

Quantitative Trait *loci* (QTL) Identification for Resistance to Mal de Río Cuarto Virus (MRCV) in Maize Based on Segregate Population

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Abstract: Mal de Río Cuarto virus (MRCV) is one of the most devastating pathogens in maize (*Zea mays* L.) causing serious yield loss in susceptible cultivars in Argentina. An effective solution to control the virus is to use resistant genes to improve the behavior of susceptible genotypes. The goals of this work are to identify simple sequence repeats (SSR) linked to quantitative trait *loci* (QTL), for resistance to MRCV, and to validate QTL found in previous research with different genetic backgrounds. Two hundred and eight F_{2:3} families derived from a cross between a susceptible inbred line, B73, and a resistant inbred line, LP116, were evaluated across three environments in the disease-endemic region. Disease incidence (INC), disease severity (SEV) and disease severity index (DSI) were employed to evaluate F_{2:3} families. Based on this segregant population, ninety-eight simple sequence repeat (SSR) markers selected from 173 markers were used. The means of all measured traits followed near-normal distribution; therefore these traits may be considered as quantitatively inherited characters. For INC, SEV and DSI all estimates showed a moderate heritability ranging from 0,33 to 0,72. Some quantitative trait *loci* (QTL), each explaining around 10% of phenotypic variance were found on chromosome 1, 6, 8 and 10. The identification of QTL conferring resistance to MRCV may contribute to breeding programs seeking to protect the crop through improved genetic resistance.

Keywords: Mal de Río Cuarto virus (MRCV), Quantitative trait *loci* (QTL), Segregant population, *Zea mays* L.

INTRODUCTION

Maize (*Zea mays* L.) is the natural host of several viruses, but only some of these cause diseases that affect yield [1]. The Mal de Río Cuarto (MRC) disease is one of the most important viral diseases causing devastating yield loss in maize in Argentina [2]. This disease was detected for the first time in the late 60s in maize crops growing in Río Cuarto, southern Córdoba, Argentina. At present, it is distributed over almost all the corn growing areas in Argentina; however the area where the disease is endemic is the Río Cuarto region [3]. The main symptoms in maize are dwarfing, internode shortening, small ears with few or no kernels, and enations as a distinctive feature [3, 4].

The causal agent, *Mal de Río Cuarto virus* (MRCV) belongs to the *Fijivirus* genus, family *Reoviridae* [3]. A close relationship was reported between the Mal de Río Cuarto virus (MRCV) and the Maize rough dwarf virus (MRDV) that is found in Europe and the East Mediterranean [4]. MRCV is transmitted in a persistent, propagative manner by the planthopper *Delphacodes kuscheli* Fennah (Homoptera: *Delphacidae*) [5]. Wheat, oat and weeds are natural reservoirs of the virus. MRC disease epidemics occur when large populations of planthopper migrate from winter cereals to the emerging maize crop [6].

Conventional controlling approaches use chemical pesticides; however this could result in environmental pollution, and is not efficient enough. One of the effective ways to solve this problem is to breed resistant genotypes. The conventional breeding method is time-consuming, and the available resistant resources are limited. An alternative solution is to use resistant genes from resistant plants to improve susceptible cultivars [7].

The genetic diversity of maize has been helpful for the identification of resistant germplasm and the exploration of disease resistance genes, which are important in breeding programs [8]. The development of DNA-based markers provides a powerful alternative method for the dissection of complex traits [9]. DNA markers linked to MRCV resistance genes have been identified in several studies. In Argentina, previous research reported two [10] five [11] and four [12] quantitative trait *loci* (QTL) for resistance to MRC disease. In China [7] two QTL regions were identified associated with disease severity index (DSI) for Maize rough dwarf disease (MRDD).

In the present study, an early generation F_{2:3} derived from the cross of B73 and LP116 was analyzed. The objectives of this study were to identify simple sequence repeats (SSR) linked to quantitative trait *loci* (QTL) contributing to resistance to MRCV, and to validate QTL found in previous research with different genetic backgrounds.

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MATERIALS AND METHODS

Plant Materials

A susceptible inbred line (B73, belonging to heterotic group Iowa Stiff Stalk Synthetic) and a resistant inbred line (LP116, developed in INTA Pergamino) were used as parents to obtain a segregant population. A total of 208 F_{2:3} families obtained through single seed descent were employed in this study.

Field Evaluation

Trials were carried out in field experiments of the Universidad Nacional de Río Cuarto, at the Río Cuarto location (33° 06' 16.08" S; 64° 17' 54.55" O) during growing season 2010/11 and La Aguada location (32° 58' 17.25" S; 64° 38' 58.43" O) during growing seasons 2010/11 and 2011/12. Each year-location combination was used to define three environments. Río Cuarto 2010/11 (E1), La Aguada 2010/11 (E2) and La Aguada 2011/12 (E3). A randomized complete block design with two replications was used at each environment. Each trial was conducted under natural infection establishing the plots where the preceding crop was winter oat, which constitutes a reservoir for both vector and virus.

Phenotypic Data

Symptoms were measured visually on each plant using a scale [6]. This rating allowed quantification of the resistance to MRCV by means of three traits. Such traits are disease incidence (INC), disease severity (SEV) [12], and disease severity index (DSI) [7, 13, 14].

Genotypic Data

Plant material from the parents and individual F₂ plants was used for DNA extraction. Total genomic DNA was isolated using a CTAB method [15]. DNA quantity and quality were measured with a Photometer (NanoDrop® ND-1000 Technologies Inc., Nilmington, DE, USA). SSR markers and primer sequences were obtained from the Maize Database website (<http://www.maizegdb.org>), and were chosen according to their bin location by providing a good coverage of the genome. One hundred and seventy-three SSRs were used for screen polymorphism between the two parents, and then ninety-eight markers that had polymorphism were used to analyze the F_{2:3} families.

Data analysis

The INC, SEV and DSI traits at individual environments and across environments were analyzed by ANOVA using the MIXED procedure of SAS software [16]. The F_{2:3} families (genotypes) and environments were considered random effects, being defined as follows:

$$Y = \mu + E + B(E) + G + G \times E + e$$

where Y is the response variable, μ is the overall mean, E is the environment effect, $B(E)$ is the block within environment

effect, G is the genotype effect, $G \times E$ is the genotype by environment interaction effect and e is an error term.

Estimates of variance components and broad-sense heritabilities of traits were calculated on an F_{2:3} family mean basis for each environment and across environments. Estimates of variance components, including genotypic variance (σ_g^2), genotype \times environment interaction (σ_{ge}^2) and residual (σ_e^2), were calculated by equating the mean square to their expected values. A broad-sense heritability estimate based on variance component was calculated [17] for each environment:

$$H = \sigma_g^2 / (\sigma_g^2 + \sigma_e^2 / r)$$

and across environments by:

$$H = \sigma_g^2 / (\sigma_g^2 + \sigma_{ge}^2 / e + \sigma_e^2 / re)$$

where e is the number of environments, and r is the number of replications in each environment.

Two-sided 90% confidence intervals were also computed to determine the precision of heritability value estimated. The 90% confidence limits for h^2 were defined according to [18].

QTL Analysis

A linkage map was constructed using MapMaker/EXP 3.0 [19]. Simple interval mapping (SIM) [20] and composite interval mapping (CIM) [21] were employed using PlabQTL software [22]. Phenotypic variation explained for putative QTL was estimated through a multiple regression model.

RESULTS

The incidence (INC), severity (SEV) and disease severity index (DSI) in F_{2:3} families ranged between low and medium values in the three environments, indicating high infection levels. However, the F_{2:3} families were not consistent across environments because the means were not similar among environments. The LP116 line, the resistant parent, exhibited less INC, SEV and DSI than B73 line, the susceptible parent. This difference was significant ($P < 0.05$) at each environment and across environments. The INC, SEV and DSI means of F_{2:3} families also were significantly different ($P < 0.05$) from parents at each environment and across environments (Table 1).

The mean distribution for INC, SEV and DSI of F_{2:3} families is shown in Fig. 1 (a, b and c), respectively. These distributions followed near-normal distribution; therefore the traits were quantitatively inherited characters. The parental lines were situated close to the boundaries of the distributions for incidence (Fig. 1a) and the disease severity index (Fig. 1c), so that transgressive segregants could not be observed for these two traits. For SEV trait, about ten F_{2:3} families presented a higher SEV value than the susceptible parent. This indicates a small transgressive segregation in this trait (Fig. 1b).

All traits evaluated for disease assessment showed moderate heritability estimates, ranging from 0.33 to 0.72, both

Table 1. Means (\pm S.E.) of disease assessment traits of parents LP116 and B73 and of a derived mapping population of 208 F_{2:3} families; measured at each individual environment and across environments.

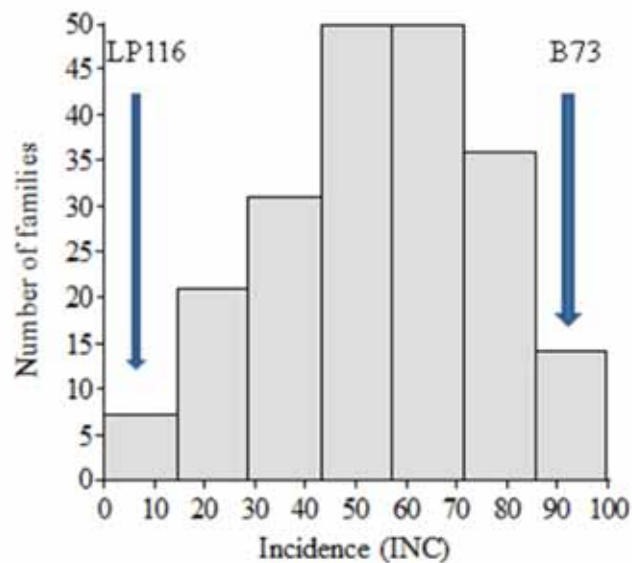
Trait*	Genotype	Environment						Across Environments	
		E1		E2		E3			
INC	LP116	0,00 \pm 0,00	a	0,00 \pm 0,00	a	0,00 \pm 0,00	a	0,00 \pm 0,00	a
	B73	0,84 \pm 0,06	c	1,00 \pm 0,00	c	1,00 \pm 0,00	c	0,95 \pm 0,04	c
	F _{2:3}	0,47 \pm 0,02	b	0,61 \pm 0,02	b	0,58 \pm 0,02	b	0,55 \pm 0,01	b
	LSD	0,25		0,28		0,33		0,17	
SEV	LP116	-		-		-		-	
	B73	1,99 \pm 0,13	b	2,81 \pm 0,01	b	2,61 \pm 0,06	a	2,47 \pm 0,16	b
	F _{2:3}	1,56 \pm 0,03	a	2,09 \pm 0,04	a	2,59 \pm 0,04	a	2,06 \pm 0,02	a
	LSD	0,34		0,39		0,33		0,24	
DSI	LP116	0,00 \pm 0,00	a	0,00 \pm 0,00	a	0,00 \pm 0,00	a	0,00 \pm 0,00	a
	B73	55,67 \pm 7,67	c	93,67 \pm 0,34	c	87,00 \pm 2,00	c	78,78 \pm 7,69	c
	F _{2:3}	24,89 \pm 1,96	b	43,08 \pm 1,50	b	49,35 \pm 1,89	b	39,11 \pm 0,93	b
	LSD	17,36		24,45		31,06		15,14	

* Disease assessment. INC, disease incidence; SEV, disease severity; DSI: Disease severity index.

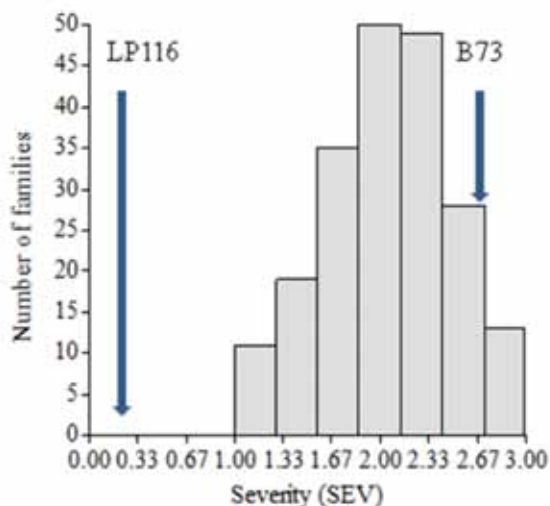
\pm Standard error

LSD: Least significant difference for comparing the means of individual F_{2:3} families at P=0.05.

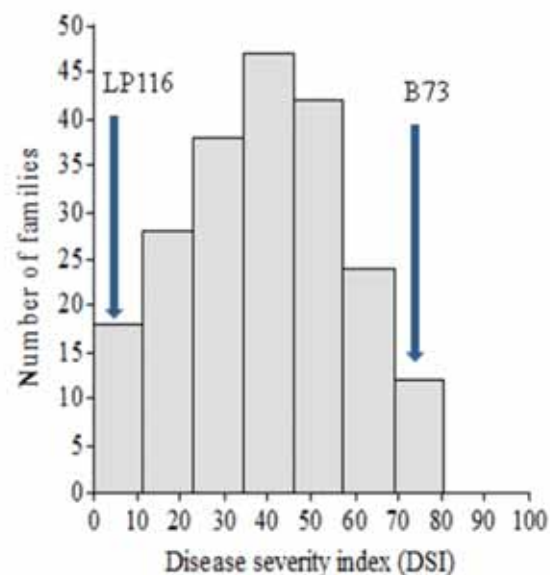
Column means followed by the same letter were not significantly different at the 0.05 probability level, according to a LSD test.



(a)



(b)



(c)

Fig. (1). Distribution of F_{2.3} families for incidence (INC) (a), severity (SEV) (b) and disease severity index (DSI) (c) in three environments. Parental mean values are indicated.

Table 2. Estimates of variance components, broad-sense heritability, and confidence intervals (CI) for incidence (INC), severity (SEV) and disease severity index (DSI) to MRC in 208 F_{2.3} families measured at each individual environment and across environments.

Parameters	Environment			Across Environments
	E1	E2	E3	
Variance Components				
Trait: INC				
σ^2_g	0,048 ± 0,008	0,058 ± 0,009	0,097 ± 0,014	0,022 ± 0,005
σ^2_e	0,057 ± 0,006	0,064 ± 0,006	0,077 ± 0,008	0,066 ± 0,004
σ^2_{ge}	-	-	-	0,045 ± 0,006

Table 2. contd...

Parameters	Environment			Across Environments
	E1	E2	E3	
Variance Components				
Heritability				
<i>H</i>	0,63 ¹	0,65 ¹	0,72 ¹	0,44 ²
CI 90%	0,508 - 0,715	0,534 - 0,731	0,626 - 0,783	0,298 - 0,558
Width ³ (%)	20,7	19,7	15,7	26,0
Trait: SEV				
σ^2_g	0,105 ± 0,031	0,215 ± 0,040	0,145 ± 0,031	0,043 ± 0,014
σ^2_e	0,290 ± 0,033	0,290 ± 0,032	0,220 ± 0,027	0,271 ± 0,018
σ^2_{ge}	-	-		0,102 ± 0,020
Heritability				
<i>H</i>	0,42 ¹	0,60 ¹	0,57 ¹	0,33 ²
CI 90%	0,212 - 0,573	0,455 - 0,702	0,402 - 0,689	0,141 - 0,472
Width ³ (%)	36,0	24,7	28,6	33,0
Trait: DSI				
σ^2_g	183,836 ± 34,592	358,306 ± 68,775	806,360 ± 117,365	146,244 ± 35,284
σ^2_e	278,663 ± 27,391	558,901 ± 55,204	681,891 ± 66,865	506,600 ± 28,866
σ^2_{ge}	-	-		303,340 ± 41,515
Heritability				
<i>H</i>	0,57 ¹	0,56 ¹	0,70 ¹	0,42 ²
CI 90%	0,433 - 0,672	0,423 - 0,667	0,609 - 0,774	0,267 - 0,540
Width ³ (%)	24,0	24,5	16,5	27,2

± Standard error.

¹ $H = \sigma^2_g / (\sigma^2_g + \sigma^2_e / r)$; r = replication number.² $H = \sigma^2_g / (\sigma^2_g + \sigma^2_{ge} / e + \sigma^2_e / re)$; r = replication number y e = environment number.³ Expressed as the ratio (%) of the confidence interval width relative to the heritability point estimate.**Table 3. Parameter associated with QTL for incidence (INC), severity (SEV) and disease severity index (DSI) to MRC, in a mapping population of 208 F_{2,3} families derived from the cross B73 x LP116 across environments.**

Trait	QTL Position ¹	Environment ²	LOD Score ³	Additive Effect	R ²
INC	1.03 (bnl1866)	E2	6,78a	-0,29	14,2
	1.03 (umc1021)	E1	6,02ab	-0,07	12,6
	1.07 (bnlg1025)	E2	7,13a	-0,26	14,9
	8.08 (phi080)	E1	3,35ab	0,09	7,3

Table 3. contd...

Trait	QTL Position ¹	Environment ²	LOD Score ³	Additive Effect	R ²
	10.04 (bnlg 1074)	E2	9,08a	-0,27	18,5
SEV	1.02 (bnlg 1007)	E1	3,65a	-0,59	8,8
	1.03 (umc 1021)	E2	3,39ab	-0,36	8,0
	4.05 (bnlg 1217)	E3	2,5ab	-0,14	5,3
	6.05 (bnlg 1443)	E1	3,44b	-0,03	8,6
	8.08 (phi 080)	E1	2,72a	0,27	6,7
DSI	1.03 (bnlg 1866)	E2	5,10b	-0,49	10,8
	1.03 (umc 1021)	E1	3,96ab	-0,12	8,5
	1.07 (bnlg 1025)	E2	4,12ab	-0,35	8,8
	8.08 (bnlg 640)	E2	2,62b	-0,28	5,7
	10.03 (bnlg 640)	E2	2,62b	-0,28	5,7

¹Chromosomal location: bin and nearest marker.

²Combinations locality-year, E1: Río Cuarto 2010-11, E2: La Aguada 2010-11; E3: La Aguada 2011-12

³Likelihood of odds (LOD) score. a and b QTL detected using CIM and SIM method, respectively.

R²: Phenotypic variation explained by the QTL.

in individual environments and across environments (Table 2). The differences in the INC, SEV and DSI of F_{2:3} families across all environments were due to significant genotype-environment interaction and the moderate heritability value estimated. For disease severity index and incidence the heritability ranged from 0.42 to 0.70. While for severity trait the higher heritability estimates were presented in E2 (Table 2).

Ninety-eight SSR markers that were polymorphic between parents were used to analyze the mapping population. The chromosomal locations of marker intervals, effect of each QTL and determination coefficients on incidence (INC), severity (SEV) and disease severity index (DSI), for each environment are presented in Table 3. Four QTL regions were identified for INC trait. Three regions were associated with alleles for MRC resistance from LP116, while the QTL detected in bin 8.08 was associated with alleles for MRC resistance, which came from susceptible parent B73. These regions explained between 7% and 19% of the phenotypic variation. Five QTL regions were found for SEV trait in bins 1.02, 1.03, 4.05, 6.05 and 8.08. Four QTL associated to DSI with a LOD score >2.5 were found in bins 1.03, 1.07, 8.08 y 10.03. These QTL showed additive negative effects, which were associated with alleles for MRC resistance came from resistant parent LP116. Phenotypic variation explained by these QTL ranged between 5 and 11%.

QTL analysis across environments presented regions associated with the three traits evaluated. The QTL were found in chromosomes 1 (bins 1.03; 1.07) and 10 (bins 10.04; 10.06). Four QTL were identified with CIM method employing eight, five and seven cofactors for DSI, INC and SEV traits, respectively. Three of these regions were also identified using SIM method. All QTL regions showed additive negative effects, therefore they were associated with alleles for MRC resistance from LP116. Phenotypic variation

explained for each QTL ranged between 5 and 11% (Table 4).

DISCUSSION

MRC, like all viral diseases, requires interactions among a competent vector, the virulent pathogen, a susceptible host and a suitable environment [2]. Transmission under controlled conditions in the laboratory offers the advantage of consistently, achieving high and uniform rates of pathogen transmission that simplifies phenotypic analysis of populations segregant for resistance [23], but it is not suitable in MRC disease because it is transmitted in a persistent propagative manner. However, in the present study natural infection was used and a good disease pressure was obtained for the evaluation from F_{2:3} families.

Heritability estimates were performed in broad-sense because they are based on differences among F_{2:3} families that reflect both additive and non additive genetic differences. For DSI trait, the estimate heritability was coincident with results obtained in a F_{2:3} segregant population with a different genetic background to the present study [13]. A higher heritability value for DSI of Maize rough dwarf disease (MRDD) was obtained in China [7]. While in a RIL population smaller heritabilities for INC, SEV and DSI traits were obtained [12]. In our assay like in previous studies [12, 13] a high genotype-environment interaction was observed. This resulted in an inconsistent response of F_{2:3} families across environments.

Plants have developed a wide range of methodologies to cope with the invading pathogens causing diseases [8]. Most complex traits, as MRC in maize and other viral diseases; are controlled by polygenes or QTL with small individual effects [12, 24, 25]. Therefore the combination of several quantita-

Table 4. Parameter associated with QTL for incidence (INC), severity (SEV) and disease severity index (DSI) to MRC, in a mapping population of 208 F_{2:3} families derived from the cross B73 x LP116 across environments.

Trait	QTL Position ¹	LOD Score ²	Additive Effect	R ²
INC	1.07 (bnlg1025)	3,68ab	-0,49	7,9
	10.04 (bnlg1074)	2,59ab	-0,38	5,6
	10.06 (umc1045)	4,56ab	-0,53	9,7
	1.03 (umc1021)	5,04a	-0,28	10,8
SEV	10.04 (bnlg1074)	4,04 b	-0,35	8,8
DSI	1.03 (bnlg1866)	3,90ab	-0,57	8,3
	1.07 (bnlg1025)	3,59ab	-0,15	7,7
	10.04 (bnlg1074)	3,10ab	-0,17	6,7

¹Chromosomal location: bin and nearest marker.

²Likelihood of odds (LOD) score. a and b QTL detected using CIM and SIM method, respectively.

R²: Phenotypic variation explained by the QTL.

tive resistance genes is necessary to achieve a high level of resistance in a genotype [26]. Improvement for MRC resistance should be made by combining resistance genes from different sources and by simultaneous selection for resistance and desirable agronomic traits across several environments [27].

In previous studies, two QTL for MRC resistance were detected on chromosomes 1 and 8 in an early generation derived from the cross between Mo17 and BLS14 [10], while Kreff *et al.* [11] identified five regions associated to MRC symptoms in one early generation derived from different parents. In the present study we have detected three QTL on bins 1.03, 1.07 and 10.04, which were also reported using different genetic backgrounds [10, 11]. By comparing the present results against those previously reported in a late selfing generation [12], it was possible to observe that a QTL associated with INC and SEV was detected in the same genomic regions (bin 8.08), in both early and late generations. On bin 10.03, where a QTL-MRC linked to marker bnlg640 for DSI was mapped, a QTL conferring resistance to maize chlorotic dwarf virus was previously identified [24]. The agreement of findings between these mapping populations may be considered an indication of the existence of these QTL and may encourage the undertaking of further research on these regions. In the present study QTL were found in different genome regions for INC and SEV. Dintinger *et al.* [28] suggest that this is due to the fact that they involve different resistance mechanisms in the resistance to virus.

This research and previous studies with different mapping populations, different genetic backgrounds and environments identified QTL for resistance to MRC in chromosomes 1, 3, 4, 6, 8 and 10 [10-12].

In summary, the resistance to MRCV observed in F_{2:3} families were controlled by few genes with small individual effects. In addition, several QTL conferring resistance to MRC disease were mapped on chromosomes 1, 6, 8 and 10 at the same regions where other virus resistance has been reported. The identification of QTL conferring resistance to MRC disease suggests that maize germplasm may contribute

to breeding programs seeking to protect the crop through improved genetic resistance. For instance, introgression QTL regions on chromosomes 1, 6, 8, and/or 10 into elite genotypes could improve resistance to MRC and other viral diseases which cause significant economic losses. The most effective and sustainable means to the prevention of virus diseases is through the deployment of genetic resistance [26]. This approach, in combination with an integrated management, will help to set criteria for sustainable control of MRC in maize.

CONFLICT OF INTEREST

The authors declare that this article content has no conflict of interest.

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