Resistance to Maize Streak Virus in the MSR Pool 9 x CML312 Population, Hunting for non-*Msv1* Resistance Loci

Mark Jones[1], Margaret Redinbaugh[1,2], Ana Nikolic[3], Kevin Pixley[4], Jean-Marcel Ribaut[4], and Richard Pratt[5]

[1]USDA-ARS Corn and Soybean Research Unit, Wooster, Ohio 44691; [2]Department of Plant Pathology, Ohio State University, Ohio Agricultural Research and Development Center (OSU/OARDC), Wooster, OH 44691; [3]Maize Research Institute "Zemun Polje", 11185 Zemun Polje, Belgrade, Serbia; [4]CIMMYT, 06600 Mexico, D.F. MEXICO; and, [5]Department of Horticulture and Crop Science, OSU/OARDC, Wooster, OH 44691.

Introduction

Maize Streak, caused by *Maize streak virus* (MSV), is an important disease that affects sub-Saharan Africa. The use of resistant germplasm is an effective method of controlling yield losses caused by MSV. Inbred lines and populations with high levels of resistance to MSV have been developed with most carrying the *Msv1* locus on chromosome one (bin 1.05). Nine S_5 lines developed from the MSR pool-9 population by the CIMMYT MSV Resistance Breeding Program in Zimbabwe were highly resistant to MSV, but lacked alleles associated with *Msv1* at the SSR markers bnlg439, bmc1016 and bmc2295. This QTL mapping study was conducted to study the resistance in the MSV resistant inbred line MSR176 derived from the MSR pool-9 population.

Maize streak virus-infected plant.

Phenotyping and genetic analysis

MSV infection was rated in $F_{2:3}$ families from the MSR176 x CML312 or MSV-resistant S_5 plants from the MSR pool-9 at CIMMYT-Harare. Plots were artificially inoculated with viruliferous leafhoppers, then rated for disease on two dates using a 1 (no symptoms) to 5 (severe streaking) scale. Data presented are for the mean (a) and lattice adjusted mean (b) (Fig. 1 and 4). SSR markers were used to genotype 248 F_2 and 9 S_5 plants at 58 loci on ten chromosomes. Composite interval mapping (QTL Cartographer ver. 2.5) was used for QTL analysis.

Fig. 1. Graphical Genotypes of MSV Resistant-S_5 and F_2 Progenies.

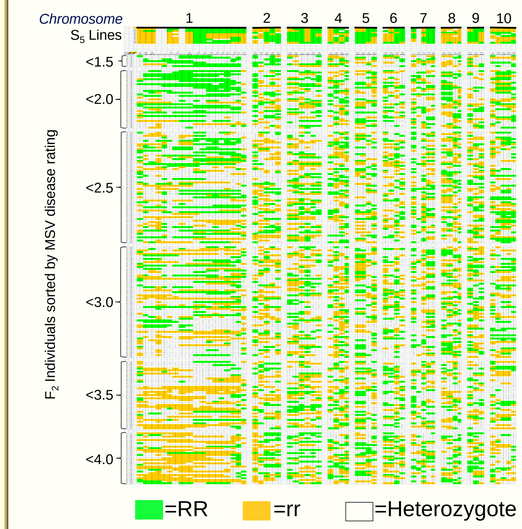
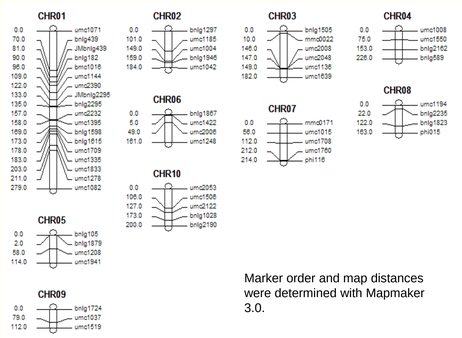
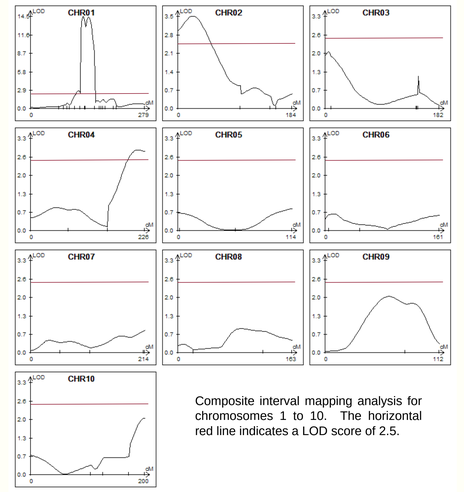


Fig. 2. Genome coverage for SSR markers.



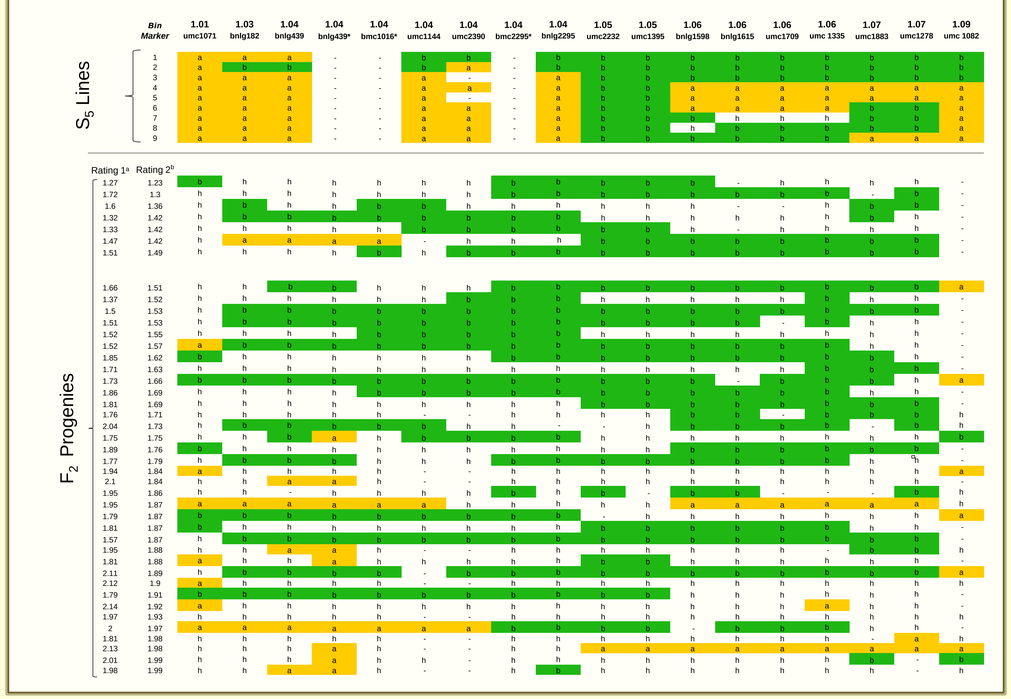
Marker order and map distances were determined with Mapmaker 3.0.

Fig. 3. Composite interval mapping.



Composite interval mapping analysis for chromosomes 1 to 10. The horizontal red line indicates a LOD score of 2.5.

Fig. 4. Chromosome 1 Genotypes in MSV Resistant-S_5 and F_2 Progenies.



Results and Discussion

Analysis of the F_2 population indicated a major QTL with a LOD peak of 14 between umc2390 and umc2232 on chromosome 1 that was centered on bnlg2295 (Fig. 3). This locus is coincident with *Msv1* found in previous studies. Additional minor QTL were found on chromosome 2 (bin 2.02-bin 2.03) between bnlg1297 and umc1185 (LOD 3.5) and chromosome four (bin 4.08) between bnlg2162 and bnlg589 (LOD2.9). These two loci have been found to contribute to MSV resistance in other populations. There was significant marker segregation distortion at markers bnlg439, bnlg1016, and umc1144 in bins 1.03 and 1.04 associated with an excess of heterozygotic plants. Similar distortion has been reported in this genomic region in other studies using several marker types and populations.

Seven of the S_5 lines expressing high levels of MSV resistance were homozygous for the susceptible parent allele at marker bnlg2295, the marker with the highest LOD score for resistance in the F_2 population. These resistant S_5 lines were homozygous for the resistant parent marker allele at the next proximal marker (umc2232) which mapped 22 cM from bnlg2295. Recombination between the markers bnlg2295 and umc2232 and the selection of MSV resistant lines in the inbred line development process could develop lines carrying *Msv1* and the S_5 marker genotype. It is also possible that these lines do lack *Msv1* and the cumulative effect of minor (non-*Msv1*) resistance genes results in the phenotypes seen in these resistant S_5 lines. Such an additional source of resistance may have significance in gene deployment strategies designed to prevent loss of *Msv1* resistance.