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Quantitative Trait Loci for Resistance to *Stenocarpella maydis* and *Fusarium graminearum* Cob Rots in Tropical Maize

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Stenocarpella maydis *and* Fusarium graminearum *are the predominant species causing maize (*Zea mays *L.) cob rots in the tropics and sub-Saharan Africa. Developing varieties resistant to cob rots is an alternative strategy that is practical and provides better insurance for small-scale farmers. The subjectivity of scoring and the varying virulence responses of these pathogens to environmental conditions make selection for resistance difficult. The objectives of this study were to map quantitative trait loci (QTL) associated with resistance to* S. maydis *and* F. graminearum *and to analyze the possibilities of utilizing these QTL for marker-assisted selection (MAS). Stable QTL mapped were Fg_4,2 (r^2 = 0.22) and Sm_4,1 (r^2 = 0.16) associated with resistance to* F. graminearum *and* S. maydis, *respectively, on chromosome 4. Another QTL associated with resistance to* F. graminearum *was Fg_5 (r^2 = 0.30) on chromosome 5. A QTL with pleiotropic effect was detected on chromosome 1, 22 cM from umc1269 marker (r^2 values of 13% and 22% for resistance to* S. maydis *and* F. graminearum, *respectively). Additive effects ranged from -0.14 to -0.35 for associated QTL of both pathogens, and all mapped QTL were more than 5 cM from the nearest molecular marker utilized in the study. Therefore, there is need to utilize the maize genomic map to identify and test several markers, < 5 cM, near the detected QTL, in order to locate more reliable molecular markers for utilization in MAS.*

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INTRODUCTION

Stenocarpella maydis and Fusarium graminearum are the predominant maize cob rot pathogens in the temperate and tropical regions of the world, causing yield losses and reducing grain quality as a result of mycotoxin contamination (Kapindu et al. 1999; Bigirwa et al. 2007). Deployment of resistant genotypes is the most cost-effective management strategy for cob rots. It is practical, economical, and provides better insurance for the small-scale farmers, especially in resource-poor farming. Breeding of tolerant genotypes is, however, compounded by reliability of selection mechanisms, mainly because of weather dependency of pathogenesis and unreliability of inoculation methods. Thus to obtain reliable results, precise timing of artificial inoculation is essential (Chambers 1988; Ali et al. 2005). In addition, the subjectivity of assessment and dependence of pathogenesis on environmental conditions make selection for resistance difficult (Bertland et al. 2007). As such, development of alternative and more reliable screening approaches is worthwhile.

Completion of sequencing of the maize genome and the development of a wide array of molecular markers today open new frontiers for more accurate and in some cases cost-effective selection systems based on marker-assisted selection (MAS) (Campos et al. 2004; Bertland et al. 2007). Additionally, molecular markers when identified can enable researchers to dissect quantitative traits into discrete components, allowing pyramiding of beneficial alleles (Ribaut and Raggot 2007). Marker-assisted selection is most suited for introgression of well-characterized loci. For many traits, such loci are inherited quantitatively and thus encoded by QTL. Eighteen QTL associated with resistance to *F. graminearum* and accounting for between 7% and 35% of phenotypic variation have been mapped in temperate maize genotypes for resistance to *F. graminearum* (Ali et al. 2005). Only one QTL (on the interval, umc0282-umc1155) on chromosome 5 was identified consistently in more than one environment. Little is known about mapping for QTL linked for resistance to *S. maydis*. The reliability of estimates of QTL position and effect in cases when the analysis is based on biparental crosses is sometimes low. This is because such QTL may often represent only a small fraction of the phenotypically relevant variation in a species and are not consistent across mapping populations (Holland 2007). Moreover, detection of QTL and accurately estimating their effects are more difficult for traits of low heritability (Ribaut and Raggot 2007). There has been limited effort to map resistance loci to multiple pathogens such as *S. maydis* and *F. graminearum*. Yet dual infection of maize cobs by both pathogens is common, and breeders

often face a challenge of selecting against both pathogen. In this study, simple sequence repeat (SSR) molecular markers were targeted to specific regions of the seven maize chromosomes identified by Wisser et al. (2006) as linked with QTL for cob and stalk rots when multiple pathogens were used. Biparental crosses were used to map QTL associated with resistance to *S. maydis* and *F. graminearum* in tropical accession. The objectives were to map QTL associated with resistance to *S. maydis* and *F. graminearum* and to investigate the possibilities of utilizing these QTL for marker-assisted selection.

MATERIALS AND METHODS

Experimental Design and Germplasm Used

The phenotypic evaluation was carried out in three sites in Uganda: Burindi (1° 25′ N, 31° 21′ E; altitude 1140 m), Masaka (0° 20′ S, 31° 44′ E, altitude 1315 m), and Namulonge (0° 32′ N, 32° 35′ E; altitude 1150 m). All three environments experience a bimodal rainfall pattern, with the first season, A, from March to August and the 2nd season, B, from September to December/January. The evaluation was done on 123 $F_{2:3}$ maize families obtained from a biparental cross between resistant line (WL118-10) and susceptible line (CZL- 8) to both *Fusarium graminearum* and *Stenocarpella maydis*. The 123 F_2 plants were selfed and the seeds planted as $F_{2:3}$ families following a randomized complete-block design with two replications. Plants were spaced at 75 cm × 30 cm in 5 m long rows with 17 plants per row. Two seeds were initially planted per hill, but seedlings were subsequently thinned to one plant per hill, 4 weeks after germination. Standard cultural practices, such as weeding and appropriate fertilizer applications, were followed in all the experiments.

Pathogen Culture, Inoculation and Assessment of Disease

Inoculum preparation and inoculation of the test genotypes was done according to Tembo et al. (2013). At least seven plants were inoculated for each treatment per plot. Each inoculation treatment per plot was harvested separately, plot number noted, and the inoculated cobs bulked. For each of the two treatments, a visual estimate of disease severity, assessed as percentage of the colonized cob from the point of infection, was made and the mean severity rating computed. The qualitative scale for *S. maydis* severity ratings was 1 = 0%–25% cob rotted; 2 = 26%–50%; 3 = 51%–75%; 4 = 76%–99%; and 5 = 100% (completely rotten) (Kapindu et al. 1999). For *F. graminearum,* the scale used was 1 = 1%–% cob rotted; 2 = 3%–10%; 3= 11%–25%; 4 = 26%–50%; 5 = 51%–75%; and 6 = 76%–100% cob rotted (Reid et al. 1992). Inspection on the use of percentage data and further transformation

revealed violation of analysis of variance (ANOVA) preconditions, accounting for the use of qualitative scale data in subsequent phenotypic analysis. With qualitative scale data ANOVA preconditions were met.

Genotyping and Construction of a Linkage Map

DNA for genotyping the mapping F_2 population was obtained from young leaf samples, two to three weeks old (advanced from a cross of WL 118-10 and CZL-8) and then tagged. DNA was extracted from the ground leaf material using the cetyltrimethylammonium bromide (CTAB) method (Hoisington et al. 1994). The seed for each test plant was later planted ear to row to obtain corresponding $F_{2:3}$ families for phenotypic evaluation.

Fifty-six SSR primer pairs were purchased from University of Cape Town, Department of Molecular and Cellular Biology (Cape Town, South Africa). SSR marker names and primer sequence information were obtained from SSR maize databases available at http://www.maizegdb.org/ssr.php. These were selected from targeted regions of the maize genome linked to QTL for resistance to cob and stalk rots (Wisser et al. 2006). The primers were used as part of the PCR reaction mixture. The final concentrations of reaction components were as follows: 0.2 μM each of SSR forward and reverse primers, 1× PCR buffer, 2.0 mmol MgCl2/L; 0.2 mmol/L each of dATP, dCTP, dGTP, and dTTP; 0.16 U *Taq* polymerase (BioLabs); and 30 ng genomic DNA and distilled sterile water to a total volume of 20 μL. The PCR conditions and cycling profiles for PCR were based on a "touchdown" protocol (Ali et al. 2005). Initial testing involved screening for polymorphism between the parental DNA (WL-118-10 and CZL-8), and only polymorphic SSR markers were subsequently used for genotyping the 123 tagged F_2 plants. The PCR products were separated on a 6% horizontal polyarcylamide gel electrophoresis (Southern 2009).

The linkage map was constructed with QTL (linkage/ association) in GenStat 14 using the sampling clustering tool (Payne et al. 2011). Assignment of linkage groups to the respective chromosomes was based on similarity to the intermated B73 × Mo17 (IBM) corn map (http://www.maizegdb.org).

Data Analysis

Analysis of phenotypic variance was performed using GenStat 14 to assess if there were significant differences in reaction to *F. graminearum* and *S. maydis* among the $F_{2:3}$ family genotypes. To detect QTL, composite interval mapping (CIM) was performed using QTL Cartographer version 2.5_009 (Wang et al. 2011). Initially, genotypic and phenotypic data were imported into QTL Cartographer from excel data sheet. Analysis was then performed using CIM and was set at the likelihood of odds (LOD) score threshold of 2.5 with the walking distance of one centi-morgan and 500 permutations. No QTL with pleiotropic effects was detected at LOD score of 2.5. The LOD

score was then adjusted to 2 to increase the chances of further detecting any QTL with pleiotropic effects for resistance to *F. graminearum* and *S. maydis*. The degree of dominance for the detected QTL was computed as d/[a] (Rabiei 2007), where d is the dominance gene effect and [a] is the absolute value of the additive gene effect of the putative QTL.

RESULTS

Reaction of Genotypes to *S. maydis* and *F. graminearum*

There were significant differences among genotypic reactions to *S. maydis* and *F. graminearum* within and across locations (Table 1). The genotype × location interaction for both pathogens was significant but of a lesser magnitude than the genotypic effects.

Genetic Linkage Map Construction and QTL Detection

Fourteen SSR markers, which were polymorphic between the test parents (WL 118-10 and CZL-8), were used to generate a linkage map (Figure 1). A map with four linkage groups was constructed and had a total of 12 SSR markers. Polymorphic markers umc2375 and umc2222, located on chromosomes 6 and 7, respectively, were not accounted for on the linkage map as they did not associate with any of the other markers utilized. Chromosome 4 and 5 had four markers each, where as chromosomes 1 and 3 had two markers each.

TABLE 1 Mean squares for genotype-location reaction to challenge infection of an $F_{2:3}$ population derived from a cross between two parents WL 118-10 and CZL-8 by *F. graminearum* and *S. maydis* in three locations of Uganda at Namulonge, Masaka, and Bulindi during the second season of 2011

Location	Source	Df	*F. graminearum*	*S. maydis*
Bulindi	Rep	1	0.15	0.02
	Genotype	122	1.73***	1.88***
	Error	122	0.63	0.7
Masaka	Rep	1	0.02	1.36
	Genotype	122	1.58***	1.71***
	Error	122	0.81	0.91
Namulonge	Rep	1	0.42	0.21
	Genotype	122	1.41***	1.23**
	Error	122	0.81	0.82
Across locations	Location	2	13.07**	5.45
	Rep	3	0.19	0.55
	Genotype	122	2.70***	2.34***
	Genotype × Location	244	1.03**	1.26***
	error	366	0.74	0.81

, * significant at $P \leq 0.01$ and $P \leq 0.001$, respectively.

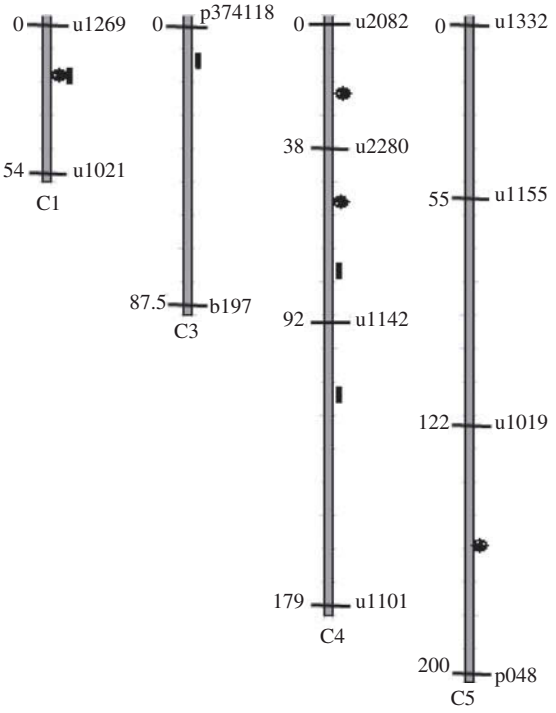

FIGURE 1 Linkage map of 12 utilized SSR markers. Distances between markers are in cM. Circular and rectangular shape depicts approximate positions where QTL associated with *F. graminearum* and *S. maydis,* respectively, were detected. Similar positioning of a circular and rectangular shape on chromosome 1 depicts a position where QTL for resistance to *S. maydis* and *F. graminearum* was detected. Note: u= umc, b= bnlg and p= phi.

Single Marker Linkage and QTL Detection

The single-marker analysis revealed that the markers that accounted for the largest fraction of the phenotypic variations across locations were marker umc2280 for *F. graminearum* and umc1142 for *S. maydis,* explaining 16.5% and 24.5% of the phenotypic variation, respectively (Table 2). The results indicated that linkage to *S. maydis* for marker umc1269, umc1142, and umc2222 had the most consistent results across all locations for *S. maydis* infection, with at least an r^2 of 8% in each location. The SSR umc2280 had the most consistent outcome for *F. graminearum* reaction across locations, with an r^2 value of at least 6% for each location (Table 3). The total phenotypic variation explained by all the markers across all locations for *F. graminearum* and *S. maydis* was 21% and 39%, respectively. The total number of QTL mapped for disease reaction in Namulonge, Bulindi, and Masaka as computed by CIM was 2, 8, and 4, respectively (Tables 3–5). Across locations a total of six QTL were mapped (Table 6). An extra QTL for resistance to *S. maydis* and *F. graminearum* was detected on chromosome 1 at a position

TABLE 2 Phenotypic variation explained (r^2) by each marker in each and across locations for *F. graminearum* and *S. maydis* (analyzed by single-marker analysis) evaluated in second season of 2011

		F. graminearum				*S. maydis*			
Chrm.	SSR Marker	Across Locations	Namulonge	Masaka	Bulindi	Across Locations	Namulonge	Masaka	Bulindi
1	umc1269	12.41****	9.4***	3.14	8.68***	20.67****	8.8***	10.49***	13.59****
1	umc1021	4.02*	2.04	1.32	2.34	7.8**	2.54	3.58*	5.22*
3	phi374118	11.17***	6.46**	11.57***	2.31	15.46****	5.96**	10.06***	5.84**
3	bnlg197	2.66	3.06	0.25	2.08	4.78*	1.06	0.16	8.94***
4	umc2082	3.65*	2.23	1.79	2.73	3.76*	1.53	2.28	2.25
4	umc2280	16.56****	13.37****	6.18**	8.86**	13.24****	11.37***	4.49*	5.19*
4	umc1142	14.52****	15.32****	2.99	7.61**	24.53****	16.82****	9.72***	10.27***
4	umc1101	5.17*	4.09*	0.79	4.03*	8.29**	3.31*	0.8	10.64***
5	umc1332	0.96	0.86	0.11	0.84	2.14	0.08	0	6.2**
5	umc1155	0.02	0.25	0	0.22	1.53	0.68	0.8	1.04
5	umc1019	6.52**	5.71**	2.32	3.38*	4.63*	1.34	2.62	2.84*
5	phi048	4.26*	0.91	5.16**	2.64	7.02***	3.11*	1.43	6.68**
6	umc2375	2.52	4.25*	0.13	1.29	1.8	2.04	0.1	1.14
7	umc2222	9.67***	8.1**	4.38*	4.43*	20.00****	11.65***	9.73***	8.18**

*, **, ***, **** r^2 significant at P = 0.05, P = 0.01, P = 0.001 and P = 0.0001 respectively.

TABLE 3 QTL associated with resistance to *F. graminearum* and *S. maydis* derived from a biparental cross between the parents WL 118-10 (resistant) and CZL-8 (susceptible) evaluated in Namulonge in 2011 (B) season, September to January cropping season

		F. graminearum							*S. maydis*					
Marker Interval	QTL[a]	pos.cM[b]	LOD[c]	r^{2d}	a[e]	d[f]	d/IaI[g]	QTL[a]	pos. cM[b]	LOD[c]	r^{2d}	a[e]	d[f]	d/IaI[g]
umc2280 - umc1142	FgN_4,1	26.4	4	0.22	−0.45	−0.11	−0.24	SmN_4,1	46.	4.4	0.18	−0.57	0.48	0.84
umc1142 - umc1101								SmN_4,2	6.6	4.3	0.19	−0.56	−0.51	−0.91

[a]QTL have been named from the trait abbreviation, followed by locational abreviation and then by the chromosome number where detected. Where more than one QTL was detected on a chromosome for a particular trait, the second number is added to show the order and the closest to zero gets position 1. Example SmN_4,2- means the QTL associated with resistance to *S. maydis* was mapped for disease reaction in Namulonge on chromosome 4 and it is in the second position (in terms of distance from 0 on linkage map) to another detected QTL on the same chromosome.
[b]The positions of the QTL are measured from the distance of the marker listed first for that interval.
[c]Logarithm of odds likelihood equivalent to $-Log_{10}$ likelihood.
[d]Amount of phenotypic variance explained by the detected QTL.
[e]Additive gene effect of detected QTL.
[f]Dominance gene effect of detected QTL.
[g]Degree of dominance.

TABLE 4 QTL associated with resistance to *F. graminearum* and *S. maydis* derived from a biparental cross between the parents WL 118-10 (resistant) and CZL-8 (susceptible) evaluated in Bulindi in 2011 (B) season, September to January cropping season

	F. graminearum							*S. maydis*					
Marker Interval	QTL[a]	pos. cM[b]	LOD[c]	r^{2d}	a[e]	d[f]	d/IaI[g]	QTL[a]	pos. cM[b]	LoD[c]	r^{2d}	a[e]	d[f]	d/IaI[g]
umc1269-umc1021	FgB_1	26	6	0.48	−0.21	−0.23	−1.1							
phi374118 - bnlg197	FgB_3	46.5	4.3	0.39	−0.16	0.31	1.94	SmB_3	47	3.1	0.45	−0.48	0.86	1.79
umc2280 - umc1142	FgB_4,1	26.2	5.7	0.47	−0.32	−0.00	0	SmB_4,1	32	4.5	0.38	−0.12	−0.60	−5
umc1142 - umc1101	FgB_4,2	41.4	4	0.46	−0.10	−0.26	−2.6	SmB_4,2	41.4	6	0.47	−0.42	0.22	−0.52
umc1019 - phi048	FgB_5	39.6	5.4	0.38	−0.24	0.01	0.04							

[a]QTL have been named from the trait abbreviation, followed by locational abreviation and then by the chromosome number where detected. Where more than one QTL was detected on a chromosome for a particular trait, the second number is added to show the order and the closest to zero gets position 1. Example SmN_4,2- means the QTL associated with resistance to *S. maydis* was mapped for disease reaction in Namulonge on chromosome 4 and it is in the second position (in terms of distance from 0 on linkage map) to another detected QTL on the same chromosome.

[b]The positions of the QTL are measured from the distance of the marker listed first for that interval.

[c]Logarithm of odds likelihood equivalent to -Log_{10} likelihood.

[d]Amount of phenotypic variance explained by the detected QTL.

[e]Additive gene effect of detected QTL.

[f]Dominance gene effect of detected QTL.

[g]Degree of dominance.

TABLE 5 QTL associated with resistance to *F. graminearum* and *S. maydis* derived from a biparental cross between the parents WL 118-10 (resistant) and CZL-8 (susceptible) evaluated in Masaka in 2011 (B) season, September to January cropping season

		F. graminearum							*S. maydis*					
Marker Interval	QTL[a]	pos. cM[b]	LOD[c]	r^{2d}	a[e]	d[f]	d/IaI[g]	QTL[a]	pos. cM[b]	LOD[c]	r^{2d}	a[e]	d[f]	d/IaI[g]
phi374118 - bnlg197	FgM_2	35.2	2.8	0.33	−0.42	0.75	1.78	SmM_3	18.2	4.3	0.47	−0.59	1.53	2.59
umc2280 - umc1142								SmM_4	30.3	2.6	0.42	−0.21	1.30	6.20
umc1019 - phi048	FgM_5	31.2	2.9	0.28	−0.16	−0.53	−3.31							

[a]QTL have been named from the trait abbreviation, followed by locational abreviation and then by the chromosome number where detected. Where more than one QTL was detected on a chromosome for a particular trait, the second number is added to show the order and the closest to zero gets position 1. Example SmN_4,2- means the QTL associated with resistance to *S. maydis* was mapped for disease reaction in Namulonge on chromosome 4 and it is in the second position (in terms of distance from 0 on linkage map) to another detected QTL on the same chromosome.

[b]The positions of the QTL are measured from the distance of the marker listed first for that interval.

[c]Logarithm of odds likelihood equivalent to -Log_{10} likelihood.

[d]Amount of phenotypic variance explained by the detected QTL.

[e]Additive gene effect of detected QTL.

[f]Dominance gene effect of detected QTL.

[g]Degree of dominance.

TABLE 6 QTL associated with resistance to *F. graminearum* and *S. maydis* derived from a biparental cross between the parents WL 118-10 (resistant) and CZL-8 (susceptible) evaluated across three locations (Namulonge, Masaka and Bulindi) in 2011 (B) season, September to January cropping season

	F. graminearum						*S. maydis*						
Marker Interval	QTL[a]	pos. cM[b]	LOD[c]	r[2d]	a[e]	d[f]	d/IaI[g]	QTL[a]	pos. cM[b]	LOD[c]	r[2d]	a[e]	d[f]	d/IaI[g]
umc1269-umc1021	Fg_1	22	2.4	22	−0.10	−0.21	−2.10	Sm_1	22	2.0	13	−0.19	0.04	0.21
phi374118 - bnlg197								Sm_3	13	5.4	32	−0.35	0.73	2.09
umc2082 - umc2280	Fg_4,1	22	3.9	20	−0.25	−0.15	−0.60							
umc2280 - umc1142[L]	Fg_4,2	18	3.7	22	−0.25	−0.11	−0.44	Sm_4,1	30	2.8	16	−0.24	−0.00	0
umc1142 - umc1101								Sm_4,2	32	2.6	19	−0.31	0.19	0.26
umc1019 - phi048[l]	Fg_5	39	3.9	30	−0.14	−0.30	−2.14							

Note: L-QTL identified between two marker intervals to be stable in at least two locations.

[a]QTL have been named from the trait abbreviation, followed by locational abreviation and then by the chromosome number where detected. Where more than one QTL was detected on a chromosome for a particular trait, the second number is added to show the order and the closest to zero gets position 1. Example SmN_4,2- means the QTL associated with resistance to *S. maydis* was mapped for disease reaction in Namulonge on chromosome 4 and it is in the second position (in terms of distance from 0 on linkage map) to another detected QTL on the same chromosome.

[b]The positions of the QTL are measured from the distance of the marker listed first for that interval.

[c]Logarithm of odds likelihood equivalent to -Log_{10} likelihood.

[d]Amount of phenotypic variance explained by the detected QTL.

[e]Additive gene effect of detected QTL.

[f]Dominance gene effect of detected QTL.

[g]Degree of dominance.

22 cM distant from the first marker, umc1269. Two mapped QTL across locations, Fg_4,2 and Sm_4,1, positioned between interval umc2280-umc1142, were identified to be stable for linkage to *F. graminearum* and *S. maydis*, respectively in at least two locations (Tables 3–5). Another mapped QTL, Fg_5, positioned between umc1019-phi048, was also identified to be stable in two locations (Tables 3–5).

DISCUSSION

Mapping of QTL linked to trait of interest is a crucial step toward employing marker-assisted selection in breeding for any trait. In this study, QTL were successfully mapped for *S. maydis* and *F. graminearum* cob rots in maize. A total of seven QTL were mapped across locations, three associated with *F. graminearum,* the other three with *S. maydis,* and one associated with both pathogens (Table 6). Most of these mapped QTL were on chromosome 4, which is associated with resistance to cob rot pathogens and many other diseases of maize (Wisser et al. 2006). It therefore appears that linkage group 4 holds many loci that have coevolved to adapt maize plants for resistance to biotic stresses. Indeed the early studies on monocrops demonstrate that at disease resistance loci, homologies of up to 70% may occur as was reported for rice, maize barley, and wheat (Spielmeyer et al. 1998).

The amount of phenotypic variations explained by the mapped QTL ranged from 13% to 32%. The additive effects of all QTL in each and across locations were negative. Across locations, the additive effects of associated QTL to the two pathogens ranged from -0.14 to -0.27 for *F. graminearum* and -0.24 to -0.35 for *S. maydis*. The finding on negative additive effects implies that the substitution effect of a non-favorable allele (susceptible) with a favorable allele (resistant) reduced severity at that locus. A QTL with probable pleiotropic effects (associated with resistance to both *S. maydis* and *F. graminearum*) was detected on chromosome one 22 cM from marker umc1269, and this explained 13% and 22% phenotypic variation for resistance to *S. maydis* and *F. graminearum*, respectively. However, the fact that the degree of dominance (for QTL with pleiotropic effects) is negative for *F. graminearum* and positive for *S. maydis* may make employing of the QTL for dual resistance breeding to both pathogens difficult. In addition, the mapped QTL was not stable in any of the locations.

While further studies are needed to validate the mapped QTL in other populations, much emphasis should focus on Fg_4,2, Sm_4,1, and Fg_5, which were identified to be stable in at least two environments (Tables 3–6). The degree of dominance for mapped QTL, Fg_4,2, Sm_4,1, and Fg_5, was -0.44, 0, and -2.14, respectively. Only Fg_5 was a major QTL ($r^2 = 0.30$) and it exhibited negative overdominance, suggesting that favorable alleles at this locus contributed more toward resistance above the mean score of the resistant parent. Previous studies on *F. graminearum* mapped only one

stable QTL on chromosome 5 located in bin 5.05 when compared with the intermated B73 × Mo17 (IBM) (Ali et al. 2005). In this study, a similar stable QTL, Fg_5, was mapped between bin 5.06 and 5.07 when compared with the intermated B73 × Mo17 (IBM) maize map (http://www.maizegdb.org). Therefore, there is a possibility that mapped QTL Fg_5 and the previously stable mapped QTL (Ali et al. 2005) are the same. The difference in mapped positions may be attributable to different materials used in the study (Collard et al. 2005). Marker-assisted selection works best when mapped QTL are tightly linked to the markers and the tighter the linkage, the higher the probability for a marker to be inherited together with the detected QTL. For the marker to be efficient, a distance of less than 5 cM between the marker and the QTL is recommended (Collard et al. 2005; Bertland et al. 2007). In this study, the closest marker to a mapped stable QTL was umc2280, which was positioned at 18 cM to the left of Fg_4,2 QTL ($r^2 = 0.22$). Fine mapping of linked QTL is therefore essential to help identify closer markers (< 5 cM) for utilization in MAS.

CONCLUSION

While this study provides useful information on approximate position and effects of mapped QTL, especially stable ones (QTL Fg_4,2, Sm_4,1, and Fg_5) associated with resistance to *F. graminearum* or *S. maydis*, the linked markers are not close enough to the QTL to be used effectively in MAS. As such, there is need to fine map these QTL using say single nucleotide polymorphism (SNP) as the next step. In the interim, further fine mapping using SSRs may help identify markers that will facilitate the introgression of these detected QTL for utilization in MAS. Additionally, there is a need to further validate these putative QTL in other populations. Such efforts will allow for detection and verification of additional QTL that could be of value for MAS.

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