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Quantitative Trait Loci Analysis of Novel Fusarium Head Blight Resistance in Tokai 66

[1]S. Malla, [2]A.M.H. Ibrahim, [3]Y. Yen, [1]W. Berzonsky, [1]K.D. Glover and [1]J. Stein
[1]Department of Plant Science, South Dakota State University, Brookings, SD 57007
[2]Department of Soil and Crop Sciences, Texas A and M University, College Station, TX 77843
[3]Department of Biology and Microbiology, South Dakota State University, Brookings, SD 57007

Abstract: Problem statement: Identification of Quantitative Trait Loci (QTLs) in durable resistant genotype is important for marker-assisted breeding. The study was conducted to identify QTLs linked to FHB resistance in 'Tokai 66', a Japanese genotype. **Approach:** A cross was made between Tokai 66 and Jagalene and the single seed descend was used to advance the population. The $F_{2:4}$ and $F_{2:5}$ populations were evaluated by artificially inoculating disease in a mist-irrigated nursery in 2006 and 2007. Disease incidence, severity, FDK and DON content were evaluated in the 128 RILs. Map Manager QTX was used to prepare the linkage map and QTL Cartographer 2.0 was used to identify QTLs for FHB responses from the field data respectively. **Results:** Four QTLs for FHB responses were detected, of which one QTL each for severity and FDK were stable across two years and were located at the 5B and 3BSc, respectively. Both QTLs conferred resistance to FHB. Two unstable QTLs were detected at the 5B and 3D for FDK and DON content, respectively using the field environment. The 5B QTL for FDK contributed to susceptibility, whereas the 3D QTL for DON content contributed to resistance. **Conclusion:** The 5B QTL associated with resistance to severity and 3B QTL associated with resistance to FDK could be utilized by winter wheat breeding programs selection to enhance FHB resistance.

Key words: Fusarium Head Blight (FHB), Tokai 66, Quantitative Trait Loci (QTL), Diversity Array Technology (DART)

INTRODUCTION

Fusarium head blight, caused by *Fusarium graminearum* Schwabe, is an economically important disease of wheat and other small grains in warm and humid areas (Stack and Mcmullen, 1985; Tuite *et al*., 1990). Nganje *et al*. (2002) estimated $ 2.7 billion loss in wheat and barley (*Hordeum vulgare* L.) farming due to the FHB epidemics in the US during the period of 1998-2000. These losses were attributed to a combination of factors, including decrease in yield due to shriveled grains, reduction in grain quality, mycotoxins contamination in the infected seeds and cost of transportation of grains from disease-free areas.

Breeding for resistance has been a predominant approach to manage the disease. There are five types of physiological resistance for FHB in the host listed in the literature, namely resistance to (i) disease penetration (type I), (ii) floral spread (type II), (iii) Fusarium Damaged Kernel (FDK) (type III), (iv) yield reduction (type IV) and (v) mycotoxin accumulation (type V) (Schroeder and Christensen, 1963; Mesterhazy, 1995). Studies have shown that the different types of resistance were positively associated, though not very highly and suggested that the selection for one type of resistance would indirectly enable selection for other types of resistance (Lemmens *et al*., 2005). Reduction in mycotoxins, especially Deoxynivalenol (DON), is paramount as DON adversely affects the health of humans and livestocks (Yoshizawa and Morooka, 1973; Aakre *et al*., 2005). The prime emphasis in breeding for resistance to FHB is to develop genotypes with low disease symptom and very low level of mycotoxins.

Use of molecular makers and consequently marker assisted selection would reduce the confounding effects of the various environmental factors and increase gain from selection for FHB resistance. Buerstmayr *et al*. (2009) reviewed the FHB response QTLs and reported that QTLs for FHB response have been identified in all wheat chromosomes except 7D. The Chinese line 'Sumai3' and its derivatives are mostly utilized in breeding programs around the world as they displayed stable resistance to disease spread in the spike (Bai and Shaner, 1994; Mesterhazy, 2003). Mapping studies of Sumai3 and its derivatives had

Corresponding Author: A.M.H. Ibrahim, Department of Soil and Crop Sciences, Texas A and M University, College Station, TX 77843 Tel: (979) 845-8274

showed that they contain QTLs on different chromosomes for resistance to spread and FDK but only a single stable QTL for mycotoxin resistance. The most prominent QTL in this Chinese source is Fhb1, formerly Qfhs.ndsu-3BS (Liu *et al*., 2006), expressing resistance to spread (Waldron *et al*., 1999; Bai *et al*., 1999). The 3BS region was also reported to contain QTLs providing resistance to FDK and DON accumulation in Sumai3 and its derivatives (Lemmens *et al*., 2005; Yang *et al*., 2005).

Resistance to FHB has been reported from different regions of the world. Despite contributing stable FHB resistance in the crosses, Sumai3 and its derivatives resulted in poor yield and low grain quality (Buerstmayr *et al*., 2002). Breeding programs have meticulously been searching for germplasm that will contribute to high resistance for FHB as well as transfer good agronomic traits into resulting crosses. Additionally, significant additive gene action in FHB resistance has indicated that gene pyramiding could be realized by combining different sources of resistance (Snijders, 1990). A genotype from Japan, 'Tokai 66', has shown resistance to FHB in field evaluation (Zhang *et al*., 2008). Tokai 66 is a novel source of resistance, though Liu and Anderson (2003) suggested that Tokai66 contained one out of 5 alleles similar to Sumai3 at the 3BS region. This novel resistance in Tokai 66 is not yet clearly understood. The objective of the study, therefore, were to (i) identify QTLs linked to disease index (type I and II resistance), FDK (type III resistance) and DON content (type V resistance) and to (ii) determine the novelty of these Tokai 66 QTLs in comparison to other available resistance sources.

MATERIALS AND METHODS

Plant material and field evaluation: A total of 128 $F_{2:5}$ and $F_{2:6}$ Recombinant Inbred Lines (RILs), atleast eight seeds per line, developed using the Single Seed Decent (SSD) procedure, from the cross Tokai66/'Jagalene' were evaluated in field environments in 2006 and 2007 at Brookings, SD (Latitude = 44°16'14", Longitude = -96°46'18"). Tokai66 is a Japanese spring wheat with unknown moderate resistance to FHB, while Jagalene is popular Hard Red Winter wheat (HRW) that is highly susceptible to FHB in South Dakota (Table 1). The RILs were transplanted into the field in May 2006 and 2007. The plot size was a 12.7 cm row in both greenhouse and field. Seedlings were vernalized for eight wk at 4°C prior to transplanting. The $F_{2:5}$ and $F_{2:6}$ RILs nurseries were laid out in the field as a randomized complete block design with two replications.

Table 1: Pedigree and seed sources of the parental genotypes used in this study

Parents	Pedigree/PI	Source
Tokai 66	PI382161	Japan
Jagalene	Abilene/Jagger	Agripro Inc.

Inoculum (corn spawn and conidial suspension) preparation and application and disease rating (disease index, Fusarium Damaged Kernel (FDK) and DON content) were followed as described in (Malla *et al*., 2009). Disease index, calculated as the product of disease incidence and disease severity, was highly correlated with severity ($r \geq 0.99$, $p < 0.01$) in both years. Therefore, disease index was not used in the analysis.

Genotyping:

Diversity Array Technology (DArT): Leaf samples were collected from plants at the seven-leaf stage. DNA was extracted from the leaf samples following the CTAB method as described by Triticarte Pty. Ltd. (http://www.triticarte.com.au/content/DNA-preparation.html). Triticarte Pty. Ltd. (Canberra, Australia; http://www.triticarte.com.au) screened parents and RILs according to the method described by Wenzl *et al*. (2004) and Akbari *et al*. (2006). Each genotype was scored as either present "1" or absent "0" for DArT markers. Triticarte Pty. Ltd. assigned the prefix "wPt" (w stands for wheat, P stands for PstI and t stands for TaqI) followed by a unique four digit identification number for each of the DArT markers and the same nomenclature of DArT markers is used in this publication.

Single Sequence Repeat (SSR): DNA was extracted from leaf samples following Saghai-Maroof *et al*. (1984) with minor modifications. The polymerase chain reaction mixture (13 μL) contained 0.05 μM of forward-tailed primer (5' to 3', ACG ACG TTG TAA AAC GAC), 0.05 μM 6-FAM/VIC/NED/PET-labelled M13 primer (5' to 3', ACG ACG TTG TAA AAC GAC, Applied Biosystems), 0.10 μM reverse primer, 200 μM of deoxynucleotide, 2.5 mM $MgCl_2$, 1.5 unit Taq polymerase, 200 ng of template DNA and 1X Ammonium Sulfate Buffer. After heating the mixture to 95°C for 5 min., the PCR reaction occurred over 35 cycles. The first five cycles consisted of denaturing at 96°C for 1 min, 68°C (-2°C/cycle) for 5 min, 72°C for 1 min, followed by five cycles of 96°C for 1 min, 58°C (-2°C/cycle) for 2 min, 72°C for 1 min and the remaining 30 cycles consisted of 96°C for 1 min, 50°C for 1 min, 72°C for 1 min with a final extension step of 72°C for 5 min. PCR products were scanned with GeneScan-500 LIZ as an internal size standard

(Applied Biosystems) in an Applied Biosystems 3730 DNA Analyzer (Applied Biosystems) and the results were analyzed with GeneMarker software (Softgenetics LLC.).

QTL analysis: Marker map distance was constructed with Map Manager QTX (Manly *et al*., 2001) setting search linkage criterion at p<0.05 and using Kosambi map functions. RECORD (Van Os *et al*., 2005) was used to reorder the marker position within the linkage group. Simple interval mapping with p<0.001was used to detect QTL in the Map Manager QTX. The linkage groups containing significant QTL were again reanalyzed in Windows QTL Cartographer 2.0 (Wang *et al*., 2007) software. Composite interval mapping (Zeng, 1994) with a background control of 5 markers and a 10-cM window size was used for QTL analysis in the Windows QTL Cartographer. (Voorrips, 2002) was used to draw the linkage map for the markers.

Statistical analysis: An analysis of variance for single and combined years was conducted for disease incidence, severity, index and FDK. Proc GLM of SAS Institute Inc. (2008) was used to analyze the data. Broad-sense heritability (h^2) was estimated by:

$$h^2 = \sigma_g /(\sigma_g+\sigma_{gy}/y+\sigma_e/ry)$$

Where:
σ_g = Genotypic variance component
σ_{gy} = Genotype X year interaction variance component
σ_e = Residual error variance component
r = Replications
y = Years

The 90% confidence interval of heritability was calculated as described by (Knapp *et al*., 1985). Pearson's correlation coefficient was computed to test the association between disease incidence, severity, index, FDK and DON content using Proc Corr of SAS Institute Inc. (2008).

RESULTS

Statistical result: Correlation coefficients of lines between years were significant for incidence (r = 0.32, p<0.01), severity (r = 0.61, p<0.01) and FDK (r = 0.72, p<0.01). However, there was no correlation (r = 0.11, p = 0.50) between DON content in either year. There were significant correlations between severity and FDK (r = 0.38, p<0.01), severity and DON content (r = 0.28, p<0.01) and FDK and DON content (r = 0.28, p<0.01). Incidence was significantly correlated with severity (r = 0.40, p<0.01) but not with FDK (r = 0.12, p = 0.18) and DON content (r = 0.14, p = 0.16).

Since line means were correlated between the years for incidence, severity and FDK and that the test for homogeneity of variance was significant, data across the years were pooled for combined analysis. There were significant differences among genotypes combined over years. The genotype-by-year interactions for the above traits were also significant (Table 2). Broad-sense heritability was highest for FDK (0.45) and lowest severity (0.38).

The RILs distributions were shown for incidence, severity, FDK and DON content in Fig. 1-4, respectively in the field environment averaged across the two years. The distribution was skewed toward susceptibility for incidence as the mean incidence of the RILs was 98.7%. The resistant and susceptible parents had 93.4 and 100.0%, respectively for disease incidence. This indicated that both the parents and lines were highly susceptible to FHB initial infection. Continuous distribution was observed for severity, FDK and DON content inferring that the traits were quantitatively inherited. The average severity in the RILs was 52.0% with the lowest severity being 22.8% and the highest severity being 84.0%. The mean FDK and DON content in the RILs were 55.4% with a range of 10-85.0 and 8.5 ppm with a range of 1.2-29.0 ppm, respectively. Few lines in the study had either lower or higher values than the two parents for severity, FDK and DON content which indicates both positive and negative transgressive segregation.

Table 2: Analysis of variance and heritability estimates for incidence, severity and Fusarium Damaged Kernels (FDK) averaged across two years (2006 and 2007) in a field at Brookings, SD

		2006-07		
Source	DF	Incidence	Severity	FDK
BLOC (Year)	2	6.31^{NS}	124.89^{NS}	137.94^{NS}
Year (Y)	1	93.64*	647.89*	512.26*
Genotype (G)	127	25.28^{NS}	471.39**	503.59**
Y×G	96	15.33*	292.52**	276.96**
Error	149	19.59	124.31	125.90
h^2 (90% CI)		0.39 (0.53, 0.22)	0.38 (0.52, 0.20)	0.45 (0.57, 0.29)

*, **: Significant at 0.05 and 0.01 probability levels, respectively; ^{NS}: Non-Significant

Table 3: QTLs for resistance to FHB severity (type II) as detected by composite interval mapping in a Tokai 66/Jagalene population in a field environment at Brookings, SD

				2006			2007			2006 and 2007		
	Chromosome	Marker interval	Region Length (cM)	LOD	Additive effects	R^2 (%)	LOD	Additive effects	R^2 (%)	LOD	Additive effects	R^2 (%)
Severity	5B	wPt-9006-wPt-6348	30.3	3.5	-6.4	14.3	8.0	-9.46	32.4	8.7	-7.6	25.7
FDK†	3B	wPt-5310-wPt-7024	39.5	5.9	-8.3	27.3	5.1	-11.1	47.9	5.3	-9.6	38.4
	5B	wPt-8132-wPt-8637	9.8	-	-	-	3.4	6.2	12.2	-	-	-
DON	3D	wPt-0524-wPt-3815	60.7	-	-	-	3.3	-2.9	18.1	-	-	-

†FDK: Fusarium Damaged Kernel; DON: Deoxynivalenol

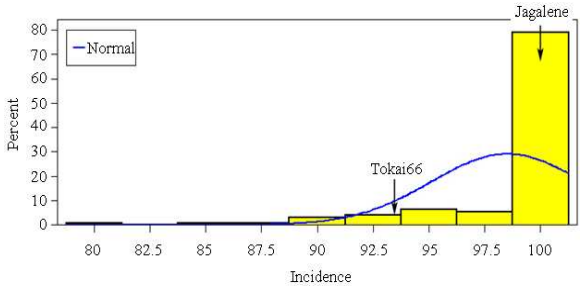

Fig. 1: Histogram of 115 RIL lines for incidence averaged across two years (2006-2007) in a field environment at Brookings, SD

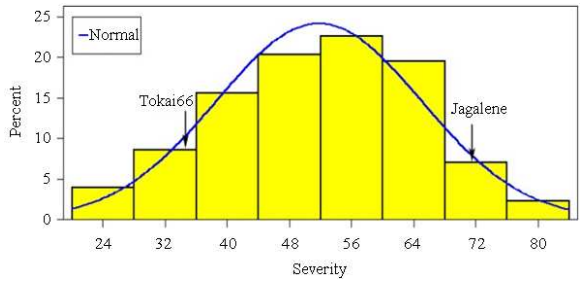

Fig. 2: Histogram of 115 RIL lines for disease severity averaged across two years (2006-2007) in a field environment at Brookings, SD

Markers and linkage map: A total of 249 polymorphic DArT (244), SSR (4) and STS (1) markers were used to construct the linkage map. Sixty-nine out of 244 DArT markers were not mapped to any chromosomal location. The 'distribution' option in Map Manager QTX was used to assign the unmapped markers into different linkage groups. There were 30 formed linkage groups (Fig. 5-9). Some of these linkage groups were vertically stacked based on the marker position in the chromosomal map of Akbari *et al*. (2006); Crossa *et al*. (2007); Mantovani *et al*. (2008) and Semagn *et al*. (2006). The longest linkage map distance was 414.4 cM for 3B linkage group, whereas the shortest linkage length was 1.7 cM for 5D linkage group. The average length of the linkage group was 83.9 cM.

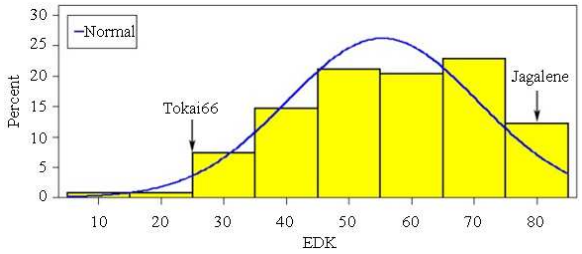

Fig. 3: Histogram of 115 RIL lines for Fusarium Damaged Kernels (FDK) averaged across two years (2006-2007) in a field environment at Brookings, SD

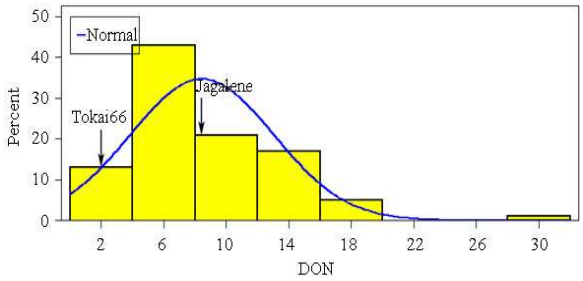

Fig. 4: Histogram of 115 RIL lines for Deoxynivalenol (DON) content averaged across two years (2006-2007) in a field environment at Brookings, SD

QTL mapping and analysis: Simple and composite interval mapping showed that there were four QTLs at three chromosomes for severity, FDK and DON content (Table 3). Consistent resistant QTL at 5B was observed for severity in both years. Likewise, a stable and resistant 3BSc QTL for FDK was observed in both years. A second susceptible 5B QTL was recorded for FDK and the QTL was unstable as it was observed only in one of the two years. An unstable resistant QTL for DON content was observed at 3D in 2007.

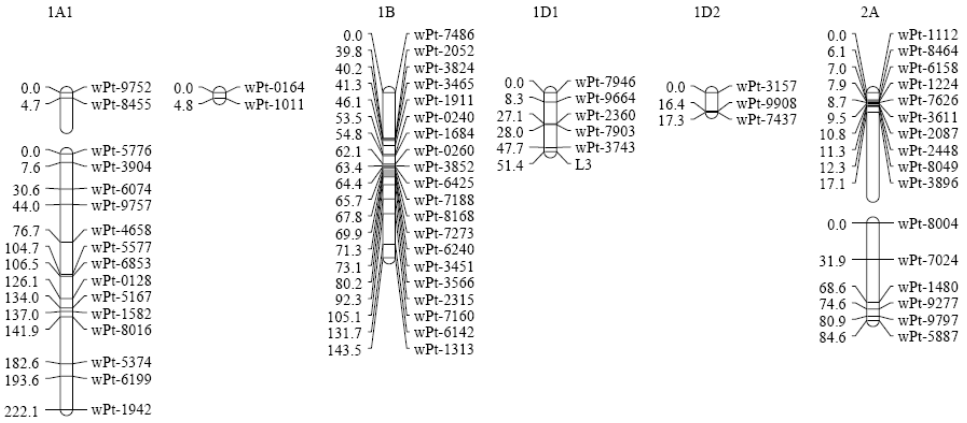

Fig. 5: Linkage map for specific chromosome (designated on top). L3 = DArT CloneID # 117430

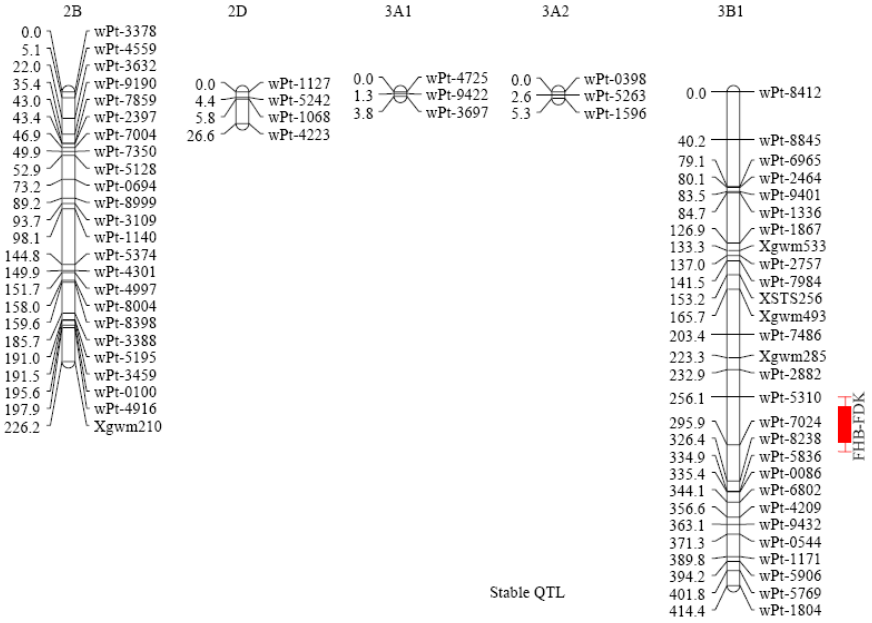

Fig. 6: Linkage map for specific chromosome (designated on top) and QTL position shown by red color on the right of the linkage map

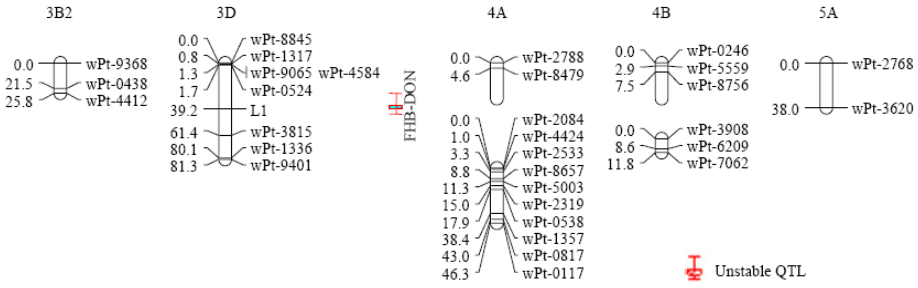

Fig. 7: Linkage map for specific chromosome (designated on top) and QTL position shown by red color on the right of the linkage map. L1 = DArT CloneID # 117472

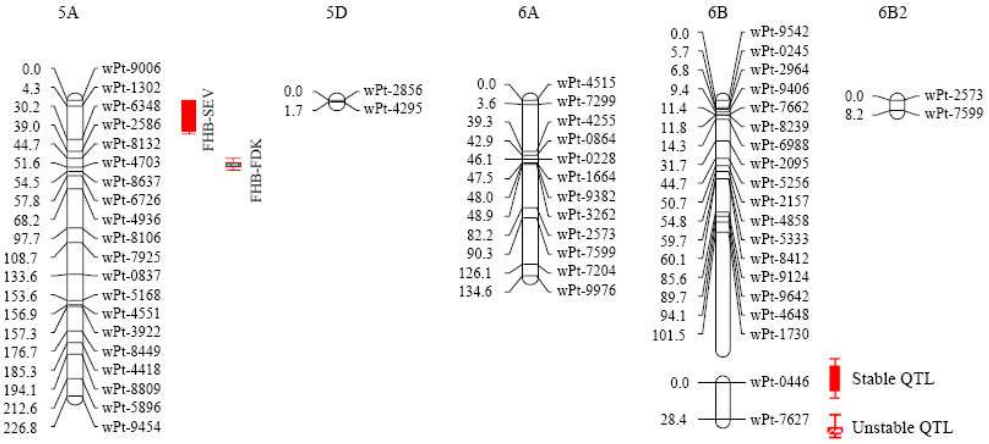

Fig. 8: Linkage map for specific chromosome (designated on top) and QTL position shown by red color on the right of the linkage map

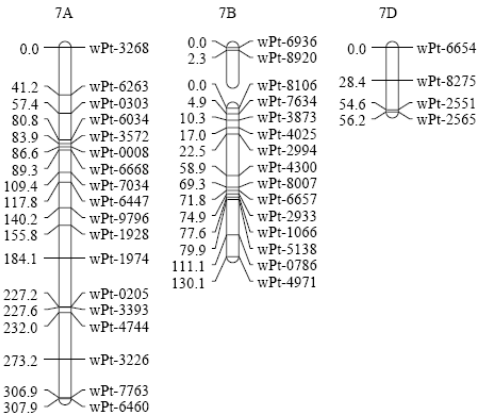

Fig. 9: Linkage map for specific chromosome (designated on top)

DISCUSSION

The mean disease incidence in this study was very high. Both Tokai66 and Jagalene showed highly susceptible reaction to incidence which suggested that the resistant parent, Tokai66, lacks type I resistance. This finding disagrees with Zhang *et al*. (2008). Due to high incidence, the distribution was skewed towards susceptibility.

Correlation was observed among severity, FDK and DON content. The result was in agreement with earlier findings where disease index, FDK and DON content were correlated (Malla, 2005). The significant correlation among the traits suggested that selecting for one trait would indirectly lead to gain in other traits. Likewise, there was correlation between years for severity and FDK but not for DON content. This highlights the complex nature of DON content expression and evaluation across years. This inconsistency in DON content across years could also be attributed to error in the sampling technique and/or small sample size (20 g). For DON content analysis, seeds were combined from two replications for each line and a sample was drawn from the composite. Furthermore, the lines varied in the amount of seed production across years, with some lines having only 5 g of ground flour available for DON content analysis. It has been reported that different isolates of the *F. graminearum* tend to vary in the production of the DON content in the seed (Mesterhazy, 2002). Ten isolates of *F. graminearum* were used in this study. A change in the dominance of *F. graminearum* isolates across the two years might have also lead to variability in DON content in the RILs in 2006 and 2007.

DArT markers detected one stable QTL for severity. This stable 5B QTL should be in a similar region where 5B QTL from Wangshuibai, a Chinese source of resistance, was reported (Akbari *et al*., 2006; Jia *et al*., 2005; Somers *et al*., 2004). In a haplotype study, Liu and Anderson (2003) reported that Sumai3 and Tokai66 contained the same 3BS allele when genotyped with the XBARC133 marker, whereas Wangshuibai did not contain any Sumai3 allele. We were not able to detect a 3BS QTL for severity in Tokai66. In another mapping study involving Tokai 66 X Y1193-6 phenotyped under greenhouse condition (spikes were point inoculated) for three seasons and genotyped with four SSR markers, Xgwm389, Xgwm493, Barc147 and STS3B-206, could not detect QTL at 3BS region for severity, FDK and DON content traits (Y. Yen, South Dakota State University, personal

communication). These findings suggested that Tokai66 is more similar to Wangshuibai than Sumai3 with regards to disease severity.

This study detected one stable and another unstable QTLs for FDK. The stable FDK QTL in Tokai 66 in this study was detected at 3BSc region exactly where Wangshuibai was also reported to have a QTL for severity (Zhou *et al*., 2004). In 2007, simple interval mapping showed a QTL for FDK at 3BS in addition to a QTL at 3BSc in Tokai 66. Simple interval mapping also showed a 3BS QTL for DON content in 2007. When data was analyzed through composite interval mapping, the QTL at the 3BS region for FDK and DON content was not detected. It can be inferred that the QTL at the 3BS region for FDK and DON content might not have a large effect, or in the case of FDK, the large effect of the 3BSc QTL might have masked the effect of the 3BS QTL. This study suggested that there might be a small effect putative QTL at the 3BS region for FDK and DON content, though the major QTL for FDK was found in the 3BSc region. An unstable and susceptible QTL was detected for FDK at the 5B region, though the QTL region was little farther from the 5B QTL for severity. Thus, a stable FDK QTL in Tokai66 similar to the region of a severity QTL in Wangshuibai again highlighted that Tokai66 is more to Wangshuibai than to Sumai3 with regard to FHB resistance.

An unstable QTL was detected for DON content in the 3D region due to complexity of DON content evaluation. Since DON content was not correlated between the years, the QTL was not detected in both years. The unstable QTL at 3D was not reported previously (Buerstmayr *et al*., 2009). This study suggested that a novel putative QTL for DON content was at the 3D region.

CONCLUSION

One stable QTL was detected for each of severity and FDK. The stable and resistant allele at 5BL for severity explained 25.7% of the phenotypic variability for FDK resistance. Similarly, a stable and resistant allele at the 3BSc for FDK explained 38.4% of the phenotypic variability. Both alleles were different from the Sumai3 allele and can, thus, be utilized through marker assisted selection in wheat breeding programs.

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