Euphytica
DOI 10.1007/s10681-016-1725-z

Quantitative trait loci for *Fusarium* and *Gibberella* ear rot resistance in Argentinian maize germplasm

Gerardo M. Giomi · Enrique D. Kreff · Juliana Iglesias · Carolina M. Fauguel · Mariana Fernandez · Maria Silvina Oviedo · Daniel A. Presello

Received: 12 February 2016 / Accepted: 17 May 2016

Abstract *Fusarium verticillioides* and *F. graminearum* cause ear rots in maize (*Zea mays* L.) that reduce yield and contaminate the grain with mycotoxins produced by the fungi. To map QTLs for resistance to these ear rots, a F_5 mapping population, consisting of 298 recombinant inbreds obtained by randomly selfing of the cross between LP4637 (moderately resistant) and L4674 (susceptible), was genotyped with 250 single nucleotide polymorphism markers and phenotyped 2 years for disease severity after silk inoculation with conidial suspensions of *F. verticillioides* and *F. graminearum*. Four QTLs were mapped in chromosomes 2, 3 and 5, bins 2.03, 3.05, 3.07 and 5.07, explaining ranges of 11.2–11.8, 3.4–5.1, 6.2–7.6 and 3.8–5.0 of phenotypic variances (%), respectively, depending on year and fungus. Additive effects of each QTL ranged from 5.0 to 11.9 % of ear area covered by mold and no epistatic interactions were observed. The four QTLs were effective for both *Fusarium* species and environments indicating that LP4637 is a source of broad resistance to *Fusarium* stable across environments. These results are consistent with previous research reporting QTLs for ear rot resistance in the same chromosome regions from sources of resistance growing in North America, Africa, Europe and China.

Keywords Maize · *Gibberella* ear rot · *Fusarium* ear rot · Quantitative trait loci

G. M. Giomi · J. Iglesias · C. M. Fauguel · M. Fernandez · D. A. Presello (✉)
Instituto Nacional de Tecnología Agropecuaria, Estación Experimental Pergamino, CC 31 2700 Pergamino, Buenos Aires Province, Argentina
e-mail: presello.daniel@inta.gob.ar

G. M. Giomi
Consejo Nacional de Investigaciones Científicas y Técnicas and the Secretaría de Ciencia y Técnica, CC 31 2700 Pergamino, Buenos Aires Province, Argentina

E. D. Kreff
Pioneer Argentina, RP178 2700 Pergamino, Buenos Aires Province, Argentina

M. S. Oviedo
Instituto Nacional de Tecnología Industrial, Centro Villa Regina, Reconquista 54 8336, Villa Regina, Rio Negro Province, Argentina

Introduction

Fusarium verticillioides (Sacc.) Nirenberg (synonym *F. moniliforme* Sheldon) and *F. graminearum* (Schwabe) cause *Fusarium* and *Gibberella* ear rots, respectively, in maize (*Zea mays* L.) These diseases reduce yield (Presello et al. 2008) and cause grain contamination with mycotoxins, including fumonisins produced by *F. verticillioides* and trichothecenes and zearalenone produced by *F. graminearum*. Since *Fusarium* mycotoxins affect human (Kumi et al.

Published online: 27 May 2016

2014) and animal (Nowak et al. 2015) health, there is concern to reduce contamination of food and feed. Most of grain contamination occurs in field conditions whenever the environment stimulates fungal growth and mycotoxin production. Ear rot effects may be reduced by developing and using less susceptible hybrids. High genotypic correlations between ear rot symptoms and grain mycotoxin concentration were observed in several host ranges (Robertson-Hoyt et al. 2006a; Presello et al. 2007) and QTLs were mapped at the same chromosome regions for both traits (Robertson-Hoyt et al. 2006b). These results indicate that disease severity accounts for most of grain mycotoxin concentration and selection for disease resistance would produce indirect responses for grain mycotoxin accumulation. Specific host mechanisms preventing mycotoxin accumulation regardless of the amount of disease also seem to exist (Presello et al. 2011) and further genetic progress might be achieved by understanding and using such mechanisms.

Sources of broad resistance to *Fusarium* are available and phenotypic selection is effective to develop more resistant genotypes (Presello et al. 2005). However, a marker assisted selection approach is advisable to accelerate genetic progress (Robertson-Hoyt et al. 2006b). Several QTLs for ear rot resistance have been mapped in different maize populations from Africa, America, Asia and Europe (Pérez Brito et al. 2001; Liakat Ali et al. 2005; Robertson-Hoyt et al. 2006b; Zhang et al. 2007; Ding et al. 2008; Rey et al. 2009; Martin et al. 2012; Tembo et al. 2014). In Argentina, QTLs for ear rot resistance were reported in an inbred that had been released from Stiff Stalk Synthetic (Rey et al. 2009) but there is scarce information on QTLs from inbreds released from ancient local germplasm. The origin of Argentinian ancient germplasm is not well known (Olmos et al. 2014) and most of the available information seems to indicate that it was originated from indigenous populations grown in Southern America with introgression of Italian varieties, which had been originated from maize sources introduced into Europe from the Caribbean Region (Luna et al. 1964).

Mapping in new sources of germplasm and environments is important to identify new QTLs and validate those genome regions already known as involved for breeding traits. This research aimed to map QTLs for resistance for *Fusarium* and *Gibberella* ear rots in an Argentinian source of resistance.

Materials and methods

Genetic materials

To select the parents of the mapping population, single rows of 650 F_7-to-F_9 inbreds representing a wide range of variability of the Argentinian temperate maize germplasm, were evaluated in Pergamino 2004/2005, for resistance to ear rot after inoculation of P319, a highly aggressive isolate belonging to Section *Liseola* (Iglesias et al. 2010; Presello et al. 2011). Twenty of the most resistant and some susceptible inbreds were selected. In 2005/2006, the selected inbreds were tested for disease resistance and fumonisin accumulation after inoculation with P364, a high fumonisin producing isolate belonging to *F. verticillioides* obtained from diseased maize kernels collected in Pergamino (Iglesias et al. 2010). The aggressiveness of P364 is representative of that expressed for most isolates in the north of the Province of Buenos Aires. The F_9 inbred LP4637, exhibiting the lowest percentages of visibly diseased ear area and grain fumonisin accumulation, was chosen as resistant parental of the mapping population. On the other hand, L4674 showed high susceptibility and fumonisin accumulation and was chosen as susceptible parent (results not shown).

LP4637 was obtained by selfing the hybrid LP561 × LP611. Both inbreds were originated from ancient Argentinian with minor introgression of Caribbean germplasm. A previous study based in molecular markers, revealed that the genome of LP4637 is still composed by both germplasm backgrounds (Olmos et al. 2014). Susceptible inbred L4674 was released from a commercial hybrid.

A F_5 mapping population consisting of 298 random inbreds was developed by selfing LP4637 × L4674. Selfing was conducted by standard procedures in summer and winter nurseries.

Genotyping and linkage map construction

Genotyping was performed by Pioneer Hi-Bred International, Inc. Ten g of leaf tissue from seedlings of the F_5 recombinant inbred were lyophilized (Virtis 35L GPFD 24DX48). Lyophilized samples were used for DNA extraction and PCR reactions. Genotyping of parental and recombinant inbreds was performed with the Illumina GoldenGate high throughput assay (Fan

et al. 2003) on 768 bi-allelic single nucleotide polymorphism (SNP) markers.

Locus orders and recombination frequencies were estimated using Mapdisto version 1.7 (Lorieux 2007). Recombination frequencies were then transformed into cM by Kosambi's mapping function. Segregation distortion for each locus was tested with Chi square tests using the same software.

Phenotyping

The parental and the 298 F_5 recombinant inbreds were tested in 2008/2009 and 2009/2010 for resistance to *Fusarium* and *Gibberella* ear rots in Pergamino, Province of Buenos Aires, Argentina (latitude/longitude 33°53′23″S/60°34′24″W) in complete block random design experiments with two replicates. Each plot consisted in a single 5-m row. Rows were separated 0.7 m each other and sown at a rate of 5 plants per m. The experiments were fertilized at planting with 155 kg N, 56 kg P_2O_5 and 20 kg S.

All plants from each experiment were inoculated into the silk channel 4 days after silking following Reid et al. (1996). The inoculum for *F. verticillioides* was produced from the isolate P364. For *F. graminearum*, the inoculum was produced from the isolate IV–II-3 that had been obtained from wheat and tested for deoxinivalenol production (Sampietro et al. 2010). Isolates were grown separately in a liquid medium following Reid et al. (1996). After 2 weeks, the cultures were filtered through cheesecloth to remove mycelium and conidial concentrations were adjusted to 1×10^6 conidia ml^{-1} with sterile water. For inoculation, 2 ml of conidial suspension were injected into the silk channel 4 days after silking following Reid et al. (1996).

Ears were manually harvested at maturity (20 % moisture, approximately) and disease severity was visually assessed as the percentage of each ear covered by mold. Means of disease severity were computed for each plot and used as phenotypic data.

Statistical analysis

To estimate components of variance and heritability, analyses of variance within fungal species were conducted using the mixed procedure from SAS statistical software (1999). The model (Eq. 1), included genotype and replicate within year as random, and year as fixed effects.

$$y = X\beta + Z\mu + \varepsilon, \quad (1)$$

where y is the vector of phenotypic data, β is the vector of environmental fixed effects, u and ε are the effects vectors of random effects associated to genotype, genotype-by-environment interaction and residuals, respectively. X and Z are the matrixes of coefficients associated to fixed and random effects, respectively.

Components of variance for means of disease severity for each fungus were estimated with the residual maximum likelihood (REML) method following Patterson (1997). Broad sense heritability (Eq. 2) was estimated following Hallauer and Miranda Filho (1988)

$$H^2 = \sigma_G^2 \Big/ \left[\sigma_G^2 + \left(\frac{\sigma_{GE}^2}{\eta}\right) + \left(\frac{\sigma_e^2}{r\eta}\right)\right], \quad (2)$$

where σ_G^2 is genotypic variance, σ_{GE}^2 is genotype-by-environment interaction variance, σ_e^2 is error variance, r is replicates nested within environment, η is environment.

For multi-trait multi-environment (MTME) QTL (joint QTL) analysis, phenotypic data were analyzed using a mixed model approach according to Malosetti et al. (2008). The MTME data set consisted of I genotypes, evaluated in J environments with K traits (disease severity after inoculation of each fungal species) and L blocks, I = 298, J = 2, K = 2 and L = 2. A N-by-1 vector (y) was defined, with N = IJKL. Vector y contains all the observations sorted by trait within environment and within genotype in each block. Random genetic effects were assumed to be normally distributed u ~ N(0, G), being the G matrix a block diagonal with blocks of 4-by-4 variance–covariance parameters. In the model (Eq. 1), ε is the vector of non-genetic residuals associated with each observation and normally distributed ε ~ N(0, R) being R the residual variance. The phenotypic co-variance was V(y) = ZGZ′ + R. Several G and R matrixes were tested and the best model was chosen on the basis of a goodness of fit Akaike information criterion (AIC). The AIC values showed that the model based on unstructured matrix G and Toeph (2) matrix R performed better than the rest of the models. This model was used to calculate the best empirical linear unbiased estimators of fixed effects and the best linear unbiased predictors (BLUPs) of random effects from REML-estimated

variance components (Mrode 2005; Piepho et al. 2008).

QTL analysis

The BLUPs for the data set where used for joint QTL mapping with a mixture model (Eq. 3). Mixture models are more efficient than mixed models for non-saturated mapping (Jiang and Zeng 1995).

$$y = X\beta + \varepsilon, \quad (3)$$

where X is the matrix of coefficients associated to fixed effects, β is the vector of fixed effects, ε is the vector of random effects associated to residuals.

Unlike mixed models, mixture models assume that the residual effects εJK are correlated among traits within individuals with covariance Cov(εJK, εJL), and are independent among individuals (Jiang and Zeng 1995).

Analysis of QTLs was conducted in two steps, following Alvarez Prado et al. (2012), with WinQTL Cartographer V2.5 (Wang et al. 2011). Firstly, a genome scan was performed with tests for joint QTL considering trait and environment correlations. Secondly, a multiple-QTL model was run testing the effects and significance of each QTL into a unique model and possible interactions among them.

The multi-trait mapping procedure, which implements the composite interval mapping (MT-CIM), was used for the first step. In the CIM, stepwise regression analysis of Model 6 from WinQTL Cartographer V2.5 was used. A threshold of 0.05 for input and output was established for selecting the putative QTLs to be used as cofactors. We used a window size of 10 cM for removing temporarily the marker effects when scanning the chromosome. A LOD score of 6.0 was used as a threshold to declare the presence of a putative QTL, with scanning intervals of 2 cM between markers and a putative QTL. The QTLs identified in the first step were included in the initial model for multi-trait multiple interval mapping procedure (MT-MIM), following Alvarez Prado et al. (2012).

Inbreeding produces high frequencies of genes in homozygous status. Since in this mapping population the number of SNPs in heterozygous status was too low to estimate dominance effects accurately, markers in heterozygous status were scored as missing data for QTL analysis. Simulation studies (Takuno et al. 2012), demonstrated that the performance of QTL mapping in F_4 could be comparable to that in F_6 or F_7 by removing dominance effects. An obvious disadvantage of this method is a reduction of sample size. Nevertheless, elimination of bias of dominance estimates increases the power to detect QTLs and this procedure is currently used for mapping bi-parental populations (Huang et al. 2011).

Results

Analyses of variance revealed significant effects of genotype, fungus and genotype-by-fungus interaction in both years. Mean squares of genotype-by-fungus interaction were minor compared to those of principal effects (results not shown) indicating that the ranking of genotypes for disease severity tended to be stable across years and fungal species. Recombinant inbreds exhibited a wide range of phenotypic variability for disease severity after inoculation with each

Table 1 Means of ear rot severity and component of variance for *Fusarium* and *Gibberella* ear rots in a mapping population of F_5 maize inbreds attained from LP4637 × L4674

Genotype/variance component	*Fusarium* ear rot		*Gibberella* ear rot	
	2008/2009	2009/2010	2008/2009	2009/2010
Mean of disease severity[a]				
LP4637	4.1	5.3	3.2	6.4
L4674	84.2	88.1	85.3	87.9
Range in recombinant inbreds	4.2–85.9	6.1–88.1	4.2–88.3	4.9–.88.3
Components of variance and heritability				
Genotype	249.71		267.85	
Genotype by environment	62.16		85.77	
Heritability	0.76		0.72	

[a] Percentage of the ear visibly covered by mold

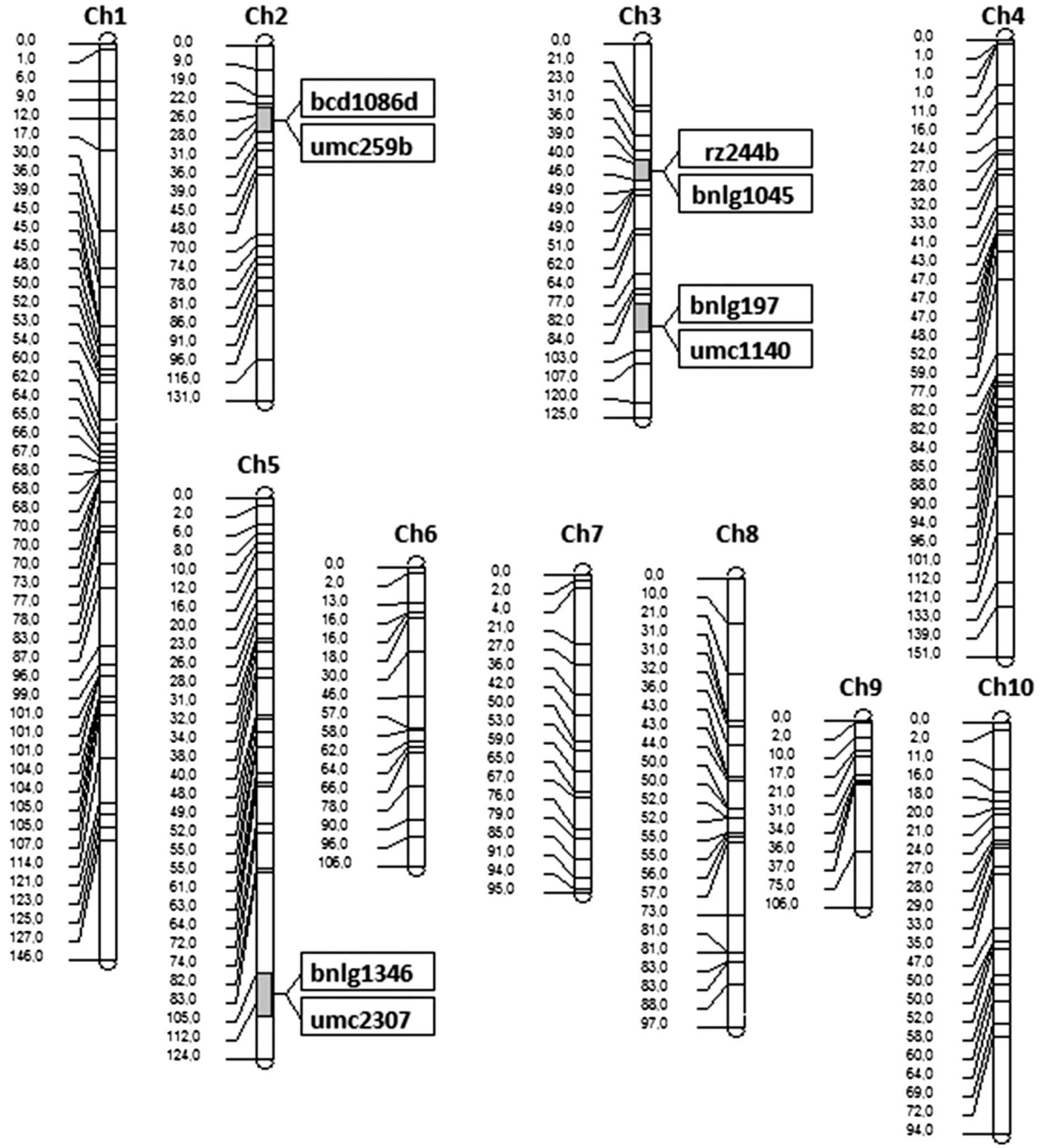

Fig. 1 Chromosomal location of four QTLs detected for resistance to *Fusarium* and *Gibberella* ear rots in a mapping population of maize. The QTLs are represented by *bars* with a connector to the closest molecular markers on the IBM2 2008 neighbors 2 reference map available in www.maizegdb.org

fungus in both years (Table 1). No transgressive segregation was observed. Heritability estimates ranged from 0.72 for *Gibberella* to 0.76 for *Fusarium* ear rot. These results indicate that genotypic variability for resistance to both ear rots was important in the mapping population. Since expected homozygosity in

Table 2 Bin, additive effects, coefficient of determination and percentage of phenotypic variance explained by four QTLs for *Fusarium* and *Gibberella* ear rot resistance identified by multi-trait multiple interval mapping in a mapping population of F_5 maize inbreds attained from LP4637 × L4674

Parameters	Years	Bin			
		2.03	3.05	3.07	5.07
Fusarium ear rot					
Additive effect[a]	2008/2009	−11.9	−8.0	−9.8	−7.9
	2009/2010	−10.4	−5.6	−7.5	−5.9
R_P^{2b}	2008/2010	11.2	5.1	7.6	5.0
	2009/2011	11.8	3.4	6.2	3.8
Gibberella ear rot					
Additive effect	2008/2009	−11.3	−7.8	−10.9	−5.1
	2009/2010	−10.4	−5.6	−7.4	−5.0
R_P^2	2008/2009	13.1	6.2	12.2	2.7
	2009/2010	11.9	3.5	6.1	2.8

[a] Computed from means of disease severity (percentage of the ear visibly covered by mold)

[b] Percentage of phenotypic variance explained

generation F_5 is 0.9375, heritable variance mostly depends on additive genetic effects and estimates should be close to narrow-sense heritability. Percentages of disease severity after inoculation with each fungus exhibited high genotypic correlations in both years ($r_G = 0.80$ and 0.89 in 2008/2009 and 2009/2010, respectively).

From the 768 SNPs tested in the mapping population, 250 exhibited polymorphism for parental inbreds and no segregation distortions. Based in polymorphic marker, the length of the genetic map was 1178 cM with an average distance between markers of 4.7 cM. Marker saturation was similar or higher than that used to map *Fusarium* ear rot (Zhang et al. 2007; Ding et al. 2008) and *Gibberella* ear rot (Martin et al. 2012) in different mapping populations.

Four QTLs were consistently identified in the 2 years for *Gibberella* and *Fusarium* ear rot resistance in chromosomes 2, 3 and 5 (2.03, 3.05, 3.07 and 5.07). For the other maize chromosomes no QTL was found (Fig. 1). No epistatic effects among QTLs were observed. The QTL in chromosome 2 expressed the larger percentages of phenotypic variability and additive effects for both diseases and years (Table 2). The joint model considering all four QTLs explained 35.6, 25.4, 30.3 and 26.3 % of phenotypic variability for disease severity after inoculation with *F. graminearum* and *F. verticillioides* in 2008/2009 and 2010/2011, respectively.

Discussion

High correlations between disease severity after inoculation with *F. graminearum* and *F. verticillioides* and consistency of QTL identification across environments and fungal species indicate that LP4637 is a source of broad resistance to *Fusarium* spp. stable across environments. Broad and stable resistance to *Gibberella*, *Fusarium* and *Aspergillus* ear rots, was reported in several sources of germplasm and environments (Presello et al. 2006; Robertson-Hoyt et al. 2007). In most maize growing regions, several ear rotting species coexist in the field and fungal prevalence, which mostly depends on environmental conditions, cannot be accurately forecasted at planting time. Thus, availability of QTLs for broad resistance is important to develop hybrids capable to reduce losses regardless of the prevalence of each ear rotting pathogen.

Ear rot resistance is a complex trait controlled by several genes with minor effects and genetic progress is feasible after accumulation of those genes (Presello et al. 2005). In this mapping population, the joint

model explained up to 35.6 % of phenotypic variability, which indicates that resistances to these ear rots in LP4637 are also complex traits depending on many genetic factors. Remaining non-explained variability might due to a low marker saturation in the mapping population rather than inaccuracy of phenotyping considering that G was three–four times higher than G × E (Table 1) and no specific QTLs for a particular disease or year were observed.

Previous studies revealed that these genome regions are important for resistance to ear rots caused by *Fusarium* in several sources of germplasm and environments. Quantitative trait loci for resistance to *Fusarium* ear rot were reported in bin 2.03 in maize from Mexico (Pérez Brito et al. 2001) and China (Zhang et al. 2007). Surrounding regions, bin 2.08, also seem to be important in temperate populations growing in the Corn Belt of US (Robertson-Hoyt et al. 2006b) and China, bins 2.01 and 2.02 (Zhang et al. 2007). Quantitative trait loci for *Fusarium* ear rot resistance in chromosomes 3 and 5 were reported in bins 3.04, 3.08, 5.04 and 5.07 from sources of resistance growing in China (Zhang et al. 2007; Ding et al. 2008; Chen et al. 2012), and in bins 3.06, 5.05 and 5.06 from the US (Robertson-Hoyt et al. 2006b). For *Gibberella* ear rot resistance, QTLs were reported in bins 2.04, 2.06 and 2.08, 3.02, 3.04, 3.05, 3.06, 5.04, 5.05 and 5.06 in sources of resistance from Canada, Germany and Uganda (Liakat Ali et al. 2005; Martin et al. 2011, 2012; Tembo et al. 2014). The same genome regions seem to be also important for *Aspergillus* ear rot resistance (Robertson-Hoyt et al. 2007; Mideros et al. 2014; Warburton et al. 2015). The most important QTL mapped in bin 2.03. Only few studies reported QTLs for *Fusarium* and *Gibberella* ear rots in this bin. On the other hand, no QTL were found in some important genome regions, such as those in chromosome 4 between bins 4.02 and 4.05 (Xiang et al. 2010).

Map overlapping of all four QTLs in this Argentinian mapping population with those mapped in a range of sources of resistance, environments, and pathogenic ear rotting species indicates that these are important chromosome regions that might be targeted for breeding of ear rot resistance in maize.

Acknowledgments This research was supported by Grants from the Instituto Nacional de Tecnología Agropecuaria (PNCER-1332), Secretaría de Ciencia y Técnica (PICT-PAE36047 N° 77/07) and Consejo Nacional de Investigaciones Científicas y Técnicas, Argentina. Genotyping was conducted by Pioneer Hi-Bred International, Inc. The authors thank to Santiago Alvarez Prado and Gerardo Cervigni for assistance in statistical analysis.

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