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Identification of Fusarium Head Blight Resistance QTLs in a Wheat Population Using SSR Markers

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Abstract: Fusarium head blight (FHB) caused by *Fusarium graminearum* is a destructive disease of wheat. Breeding for FHB resistance by conventional breeding methods is feasible but laborious and expensive. Detection of DNA markers associated with FHB resistance quantitative trait loci (QTL) will accelerate breeding programs. This study was conducted to identify simple sequence repeat (SSR) markers linked FHB resistance QTLs in wheat. A population of 167 $F_{2:3}$ from the cross Wangshuibai (resistant)/Falat (susceptible) was used. The Type II resistance (spread of pathogen within a spike) was evaluated on F_3 families derived from F_2 plants in the greenhouse. Two QTLs were detected that together explained 25.1% of the phenotypic variation. A QTL region on chromosome 3B explained 16% of the variation. Additional QTL was located on chromosomes 2A accounting for 9.1% of the phenotypic variation.

Key words: FHB resistance, wheat, QTLs, SSR markers

INTRODUCTION

Wheat head scab, also called Fusarium head blight (FHB) caused by *Fusarium graminearum* is an economically important disease of wheat and other cereals in semi-humid areas (Parry *et al*., 1995; McMullen *et al*., 1997). It causes severe yield loss and a reduction in grain quality. In addition, FHB contaminates cereal products with mycotoxins such as deoxynivalenol (DON), which are toxic to human and animals (McMullen *et al*., 1997; Bai *et al*., 2001). Cultivation of genetically resistant cultivars is the most effective way of controlling the disease (Bai and Shaner, 1994; Mesterhazy, 1995; Parry *et al*., 1995).

Different studies in wheat have shown that few genes may be involved in FHB resistance. Yao *et al*. (1997) reported 3 resistance genes in Sumai 3. In Wangshuibai, FHB resistance was reported to be controlled by 2 genes (Bai *et al*., 1990). Singh *et al*. (1995) identified 3 resistance genes in Frontana, however, in other study it was shown that Frontana and Ning 7840 (derived from Sumai 3) each possess 2 resistance genes (Van Ginkel *et al*., 1996).

Two major types of resistance (Type I and Type II) have been proposed by Schroeder and Christensen (1963). Type I resistance is resistance to initial infection and Type II is resistance to spread of fungus within the spike. Another types of FHB resistance have been described by Mesterhazy (1995). Accurate assessment of Type I resistance is difficult because it is highly influenced by environmental conditions and the amount of inoculum actually applied is difficult to quantify (Bai and Shaner, 1994; Rudd *et al*., 2001). Evaluation for Type II resistance is most often done in the greenhouse under controlled conditions which decreases environmental variation and confounding effects prevalent in FHB phenotypic evaluation (Bai *et al*., 1999; Waldron *et al*., 1999).

Evaluation of FHB resistance is tedious, time consuming and expensive. Molecular markers have been used to estimate number and location of genes involved in FHB resistance and could complement classical plant breeding. In wheat random amplified polymorphic DNA (RAPDs), restriction fragment length polymorphisms (RFLPs), amplified fragment length polymorphisms (AFLPs) and microsatellite or simple sequence repeats (SSRs) have been used to detect FHB resistance genes (Bai, 1995; Bai *et al*., 1999; Waldron *et al*., 1999; Anderson *et al*., 2001; Gervais *et al*., 2003; Zhou *et al*., 2004; Schmolke *et al*., 2005). Waldron *et al*. (1999)

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identified five QTLs for FHB resistance by analyzing RFLPs in a Sumai 3/Stoa recombinant inbred lines population (112 F_5-derived RILs). Two major QTL were identified on chromosome 3BS of Sumai 3 and chromosome 2AL of Stoa. The RFLP marker with high effect in 3BS region explained 15.4% of the phenotypic variation. Anderson *et al.* (2001) using recombinant inbred lines from crosses ND2603/Butte 86 and Sumai 3/Stoa identified 3 QTLs on chromosomes 3BS, 2AL and 6BS. The QTLs on chromosome 3BS explained 41.6 and 24.8% of the variation in Sumai 3/Stoa and ND2603/Butte 86 populations, respectively. The association of several SSR markers with a major QTL for FHB resistance on chromosome 3BS was confirmed by other groups (Buerstmayr *et al.*, 2002; Zhou *et al.*, 2002, 2003; Zhang *et al.*, 2004; Mardi *et al.*, 2005). Gervais *et al.* (2003) evaluated a set of 194 recombinant inbred lines from a cross Renan (resistant)/Recital (susceptible) using SSR, AFLP and RFLP markers and reported that wheat chromosomes 2A, 2B, 3A, 3B and 5A carry FHB resistance genes. Steiner *et al.* (2004) identified two major QTLs for FHB resistance on chcromosomes 3A and 5A in Frontana that collectively explained 25% of phenotypic variation.

Currently the majority of the FHB resistant breeding lines and cultivars have Sumai 3 and/or Frontana in their pedigrees. Bai *et al.* (2003) reported that Wangshuibai is not closely related to Sumai 3. In a recent study looking at genetic diversity of chromosome 3BS, Liu and Anderson (2003) found that Wangshuibai has no alleles in common with Sumai 3 for any molecular markers around the QTL detected on the short arm of chromosome 3B. Given these reports and high level of resistance in Wangshuibai, it seems logical to contemplate the possibility of Wangshuibai carrying different resistance genes than Sumai 3. In this study analysis of QTLs governinng FHB reistance was carried out using a $F_{2:3}$ population from a cross between Chinese variety Wangshuibai as resistant parent and Falat as susceptible parent. Falat is a spring wheat that possesses well-adapted agronomic characters for cultivation but it is susceptible to FHB. The objectives of this research were to determine the chromosomal location of FHB resistance QTLs and to identify SSR markers linked to the FHB QTL(s).

MATERIALS AND METHODS

Plant material: A population of 167 $F_{2:3}$ lines was developed from the cross Wangshuibai/Falat. Wangshuibai is a FHB resistant spring wheat cultivar from China. It has high resistance to spread of scab within an inoculated spike. Falat is a spring wheat and susceptible to spread of scab within the spike. Seeds from F_1 plants were planted in the greenhouse at the University of Tabriz, Iran. Two hundred random F_2 seeds were planted in large plastic containers. To produce F_3 Plants, approximately 20 seeds from each F_2 plant were planted in three 23 cm pots (usually 6-7 seeds per pot). The pots were arranged randomly on benches in the greenhouse. The soil texture was sandy loam. The plants were grown with a 16 h photoperiod, watered as needed and fertilized twice, firstly at the 4-5 leaf stage and for the second time at the stem elongation stage. The F_3 Families were used to determine the phenotypes of F_2 plants for response to FHB.

FHB evaluation: Thirty plants of each parent (Wangshuibai and Falat) and 167 $F_{2:3}$ families were tested for spread of FHB within a spike in a greenhouse at University of Tabriz, Iran, in 2005. The inoculum of *F. graminearum* contained a mixture of five isolates. At anthesis, 15 F_3 plants derived from each F_2 plant were inoculated with a 10 microlitre droplet (20000 conidia mL^{-1}) of conidial suspension placed directly into a single floret of a spikelet near the center of the spike, following procedures described by Stack (1989) and Anderson *et al.* (2001). This method bypasses primary infection and targets Type II resistance (Waldron *et al.*, 1999). The inoculated plants were covered with humid plastic bags for 3 days to maintain high humidity for initial fungal infection. Then they were transferred to a section of the greenhouse with 70% relative humidity. Greenhouse temperature averaged 25°C during the day with a range of 19 to 30°C and 19°C at night with a range of 17 to 21°C. Three weeks after inoculation, percentage of scabbed spikelets (PSS) on inoculated spikes was determined as disease severity. Average PSS values for all F_3 plants derived from the same F_2 plant were used to represent the resistance level of the original F_2 plant (Zhou *et al.*, 2003).

DNA isolation and bulked segregant analysis: Leaf samples were harvested from F_2 plants and both parents (Wangshuibai and Falat) and used for DNA isolation using the CTAB method described by Saghai-Maroof *et al.* (1984). Based on greenhouse phenotypic evaluation, DNAs of 15 most resistant and susceptible F_2 plants were pooled in equal quantities to construct a resistant (R) and a susceptible (S) bulk, respectively. Parents and the two bulks were screened for polymorphism with 341 SSR markers, 166 from Roder *et al.* (1998) and 175 from Cregan *et al.* (2001). Polymorphic primer pairs between two parents, as well as the two bulks were used to screen individuals of the

bulks. Those primers with a significant difference in allele frequency between the individuals of the R and S bulks, were used for whole population screening.

PCR amplification was conducted according to Roder *et al.* (1998) with the exception that the reaction volume was reduced to 10 μL. Amplified PCR products were run in 8% polyacrylamide sequencing gels. Gels were stained with ethidium bromide and visualized with a UV transilluminator.

Statistical analysis: The distribution of PSS data was tested and square root of the percentage score was used to transform non-normal distribution of data into normal distribution (SAS version 8, SAS Institute Inc, 1999). Associations between markers and PSS were made with both transformed and untransformed data in the QTL analysis. Since the nature of the association did not change substantially, the results with untransformed data are presented. For each segregating marker in the population, segregation distortion was tested using a Chi-square test ($\propto$ = 0.01). A linkage map was constracted using Map Manager QTXb20 (Manly *et al.*, 2001). Recombination fractions were converted into map distances in centiMorgans (cM) using the Kosambi mapping function (Kosambi, 1944). Initially, simple regression analysis in Map Manager was used to identify markers significantly associated with FHB resistance in the population. QTL detection was performed by composite interval mapping (CIM) using QTL Cartographer software (Basten *et al.*, 2001). A LOD threshold of 3 was selected for CIM. For each QTL , the position, the additive effect and the percentage of phenotypic variation explained were estimated.

RESULTS

FHB evaluation: Phenotypic values of disease severity significantly differed between two parents ($t = 13.17$, $p < 0.0001$). Disease severity of the two parents was 12.4% for Wangshuibai and 78.6% for Falat. The F_3 families displayed a continuous distribution for FHB infection severity. The two parents were situated on either end of the distribution. Mean disease severity in the F_3 families ranged from 9.5 to 85.7%, showing large phenotypic variation in the population (Fig. 1).

SSR marker analysis: Out of 125 polymorphic markers (36.7%) between two parents, 16 showed also polymorphism between the two R and S bulks. Using these 16 markers, the individuals in the two bulks were genotyped to confirm the initial bulked segregant

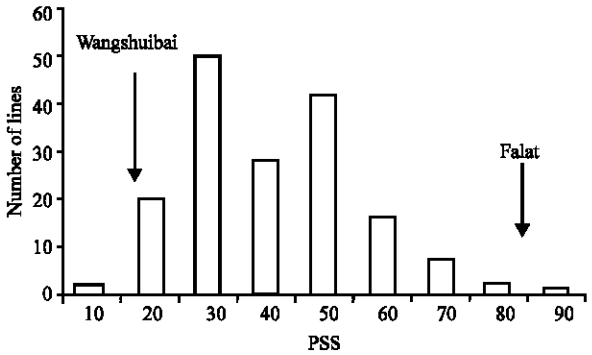

Fig. 1: Histogram of percentage of scabbed spikelets (PSS) for FHB severity in F_3 lines of Wangshuibai/Falat. The disease severity in the two parents is indicated

Table 1: QTLs identified for FHB resistance in Wangshuibai/Falat population using composite interval mapping

QTL	Map interval	Chromosome	LOD	R^2(%)	Additive effect
Qfhb3B	Xbarc 133-Xgwm 493	3BS	5.7	16.0	10.72
Qfhb2A	Xbarc 353.1-Xgwm 372	2AL	3.6	9.1	7.5

analysis. Finally 8 markers were confirmed to reveal significant polymorphism and were screened across the whole population. In addition, SSR markers which revealed polymorphism between two parents and closely linked to the 8 above mentioned markers according to wheat genetic maps (Roder *et al.*, 1998; Cregan *et al.*, 2001), were also screened across the whole population. Chi-square test for each segregating marker in the population showed that observed frequencies were in agreement with expected ratio 1:2:1. A partial linkage map was constructed using Map Manager.

QTL analysis: Preliminary analysis using regression analysis showed that 10 SSR markers were significantly associated with FHB resistance ($p < 0.01$) in the population (data not shown).

QTL analysis by CIM detected 2 QTLs on chromosomes 3BS and 2AL (Table 1 and Fig. 2). The chromosomal locations of marker intervals, additive effect of each QTL and coefficients of determination are presented in Table 1. A linkage group of 4 SSR markers defined a segment of 20.2 cM near the end of chromosome 3BS. All of the markers in the group were significantly associated with FHB resistance with a logarithm of odds value greater than 3.9. The most likely QTL position on chromosome 3BS was at the interval of Xbarc 133 and Xgwm 493 markers (Fig. 2a), which were the two markers with the greatest coefficient of determination. This major QTL with a LOD score of 5.7 explained 16% of the phenotypic variation for FHB resistance. Five SSR

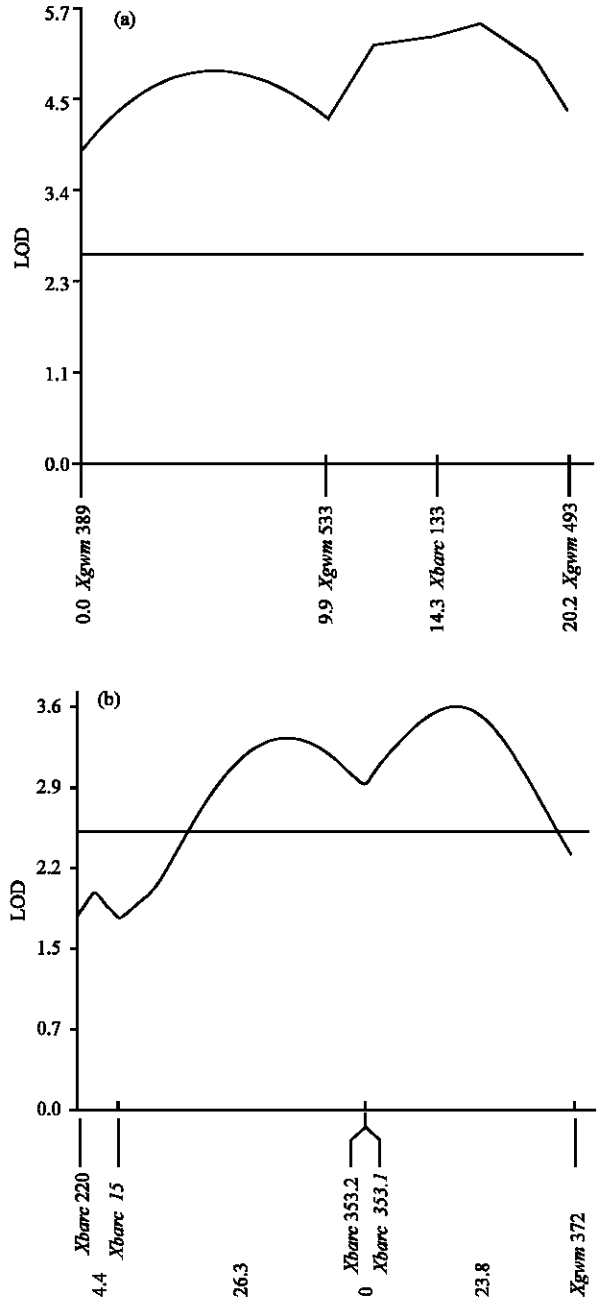

Fig. 2: Composite interval analysis of QTLs for Fusarium head blight resistance on linkage groups corresponding to chromosomes 3B (a) and 2A (b)

markers formed another linkage group covering 54.5 cM on chromosome 2AL. The location of the QTL on chromosome 2AL was in the Xbarc 353.1 and Xgwm 372 markers interval with a LOD of 3.6 and accounted for 9.1% of the variation (Fig. 2b). For these two detected QTLs the allele conferring FHB resistance originated from the resistant parent Wangshuibai.

DISCUSSION

FHB assessment: Accurate phenotypic evaluation is a prerequisite for QTL mapping. Environmental factors such as humidity and temperature are critical factors for initiation and development of FHB infection (Bai and Shaner, 1994). In this study we provided favorable moisture and temperature conditions in a controlled greenhouse to minimize environmental variation. Since resistance to spread of disease within a spike (Type II) is a major type of resistance (Schroeder and Christensen, 1963), we used a single floret inoculation method to assess this type of resistance. Inoculating a single spikelet in a spike eliminates differences in disease incidence among test lines and simplifies the complicated disease system (Bai *et al*., 1999).

The highly significant difference between parental lines for FHB response led to large variation in the mapping population. For FHB resistance, F_3 families showed a continuous distribution that characterized by two peaks. Similar distributions for FHB severity was reported by Waldron *et al.* (1999), Bai *et al.* (2002) and Gervais *et al.* (2003) who especulaed that resistance to FHB in wheat is controlled by one or few major genes and several minor genes.

QTL mapping: QTL analysis of FHB resistance in the $F_{2:3}$ population from the cross Wangshuibai/Falat revealed 2 QTLs significantly associated with the resistance. They were located on chromosomes 3BS and 2AL. The first QTL was located on the short arm of chromosome 3B in the interval flanked by SSR markers Xbarc 133 and Xgwm 493, explaining 16% of phenotypic variation. Shen *et al.* (2003) reported an important QTL for Type II resistance in Ning 894037, another Chinese FHB resistance source, in exactly the same region on chromosome 3BS. Similar results concerning the 3BS QTL were reported by other researchers (Buerstmayr *et al*., 2002; Zhou *et al*., 2003; Zhang *et al*., 2004). Several QTL mapping studies on FHB resistance using Sumai 3 and its derived lines have reported a major QTL on chromosome 3BS (Waldron *et al*., 1999; Andeson *et al*., 2001; Zhou *et al*., 2002; Buerstmayr *et al*., 2002). It has been reported that Wangshuibai is not closely related to Sumai 3 (Bai *et al*., 2003). Liu and Anderson (2003) Showed that Wangshuibai has no alleles in common with Sumai 3 for several SSR markers around the QTL detected on chromosome 3BS. Therefore it is likely that there are different genes or different alleles of the same locus for FHB resistance on 3BS in these varieties. Further research needs to be done to determine the relationship between QTL identified for FHB resistance on 3BS in different resistance sources.

The second QTL on chromosome 2AL at the interval of *Xbarc 353.1* and *Xgwm 372* explained 9.1% of phenotypic variation. A similar QTL position was reported by Waldron *et al.* (1999) and Anderson *et al.* (2001) in moderately susceptible variety Stoa and by Gervais *et al.* (2003) in resistant variety Renan which is possibly the same QTL or a closely related allele.

Collectively these 2 QTLs explained 25.1% of phenotypic variation. The unexplained variation in FHB resistance may be due to QTLs that remain undetected due to their minor effects or because the markers on that regions were not assessed in the present study. Epistatic interaction between QTLs may also contribute to the unexplained variation.

We used SSR markers coupled with bulked segregant analysis (BSA) to identify the QTLs controlling FHB resistance. SSR markers are codominant, relatively easy to perform and are suitable marker system for practical breeding applications. The BSA method is efficient for gene mapping because it allows a considerable research-saving compared to comprehensive genotyping. We used $F_{2:3}$ mapping population to detect QTLs associated with FHB resistant genes. Zhou *et al.* (2003) reported that marker assisted selection for FHB resistance in the F_2 can be as effective as that in homozygous generations when codominant markers are used. Therefore marker assisted selection for the major QTLs in other early generations and selecting homozygous individuals can accelerate the breeding practice and significantly increase selection accuracy.

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