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Mapping of QTLs associated with recovery resistance to streak virus disease in maize

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ABSTRACT

Maize streak virus (MSV, genus *Mastrevirus*, family *Geminiviridae*), vectored by the leafhoppers (*Cicadulina* sp.), is the most economically important viral disease of maize endemic to Sub-Saharan Africa and its offshore islands. Yield losses due to MSV are mainly controlled through use of resistant varieties. 'Recovery' (ability of plants to reduce symptom severity), is one of the types of resistance being used to develop MSV resistant cultivars through breeding. This study was conducted to map Quantitative Trait Loci (QTLs) associated with recovery resistance to MSV in a mapping population comprising 250 S1 lines derived from a cross between two MSV resistant lines. The population was genotyped using single nucleotide polymorphism (SNP) markers at 269 loci. A genetic map of 11 linkage groups was constructed comprising 230 SNP markers. Four QTLs, two putative QTLs with significant effect on chromosome 3 and two other QTLs with reduced effect on chromosomes 7 and 9, were identified from the population. The two QTLs on chromosome 3 together accounted for 47 to 51% of the total phenotypic variance while the other two QTLs accounted for 28 to 32% of the total variation. These QTLs originated from the two parents of the mapping population had both additive and dominance effects but interaction among the four loci was not significant. Further validation of these QTLs associated with recovery resistance in other diverse populations will lead to the development of new genomic resources to enhance breeding for MSV resistant maize.

1. Introduction

Maize is a major staple crop in sub-Saharan Africa (SSA) mainly produced by smallholder subsistence farmers. Streak disease caused by the Maize streak virus (MSV, genus *Mastrevirus*, family *Geminiviridae*) is one of the most economically important diseases of maize, endemic to sub-Saharan Africa and its offshore islands. MSV is transmitted by several species of leafhoppers of the *Cicadulina* spp., particularly, *C. mbila* and *C. triangula*, being the most important vector species involved in MSV transmission (Bosque-Pérez, 2000). MSV infection results in chlorotic streaks parallel to the veins due to the destruction of the chloroplast in the leaf lamina resulting in necrotic stripes and wilting of affected portions. In severe cases leaves become totally chlorotic leading to severe necrosis and premature death of the plant before flowering. Affected maize plants may become stunted in growth and have reduced cob size with smaller grains and ears (Engelbrecht, 1982; Rodier et al., 1995; Shepherd et al., 2010). MSV incidence in the fields is unpredictable and varies between years to year resulting in up to 100% yield losses in epidemic years (Martin and Shepherd, 2009). Epidemics resulting in devastating losses of maize harvest due to MSV have been reported in at least twenty African countries (Thottappilly et al., 1993; Wambugu and Wafula, 1999; Bosque-Pérez, 2000; Martin and Shepherd, 2009).

Management of MSV has been difficult owing partly to the unpredictability and sporadic nature of disease appearance and also due to the susceptibility of locally adapted maize cultivars. Resistance breeding has thus been considered as an economical, eco-friendly and efficient method of control and prevention of yield loss due to MSV (Magenya et al., 2009; Martin and Shepherd, 2009). Different varieties of maize have shown differential responses to MSV infection resulting in varying levels of disease severity and incidence (Bosque-Perez et al., 1998). Maize lines expressing complete and partial resistance to MSV have been identified from various breeding populations (Soto et al., 1982; Rodier et al., 1995; Olaoye, 2009). Studies on the genetics of resistance to MSV have indicated that maize lines possessing complete resistance are controlled by few major genes and inheritance of this is

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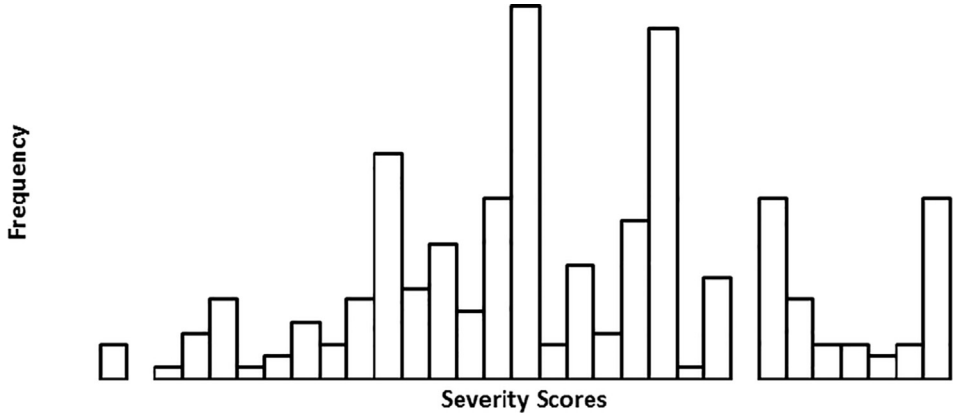

Fig. 1. Frequency distribution of average maize streak symptom severity scores of 250 S1 lines derived from a mapping population.

simple while partial resistance is controlled by several genes and are quantitatively inherited with additive gene action (Kyetere et al., 1999; Pernet et al., 1999a). Prior studies using restriction fragment length polymorphism (RFLP) markers have identified Quantitative Trait Loci (QTLs) controlling resistance to MSV from diverse maize mapping populations developed from various sources of resistance (Welz et al., 1998; Kyetere et al., 1999; Pernet et al., 1999a, 1999b). A major consensus QTL designated *msv1* was detected on the short arm of chromosome 1 in CML lines developed at CIMMYT, having a major effect on the MSV resistance trait and controlling between 48 and 76% of phenotypic variation, and further studies by finemapping using SNPs delimited *msv1* to an interval of 0.87 cM on chromosome 1 at 87 Mb (Sudha Nair et al., 2015). Other putative QTLs were also identified in these studies on chromosomes 3, 9 and 10 most of which were specific to each of the mapping populations. Additional fine mapping studies by Lagat et al. (2008) identified SSR markers located within the *msv1* region. All these studies have identified a consensus major QTL controlling resistance by a single gene with partial dominant effect on chromosome 1.

Since 1975, MSV resistance breeding programs at the International Institute of Tropical Agriculture (IITA) has been using TZ-Y derived lines as resistant sources (Bosque-Pérez, 2000). Resistance lines express very few streak symptoms (< 5–30% of the leaf area compared to susceptible lines with streak symptoms > 75%) or the resistant plants initially produce severe symptoms (streaks on > 75% of the leaf lamina) but leaves emerging post infection show symptom remission, termed as recovery resistance (Salaudeen et al., manuscript in preparation). A new mapping population of 250 S1 lines ($F_{2:3}$) was developed from a cross between MSV resistant parents, TZIL07A01005 and TZIL07A01322, and they were phenotyped for MSV under screen house conditions by inoculating plants with viruliferous leafhoppers (Salaudeen, 2012). All the inoculated lines were susceptible to MSV and showed severe symptoms at the early stage (2 weeks post inoculation), however, based on variation in symptom severity due to recovery resistance at later stage of the growth (4–6 weeks post inoculation), 24 lines were categorized as highly resistant, 37 as resistant, 46 as moderately resistant, 57 susceptible and 86 were highly susceptible (Salaudeen, 2012). Host resistance response in the highly resistant and resistant lines was found to be 'recovery type'. The objectives of the present study were to genotype this mapping population using single nucleotide polymorphism (SNP) markers to identify QTLs linked to MSV recovery resistance.

2. Materials and methods

2.1. Plant material and MSV phenotype data

The 250 S1 lines ($F_{2:3}$) used in this study was the F2 mapping population developed from a cross between TZIL07A01005 and TZIL07A01322 in the Maize Breeding Unit, IITA, Ibadan, Nigeria. Both parents are adapted drought tolerant line with good agronomic traits and resistance to MSV and other foliar diseases. These lines were phenotyped for MSV under screenhouse conditions by Salaudeen (2012). Briefly, phenotyping experiment was arranged in alpha-lattice design with two replications in pots in a screenhouse and they were inoculated one week after planting with laboratory reared viruliferous *Cicadulina triangular* colony. Infection incidence (percent infected plants) was assessed two weeks after inoculation. Subsequently, disease severity on each plant was assessed using a 1 to 5 symptom severity rating scale (0 = no symptoms, 1 = < 10% of the leaf area covered with streaks, 2 = 11–25% of the leaf area covered with streaks, 3 = 26–50% of the leaf area covered with streaks, 4 = 51–75% of the leaf area covered with streaks, 5 = > 75% of the leaf area covered with streaks) at weekly intervals for 6 weeks. The Area Under the Disease Progress Curve (AUDPC) was estimated as detailed in Ariyo et al. (2002) from the severity data for each score and also for the mean of all the observations, and a frequency histogram of mean severity scores was drawn to show the distribution of MSV severity scores within the population (Fig. 1). Normality test, Skewness and Kurtosis tests were performed on average severity score data.

2.2. Sample collection, DNA extraction, and SNP genotyping

About 8 to 10 young leaves were collected from each test line, packed in paper envelopes and then stored at −80 °C for about one hour, after which they were lyophilized using a free zone 18 L console dry system (Labconco Inc., Missouri, USA). Lyophilized samples were subsequently stored at −20 °C until analyzed. Genomic DNA of each sample was isolated from lyophilized leaf samples using a CTAB-based protocol modified from Saghai-Maroof et al. (1984). Genotyping using 269 single nucleotide polymorphism (SNP) markers was performed using the Kompetitive Allele Specific PCR (KASP) method on Kbiosciences' KASPar assay platform from LGC Genomics (Semagn et al., 2014; http://www.lgcgenomics.com) to identify QTLs linked to MSV recovery resistance. It is a singleplex compatible SNP assay technique that uses a florescent allele specific oligo extension method based on FRET (Förster resonance energy transfer) for SNP detection (Semagn et al., 2014).

2.3. Construction of genetic linkage map

The complete data obtained from genotyping the S1 lines were used to construct a genetic linkage map using JoinMap4 (Van Ooijen, 2006). Markers were assigned to linkage groups with independent LOD values > 3.0, recombination frequency < 0.49 and a maximum threshold value of 5 for the jump. Regression mapping algorithm was

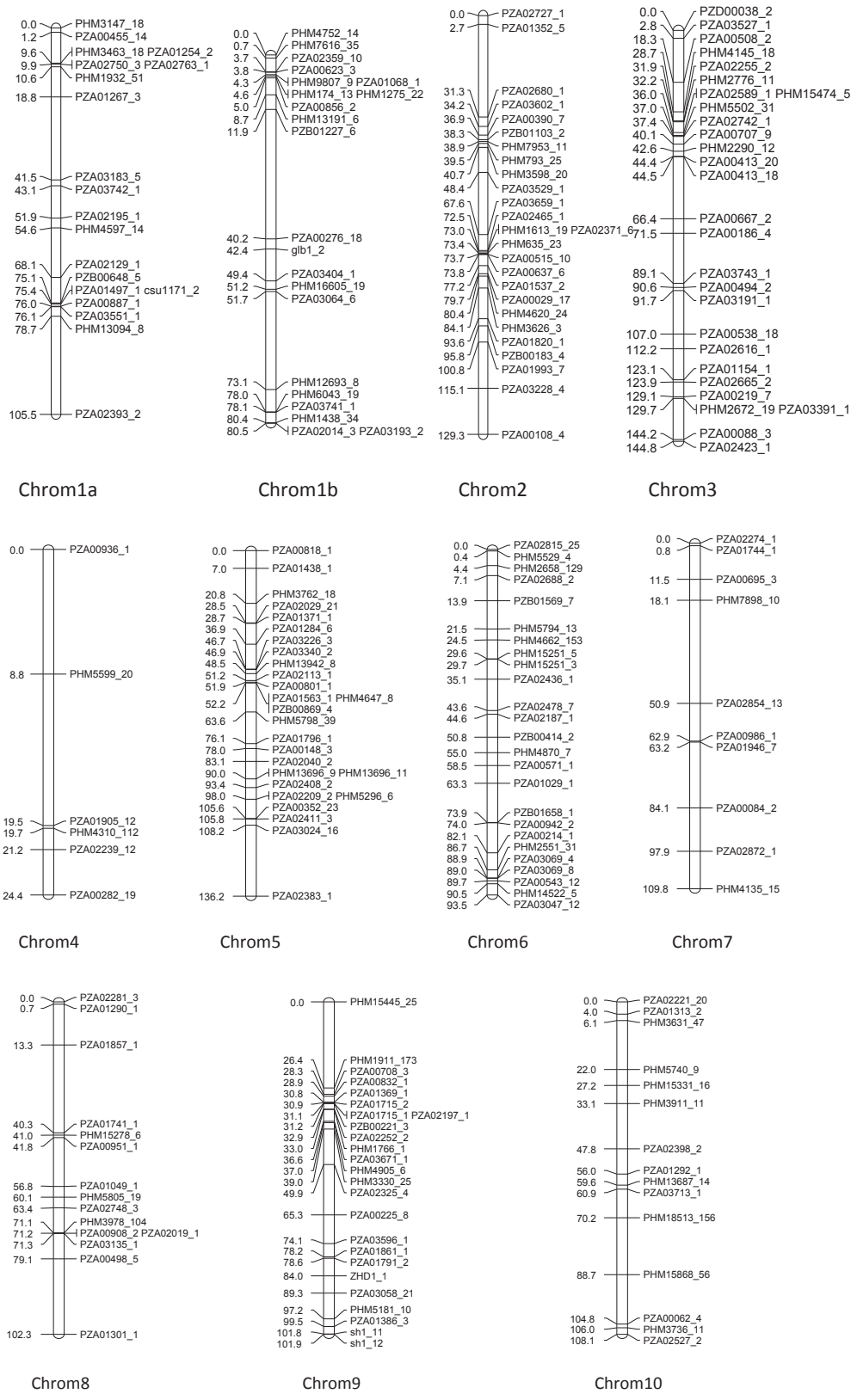

Fig. 2. Linkage map of F2 MSV mapping population showing linkage groups 1–11 (Chromosomes 1–10). SNP names are written on the right while the cumulative map distances (cM) are shown on the left.

used to order the markers and Kosambi mapping function was used to transform estimates of recombination frequency to map distances in centimorgans (cM). Markers that had insufficient linkage data were excluded from the final linkage map. Segregation distortion from the expected Mendelian segregation ratio (1:2:1 for an F2 population) was tested using the genotype frequencies at each SNP locus and chi-square test for significance of the segregation ratio at each SNP locus was calculated. The order of SNP loci was checked against the maize

Table 1
Characteristics of Linkage map.

Linkage group	Chromosome	Number of markers	Marker interval (cM)	Length (cM)
1	1a	20	5.55	105.5
2	1b	22	3.83	80.5
3	2	26	4.97	129.3
4	3	28	5.36	144.8
5	4	6	4.05	24.4
6	5	37	3.69	136.6
7	6	25	3.74	93.5
8	7	10	10.98	109.8
9	8	15	6.81	102.3
10	9	25	4.25	101.9
11	10	16	6.82	108.1
Total		230		1136.7
Average		20.91	20.91	20.91

reference map (B73 RefGen_V2) on the maize genetics and genomics database (www.maizegdb.org) to ascertain the real locations of SNPs in the genome. The linkage groups from JoinMap were rearranged into chromosomes according to their order on the reference map.

2.4. Quantitative Trait Loci (QTL) analysis

QTL analysis was performed using *R/qtl*, an add-on package for R program (Broman et al., 2003). Markers that had the same location on the linkage map were rearranged using the *jittermap* function in R/qtl. QTLs were analyzed using single marker EM algorithm (Lander and Botstein, 1989), Haley-Knott regression (Haley and Knott, 1992) and composite interval mapping (CIM) model (Jiang and Zeng, 1995). Permutation analysis was used in both the HK regression and CIM models to get the best LOD score at which a QTL was identified. The mean permutation LOD was used as the threshold LOD for declaring QTL loci. The genetic effects of the QTL – additive and dominance effects – were calculated. The *fitqtl* command was used to compute an ANOVA to test for the QTL significance and also to determine pairwise and multiple interactions between QTLs.

3. Results

3.1. Population phenotype

All 250 S1 lines were susceptible to MSV (100% incidence), however, the frequency distribution for average severity scores of the S1 lines was unimodal and approximately bell shaped, indicating an normal distribution (Fig. 1) (Salaudeen, 2012). Kurtosis and Skewness tests (−0.40 and 0.05, respectively) were not significant denoting an approximately symmetric normal curve. This gives an indication that several genes are involved in conferring recovery resistance to MSV in maize.

3.2. Genotyping data and segregation distortion

A total of 1234 SNPs were obtained with various degrees of consistency in replicate samples. Further filtering of these SNPs based on successful calls across parents and consistency in biological replicates (line replicates) resulted in over 430 polymorphic SNP but more SNP were discarded due to ambiguity in differentiating the homozygote and heterozygote genotypes as well as lack of reproducibility of result in replicates leaving 269 informative SNP which were used to genotype the entire mapping population. However, only 230 SNP passed the QC for linkage map construction and QTL analysis after removing failed, distorted or SNPs with high level of missing data. An initial screening of the two parental lines with SNP markers identified 269 markers that were polymorphic on the parents of the mapping population that were subsequently used to genotype the 250 S1 lines derived from the F2 population. All markers were scored as 'A' for progenies that were homozygous for the allele of parent TZIL07A01005, 'B' for progenies that were homozygous for the allele of parent TZIL07A01322 and 'H' for heterozygote genotypes. Chi-square test for segregation distortion from the Mendelian ratio revealed that approximately 71% of the 269 SNPs used did not deviate from the expected Mendelian segregation ratio 1:2:1. Severe segregation distortion was observed for 22 SNP loci at $p \leq 0.0001$ with the remaining 56 SNP loci having moderate distortions ($0.005 \leq p \leq 0.5$). Most of the SNP markers having high segregation distortions were still mapped to linkage groups without distorting the linkage maps consequently increasing the linkage map density. However, 15 SNPs that had high level of segregation distortionwere not mapped to any linkage group and they were excluded from linkage analysis. The inclusion of the markers with significant distortions in the linkage group did not affect the marker order or the distances between markers as the markers were randomly distributed throughout the linkage groups.

3.3. Genetic linkage analysis

A total of 269 polymorphic SNPs were used to genotype 250 S1 lines and the data was used for linkage analysis. Amongst these, only 230 SNPs were used to construct the linkage map because 39 of the SNPs could not be assigned to any linkage groups. This linkage analysis using 230 SNPs gave a linkage map with a total length of 1136 cM, comprising of 11 linkage groups (Fig. 2). The lengths of linkage groups varied from 24.4 cM on linkage group 5–144.8 cM on linkage group 4 (Table 1) with an overall average length of 103.34 cM. Marker intervals ranged between 3.69 cM on linkage group 6–10.9 cM on linkage group 10 with an overall average marker interval of 5.46 cM. The linkage map from this study appeared to be slightly shorter compared with linkage maps obtained from previous studies for MSV resistance using RFLP markers. However, the density of SNP linkage map established in this study is higher at 5.46 cM than the earlier maps for MSV resistance (Welz et al., 1998; Pernet et al., 1999a, 1999b). The linkage groups were also rearranged into chromosomes for QTL analysis according to their order in the maize genome and as inferred from maize genome database. The order of SNP loci on the linkage map agrees with their order on the maize genome reference map (www.maizegdb.org).

The number of linkage groups obtained was more than the haploid number of chromosomes in the maize genome. This was due to insufficient linkage among SNP loci on the linkage group corresponding to chromosome 1, causing the chromosome to be split into two groups in the linkage map, resulting in a total of 11 linkage groups. The two linkage groups were renamed chromosome 1a and 1b (Table 1). This split suggests that the markers used in genotyping were not enough to give a dense linkage map with better coverage for chromosome 1. The number of markers on each linkage group ranged from 6 on linkage group 5 to 37 on linkage group 6. The number of markers did not correspond to the length of the linkage group. Larger groups with many markers had shorter marker intervals, were denser and had shorter lengths than some groups with fewer markers (Table 1).

3.4. QTL analysis

QTL analysis using each severity score recorded weekly for six weeks, average values of all severity scores and AUDPC values, found four SNP loci to be consistently associated with recovery resistance to MSV. Two of the QTLs were found on linkage group 4 (chromosome 3) at positions 37.0 cM and 112.2 cM corresponding to the SNP loci PHM 5502_31 and PZA02616_1, respectively (Fig. 3). These SNPs were found in the maize genome reference map - B73 RefGen_V2 on chromosome 3 approximately in bins 3.04 and 3.08, respectively (Table 2). PZA02616_1 was detected with CIM method at LOD score of 3.4 while PHM5502_31 was detected with Haley-Knott (HK) regression method at

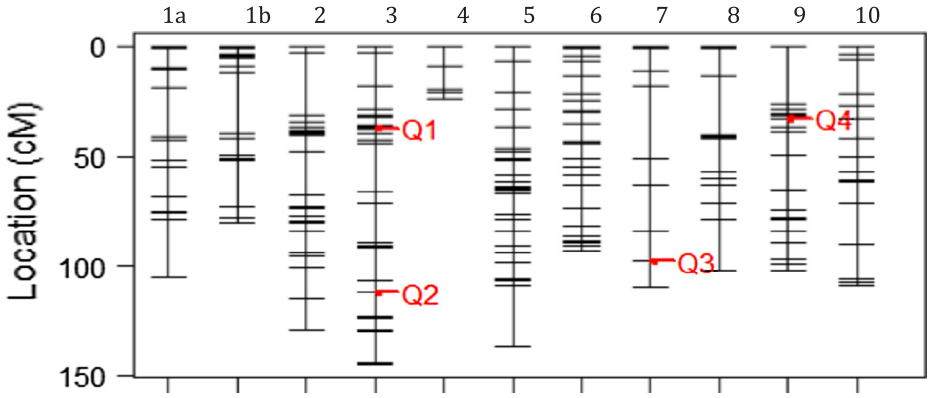

Fig. 3. Linkage map and the position of QTLs. Q1 corresponds to SNP PHM5502_31, Q2- PZA02616_1 and Q3 - PZA02872_1; Q4 - PHM1766_1.

LOD score of 2.9. A genome wide mean permutation LOD score of 3.01 was used as the threshold value to declare the significant QTL from the CIM method for SNP loci PZA02616_1 whereas a mean permutation LOD value of 2.4 was used to declare the significant QTL from the HK method for SNP PHM5502_31. The two SNP loci PZA02616_1 and PHM5502_31 were found to be highly significant based on ANOVA at $p = 0.001$ and $p = 0.01$, respectively. SNP PZA02616_1 was the most significant QTL accounting for 51% of the variation in MSV severity score while PHM5502_31 was the second significant QTL accounting for 47% of phenotypic variations. Interaction between the two SNPs tested by ANOVA was not significant.

The SNP marker PZA02616_1 originated from parent TZIL07A01005 while PHM5502_31 QTL originated from parent TZIL07A01322 (Fig. 4). QTL effect analysis revealed that PZA02616_1 gave an additive genetic effect whereas PHM5502_31 gave a dominance effect. Two significant QTLs with lesser effects were also found on linkage groups 8 at 97.9 cM and 10 at 33.0 cM with LOD 2.4 from HK regression method and 2.3 from CIM, respectively. They correspond to chromosomes 7 and 9 at SNPs PZA02872_1 and PHM1766_1, respectively. The SNP marker PZA02872_1 originated from the parent TZIL07A01005 and the SNP marker PHM1766_1 was detected in both parents (Fig. 4). The two QTLs explained 37% and 29% of the total phenotypic variance, respectively. Multiple QTL models for pairwise interactions as well as full models did not detect any significant interactions between the two SNPs.

4. Discussion

Resistance to maize streak virus disease is an essential trait required in breeding for improved maize varieties targeted to regions in Africa (Bosque-Pérez, 2000; Pingali and Pandey, 2000). A number of conventional maize breeding programs have identified several maize lines of diverse origins that possess resistance to MSV (Efron et al., 1989). The form of resistance in several resistant sources has been found to be polygenic with both major and minor genes of varied effects (Efron et al., 1989; Rodier et al., 1995; Bosque-Perez et al., 1998; Sudha Nair et al., 2015). Identification of the genes responsible for resistance to MSV is an important part of the process of breeding for new maize varieties with new and more durable levels of resistance. This study focused on identification of QTLs associated with 'recovery' resistance to the maize streak disease and identify potential markers-linked to MSV resistance for marker assisted selection.

Breeders usually cross two resistant parents to exploit the potential contribution of beneficial resistance alleles originating from them to generate transgressive segregation that can lead to the development of new maize inbred lines with much higher levels of resistance to MSV and desirable agronomic traits. The two resistant parents were thus crossed in the current study to identify markers associated with complementary resistance alleles originating from the two parents to create better inbred lines. The 250 S1 lines derived from this bi-parental cross were genotyped using SNPs, which have become markers of choice for use in linkage map-based QTL analysis and to build dense linkage maps, by Kompetitive allele specific PCR (Jones et al., 1997; Semagn et al., 2014), and phenotyped at the seedling stage under artificial infection using viruliferous vectors (Salaudeen, 2012). The results of linkage analyses identified four QTLs, with two putative QTLs having large effects on chromosome 3 and two others having small effects on chromosomes 7 and 9. These QTLs originated from mapping population had both additive and dominance effects but interaction among the four loci was not significant. All QTLs identified in this study were detected with across the six severity scoring dates, indicating that the QTLs were consistently associated with resistance to MSV at all stages of infection.

In contrast, previous QTL studies evaluated populations developed by crossing MSV susceptible and MSV resistant parents and identified a

Table 2
Details of QTLs and its effects on recovery resistance in maize inbred lines.

QTL name	QTL location (cM)	Linkage group[a]	Position (cM)	LOD score	Effect (additive or dominant)	Phenotypic variance explained (%)	Flanking markers	Physical position of QTL (Mbp)[d]
Q1: PHM5502_31	37.0	3	68,056,867	3.04[b]	Dominant	47	PZA00508_2, PZA00667_2	18.2, 66.4
Q2: PZA02616_1	112.2	3	211,720,827	3.08[b]	Additive	51	PZA00084_2, PHM4135_15	84.1, 109.8
Q3: PZA02872_1	97.9	7	13,174,365	2.4[c]	Dominant	37	PHM2776_11, PZA01154_15	32.2, 123.1
Q4: PHM1766_1	33.0	9	140,774,640	2.3[c]	Additive and Dominant	29	PHM1911_173; sh1_12	26.3, 101.8

[a] Linkage group (see Fig. 3).
[b] LOD $P < 0.01$.
[c] LOD $p = 0.5$.
[d] Schnable et al. (2009).

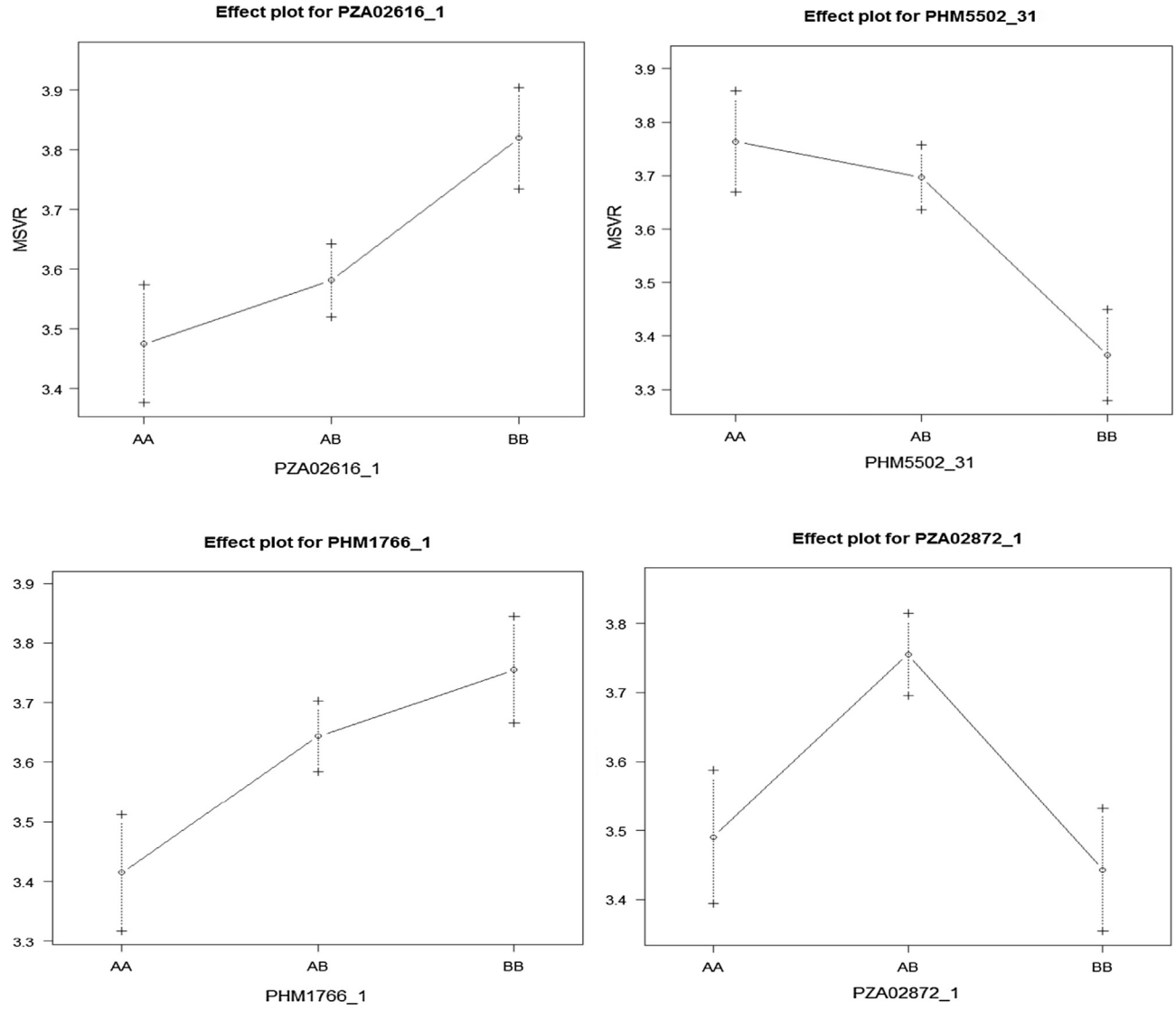

Fig. 4. Effect plots for all QTLs showing the contribution of each parent. Parental line TZIL07A01005 is designated the 'A' parent and the TZIL07A01322 as the 'B' parent.

major QTL on the short arm of chromosome 1 designated as *msv1* in maize line D211 (Rodier et al., 1995; Pernet et al., 1999a), CML202 (Welz et al., 1998), Tzi4 (Kyetere et al., 1999), CIRAD390 (Pernet et al., 1999b) MAL13 (Lagat et al., 2008) and CML206 (Sudha Nair et al., 2015). The *msv1* was found to be the major gene accounting for 50–70% of the phenotypic variance due to MSV resistance even with different resistant parental lines in F2 mapping populations (Welz et al., 1998; Kyetere et al., 1999; Pernet et al., 1999a, 1999b; Sudha Nair et al., 2015). However, *msv1* was not found in the populations evaluated in this study. As the parents used in the current study had different genetic backgrounds, the observed resistance could arise from combination of different sets of alleles originating from these parents. All the resistant and highly resistant S1 lines identified in this study showed severe MSV symptoms (severity rating > 4) initially and subsequently emerged leaves had moderate to mild streak symptoms (severity rating score < 3) (Salaudeen, 2012). The recovery type of resistance is conditioned by many genes that render resistance to MSV durable. Recovery resistance improved grain yield as the upper leaves emerging later in the season that contribute most to the photosynthate of the developing ears are not adversely affected by MSV (Subedi and Ma, 2005; Asea et al., 2012).

The QTLs on chromosome 3 appeared to be similar to those identified by Pernet et al. (1999a) in bins 3.03 and 3.09 that were involved in both early and late resistance to MSV, respectively. The other two SNP QTLs – PZA02872_1 and PHM1766_1 on chromosome 7 and 9, respectively, seemed to be unique to the current mapping population. Resistance QTLs identified in this study originated from the two parental lines with the parent TZIL07A01005 contributing to the QTL with the highest effect (Fig. 4). As this QTL and another one showed additive to partial dominance gene effects, fixing the favorable alleles of these QTLs via marker-assisted selection may facilitate the development of inbred lines with higher levels of resistance to MSV.

This study has identified four QTLs linked to MSV resistance originating from two MSV resistant parents. It is hoped that this finding will allow the accumulation of resistant genes in new MSV resistant lines to enhance the levels of MSV resistance. However, further research is needed to validate the QTLs identified from this study, including any $G \times E$ effect and fine mapping by improving the marker density. Molecular markers can be designed based on these SNPs loci and used to screen germplasm resistance to MSV and for introgressing the resistant loci into new varieties to ensure effective field protection against MSV.

5. Conclusion

This study identified two putative QTLs on chromosome 3, at positions 37.0 cM and 112.2 cM corresponding to the SNP loci PHM 5502_31 and PZA02616_1, accounting for 47 to 51% of the total phenotypic variance observed in maize lines with recovery resistance to streak virus disease. Two other QTLs on chromosomes 7 and 9 accounted for 28 to 32% of the total variation with recovery resistance to streak disease in maize. Further studies are necessary to validate two major QTLs detected on chromosome 3 in unrelated populations and fine mapping for utilization in maize improvement programs.

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