Identification of quantitative trait loci for resistant to maize ear rot caused by *Fusarium moniliforme* Sheldon and common rust caused by *Puccinia sorghi* in Argentinian maize germplasm.

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Maize ear rot caused by *Fusarium moniliforme* Sheldon and common rust caused by *Puccinia sorghi* are two of the most important diseases in maize in Argentina. Variability for resistance in both diseases is observed between the two heterotic pools commonly used in maize breeding in Argentina. General resistance to ear rot has been reported and general or partial and race-specific resistance are available for rust. The objective of this work was to identify quantitative trait loci (QTL´s) associated to genetic resistance to both diseases and analyze the possibility of using the QTL´s in a marker-assisted selection program to reduce the amount of materials to screen in disease nurseries. A population of 190 $F_{2:3}$ families of maize obtained in a cross between two inbreds belonging to both pools was evaluated for resistance to both diseases under artificial inoculation and natural infection respectively. One hundred and thirty simple sequence repeat (SSR) were used. Six and three putative resistant QTL´s to ear rot and rust were identified respectively using a single point linkage analysis. QTL´s explained up to 6.5% of phenotypic variation for ear rot and up to 27% for rust. These results suggest that , in relation to ear rot, marker assisted selection alone is not recommended for use on a routine basis, whereas its use, along with phenotypic scores in rust could be of value for selecting superior genotypes.

KEY WORDS: common rust, *Fusarium* ear rot, , resistance QTL´s, simple sequence repeat, SSR*, Zea mays*

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La podredumbre de la espiga de maíz causada por Fusarium moniliforme Sheldon, y la roya común, causada por Puccinia sorghi, son dos de las más importantes enfermedades del maíz en Argentina. Se observa variabilidad para la resistencia a ambas enfermedades entre los grupos heteróticos Iowa Stiff Stalk y Flint Argentino, utilizados para el mejoramiento genético del maíz. Se ha informado sobre la existencia de resistencia general a podredumbre de la espiga para podredumbre y sobre resistencia general y específica para roya. El objetivo de este trabajo fue identificar loci de resistencia cuantitativa (QTL´s) asociados a la resistencia genética a ambas enfermedades y analizar la posibilidad de usarlos en un programa de selección asistida por marcadores para reducir la cantidad de material a tamizar en viveros de enfermedades. Una población de 190 familias F2:3 de maíz originadas luego del cruzamiento entre dos líneas pertenecientes a ambos grupos heteróticos fue evaluada para la resistencia a ambas enfermedades luego de inoculación artificial para podredumbre de la espiga y natural para roya. Se usaron 130 microsatélites. Mediante un análisis de ligamiento de un punto se detectaron seis y tres probables QTL´s para resistencia a podredumbre y roya respectivamente. La cantidad de variación fenotípica explicada por cada QTL fue de hasta 6,5% para podredumbre y hasta 27% para roya. Se concluye que en relación a podredumbre, no se recomienda como rutina la selección asistida por marcadores solamente, mientras que en roya, junto a las clasificaciones fenotípicas, sería de valor para seleccionar genotipos superiores.

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PALABRAS CLAVE: roya común, podredumbre de la espiga de maíz, QTL´s para resistencia, marcadores microsatélites, maíz, Zea

INTRODUCTION

The disease maize ear rot is caused by the facultative, polyphague and toxic fungi *Fusarium moniliforme* Sheldon (perfect stage: *Giberella fujikuroi* Sawada) (Bacon & Hinton, 1996). Ear rot is one of the most important diseases in maize and it is the most important ear disease in Canada for a significant detrimental effect on both grain yield and quality (Vigier *et al*.,1997), because toxins produced by the fungi concentrate in ears, stems, grains and by products. The importance of the disease is increasing in incidence in the south of Buenos Aires province, Argentina, in the last years (Cerono, personal comm.).

Genetic resistance to ear rot appeared to be controlled by several genes (Nankam & Pataky, 1996). In some studies it was observed that resistance is influenced by maternal effects operating in the silks and pericarp (Headrick & Pataky, 1991).

Common rust is one of the most persistent diseases encountered in maize in USA and causes serious yield reductions throughout much of the corn growing area in Argentina. In this country, disease severity levels has reached up to 40% (González *et al*. ,1999) depending of the genotype, pathogen virulence and environmental conditions. Yield losses are estimated to be in the order of 0.5% for every 1% area of leaf infected. *Puccinia sorghi*, the causal agent of the disease, is a heteroic fungi, that completes its cycle on the alternative host, *Oxalis* sp. Two forms of resistance are available, general or partial, in which the size and number of pustules are reduced, and race-specific or major gene resistance (*Rp*), in which a hypersensitive reaction restricts pustule development (Groth *et al*. ,1983). Partial resistance generally is expressed through components of the infection cycle, which result in fewer and smaller lesions or fewer uredinia (Davis *et al*. ,1988; Juvik *et al*. 1994; Pataky, 1986). This kind of resistance also has resulted from longer latent periods (Gingera *et al.* ,1995). The *Rp1* locus, which is available in many hybrids, is a complex locus consisting of duplicated sequences. A high frequency of unequal crossing over has generated a large number of alleles and new allele combinations have been fixed and released. The allele most frequently used today is *Rp1-D*. However, other alleles at *Rp1* as well as the c allele at *Rp-3* have utility. Resistance due to alleles at *Rp1* and *Rp3* is usually dominant (Pfeffer &Thro, 1999). Simply inherited resistance may result in selection for virulent races. For example, biotypes of *Puccinia sorghi* virulent on *Rp1-D* resistant hybrids were recently identified in the Midwestern United States where the *Rp1-D* gene has been used successfully for the past 15 years to provide resistance in sweet corn to common rust (Pataky &Tracy, 1999).

Partial resistance may be more durable than simply inherited resistance (Vanderplank, 1968). Both dominant and additive genetic variances have been associated with this type of resistance (Kim & Brewbaker, 1977; Randle *et al*., 1984). Traditionally, partial resistance has been more difficult to transfer than simply inherited resistance due to its presumed multigenic nature. In addition backcrossing polygenic traits from unadapted lines with poor combining ability results in inferior inbreds. Molecular mapping techniques in combination with marker assisted selection may enable breeders to identify and exploit these forms of resistance more effectively (Young, 1996).

In Argentina, most private maize breeding programs use three traditional heterotic pools: Iowa Stiff Stalk (BISS), Mo17, and Iodent, a particular pool developed from ancient local flint populations, frequently termed Flint Argentino (FA). Inbred developed from the latter usually show heterotic vigor when crossed against inbred derived from BISS and Mo17 (Cerono, pers comm.) and variability in resistance to ear rot and common rust is observed. The importance of FA in Argentina can not be neglected, since it is the basis for the development of hybrids with resistance to abiotic stresses (Cerono, pers comm.).

The objective of this work was to identify resistance QTL´s to ear rot and common rust in a population $F_{2:3}$ of maize derived from a cross between a pure BISS derived inbred and an inbred derived from the Flint Argentino pool, using molecular markers. An important advantage of this latter for the incorporation of resistance genes, is the possibility for the breeder to carry out various selections per year without having to depend on the natural occurrence of the pathogen and even without presence.

MATERIALS AND METHODS

Plant materials

The mapping population consisted in 190 $F_{2:3}$ families derived from the crosses between two maize lines: P_1 and P_2. The female parent, P_1, of the heterotic pool BISS, is considered tolerant to ear rot and non-resistant to rust. During two years of experiments with artificial inoculation with *Fusarium moniliforme* Sheldon previous to this study, P_1 had an average score of 2, in a visual scale from 1 to 9, where 1 is resistant and 9 is susceptible. The grain has a partially vitreous, orange colored endosperm. The male parent, P_2, of the heterotic pool Flint Argentino, is considered non-resistant to ear rot and resistant to rust. During two years of experiments under artificial inoculation previous to this study, P_2 was scored with 7, in the same scale mentioned above. The grain has a yellow to orange endosperm, with a greater proportion of the vitreous type, than P_1 (Cerono, personal comm.).

Initial crossings between P1 and P2 plants were made in the Experimental Station of Monsanto in Camet, Argentina. The F_1 generation resulting was selfed in Kihei Research Station, Hawai, USA, in the same year. The resulting F_2 generation was planted in Camet Argentina in the following year, and approximately 200 random plants were selfed, which, after discarding a few

poorly grained ears, originated the 190 $F_{2:3}$ families that were used in this study.

Marker analysis

For the molecular marker analysis, 130 polymorphic microsatellites (SSR´s) evenly distributed over all linkage groups of corn, were used. DNA was extracted according to Dellaporta *et al*. (1983) of a sample of 40 plantlets of each $F_{2:3}$ family and of the parents. A thermocycler was used for DNA amplification. A 10 μl reaction solution contained 5 μl of DNA (10ng) , 0.28 μl of primer (5μM), 0.78 μl 10X buffer, 0.03 μl dNTP (25mM each one), 0.47 μl MgCl2 (25 mM), 0.08 μl Taq polymerase (5U/μl), 0.90 μl loading dye and 2.46 μl HPLC H2O. The thermal cycler program consisted of 40 cycles of 94°C for 30 seconds and 60°C for 45 seconds, then a 2-minute extension at 72°C. The SSR products were resolved in a 2% acrylamide gels, stained with SyBr Green and visualized. The assignment of alleles in the population was made comparing it with the parents.

Experimental plots

All 190 $F_{2:3}$ families and their parents were sown in field plots at Camet. An incomplete augmented blocks design was used (Federer, 1961) where parental lines were placed in each block. Each plot consisted of a 5 meters long row, with 30-to 35 plants. Plants within a row were 0,15 m apart. Plots were sowed mechanically with a density of 77000 pl/ha and fertilized with 100kg/ha of diamonic phosphate at sowing (18-46-0) and 190 kg/ha of urea (0-46-0) at V6. Weeds were controlled with adequate herbicides.

Fusarium ear rot evaluation

Silks were inoculated with a conidial suspension of 1 x 10^6 spores concentration of an isolate of a fungi *Fusarium moniliforme* Sheldon. This isolate had been successfully used for many years in Monsanto. Fungi was cultivated in Petri dishes with agar-potato-glucose (GPA) and transferred to small glass containers containing sorghum grains. Conidial suspension was prepared afresh at the moment of inoculation, and inoculated into the silk channel at seven days after silk emergence (Chungu *et al*., 1996). Inoculation was repeated seven days later. Irrigation was applied during fourteen days from first inoculation.

At harvest, infection severity was scored visually at each spike (percentage affected) individually using a scale from 1 to 6, where 1= 0% of severity, , 2= 1 to 10%, 3= 11 to 25%, 4= 26 to 50%, 5= 51 to 75%, and 6=76 to 100%. At each plot, total number of spikes and number of spikes of each scale degree were determined. Then, weighted means of severity disease were calculated according to Pérez Brito *et al*., (2001).

Common rust evaluation

Plants were infected naturally in the field, and infection severity was scored visually using a scale currently used to score infection severity of *Stenocarpella macrospora* (Olatinwo *et al.,* 1999), from 1 to 9. These evaluations were made from blossom once a week during four weeks. With infection severity data, the area under curve of disease progress (AUDPC) was calculated with the objective to compare the disease progress between families. The AUDPC was calculated as follows:

$$AUDPC=\sum_{n-1} (yi+yi+1)\,(ti+1-ti),$$

where **n** represents the number of evaluations made, **y** represents infection severity expressed in %, and **t** is a time (Berger, 1988).

QTL´s analysis

Individuals were grouped in classes for each molecular marker. LSMeans and variances for lesion area were calculated and compared between classes. In order to identify genomic regions associated with ear rot and rust resistance , the $F_{2:3}$ genotypic classes for each DNA marker were contrasted with ear and rust disease response in a single marker analysis using simple regression in S-PLUS (version 6.1) (2003) (http://www.insightful.com). A significant association between a DNA marker and the disease infection percentage, and so the presence of linkage between the marker and a QTL, was declared with an alpha level of 0.05.

Additive and dominance values for each QTL were calculated. Negative values in estimated additive effects indicate that the substitution effect of a non favorable allele from the susceptible parent (P_2) by a favorable allele from the resistant parent (P_1) tends to reduce the infection severity at that locus. On the other hand, negative values in dominance effects indicate that the mean of the heterozygote class reduce the infection severity as compared with the mean of the homozygote class.The type of gene action for the QTL´s detected was analyzed with the degree of dominance: d/a, where a degree 1 is considered totally dominant, a degree of -1 is considered totally recessive, and a case of degree 0, the gene action is considered totally additive. Values above 1 or bellow -1 indicate overdominance. Two-way analysis of variance was made between QTL´s pairs to detect epistasis. The coefficient of determination (R^2) was calculated to determine the proportion of genetic variance explained for each QTL. A model of multiple regression was developed including only the markers with the highest p-value of association for the trait (Eathington *et al*., 1997). With this model, the proportion of total phenotypic variance (between genotypes) explained by significant markers was calculated. Including only those markers most closely associated with the putative QTL ´s to avoid the presence of a correlated structure among the regressors. Heritability in the broad sense was calculated according to the formulae: $H^2 = \sigma^2 g/\sigma^2 p$. The $\sigma^2 e$ (error variance) was calculated as the difference among phenotypic values of parental lines (placed in all blocks).

The proportion of genetic variance explained by the markers was estimated as the ratio between R^2 and H^2.

RESULTS AND DISCUSSION

Disease tests and data analysis

Fusarium ear rot

The distribution of $F_{2:3}$ families for percentage of spike infection is shown in Figure 1. A distribution with a trend to lower infection percentages was observed. There

were families with infection percentages lower than those of the resistant parent.

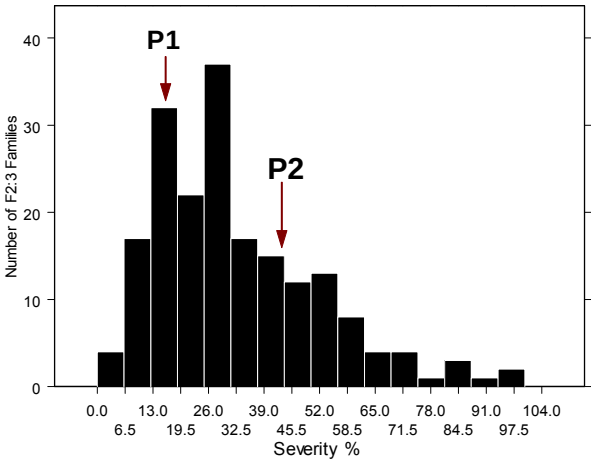

Figure 1. *Distribution of spikes infection percentages of ear rot in 190 F2:3 families. $P_{1:}$ the female parent. It is considered tolerant to ear rot and non-resistant to rust. $P_{2:}$ the male parent. It is considered non-resistant to ear rot and resistant to rust.*
Figura 1. *Distribución de los porcentajes de infección de las espigas afectadas de podredumbre de la espiga en 190 familias F2:3 .*

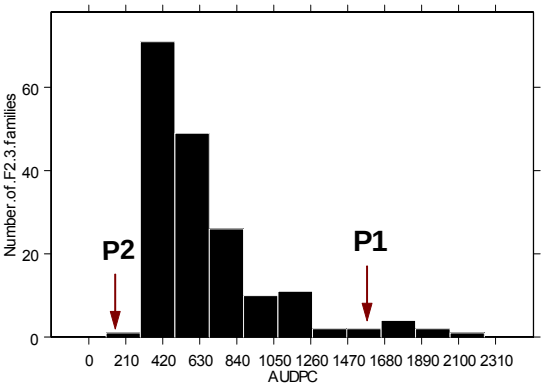

Figure 2. *Distribution of F2:3families for the area under curve of disease progress (AUDPC) (common rust). $P_{1:}$ the female parent. It is considered tolerant to ear rot and non-resistant to rust. $P_{2:}$ the male parent. It is considered non-resistant to ear rot and resistant to rust.*
Figura 2. *Distribución de familias F2:3 para el área bajo la curva de progreso de la enfermedad (ABCPE) (roya común)*

Transgressive segregation was observed. This phenomenon was observed before by maize breeders for quantitative traits although genetic basis are not well determined (Veldboom *et al*., 1994). For *Fusarium*, this phenomenon could be explained by the contribution of resistance alleles to ear rot coming from both parents.

Common rust
The distribution of F2:3 families for AUDPC is shown in Figure 2. A strong deviation of values towards dominant phenotypes is observed and can be interpreted as an effect of dominant action of genes conferring resistance to the disease. There were families with AUDPC lower than the resistant parent and higher than the susceptible parent. Transgressive segregation was observed and could be explained by the contribution of resistant alleles coming from both parents. This phenomenon was observed before for Puccinia sorghi by Hooker (1962). The broad sense heritability value of resistance to common rust of population was 0.93.

QTL´s mapping
Fusarium ear rot
Six markers with significant associations ($P< 0.05$) to resistance to ear rot were identified by comparing the phenotypic scores of the genotypic classes for each marker. Significant markers were located on four linkage groups and identified six QTL´s. One QTL was located on chromosome 1 (P=0.02), two QTL´s were located on chromosome 5 (P=0.04 and P=0.001), two QTL´s on chromosome 7 (P=0.03 and P=0.0007) and one QTL was located on chromosome 9 (P=0.02) (Figure 3). Additive values were negative except for QTL´s detected on chromosomes 1 (163 cM) and 9 (111,2 cM), and dominance values were negative too, except for the QTL located on chromosome 7 (131 cM). Genetic effects of partial and complete dominance and overdominance were observed. Epistasis was detected between six of the 15 possible pairs of markers.
The QTL´s of greater relative contribution to phenotypic individual variation, according to the R^2 was those located in chromosome 7 (6.5%), followed by those located in chromosome 5 (5.8 and 4.7%), all coming from the resistant parent (Table 1). Because additive values were negative, it is possible to infer that the resistance of QTL´s on chromosomes 1 and 9 were contributed by the susceptible parent, while at other QTL´s, it was contributed by the resistant parent. So, negative values of dominance effects indicate that in general, it is not essential to fix the resistance alleles in a particular germplasm that is being improved since the heterozygous state at these loci reduce the severity of infection.
The broad sense heritability value of resistance to ear rot of population was 0.18. This low heritability value indicates the necessity to measure the trait with higher precision. However, in experiments assayed in several environments estimated heritabilities were of low to medium values (Pérez Britos *et al.,* 2001). So, a major effort in a breeding program is needed to accumulate a large number of resistance alleles in germplasm, but as the probability of selecting superior genotypes (i.e. inbreds) is low, marker assisted selection (MAS) offers a strategy that could increase the gains made from selection. However, because of the presence of different races of the pathogen, the possible errors in detecting the position of putative QTLs, the necessity of mapping the QTL in every population where MAS will be applied, and the relatively small contribution of the QTL to the phenotypic variance in this study, indicate that MAS alone is not recommended for use on a routine basis (Knapp, 1998; Mohan *et al*., 1997). The best scheme to use would probably consist of combining the use of MAS with conventional selection methods (ie: recurrent selection) (Pérez Brito *et al*., 2001).
R^2 calculated by multiple regression accounted for 21% of phenotypic variation.

Table 1. *Localization of markers on genome, gene effects associated with the QTL's related significantly with ear rot resistance, degree of dominance and coefficient of determination (%) estimated from phenotypic and genotypic data of population of F2:3 families.*
Tabla 1: *Localización de los marcadores en el genoma, efectos génicos asociados con los QTL´s relacionados significativamente con la resistencia a la podredumbre de la espiga, grado de dominancia y coeficiente de determinación (%) estimado de los datos fenotípicos y genotípicos de una población de familias F2:3.*

Chromosome	Marker (%)	P∞	Add*	Dom**	Dom/Add***	R^2
1	Mk 1-18	0,02	5	-2,5	-0,5	3
5	Mk 5-5	0,004	-5,9	-7,4	1,2	4,7
5	Mk 5-13	0,001	-6,3	-7,3	1,2	5,8
7	Mk 7-2	0,03	-5,4	-5,2	1	2,6
7	Mk 7-6	0,0007	-7,2	1,6	-0,2	6,5
9	Mk 9-7	0,02	5,3	-1,6	-0,3	2,8
R^2 (%)##	21					

∗ genic effect of additivity (Add),∗∗ genic effect of dominance (Dom), ∗∗∗ degree of dominance, # Coefficient of determination,## Total coefficient of determination.

Table 2. *Significant epistasis values detected between 6 of 15 possible marker pairs associated with ear rot resistance by two ways ANOVA, and map position.*
Tabla 2: *Valores de epistasis significativos detectados entre 6 de los 15 pares de marcadores asociados con la resistencia a la podredumbre de la espiga, mediante ANOVA de 2 vías, y posición en el mapa.*

Markers	chromosome/ubication	P∞
Mk 1-18 - Mk 9-7	1(163 cM)- 9(111 cM)	0,004
Mk 5-5 - Mk 5-13	5(57,7 cM)- 5(99,6 cM)	9,8x10-6
Mk 5-5 - Mk 7-6	5(57,7 cM)- 7(131,6 cM)	1,05x10-6
Mk 5-13 - Mk 7-2	5(99,6 cM)- 7(60,5 cM)	3x10-5
Mk 5-13 - Mk 7-6	5(99,6 cM)- 7(131,6 cM)	9x10-7
Mk 7-2 - Mk 7-6	7(60,5 cM)- 7(131,6 cM)	0,0016

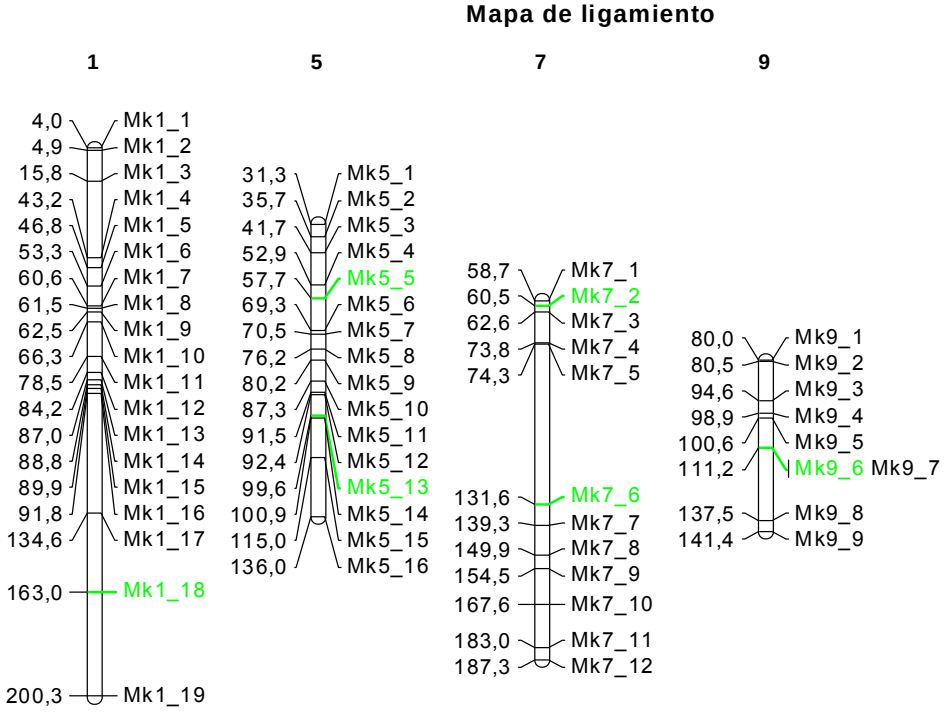

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Figure 3. *QTL´s detected for ear rot resistance associated with their linkage groups (green numbers are markers linked with QTL´s).*
Figura 3. *QTL´s detectados para resistencia a podredumbre de la espiga asociados con sus grupos de ligamiento*

Table 3. *Localization of markers on genome, gene effects associated with the QTL's related significantly with common rust resistance, degree of dominance and coefficient of determination (%) estimated from phenotypic and genotypic data of population of F2:3 families.*
Tabla 3. *Localización de los marcadores en el genoma, efectos génicos asociados con los QTL´s relacionados significativamente con la resistencia a la roya de la espiga, grado de dominancia y coeficiente de determinación (%) estimado de los datos fenotípicos y genotípicos de una población de familias F2:3.*

Chromosome	Marker	P$^{\infty}$	Add*	Dom**	Dom/Add***	R^2 (%)$^{\#}$
2	Mk2_11	0,008	-108,8	-26,61	0,24	3,9
4	Mk4_1	$2,01^{x}10^{-8}$	216,29	57,37	-0,27	16
10	Mk10_2	$5,73^{x}10^{-14}$	-268,5	-94,33	0,35	27
	R^2 (%)$^{\#\#}$	45				

* genic effect of additivity (Add),** genic effect of dominance (Dom)
*** degree of dominance, # Coefficient of determination,## Total coefficient of determination

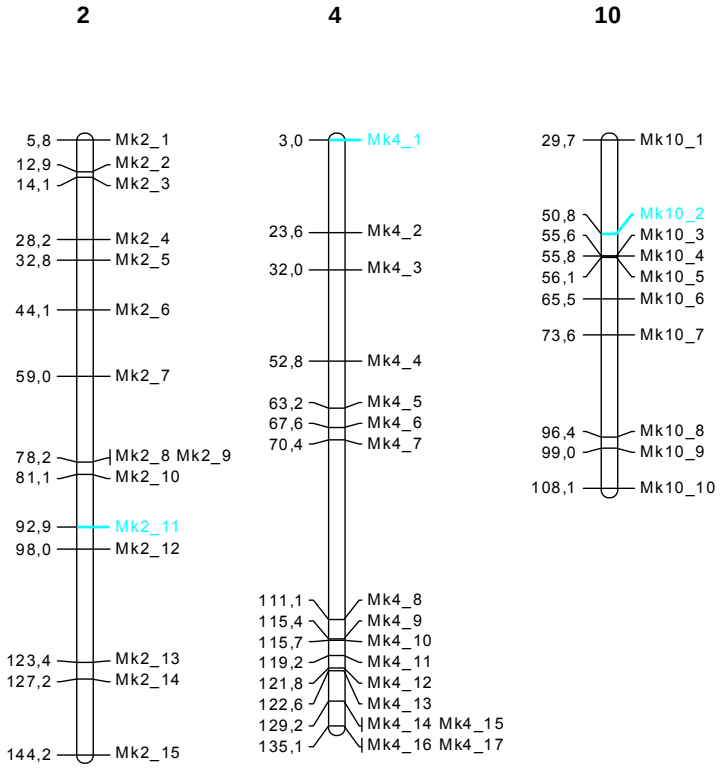

Figure 4. *QTL´s detected to common rust resistance associated with their linkage groups.(light blue numbers are markers linked with QTL´s)*
Figura 4. *QTL´s detectados para resistencia a roya común asociados con sus grupos de ligamiento.*

R^2 values associated with individual QTL´s are low, but they are within the range of values reported for QTL´s controlling resistance to fungi and insects in maize (Freymark *et al*., 1993; Pè *et al*., 1993; Groh *et al*., 1998; Khairallah *et al*., 1998; Pérez Brito *et al*., 2001).

Common rust
Three markers with significant associations (P< 0,05) with common rust were identified by comparing the phenotypic scores of the genotypic classes for each marker. Significant markers identified three QTL´s located on linkage groups 2, 4 and 10 (Figure 4). Gene effects of partial dominance towards the resistance phenotype of two QTL´s located on chromosomes 2 and 10 were observed (Table 3). Furthermore, Kerns *et al*., (1999) and Brown *et al*., (2001) found no single-gene, dominance resistance for this disease in experiments in maize to identify QTL´s associated with resistant to common rust.
Epistasis was detected between all possible pairs of markers (Table 4).
R^2 calculated by multiple regression accounted for 45% of phenotypic variation.
The QTL´s of greater relative contribution to phenotypic individual variation, according to the R^2 were those located in chromosome 10 (27%) and 4 (16%) (Table 3). Many dominant, single resistance genes have been characterized and mapped to chromosome 4 (*Rp4*) and chromosome 10 (*Rp1, RP1-G* and *Rp5*) (McMullen & Simcox, 1995; Sanz-Alferez *et al*., 1995). The region on chromosome 10 appears to be an Rp1 complex. This complex may consist of many *Rp* genes instead of the proposed *Rp1* alleles and may be capable of producing novel forms of resistance. This could be due to meiotic instability causing higher amounts of crossing over in

this region and subsequent gene conversions (Richter *et al*., 1995).

***Table 4**. Significant epistasis values detected between all possible marker pairs associated with common rust resistance by two ways ANOVA, and map position.*
***Tabla 4**: Valores de epistasis significativos detectados entre todos los pares de marcadores asociados con la resistencia a la roya común, mediante ANOVA de 2 vías, y posición en el mapa.*

Markers	chromosome/location	P^{∞}
Mk2_11-Mk4_1	2(92,9 cM)- 4(3 cM)	$6{,}66 \times 10^{-8}$
Mk4_1-Mk10_2	2(92,9 cM)- 10(50,9 cM)	$7{,}88 \times 10^{-8}$
Mk4_1-Mk10_2	4(3 cM)- 10(50,8 cM)	$1{,}7 \times 10^{-10}$

Lubberstedt *et al*. (1998) identified 20 QTL´s conferring partial resistance to common rust distributed over all 10 chromosomes, and Kerns *et al*. (1999), found 24 markers in 16 chromosomal regions significantly associated with common rust. Brown *et al*. (2001) identified nine QTL´s in six chromosomes significantly associated with rust severity. All these studies contrast with our findings, which indicate that a small number of QTL´s explained a significant amount of the variation. Specifically, this study showed that three QTL´s were associated with rust resistance. These findings, plus the identification of one QTL on chromosome 10 that explain 27% of phenotypic variation, suggest that selection using markers alone may result in a significant improvement in the level of resistance at the population level.

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