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Genetic mapping of maize stripe disease resistance from the Mascarene source

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Abstract Maize stripe virus (MStV) is a potentially threatening virus disease of maize in the tropics. We mapped quantitative trait loci (QTLs) controlling resistance to MStV in a maize population of 157 $F_{2:3}$ families derived from the cross between two maize lines, Rev81 (tropical resistant) and B73 (temperate susceptible). Resistance was evaluated under artificial inoculations in replicated screenhouse trials across different seasons in Réunion Island, France. Composite interval mapping was employed for QTL detection with a linkage map of 143 microsatellite markers. Heritability estimates across seasons were 0.96 and 0.90 for incidence and severity, respectively, demonstrating a high genotypic variability and a good control of the environment. Three regions on chromosomes 2L, 3 and 5, with major effects, and another region on chromosome 2S, with minor effects, provided resistance to MStV in Rev81. In individual seasons, the chr2L QTL explained 60–65% of the phenotypic variation for disease incidence and 21–42% for severity. The chr3 QTL, mainly associated with incidence and located near centromere, explained 42–57% of the phenotypic variation, whereas the chr5 QTL, mainly associated with severity, explained 26–53%. Overall, these QTLs explained 68–73% of the phenotypic variance for incidence and 50–59% for severity. The major QTLs on chr2 and 3 showed additive gene action and were found to be stable over time and across seasons. They also were found to be included in genomic regions with important clusters of resistance genes to diseases and pests. The major QTL on chr5 appeared to be partially dominant in favour of resistance. It was stable over time but showed highly significant QTL × season interactions. Possible implications of these QTLs in different mechanisms of resistance against the virus or the insect vector are discussed. The prospects for transferring these QTLs in susceptible maize cultivars and combining them with other resistances to virus diseases by conventional or marker-assisted breeding are promising.

Keywords Tropical maize · Quantitative trait loci · MStV · *Peregrinus maidis*

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Introduction

The Maize stripe virus (MStV)—formerly MStpV and recently abbreviated MStV—was first reported in Venezuela in 1974, where it was also known as maize hoja blanca virus (Trujillo et al. 1974). This is an important disease of maize (*Zea mays* L.) occurring in many tropical and sub-tropical countries (Tsai 1975; Nault et al. 1979; Migliori and Lastra 1980; Greber 1981). Losses caused by MStV have generally been minor, although serious outbreaks have been reported from the United States (Niblett et al. 1981), Venezuela (Lastra

and Carballo 1983), and some African countries (Rossel and Thottappilly 1985). The disease is widespread in the Mascarenes (Autrey 1983), especially in Réunion Island, where heavy attacks were regularly reported in the lowlands (Etienne and Rat 1973; Delpuech et al. 1986). The pathogenic agent was shown to be a member of the tenuivirus group (Falk and Tsai 1998), and its only known vector is the planthopper *Peregrinus maidis* (Ashmead 1890) (Homopteran: Delphacidae), which was found to be cosmopolitan and restricted to poaceous hosts. The virus is transmitted in a persistent, propagative manner (Reynaud 1988; Nault and Ammar 1989), and transovarially passed from viruliferous females to their progenies (Nault and Gordon 1988; Ammar et al. 1995). The narrow-range of host plants includes *Zea* spp., *Sorghum* spp., and wild grasses such as *Rottboellia exaltata* (Trujillo et al. 1974; Migliori and Lastra 1980; Greber 1981).

Two striping patterns were described: (1) the initially reported one, called simply maize stripe, characterized by a fine striping on lower leaves that evolves quickly into broad chlorotic bands of various width along the leaves, and also by a typical gooseneck bending of the infected plants and (2) the more recently reported one, called maize chlorotic stripe, described only in the Mascarenes and characterized by stripes, which rapidly coalesce to form a chlorotic patch on the whole area of the lamina, with the reappearance of green discontinuous parts (Autrey and Mawlah 1984; Marchand et al. 1994). The very initial symptoms of the two syndromes are identical and very close to those of the three types of chlorotic streaks due to Maize mosaic virus (MMV) (Autrey 1980) also transmitted by *P. maidis*. The evolution of symptoms on the leaves is different in plants exhibiting maize stripe from those exhibiting maize chlorotic stripe, and the two syndromes can be distinguished readily in the field (Autrey and Mawlah 1984). For both, the plants present a very severe dwarfism when infected at an early stage. They eventually dry up and die, but often they survive without forming any ear. No serological difference was found between the two pathogenic agents causing maize stripe and maize chlorotic stripe syndromes. Then, it was concluded that both were probably isolates of MStV (Autrey 1983; Gingery and Autrey 1984). The largest part of the striping pattern observed under natural conditions of Réunion Island as well as under artificial inoculation, is maize chlorotic stripe.

As the control of the delphacid populations is not effective, varietal improvement for resistance to MStV and/or to *P. maidis* is the most promising method to reduce the impact of the disease in the infested areas. Until now, neither a classical genetic approach, nor molecular-marker studies have been conducted on the genetic control of the resistance to the virus or to the vector. Resistant lines were selected in maize populations originating from Réunion Island, these being known as excellent sources of resistance to virus diseases (Hainzelin and Marchand 1986). Moreover, a large number of molecular markers are now published and available on maize for building linkage maps, and statistical methods have been designed and progressively improved for QTL detection (for a review, see Liu 1998).

In this study, we mapped QTL for resistance to MStV using a population of $F_{2:3}$ families derived from the cross between Rev81 (resistant) and B73 (susceptible). Our three main objectives were to: (1) understand the genetic foundation of resistance to MStV; (2) make inferences about mechanisms of resistance to the virus or to the vector, by dissecting this resistance; and (3) examine the perspectives of conventional or marker-assisted selection (MAS) to improve resistance in breeding programmes.

Materials and methods

Plant material

Two homozygous inbred lines, Rev81 and B73, were used as the parental material. The resistant parent Rev81, a tropical yellow semi-dent line at S_9 generation, was selected in population 'Revolution' through screenings under natural infestation for the first five cycles and under artificial inoculation for the last four. The temperate line B73 from the Iowa Stiff-Stalk Synthetic was the susceptible parent. During the 1998 austral winter, F_2 plants derived from a single F_1 plant from the Rev81 × B73 cross were selfed to produce 295 F_3 lines. The seeds obtained from the selfed F_1 plant were treated by systemic insecticide Gaucho (490 g/100 kg seeds) to avoid any virus infestation on the plant that could induce selection of the F_3 kept for the study. Leaf samples were taken from a random subset of 157 parental F_2 for DNA extraction and subsequent SSR mapping. These 157 F_2 plants produced enough seeds for field trials.

Field trials and experimental design

Four trials were conducted under artificial inoculation during different cropping seasons in the period 1999–2000, at the experimental station of Centre de Coopération Internationale en Recherche Agronomique pour le Développement (CIRAD) in Saint-Pierre, Réunion (lowland tropical environment, 140 m elevation, 20°S). They were named 99-A for January–February 1999 (hot and wet); 99-B for May–June 1999 (cool and semi-wet); 99-C for October–November 1999 (hot and dry); 00-C for August–October 2000 (cool and dry). Each experiment included 160 entries: 157 F_3 lines, the two parental inbred, and the F_1 generation as checks. The partially resistant tropical hybrid PAN 6191 (Pannar Seeds, South Africa) was used as an internal control to monitor the level of infection in the cages. The experimental design was a 20 × 8 alpha-lattice design (Patterson and

Williams 1976) with two replications. Plots consisted of single rows, 0.75 m apart and 5 m long, with 24 infested plants each. Twenty incomplete blocks of eight entries each were arranged and planted under ten individual insect-proof screenhouses for replication 1 and under a large unique insect-proof screenhouse for replication 2, so that each screenhouse or compartment of screenhouse contained two incomplete blocks plus the internal check (PAN 6191).

Artificial inoculation and disease assessment

Infestations were conducted with viruliferous planthoppers mass-reared at the CIRAD station, St-Pierre, Réunion. The MStV isolate used in this study originated from the CIRAD station and was maintained in the mass rearing cages on a genotype susceptible to MStV, but resistant to MMV to avoid contaminations by this second virus, which is present on the station and also transmitted by *P. maidis*.

Seeds of each entry tested were germinated in a greenhouse. At the three-leaf stage, the seedlings were carried into mass rearing cages where they stayed during 3 days under a massive inoculation with viruliferous planthoppers. One cage contained 16 entries corresponding to two incomplete blocks and plants of the internal check PAN 6191 placed in the centre of the cage. Planthoppers were equally dispatched between the cages in which they were regularly moved so that they spread over all the plants, avoiding inoculation escape. Then, infested plants were removed from the cages, treated with insecticide and transplanted under the insect-proof screenhouses. In the screenhouses, the plants were treated with a systemic insecticide once a week to avoid secondary infestations from insects having survived or from outside.

Symptoms were evaluated on the last fully expanded leaf of each plant using a visual 1–10 scale, with 1 corresponding to a plant without symptom; 9 corresponding to the maximal development of the disease on the leaf, with complete stunting and no ear formed, but plant still alive; and 10 corresponding to a plant dead because of the disease. These ratings were taken once per week from 7 to minimum 56 days after inoculation (dai). Plants presenting symptoms of other viral diseases were systematically discarded.

Variables description

The analysed variables, calculated on a plot basis at the uth scoring date, were the disease score (SCO_u), as the mean rating of all plants showing symptoms or not, the disease incidence (INC_u), as the proportion of plants presenting symptoms, the disease severity (SEV_u), as the mean disease score of the plants presenting symptoms. In order to integrate these variables over time, the area under the disease progress curve (AUDPC) (Jeger and Viljanen-Robinson 2001), called SCO_a for the disease score, INC_a for the disease incidence, and SEV_a for the disease severity, was calculated as $\sum_{i=1}^{n-1}\{([y_i + y_{i+1}]/2)(t_{i+1} - t_i)\}/(t_n - t_1)$, in which y_i was the proportion or the mean symptoms rating at the ith rating date; t_i is the time at the ith observation; n is the number of dates at which disease was recorded. The AUDPC was standardized, by dividing the value by the total time duration ($t_n - t_1$) of the disease progress study. The first interval of time was between 0 and 7 dai. At 0 dai, all plants were symptomless. The total length of time of the disease study was 56 dai in 99-A, 70 dai in 99-B, 63 dai in 99-C and 00-C.

SSR assays

The DNA was extracted from the lyophilized leaves of the F_2 plants as well as of the parents Rev81 and B73, and the F_1 generation plants, following the protocol of Saghai Maroof et al. (1984). The SSR primer sequences were obtained from MaizeGDB (http://www.maizegdb.org). Two hundred seventy-six SSRs showing polymorphism between the two parents were used to genotype the F_2 population. They were chosen to provide a good coverage of the genome, according to their bin locations presented in the public SSR's list of MaizeGDB.

Microsatellite amplifications were performed in a 25-µl final volume containing 25 ng DNA, 5 pmol each primer, 200 µM each dNTP, 2 mM $MgCl_2$, one-time buffer and 1 U *Taq* polymerase, using a Perkin-Elmer 9600 Thermal cycler with the following conditions: 94°C for 5 min (one cycle); 94°C for 30 s, 53°C for 1 min, 72°C for 1 min (35 cycles); 72°C for 8 min (one cycle); and continuous cycle at 4°C. Approximately 75% of the polymorphic probes were resolved in CIRAD station, Saint-Pierre, Réunion, by using simple electrophoresis separation on simple (4% SeaKem) or high-resolution agarose (4.5% MetaPhor). Markers were visualized under UV after dipping the gel 20 min in a solution of 300 µg l^{-1} ethidium bromide. Due to very small differences in alleles size, the other probes needed to be resolved with polyacrylamide gels, using radioactive labelling in CIRAD-Montpellier, France. Polymorphisms were visualized by labelling the F primer with [^{33}Pγ]-ATP using T4 polynucleotide kinase. The PCR products were added to an equal volume of stop solution and heated for 3 min at 92°C. A 5-µl aliquot of the reaction mixture was analysed by 5% denaturing polyacrylamide gel electrophoresis and autoradiography.

Field data analyses

Analyses of variance were conducted for each trait within each trial separately and across trials to estimate the environmental effect, using the SAS Mixed procedure (SAS, version 6.12; SAS Institute, Cary, N.C.,

USA). Block and family factors were considered to be random, and replication and season to be fixed. On a plot mean basis, the total variation was partitioned as follows:

$$Y_{ijkl} = \mu + S_i + Rep_{ij} + (Block/Rep)_{ijk} + F_l + (FxS)_{il} + e_{ijkl}$$

where Y_{ijkl} is the variable measured on the lth F_3 family in the kth block of the jth replication in the ith trial (season), μ, the general mean and e_{ijkl}, the residual error. The best linear unbiased predictors [(BLUPs) Henderson 1975] were obtained by adding the general mean of the trial to the solution of the random family effect.

Broad sense heritability (h^2) on an entry-mean basis was calculated at the experimental design level for each season and across the four seasons, according to Hallauer and Miranda (1981). Exact 90% confidence intervals of h^2 were calculated from Knapp et al. (1985). Phenotypic ($\hat{r}_p$) and genotypic ($\hat{r}_g$) correlation coefficients were calculated among traits in F_3 lines (Mode and Robinson 1959).

In each trial, an analysis of variance was performed on phenotypic data obtained from the internal check to estimate the effect of the cage in the experimental design, using SAS GLM procedure according to the model $Y_{ij} = \mu + Rep_i + Cage_{ij} + e_{ij}$, where $Cage_{ij}$ is the effect of the jth cage in the ith replication.

Segregation and linkage analyses

The segregation at each locus was checked for deviations from expected Mendelian segregation ratios (1:2:1 or 3:1), and the observed allele frequency for deviations from the expected 0.5 by standard χ^2 tests. Because multiple tests were performed (corresponding to the number of SSR markers assayed), appropriate type I error rates were determined by the sequentially rejective Bonferroni procedure (Holm 1979). Using MAPMAKER, version 3.0b (Lander et al. 1987), linkage between two loci was declared significant in the two-points analysis when the LOD score ($\log_{10}$ of the likelihood odds ratio) exceeded the threshold of 3.0. After determination of linkage groups and the correct linear arrangement of loci along the chromosomes, recombination frequencies between loci were estimated by multi-point analyses and transformed into centi-Morgans (cM) applying the Haldane mapping function (Haldane 1919).

QTL analyses

The QTL analyses were performed in each season and across the four seasons on BLUPs values of the 157 families. For mapping QTLs and estimating their effects, the method of composite interval mapping (CIM) (Jansen and Stam 1994; Zeng 1994; Jiang and Zeng 1995) was employed using PLABQTL (Utz and Melchinger 1996). For each trait analysed, a set of cofactors was selected by stepwise regression for the analyses of data from each season as well as for the analysis of average data across seasons. A dominance model of inheritance was assumed but final selection was for the model that minimized Akaike's information criterion with penalty = 3 (Jansen 1993).

According to permutation tests (Doerge and Churchill 1996), an average LOD score threshold across traits of 4.0 was chosen for declaring a putative QTL significant, as well for incidence as for severity. In a F_2 population ($df = 3$), this threshold ensures a comparison-wise type I error $P_c < 0.0004$ in the mapping of QTL. Estimate of QTL position was obtained at the point where the LOD score assumes its maximum in the region under consideration. A one-LOD support interval was constructed for each QTL, as described by Lander and Botstein (1989). Two QTLs with non-overlapping support intervals were regarded as being different.

The proportion of the phenotypic variance explained by the ith detected QTL was obtained by the square of the partial correlation coefficient (R_i^2). Estimates of the additive ($\hat{a}_i^2$) and dominance ($\hat{d}_i^2$) effects of the ith QTL, and the total $\hat{\sigma}_p^2$ explained by all QTLs were obtained by fitting a model including all putative QTLs detected for the respective trait. The hypotheses of no additive or dominance effect of the QTL (H_0: $a_i = 0$ or H_0: $d_i = 0$) were tested by an F-test. The type of gene action at each QTL in the F_2 population was characterized by calculating the dominance ratio $DR = |2d_i/a_i|$ (Stuber et al. 1987): additivity for $DR < 0.2$; partial dominance for $0.2 \le DR < 0.8$; dominance for $0.8 \le DR < 1.2$; overdominance for $DR \ge 1.2$. As it was estimated from F_3 families, the dominance effect d_i given by the programme had to be multiplied by 2 to obtain the correct dominance estimation.

The QTL × season interaction variance was estimated by fitting a model to the BLUPs from each season, which included all QTLs detected in the analysis across seasons. The partition of the combined ANOVA into variations due to the QTL action, the QTL × season interaction and the residual, as well as the test of hypothesis of no significant QTL × season interaction were performed as described by Bohn et al. (1996). Putative QTLs were also examined for the presence of digenic epistatic interactions.

Results

Segregation and linkage of SSRs

Of 574 SSR probes screened on the parental lines, 276 detected polymorphism. Of those, 159 were used to genotype the F_2 population and resulted in 143 linked loci, of which 142 were codominant, and only one dominant from the susceptible parent (B73). Seven out

of the 143 marker loci showed a significant ($P < 0.01$) distortion from Mendelian segregation ratios. No marker deviated significantly ($P < 0.01$) from the expected allele frequency and the proportion of resistant parent (Rev81) genome among the 157 F_2 individuals ranged from 32.5% to 64.0%, with $\bar{x} = 51.1\%$ and SD = 6.4%, which was not significantly different from the expected 50% under Mendelian segregation. The level of homozygosity in F_2 plants, followed approximately a normal distribution and ranged from 28.4% to 63.4%, with $\bar{x} = 47.0\%$ and SD = 8.1%. A highly significant ($P < 0.001$) lower value than the expected 50% was noted at 16 loci. Finally, our SSR linkage map was in good agreement with the Maize Microsatellite–RFLP consensus map (Romero-Severson 1998) and other maize maps built with SSRs, like those from the Missouri Maize Project (http://www.agron.missouri.edu). All loci were significantly linked (LOD > 3.0) to one of the ten linkage groups and mapped to the right bin. The 143 markers spanned a map distance of 1615 cM with an average interval length of 12.2 cM (Fig. 1).

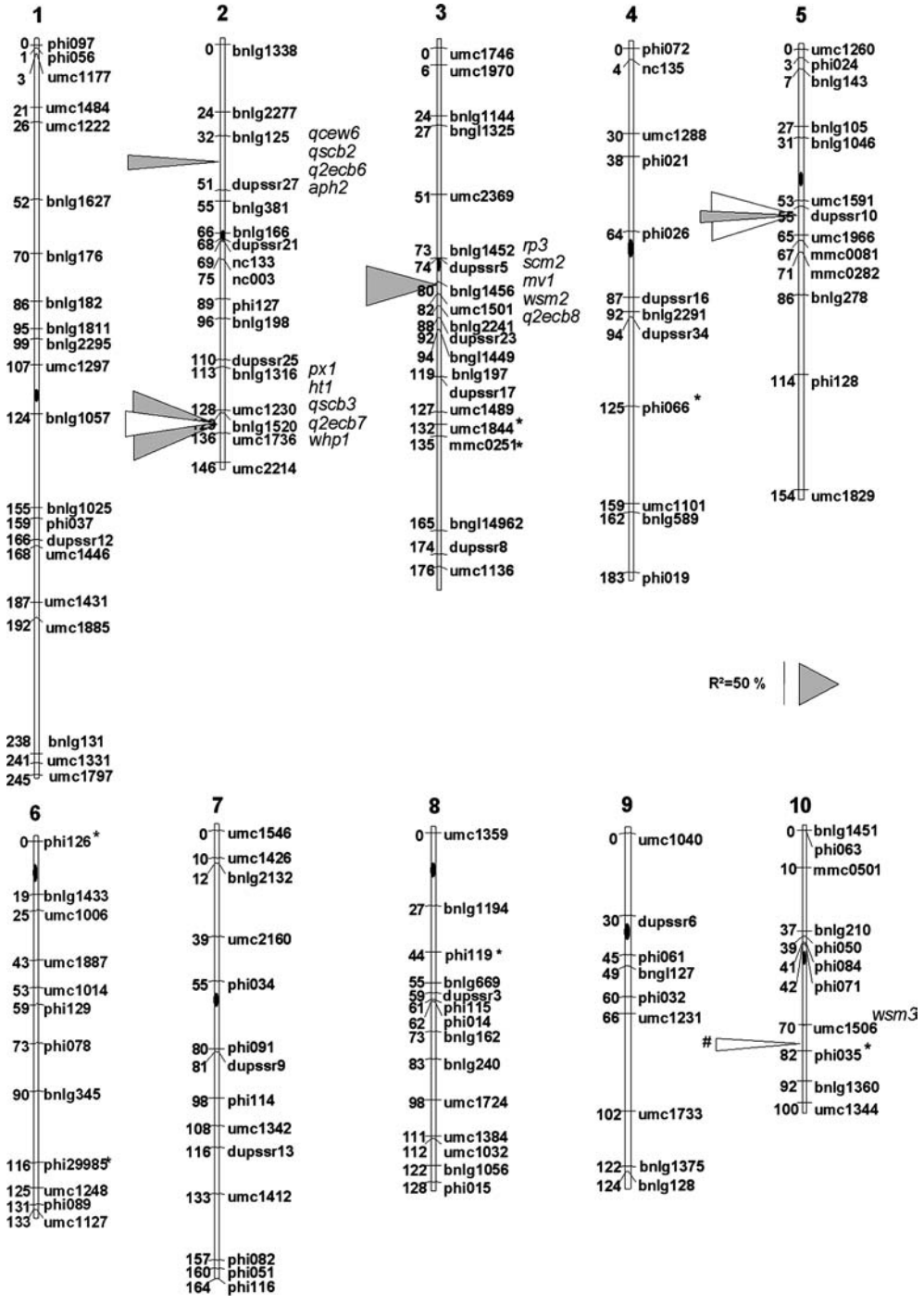
Field trait analysis

Although the artificial inoculations were successful across the four seasons, the infestation level in 99-C was lower in comparison to the other seasons, whereas the strongest one was obtained in 99-B, the 99-A and 00-C being intermediate. However, from 21 dai, 100% of the plants of the susceptible parent B73 exhibited heavy symptoms in each season, except in 99-C (3% of the B73 plants remained symptom-free in that trial), and several of them died from 42 dai. The parent Rev81 showed a high but incomplete resistance, with a maximum disease incidence level varying from 16% to 36%, depending on the season. With time, all genotypes had a tendency to reach high values of disease severity, close to the one exhibited by the susceptible B73 and with a delay in regard of that one, especially for Rev81 in which the progression of foliar symptoms was reduced. The internal check PAN6191 was partially resistant to the disease incidence and stabilized between 65% and 85%, depending on the season. Using the scoring values of PAN6191 in each cage, the ANOVA, on an individual or a multi-season basis, showed no significant effect of the cage on the level of both incidence and severity obtained on the infested plants.

In individual seasons, normality of the distribution of the residual errors was generally met for all score and incidence traits, except at 7 dai and sometimes at 14 dai, whereas it was most often met only at the AUDPC and at 28 dai, for severity traits. Across seasons, normality of the residual distribution was met for the variables SCO_a, SCO_{21}, SCO_{28}, INC_a, INC_{14}, SEV_{21} and SEV_{28}. Genotypic variance component ($\hat{\sigma}^2_F$) was highly significant for all traits across seasons and for each season individually ($P < 0.001$), except in 00-C for SCO_7 ($P < 0.01$), INC_7 ($P < 0.01$) and SEV_7 ($P < 0.05$). The seasons were significantly ($P < 0.001$) different for all score, incidence and severity variables. The genotype × season interaction variance component ($\hat{\sigma}^2_{FxS}$) was significant at $P < 0.001$ for the disease score at 7, 14, 21 and 28 dai, significant at $P < 0.05$ for the disease score at 42 dai and for the AUDPC, and non-significant for the disease score at 56 dai. It was significant at $P < 0.001$ for all the variables related to the disease severity, except SEV_7, whereas for the variables related to the disease incidence, it was significant only for INC_7 ($P < 0.001$). However, estimates of σ^2_{FxS} were much smaller than $\hat{\sigma}^2_F$, except at the earliest scoring dates for the three groups of variables, and also except at the latest ones for severity variables. This indicated that the effect of the season on the disease incidence component of the resistance was low, relative to the family effect, except for the earliest dates, whereas it has influenced the severity and score components, depending on the family genotype. Heritabilities across seasons, at the experimental design level, were high for score and incidence variables and ranged from 69% for SCO_7 to 96% for SCO_a and from 80% for INC_7 to 96% for INC_a, increasing rapidly with time after inoculation and stabilizing after 21 dai. They were moderate to high for severity variables and ranged from 62% for SEV_7 and SEV_{56} to 90% for SEV_{28} and SEV_a, increasing rapidly with time after inoculation and then decreasing after 28 dai.

Phenotypic ($\hat{r}_p$) and genotypic ($\hat{r}_g$) linear correlations between resistance traits in F_3 line families were positive and highly significant ($P < 0.001$) for all trait combinations within score, incidence, and severity group. Within those groups, some traits showed lower correlations than the other ones. The coefficients of correlation between these traits were tested (Table 1). They were extremely high between SCO_a and INC_a, as well as between SCO_7 and INC_7, so that we could consider that the incidence component of the resistance was strongly associated with disease score. Coefficients of correlations for the other combinations ranged from intermediate to very high, except those involving SEV_{28} with INC_a and INC_7, as well as those involving SEV_{56} with INC_a, INC_7, SEV_{14}, and SEV_{28}, which were of a lower value. Nevertheless, we selected INC_a and SEV_a as the most appropriate traits for representing incidence and severity components of the resistance, respectively. The means, variance components and heritability estimates for these two principal resistance characters were presented for each season individually and across the four seasons (Table 2). No significant differences among the mid-parent value and the F_1 and F_3 generation means, and consequently no significant mid-parent heterosis, were observed for these two traits.

Resistance to MStV was a quantitatively inherited character, as shown by distributions of incidence and severity BLUPs obtained per family (Fig. 2). These distributions followed near-normal distribution and were skewed toward susceptibility when the infestation level of the trial increased across the four seasons. The



parental lines were situated close to the boundaries of the distributions for incidence and outside of these boundaries for severity, so that no transgressive segregants could be noted in any direction for these two characters.

QTL analysis

The QTL analyses were performed on incidence and severity traits using genotypic data from 143 SSR loci and BLUPs obtained from individual seasons as well as across seasons. Although detailed QTL analyses were performed only for the two main reliable traits INC_a and SEV_a, incidence and severity of the disease at different dates were also examined, in order to identify genetic factors, which could be involved in the expression of resistance at earlier or later stages.

Composite interval mapping, employing four to eight cofactors for incidence and five to seven cofactors for severity, resulted in five significant QTLs located on chromosomes 2, 3, 5 and 10 (Table 3). The major QTL

◄

Fig. 1 Linkage map for 143 SSR markers based on 157 $F_{2:3}$ families derived from the cross Rev81 × B73 and approximate map position of five quantitative trait loci (QTLs) for resistance to Maize stripe virus (MStV) found from a study across four seasons. Other resistance genes and QTLs against various viral and fungal diseases and insects of maize reported in the literature are located on the *right side* of the chromosome. Numbers to the *left* of the chromosome indicate distance in centiMorgans *relative to the first marker*. The approximate position of the centromere is represented by a *black rectangle*. Marker loci with a significant distorted segregation are marked by an *asterisk*. *Tops* of *triangles* mark LR peak positions of each QTL. The QTL detected are represented by *grey* and *white triangles* for the disease incidence and severity, respectively. The width of the triangle basis is proportional to the percentage of the phenotypic variation (R^2) explained by the QTL under consideration. The *crosshatch* indicates that the allele increasing MStV resistance is contributed by the susceptible parent

in bin 2.09 was associated with the two components of the resistance, but the percentage of phenotypic variation explained by this QTL was much lower for severity than for incidence (Table 3). Another QTL with major effect was detected in bin 3.05; across season, it was specific of the incidence component of the resistance and not significant for the severity component. In contrast, a major QTL in bin 5.04 appeared to be mainly associated with severity component of the resistance, although it was significantly detected for incidence component in seasons 99-A, 00-C and across seasons. Two minor QTLs also were identified: in bin 2.02, as strictly specific of incidence and only detected across seasons; and in bin 10.06, as strictly specific of severity and detected in each individual season and across seasons. Alleles contributing to the resistance came from the resistant parent Rev81, except for the QTL in bin 10.06, which originated from the susceptible parent B73.

The percentage of phenotypic variance explained by putative QTLs was higher for the incidence than for the severity component, as shown by R^2 in individual seasons and across seasons obtained from the multiple-locus model (Table 3). Incidence component of the

Table 1 Phenotypic ($\hat{r}_p$) and genetic ($\hat{r}_g$ in *boldface*) correlation coefficients among resistance traits, estimated in a population of 157 F_3 lines derived from the cross Rev81 × B73

Trait	SCO_a[a]	SCO_7	INC_a	INC_7	SEV_a	SEV_{14}	SEV_{28}	SEV_{56}
SCO_a		0.80	0.99	0.84	0.76	0.81	0.70	0.42
SCO_7	**0.90**		0.80	0.97	0.59	0.80	0.53	0.18
INC_a	**0.99**	**0.90**		0.85	0.67	0.75	0.61	0.36
INC_7	**0.92**	**0.99**	**0.92**		0.60	0.79	0.54	0.22
SEV_a	**0.79**	**0.70**	**0.71**	**0.69**		0.84	0.96	0.64
SEV_7	**0.91**	**0.92**	**0.85**	**0.90**	**0.95**		0.80	0.34
SEV_{28}	**0.73**	**0.65**	**0.64**	**0.62**	**0.99**	**0.94**		0.49
SEV_{56}	**0.52**	**0.32**	**0.46**	**0.35**	**0.70**	**0.51**	**0.58**	

All phenotypic correlation coefficients were significant at the 0.0001 probability level

All genetic correlation coefficients exceeded two times their standard error

Selected traits for showing lowest coefficient of correlation between them, within disease score (*SCO*), disease incidence (*INC*) and disease severity (*SEV*) groups

[a] SCO_a, INC_a and SEV_a, Area under the disease progress curve for the disease score, disease incidence and disease severity; SCO_7 and INC_7 disease score and disease incidence at 7 days after inoculation;

SEV_{14}, SEV_{28} and SEV_{56} disease severity at 14, 28, and 56 days after inoculation

Table 2 Means of parent lines Rev81 and B73, mid-parent ($\bar{P}$), F_1 generation ($\bar{F}_1$), 157 F_3 families ($\bar{F}_3$) derived from maize population Rev81 × B73, and infestation check PAN6191, as well as estimates of variance components and heritabilities among F_3 families, for the area under the MStV incidence progress curve (INC_a) and the MStV severity (SEV_a), evaluated in each trial and across four trials over the period 1999–2000

Parameters	INC_a (%)					SEV_a (2–10 scale)				
	99-A[a]	99-B	99-C	00-C	Across	99-A	99-B	99-C	00-C	Across
Means[b]										
Rev81	11.1 ± 5.9	29.1 ± 6.6	13.6 ± 0.5	16.2 ± 9.6	17.5 ± 3.6	3.95 ± 0.07	5.21 ± 0.27	3.91 ± 0.30	3.36 ± 0.05	4.11 ± 0.27
B73	93.8 ± 0.0	95.0 ± 0.0	86.9 ± 7.1	93.7 ± 0.0	92.1 ± 2.1	7.79 ± 0.41	7.86 ± 0.17	7.15 ± 0.35	6.99 ± 0.42	7.51 ± 0.20
$\bar{P}$	52.4 ± 3.0	62.1 ± 3.3	50.2 ± 3.3	55.0 ± 4.8	54.9 ± 2.2	5.87 ± 0.17	6.54 ± 0.23	5.53 ± 0.32	4.97 ± 0.18	5.78 ± 0.21
$\bar{F}_1$	57.6 ± 2.2	70.0 ± 1.6	35.7 ± 12.9	55.9 ± 8.9	54.8 ± 5.5	6.25 ± 0.28	6.48 ± 0.20	5.40 ± 0.33	4.83 ± 0.08	5.74 ± 0.27
$\bar{F}_3$	49.9 ± 1.2	62.3 ± 1.0	46.6 ± 1.1	48.7 ± 1.0	51.9 ± 0.6	6.08 ± 0.02	6.53 ± 0.02	5.65 ± 0.03	5.05 ± 0.03	5.83 ± 0.02
Range	9.3–91.1	9.7–91.8	3.6–87.5	14.3–81.8	14.3–87.2	4.61–6.88	5.47–7.28	3.15–6.35	3.82–5.91	4.70–6.57
PAN6191	61.0 ± 2.6	74.0 ± 2.3	54.8 ± 2.4	67.4 ± 2.4	64.3 ± 1.4	6.46 ± 0.06	6.58 ± 0.06	5.60 ± 0.04	5.54 ± 0.06	6.04 ± 0.06
Variance components (F_3 families)[c]										
$\hat{\sigma}^2_F$	286 ± 39	227 ± 30	262 ± 34	216 ± 29	244 ± 29	0.13 ± 0.02	0.09 ± 0.01	0.14 ± 0.00	0.17 ± 0.02	0.12 ± 0.02
$\hat{\sigma}^2_{FxS}$					3 ± 4					0.02 ± 0.00
$\hat{\sigma}^2_e$	94 ± 12	60 ± 8	65 ± 9	65 ± 8	72 ± 5	0.06 ± 0.01	0.04 ± 0.01	0.10 ± 0.01	0.07 ± 0.01	0.06 ± 0.00
Heritability (F_3 families)[d]										
$\hat{h}^2$	0.86	0.88	0.89	0.87	0.96	0.83	0.81	0.74	0.84	0.90
90% C.I on $\hat{h}^2$				0.95–0.97					0.88–0.92	

[a] *99-A* Hot and wet season 1999, *99-B* fresh and semi-wet season 1999, *99-C* hot and dry season 1999, *00-C* fresh and dry season 2000, and *Across* multi-season analyses

[b] Standard errors are attached

[c] $\hat{\sigma}^2_F$, $\hat{\sigma}^2_{FxS}$, $\hat{\sigma}^2_e$ Estimates of the variances between families, of families × season interactions and residual, respectively. All variances $\hat{\sigma}^2_F$ were significant at $P < 0.001$. Variances $\hat{\sigma}^2_{FxS}$ were significant at $P < 0.001$ for severity, non-significant for incidence

[d] $\hat{h}^2$ Broad-sense heritability at the experimental design level

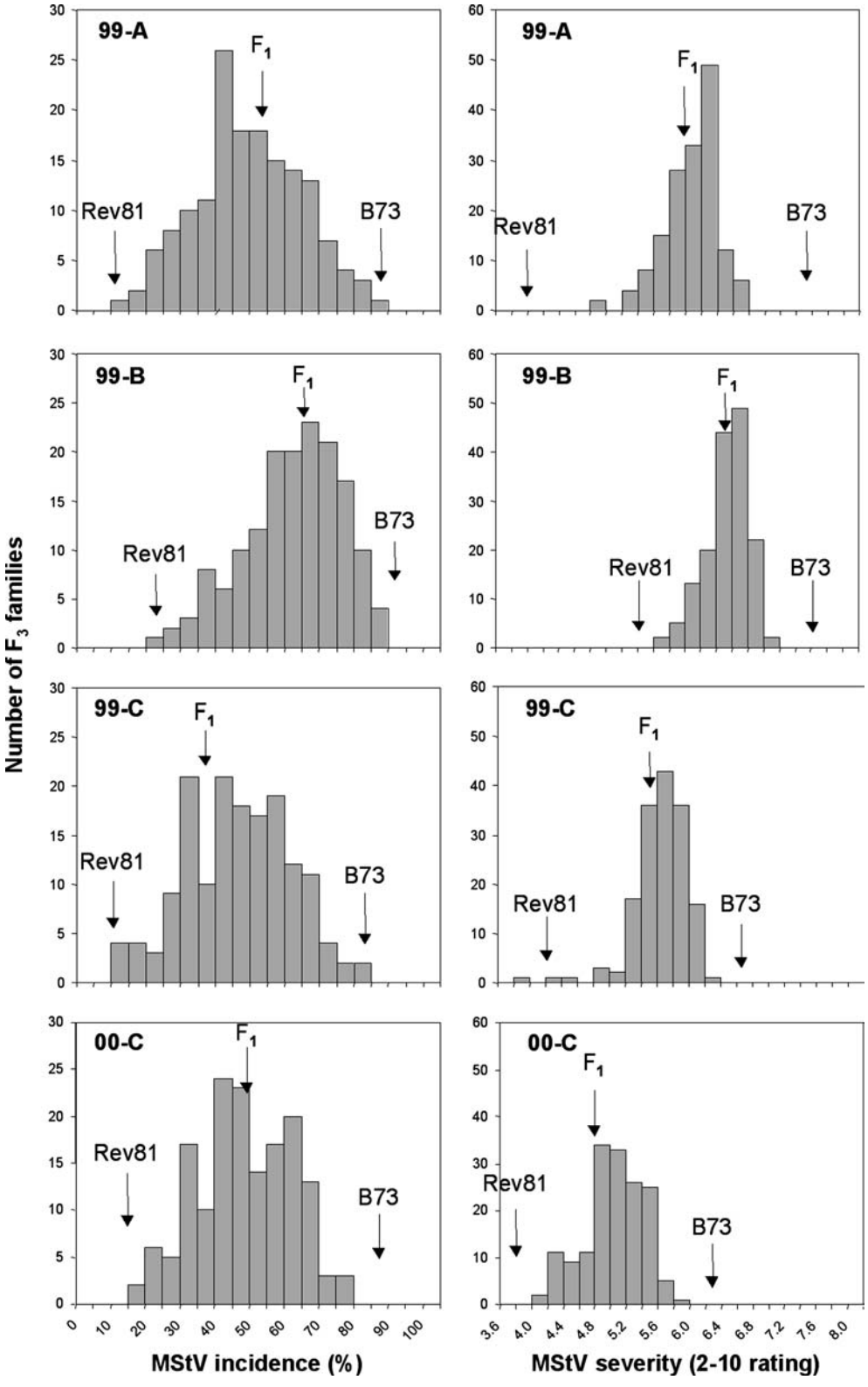


Fig. 2 Histograms for INC_a [area under the disease progress curve (AUDPC) values of MStV incidence] and SEV_a (AUDPC values of MStV severity) measured in Réunion, France, at four seasons over the period 1999–2000, for best linear unbiased predictors (BLUPs) of 157 F_3 families derived from the cross Rev81 × B73. *Arrows* indicate the means of parental lines Rev81 and B73, and of the F_1 generation

resistance was found to be an additive trait, whereas severity component was found to be partially dominant mainly due to significant dominance effects at QTL in bin 5.04.

The QTLs associated with incidence were found to be stable with time and were detected from 7 dai, except the

minor one in bin 2.02, which was detected after 21 dai; in contrast, the QTLs associated with severity were only detected from 14 to 42 dai (data not shown). For incidence, the proportions of phenotypic variance explained by the major QTLs in bins 2.09 and 3.05 reached their maximum and stabilized at 21 dai. For severity, the proportion explained by the QTL in bin 5.04 reached its maximum at 21 dai, then decreased, and became non-significant at 56 dai; the proportion explained by the QTL in bin 2.09 was maximum at 14 dai, then decreased from 14 dai to 42 dai, whereas the proportion explained by QTL in bin 10.06 was approximately constant from 21 to 42 dai (data not shown).

For incidence, the major QTL in bin 2.09 exhibited an excellent stability across the four seasons for each scoring date from 21 dai (data not shown) as well as for AUDPC (Table 3), with almost no significant QTL × season interactions; on the other hand, significant but low QTL × season interactions ($P < 0.05$) were observed at the QTL in bin 3.05 for different scoring dates (data not shown), but not for AUDPC (Table 3). For severity, significant QTL × season interactions ($P < 0.01$) were observed at the QTL in bin 5.04 for each scoring date (data not shown) as well as for AUDPC (Table 3), at the QTL in bin 2.09 for most of scoring dates (data not shown) but not for AUDPC (Table 3), and at the QTL in bin 10.06 only for 21 dai scoring date (data not shown).

Extending this model for digenic epistatic effects between QTLs did not increase the R^2 values. Nevertheless, some rare significant digenic epistatic effects were found between the QTLs in bins 2.09, 3.05 and 5.04 for incidence component of the resistance (Table 3).

Discussion

Genetic foundation of resistance to MStV

The MStV is non-mechanically transmissible and must be necessarily inoculated by planthoppers. For mapping studies such as this one, an important prerequisite is to ensure a uniform level of disease for all the families tested. These conditions being almost never met with natural infestation in the field, we chose to conduct the trials under artificial infestations in mass rearing cages. An acceptable spreading of the insect vectors over the cages and a global success of the infestation were assessed by the internal check put in every cage. The analyses of variance did not show any significant effect between cages within each trial either for incidence or severity variables. Moreover, the use of an alpha-lattice design helped to control the environmental heterogeneity, which involved the differences of infestation levels among the cages. It also permitted yielding of BLUP values, which fulfilled the primary requirement of estimating accurate genotypic values of each family of the population. High heritability values, obtained for most traits in individual trials as well as across trials, confirmed the efficiency of the artificial infestations, resulting in a very good differentiation of the families and a high repeatability of the trials.

Genetic control of MStV resistance was never studied before, and this is the first attempt for locating QTLs of resistance to this disease in a maize population. We showed that MStV resistance was quantitatively inherited, and we detected at least five significant QTLs on chromosomes 2, 3, 5, and 10, which together explained approximately between 30% and 80% of the total phenotypic variation, depending on the variable studied. The three major QTLs on chromosomes 2, 3 and 5 were identified with very high significant effects on resistance to MStV in the analyses across seasons as well as in each individual season. Moreover, they were precisely positioned on the maize genome in a confidence interval smaller than 10 cM, with a high probability of containing the true location of the QTL. These QTLs explained a higher proportion of the phenotypic variation for disease incidence than for severity. Thus, the QTLs with the largest effects, in bins 2.09 and 3.05, and the minor one in bin 2.02 together would strongly reduce the probability of infection, whereas the QTLs in bins 5.04 and 10.06 could only delay and slow down the development of symptoms on diseased plants. It is noteworthy that the QTL in bin 10.06 was specifically detected for severity and was contributed by the susceptible parent B73. Previous studies of MSV and MMV resistances in F_2 populations derived from crosses between a resistant line and B73 already indicated that such resistance alleles were coming from the susceptible line B73 (Pernet 1998; Pernet et al. 1999b).

In agreement with our results, other mapping studies on resistances to viruses in maize indicated an oligogenic type of inheritance including from one to three major QTLs with complementary effects on the resistance (McMullen et al. 1994; Welz et al. 1998; Xia et al. 1999; Pernet et al. 1999a). In many cases, the type of action of the resistance genes ranged from partial dominance to dominance (Louie et al. 1991; McMullen et al. 1994; Xia et al. 1999; Pernet et al. 1999a, 1999b). Nevertheless, oligogenic inheritance of virus resistance with additivity was already reported, for example concerning some components of MSV and MMV resistances (Pernet 1998; Pernet et al. 1999a, 1999b).

The three largest QTLs were stable for all dates and none of the two minor QTLs detected appeared to be specific of the expression of early or late resistance. Nevertheless, we cannot exclude a possible action of some minor modifier genes specifically involved in early resistance but not detected because of difficulty to evaluate with enough accuracy the symptoms at the first stages of the disease development. On the other hand, the drastic reduction of the genetic effects on the severity component of the resistance at the later scoring dates (> 42 dai) may be considered as typical of a partial resistance being overcome by the virus and resulting in

Table 3 Parameters associated with putative quantitative trait loci (QTLs) significantly affecting resistance to Maize stripe virus on INC_a and SEV_a components of the disease in 157 F_3 families of the maize population Rev81 × B73 measured in four seasons

Chr. bin[a] position	Parameters[b]	INC_a(%)					SEV_a (2–10 scale)				
		99-A	99-B	99-C	00-C	Across	99-A	99-B	99-C	00-C	Across
2.02	$a_{2.02}$					3.9**					
32-44-54	$d_{2.02}$					0.4					
	$R^2_{2.02}$					14.7					
	$a_{2.02} \times S$					ns					
	$d_{2.02} \times S$					ns					
	Action					A					
2.09	$a_{2.09}$	15.6**	14.6**	15.6**	13.1**	15.9**	0.16**	0.21**	0.21**	0.20**	0.22**
130-132-134	$d_{2.09}$	−1.4	−0.2	−1.6	−1.6	−0.8	0.04	0.02	0.10*	0.07	0.07
	$R^2_{2.09}$	62	65	60	61	76	21	42	30	21	32
	$a_{2.09} \times S$					ns					ns
	$d_{2.09} \times S$					ns					ns
	Action	A	A	A	A	A	A	A	D	A	A
3.05	a_3	12.0**	9.0**	10.8**	11.7**	12.3**	0.15**		0.15**		
78-80-82	d_3	−1.4	2.9*	1.2	1.3	0.9	0.05		0.05		
	R^2_3	54	42	43	57	63	15		18		
	$a_3 \times S$					ns					
	$d_3 \times S$					ns					
	Action	A	PD	A	A	A	A		A		
5.04	$a_{5.04}$	4.3**			3.6**	3.7**	0.23**	0.23**	0.16**	0.34**	0.26**
52-56-58	$d_{5.04}$	−0.3			−0.8	−0.7	0.09*	0.04*	0.08*	0.11*	0.10**
	$R^2_{5.04}$	18			12	13	38	26	26	53	50
	$a_{5.04} \times S$					ns					**
	$d_{5.04} \times S$					ns					ns
	Action	A			A	A	D	A	D	PD	PD
10.06	a_{10}						−0.16**	−0.10**	−0.11**	−0.12**	−0.16**
70-78-94	d_{10}						0.09*	0.04	0.10*	0.15**	0.08
	R^2_{10}						21	12	13	13	18
	$a_{10} \times S$										ns
	$d_{10} \times S$										ns
	Action						A	A	OD	OD	A
Total	R^2 [c]	71	71	68	73	83	50	59	51	55	56
	Action [d]	A	PD	A	A	A	PD	PD	OD	OD	PD
	Epistasis										
	$a_{2.09} \times d_5$	5.68**				3.35*					
	$a_{2.09} \times a_3$		−3.32**								
	$d_3 \times d_5$	−7.30**									
	$a_{2.09} \times a_5$										
Total	R^2[e]	72	71	68	73	82	44	56	50	54	58

*, ** Significant at the 0.05 and 0.01 probability level, respectively; *ns* not significant ($P > 0.05$)

[a]Chromosomes dissected into bin regions and QTL position based on the marker linkage map (Fig. 1) and SSR–RFLP consensus map (Romero-Severson 1998). Position of likelihood peak (maximum LOD) with underlined letters plus support interval in centiMorgans relative to the first SSR marker on chromosome, for analysis across seasons

[b]*a* Additive effect and *d* dominance effect of the QTL. A positive sign of additive effects reflects that parent Rev81 contributed QTL alleles increasing resistance. R^2 estimates the proportion of the phenotypic variance (percentage) explained by the detected QTL. a × S and d × S Interactions between QTL and season for the a and d effect, respectively. Gene type action as described in 'Materials and Methods': *A* additive, *PD* partially dominant, *D* dominant, *OD* overdominant

[c]Estimates of total R^2 obtained by a simultaneous fit of all putative QTL affecting the respective trait using a model with additive and dominance effects

[d]Global additive and dominance effects were obtained by summing the significant additive and dominance effects of all significant QTLs detected for the trait under consideration and then used to calculate the degree of dominance in the F_2 generation and to determine the global gene action

[e]Estimates of total R^2 obtained by a simultaneous fit of all putative QTLs affecting the respective trait using a model including additive, dominance and epistatic effects

severe symptoms observed on all diseased plants of the population after a long period. The QTL × season interactions for additivity were significant ($P < 0.01$) at each date for QTL in bin 5.04, controlling the largest part of the partial resistance to MStV severity. In contrast, the two major QTLs in bins 2.09 and 3.05 were found to be fairly stable for incidence across the four seasons. Of course, this stability does not allow concluding that these QTLs will be stable across different environments and against different MStV isolates or vector biotypes.

Some of the QTLs detected in our study are located in regions well known for containing genes of resistance against various maize pathogens and pests (McMullen and Simcox 1995). This is particularly the case of the major QTL in bin 3.05, linked to marker *bnlg1456* and mapped with high precision near the centromere of chromosome 3, very close to the position of *Wsm2*, a

gene for resistance to Wheat streak mosaic virus (McMullen et al. 1994), *Scmv2*, a gene for resistance to Sugarcane mosaic virus (Melchinger et al. 1998), and *mv1*, the major gene for resistance to MMV (Ming et al. 1997; Pernet 1998). Our results strengthen the conclusions of McMullen and Simcox (1995) that a cluster of genes for resistance to maize pathogens, especially viruses, is located in this region of chromosome 3. However, the hypothesis of a link between the co-localisation of these genes and a functional relationship in the resistance mechanism to these four viruses has never been investigated.

Resistance mechanisms to MStV

The existence of two QTL sets, partially overlapping for incidence and severity components of resistance to MStV, suggests that, at least, two main mechanisms could be involved in line Rev81. Actually, a reduction of the disease severity seems consistent with a resistance mechanism to the virus *sensu stricto*, whereas a reduction of the disease incidence may be due to a resistance mechanism against the virus as well as against its transmission by the vector. The two types of resistance are indistinguishable from the phenotypical point of view. In other respects, the concept of resistance to transmission remains vague, because of a lack of information on the mechanisms underlying this type of resistance and because of the difficulty to dissociate it from other forms of resistance to the insect vector, and even from resistance to the virus itself. Nevertheless, preliminary studies by electrical penetration graph (EPG) analysis suggested that a partial resistance to transmission is present in line Rev81, independently of virus resistance. As observed in other cases (for a review, see Jones 1987), this type of resistance could slow down the infection in the field by reducing the likelihood of inoculation and acquisition of the virus, but could be overcome by a strong inoculation. Thus, the large number of viruliferous planthoppers we used in our experiments probably overcame resistance to transmission in Rev81. However, the mechanisms of resistance to MStV in Rev81 are unknown, and there is no clear evidence that the major QTLs we found are only associated with a resistance to the virus. Therefore, we cannot exclude the hypothesis that some QTLs would be able to launch defence mechanisms effective against the virus as well as against the vector.

For increasing our knowledge of the resistance mechanisms by genetic mapping, further investigations are necessary. As MStV is not transmissible by classical methods of mechanical inoculation, and an agrobacterium technique is not yet available, other strategies must be developed for dissociating resistance to the virus from resistance to its transmission. A direct strategy would be to search for QTLs associated with EPG parameters describing the feeding behaviour of the vector (Buduca et al. 1996). Unfortunately, this appears as a difficult and time-consuming option, because it needs to release RILs and to produce a very large number of replicated EPG data. At first, an indirect but more feasible strategy would be to test our F_2 population against MMV also transmitted in a propagative manner by *P. maidis*. Such a study should help us to determine more precisely which QTL could be associated with resistance to the virus (virus-specific QTL) and to the vector or to the transmission by the vector (virus-non-specific QTL).

Perspectives for conventional and MAS breeding programmes

An important point is the possibility to use classical methods of recurrent selection for improving the MStV resistance in open-pollinated varieties. The quantitative inheritance and the high heritability of this character, which involves a relatively small number of genes with an additive type action for most of them, suggest that a simple mass phenotypic selection with artificial screening procedure should be very efficient. An S_1 line method is also a very attractive option resulting in a higher selection gain per cycle for traits with pure additive gene action, like the incidence component of the resistance. However, it is more time-consuming and risks of losing other favourable genes as well as reducing the genetic basis of a population are more important. The stability of the major QTLs in bins 2.09 and 3.05 suggests that, at least for the incidence component of the resistance, selection for MStV resistance may be efficient without multi-season tests.

When breeding for releasing resistant hybrids is a major objective, the results of our study can also be used directly in setting up a MAS scheme, to introduce MStV resistance into parental inbred lines. The screening for MStV resistance is quite difficult and time-consuming, because mass rearing of the insect vectors and artificial inoculations are needed to ensure a good and controlled level of infestation. The MAS selection could be used in order to decrease the number of phenotypic evaluations and the number of backcross cycles needed for transferring resistance-related genome segments from Rev81 into lines with good combining ability for yield but susceptible to MStV. The major QTLs in bins 2.09 and 3.05 associated with incidence would be candidates to marker-assisted transfer, but also the QTL in 5.04, associated with severity, and for which partially dominant type of gene action suggests that classical methods of recurrent selection should be less effective than for the two first ones with additive type. An important advantage of MAS compared to conventional backcrossing procedure is that it provides an efficient method to control the transfer of several major QTLs in the same time, avoiding the loss of some of them because their effects may be masked at the phenotypic level.

Another promising prospect would be to constitute maize lines with multiple resistances to virus diseases.

Our increasing knowledge of the determinisms of resistance to MMV and MStV as well as to their common insect vector *P. maidis* is of a primary importance from this point of view. The location of major QTLs of resistance to these viruses and possibly to their vector in two principal regions on chromosomes 2L and 3 near the centromere means that MAS could facilitate the combination of these resistance factors in a recipient line. In a pyramiding scheme, the QTLs of resistance to the *P. maidis*/MStV/MMV complex may be associated with the resistance to MSV, which is quite easy to screen without MAS and directly in the field under artificial infestation (Pernet et al. 1999b). The existence of possible linkages with resistance factors to other diseases or insects in the genomic regions containing the QTLs of resistance to these viruses diseases suggest that such a MAS strategy could be extended to the combination of several resistances or tolerances to biotic stresses.

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