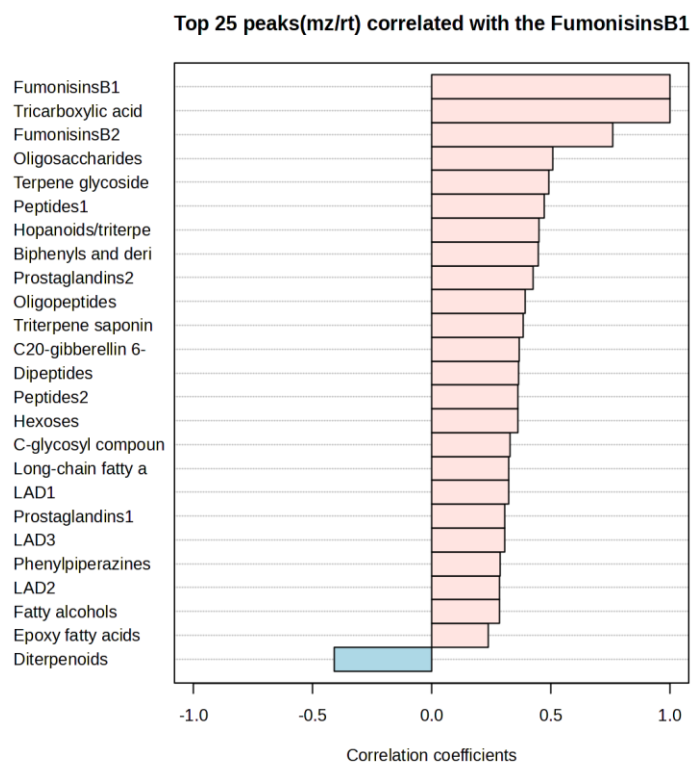


Figure 8. Metabolite classes showing the strongest correlations with fumonisin B1 are displayed. Positive correlations (pink) indicate co-accumulation, whereas negative correlations (blue) represent inverse relationships. Fumonisin isoforms, terpenoids, and peptide-related compounds showed strong positive correlations with fumonisin B1, while diterpenoids were inversely associated.

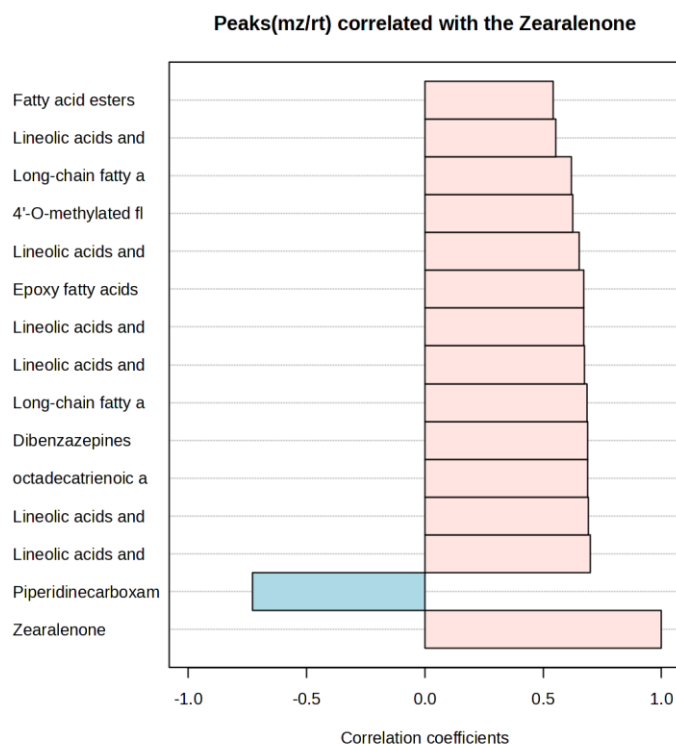


Figure 9. Correlation network of metabolites associated with zearalenone (RT/*mz*: 7.143/317.13702). The bars illustrate the strength and direction of the correlation coefficients between zearalenone and the respective metabolite. Piperidine carboxamides showed a strong negative correlation ($r = -0.73$), whereas linoleic acid derivatives, octadecatrienoic acids, epoxy and long-chain fatty acids, fatty acid esters, 4'-O-methylated flavonoids, and dibenzazepines exhibited positive correlations ($r = 0.54 - 0.70$).

6. Discussion *(provide a detailed discussion of project outcomes in terms of the original hypothesis, results obtained, existing knowledge, and impact on the maize industry)*

Maize is a cornerstone food and feed crop in Africa, particularly in Southern Africa, where it contributes substantially to food security and rural livelihoods. However, maize production is threatened by several diseases, among which Fusarium ear rot (FER), caused primarily by *Fusarium verticillioides*, and Gibberella ear rot (GER), caused mainly by *Fusarium boothii*, are particularly important. Beyond causing yield losses and reducing grain quality, these pathogens contaminate maize grain with harmful mycotoxins, including fumonisins (FUM), deoxynivalenol (DON), and zearalenone (ZEA). These toxins pose significant risks to human and animal health and create a major challenge for agricultural systems that must simultaneously maximize grain production while ensuring food safety. Although breeding resistant maize varieties remains the most sustainable and environmentally responsible management strategy, progress has been slow because resistance to both ear rot diseases and mycotoxin accumulation is quantitatively inherited, controlled by numerous genes with small effects, and strongly influenced by environmental conditions. This project, therefore, aligned well with the **MT strategic objective 4** “To support the development of practical, affordable, and environmentally sound methods to manage toxigenic fungi in maize, with particular emphasis on the introduction of resistance in local

maize cultivars and **objective 5** “To support the development of sound mycotoxin risk management practices in the maize supply chain to ensure the delivery of safe products to the consumer.

This study sought to improve understanding of the phenotypic, genetic, and biochemical factors underlying resistance to FER, GER, and their associated mycotoxins in maize adapted to South African production environments. A doubled haploid (DH) mapping population was developed from contrasting parental lines and evaluated across two seasons following artificial inoculation with both pathogens. The results demonstrated the complexity of resistance to these diseases and toxins. Disease severity and mycotoxin accumulation varied substantially depending on the inoculation method and environmental conditions experienced during each season. These findings highlight the importance of considering both disease symptoms and toxin accumulation during resistance evaluations, as visual disease severity alone does not reliably predict mycotoxin contamination.

The DH population was subsequently used to identify genomic regions associated with resistance through quantitative trait locus (QTL) mapping. Five putative QTL—qGER1, qGER2, qDON3, qDON4, and qFER5—were initially identified using interval mapping approaches. The qDON3 remained significant when evaluated using multiple-QTL mapping, reflecting the small effect sizes typical of quantitative resistance traits. The inconsistency of QTL detection across seasons, and the observation that some QTL originated from different parental sources under different environmental conditions, highlighted the challenges associated with mapping complex traits. Factors such as phenotyping methodology, environmental variation, marker density, chromosome fragmentation, and population integrity all influenced QTL detection and stability. This work represents the first reported effort to identify QTL associated with FER, GER, and mycotoxin resistance in South African-relevant maize germplasm, providing a valuable foundation for future genetic studies and marker-assisted breeding initiatives.

Recognizing that genetic mapping alone provides limited insight into the biological mechanisms responsible for resistance, the study incorporated an untargeted metabolomics approach to investigate the biochemical responses of resistant and susceptible maize lines following pathogen infection. Resistant (CML444) and susceptible (R2565Y) maize lines were sampled before inoculation and at multiple time points following infection to characterize both constitutive and induced defence responses.

The metabolomics analyses revealed clear pathogen-specific and genotype-specific responses. Infection by *F. verticillioides* influenced a broad range of metabolite classes, including terpenoids, phenylpropanoids, peptides, and lipid-related compounds, whereas *F. boothii* infection was associated predominantly with lipid and fatty-acid-derived metabolites. Importantly, resistant and susceptible

genotypes exhibited distinct metabolic reprogramming patterns over time, demonstrating that resistance involves dynamic biochemical adjustments rather than static defence mechanisms.

Among the metabolite classes identified, lipid- and fatty-acid-derived compounds emerged as particularly important components of maize defence responses. These findings support and extend previous international studies linking lipid metabolism, oxylipins, glycerophospholipids, and related pathways to *Fusarium* resistance and mycotoxin accumulation. The present study demonstrated that these metabolic responses are relevant not only to FUM-producing pathogens but also to resistance against *F. boothii* and ZEA accumulation, suggesting a broader role for lipid-mediated defence mechanisms in maize-*Fusarium* interactions.

Several metabolite classes also showed potential as biomarkers of resistance. Diterpenoids were strongly and negatively associated with FUM accumulation and consistently differentiated resistant and susceptible genotypes, suggesting their potential utility as indicators of resistance. In contrast, a metabolite class structurally similar to piperidine carboxamides was negatively associated with zearalenone accumulation and appeared to differ between resistant and susceptible lines during early infection stages. However, inconsistent patterns later in infection prevented definitive conclusions regarding its role in resistance and warrant further investigation.

Overall, the metabolomics component demonstrated the value of integrating biochemical analyses with traditional genetic mapping approaches. Metabolomics provides a functional bridge between genotype and phenotype, enabling the identification of biochemical pathways and metabolites that contribute to resistance. Such information has considerable potential for developing metabolite-assisted selection strategies and identifying early biomarkers of resistance that could reduce reliance on costly and labour-intensive disease screening trials.

Collectively, this study provides important new knowledge and resources for maize improvement programmes in South Africa and the broader sub-Saharan African region. By combining phenotypic assessments, QTL mapping, and metabolomics analyses, the research advances understanding of the genetic and molecular basis of resistance to FER, GER, and their associated mycotoxins. The findings emphasize the importance of robust phenotyping and integrated multi-omics approaches for dissecting complex resistance traits. Ultimately, this work contributes to the long-term goal of developing high-yielding maize varieties with durable resistance to ear rot diseases and mycotoxin contamination, thereby improving grain quality, protecting consumer health, and enhancing food security throughout the region.

7. Scientific Outputs (give full references and indicate poster or oral presentation for conference contributions)

Scientific papers:	Two papers are currently in preparation for peer-review publication.
Technical reports:	4 MT reports; 3 NRF reports
Articles in industry magazines:	None
Conference contributions:	Shaikh, A., Prins, R., Viljoen, A. and Rose, L.J. 2026. Integrative analysis of genetic and metabolic resistance mechanisms to Fusarium and Gibberella ear rot of maize. 54th Southern African Plant Pathology Congress. Umhlanga, 18-22 January 2026. (Oral). Rose, L.J., Janse van Rensburg, B. and Viljoen, A. 2025. Safeguarding and future-proofing South African grains against mycotoxins. National Grain Research Program Annual Research Day, Northwest University, 18-20 March 2025. Shaikh, A., Prins, R., Viljoen, A. and Rose, L.J. 2025. From Genes to Fields: Mapping Resistance in Maize to Multiple Ear Rot Fungi. National Grain Research Program Annual Research Day, Northwest University, 18-20 March 2025. Rose, L.J. Van Coller, G.J., Janse van Rensburg, B., A. Shaikh and Viljoen, A. 2025. Managing mycotoxins in South African grain: ‘‘A continuously moving target’’. Gordon Research Conferences Meeting of Mycotoxins and Phycotoxins, Stonehill College in Easton, Massachusetts United States, 15-20 June 2025. (Oral). Rose, L.J., Van Coller, G.J., Shaikh, A., Flett, B.C., Janse van Rensburg, B. and Viljoen, A. 2025. Sustainable mycotoxin management: Leveraging plant resistance knowledge. 7th International Conference of Mycotoxicology and Food Security, Hangzhou, China, 17-20 November 2025. (Oral). Shaikh, A., Prins, R., Viljoen, A. and Rose, L.J. 2025. Multi-omics of maize resistance to multiple ear rot pathogens. 7th International Conference of Mycotoxicology and Food Security, Hangzhou, China, 17-20 November 2025. (Oral). Shaikh, A., Prins, R., Viljoen, A. and Rose, L.J. 2023. Quantitative trait loci for resistance to maize ear rots and mycotoxins. 53rd Southern African Plant Pathology Congress. Golden Gate Park, 21-25 January 2024. (Poster presentation).
Human capacity development:	1 PhD student (Ayesha Shaikh) graduated in March 2026 and is currently employed at Corteva

	AgriSciences. Dr Shaikh was also a MT bursary recipient.
Technology transfer:	The markers close to the mapped QTLs need to be tested. Once achieved, these will be publicly available to breeders to improve selection of resistance maize material.
Other outputs (Procedures, Methods, Databases, etc):	Phenotypic and genotypic database of the DH populations which can be used to study various other aspects. Database of metabolites associated with FER- and GER-associated mycotoxins which can be further validated by gene knock-out technology.

8. Personnel / Management / Risk factors that influenced progress and lessons learned *(if applicable)*

- The progress of the project was severely impacted by the loss of the primary breeding partner during the initial period of this project. Finding an industry partner to assist with trial areas and maintenance took longer than anticipated due to legal and contractual agreements that needed to be finalised. This could not have been anticipated at the start of the project and had a profound impact on the execution and timeline of the project.
- The collaboration with experienced researchers particularly in the field of plant genetics was enriching and resulted in a thorough and expanded interrogation of plant resistance in the SA lines that was not limited to only genetics but also at a metabolic level.

9. Conclusions and Comments you wish to share with the Trust

None

10 Signatures of Project Leader and Authorized Manager



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Altus Viljoen
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1 July 2026