Introduction

Fusarium head blight (FHB; also known as scab disease) is caused mainly by *Microdochium nivale* and *Fusarium graminearum* in Tokachi area, Hokkaido. FHB is one of the most devastating fungal diseases of wheat (*Triticum aestivum* L.) (McMullen et al. 1997). Besides causing losses in grain yield and quality, *F. graminearum* leads to severe and harmful contamination of the grain with fungal toxins. FHB infection is favored by warm and humid climates during the flowering and maturing stages, and upward trends during growing-season temperatures are the cause of some concern in wheat-producing regions.

Host plant resistance is the most effective way to control FHB, and breeding for improved FHB resistance has thus become one of the most important targets of wheat breeding programs. Even though a number of studies have shown that the inheritance of FHB resistance is quantitative in nature, the goal for wheat breeders is to create cultivars that are regionally adapted for high yield and quality with FHB resistance. Fortunately, large genetic variations for FHB resistance in the world wheat gene pool have been reported, and several quantitative trait loci (QTL) for FHB resistance were mapped on all wheat chromosomes (Buerstmayr et al. 2013).

Currently, one of the most repeatable QTLs for FHB resistance is reported on chromosome 3BS (*Fhb1, Qfhs.ndsu-3BS*) (Anderson et al. 2001, Bai et al. 1999, Buerstmayr et al. 2002, Cuthbert et al. 2006, Pumphrey et al. 2007, Waldron et al. 1999), in addition, a nearly perfect DNA marker *Umn 10* for *Fhb1* (Liu et al. 2008) has been developed, and significant progress regarding FHB resistance has also been accomplished in a hard red spring wheat cultivar ‘Alsen’ (Bakhsha et al. 2013, Frohberg et al. 2006) that possesses *Fhb1*.

Other QTLs are mapped on chromosomes 5AS (*Fhb5, Qfhs.ifa-5A, Qfhi.nau-5A*) (Buerstmayr et al. 2003, Lin et al. 2006, Xue et al. 2011), 6BS (*Fhb2*) (Anderson et al. 2001, Cuthbert et al. 2007) and 4BL (*Fhb4, Qfhi.nau-4B*) (Somers et al. 2003, Xue et al. 2010) from Asian spring wheat sources, and 1BL (*Qfhs.lfl-1BL*) (Häberle et al. 2001).
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2009), 6AL (Qfhs.hl-6AL) and 7BS (Qfhs.hl-7BS) (Häberle et al. 2007, Schmolke et al. 2005) from European winter wheat cultivars.

It was demonstrated that the pyramiding of minor to moderate FHB resistance QTLs also provides enhanced resistance to FHB (Miedaner et al. 2006). A higher number of QTLs with minor to moderate effects have been detected in several European and North American winter wheat populations, and they were mapped on several chromosomes (Buerstmayr et al. 2009, 2013). Most of those QTLs showed minor to moderate effects on FHB resistance, depending on their genetic backgrounds. Nevertheless, they would be useful for breeding due to their improved agricultural traits compared to exotic FHB resistant sources. FHB-resistant elite wheat cultivars like ‘Glenl’ and ‘Parshall’ were reported to not possess Fhbl, which indicates that the use of new sources of FHB resistance is still meaningful for practical breeding (EIDoliefy 2015, Rosyara et al. 2009). Thus, the exploration of new genetic sources is still necessary to enhance the genetic diversity of FHB resistance.

‘Yumechikara’ is a hard red winter cultivar with wheat yellow mosaic resistance, winter hardiness and favorable bread-making quality that was developed in Hokkaido, Japan (Ito et al. 2015, Kojima et al. 2015, Tabiki et al. 2011, Terasawa et al. 2016). Repeated evaluations of the FHB resistance in Yumechikara proved its stable resistance to FHB throughout a 5-year nursery trial. Hence, an exploration of the FHB resistance in Yumechikara may provide additional sources of FHB resistance for wheat breeding. The objective of the present study was to clarify novel QTLs for FHB resistance in the advanced winter wheat bread-making cultivar Yumechikara.

Materials and Methods

Plant materials

We developed F1-derived doubled haploid (DH) lines from the cross of ‘Yumechikara/Kitahonami’, Yumechikara (Satsukei 159/KS 831957/Kitanokaori) and Kitahonami (Kitamoe/Kitakei 1660), which is a soft red winter wheat (Satsukei 159/KS 831957//Kitanokaori) and Kitahonami from the cross of ‘Yumechikara/Kitahonami’. Yumechikara

F1

Plant materials

We developed F1-derived doubled haploid (DH) lines from the cross of ‘Yumechikara/Kitahonami’, Yumechikara (Satsukei 159/KS 831957/Kitanokaori) and Kitahonami (Kitamoe/Kitakei 1660), which is a soft red winter wheat cultivar (Yanagisawa et al. 2007). The cross was made in 2003, and DH lines were developed from field-grown F1 plants using the maize pollination procedure (Inagaki and Tahir 1990).

Evaluation of FHB resistance

We evaluated their FHB resistance of 94 DH lines in a 5-year field trial (2008–2012) at the NARO Hokkaido Agricultural Research Center (Memuro, Japan) (42°53′ N, 143°05′ E). The soil type was volcanic ash soil. All DH lines and the parents were sown in mid-September. In each field trial, the experimental units contained 20 plants in a single 0.5-m row, spaced 0.72 m from the adjacent row with two replications. The plots received roughly 40 kg N ha⁻¹ at the seeding stage in the fall and an additional 60 kg N ha⁻¹ after the April snow melt. Inoculum was prepared using F. graminearum isolated in Hokkaido (Nishio et al. 2008), and colonized oat grain was incubated for 2 weeks under room temperature. Approx. 20 L/100 m² of oat grain was spread before the flowering of the DH population. A gentle mist was applied with overhead sprinklers every hour for 10 mins (from 6:00 am to 6:00 pm) to maintain high humidity from the day of the first flowering of the materials until all of the materials were scored for FHB severity. The amount of precipitation by simulated rainfall was estimated to be approx. 10 mm/day.

At 2 weeks after flowering, we scored the plants’ FHB severity on a scale of 0 to 9 per 20 plants as follows: 0 = no damage; 2 = 1–3 spikelets diseased (20%); 4 = 4–5 spikelets diseased (40%); 6 = 6–7 spikelets diseased (60%); 8 = 8–9 spikelets diseased (80%); 9 = almost all or all spikelets diseased (90% or more) based on the reported index (Ban and Suenaga 2000), and then mean scores were calculated. All tests were conducted with two replicates of each line for each year, but in 2008 the test was conducted without replication.

Molecular marker analysis

Leaf tissue (2 g) was ground in a mortar with liquid nitrogen until powdered, and then 10 ml of extraction buffer (1.5% cetyltrimethylammonium bromide [CTAB], 75 mM Tris, pH = 8.0, 15 mM Na₂EDTA, 1.05 M NaCl) was added. The resultant slurry was incubated at 65°C for 30 min (Murray and Thompson 1980). DNA was purified in 24:1 chloroform/isoamyl alcohol, precipitated from the aqueous phase by the addition of isopropanol and then washed in 70% ethanol. DNA samples were dissolved in distilled water, and the DNA concentration was determined by measuring the optical density at 260 nm.

We screened 1,224 microsatellite (SSR) markers based on the maps of Gupta et al. (2002), Guyomarc’h et al. (2002), Pestsova et al. (2000), Röder et al. (1998), Somers et al. (2004), Song et al. (2005), Sourdille et al. (2001) and Torada et al. (2006) for polymorphism between the parental cultivars Yumechikara and Kitahonami. A total of 265 primer pairs amplified one or more polymorphic bands between the parents; of these, 224 markers were used to assemble linkage groups using the mapping population.

We conducted a polymerase chain reaction (PCR) in 10-μl volumes using a GeneAmp PCR System 9700 thermal cycler (Applied Biosystems, Foster City, CA, USA). The amplification program began with 1 cycle of primary denaturation at 94°C for 5 min, followed by 42 cycles of 94°C for 1 min of denaturing, 51°C, 55°C or 61°C for annealing and 72°C for extension. One additional cycle was performed at 72°C for 5 min for the final elongation of the PCR product. Each 10-μl PCR reaction contained 10 ng of template DNA, 1 × PCR mix, 1.7 mM MgCl₂, 200 nM of each primer, 200 μM of each dNTPs and 0.2 U Taq polymerase (Promega, Madison, WI). The PCR products were separated on a Sub-Cell® Model 96/192 (Bio-Rad, Hercules, CA) electrophoresis apparatus with 4% agarose gels. The gels
were run in 1 × TBE at a constant 100 V for 2 hr and then visualized after staining with ethidium bromide for 30 min.

**Mapping and QTL analysis**

We used MAPMAKER/Exp v3.0b (Lander et al. 1987) to construct the wheat microsatellite map. The Kosambi function was applied to convert recombination fractions into map distances (Kosambi 1944). Logarithm of odds (LOD) thresholds of 3 and a distance threshold of 35 cM were used in the ordering and grouping of markers. We analyzed the QTLs for FHB resistance by performing composite interval mapping (CIM) using Windows QTL Cartographer ver. 2.5 with model 6 of the Zmapqtl procedure (Basten et al. 1996, Wang et al. 2006). For each analysis, genome-wide thresholds of LOD scores for significant (P<0.05) QTLs were determined by 1,000 permutations, and the significance of the detected QTLs was confirmed.

**Results**

**Phenotypic evaluation of FHB response**

The flowering date of parents and the mean temperatures during FHB development (from the first flowering of the DH lines to 2 weeks after the last flowering of the DH lines) in the 5-year field experiments are shown in **Table 1**. The mean values of the FHB disease index of the DH lines and their parental cultivars for the 5-year tests are shown in **Fig. 1**. The resistant parent Yumechikara showed a mean FHB severity of 3.6, ranging from 3.3 to 4.4 across the tested years, whereas the susceptible parent Kitahonami showed a mean FHB severity of 5.4, ranging from 4.8 to 6.3. The mean FHB severity of all DH lines was 5.1 and ranged from 4.5 to 5.5 across the tested years, and the results indicated a continuous variation of FHB severity with a single peak (Fig. 1).

Yumechikara showed the most extreme disease-resistance index among the progeny lines except for 2010, whereas transgressive segregation was observed in the susceptible lines, suggesting that the susceptible parent might contribute to resistance QTLs in the population. The ANOVA results indicated highly significant variations in genotypes, tested years, and genotype by tested year interactions (**Table 1**).

**Effects of QTL on FHB resistance**

Our QTL analysis revealed that a distal region of the short arm of chromosome 1B of Yumechikara was significantly associated with FHB resistance through all five tested years (**Fig. 2, Table 3**). The FHB-resistance QTL peak of Yumechikara was mapped between the low-molecular weight glutenin subunit gene Glu-B3 and the SSR marker Xbarc32, and the QTL region of approx. 10 cM included the wheat glume color gene Rg-B1 (Khlestkina et al. 2006) (**Fig. 2**). The phenotypic variations of the FHB disease index explained by the QTL ranged from 23.9% to 31.8%, and that for the combined mean was 36.4% over the 5-year trial (**Table 3**).

Another QTL for FHB resistance was detected on chromosome 3B of the susceptible parent cultivar ‘Kitahonami’. The putative resistance QTL for FHB in Kitahonami was located between the SSR markers Xgwm389 and Xwmc754, and the explained phenotypic variation ranged from 2.9% to 12.7%, and that for the combined mean was 11.2%; the effects of this QTL varied among the years as the LOD score was not significant in 2008, 2010 or 2011 (**Table 3**). The effect of the QTL on chromosome 3BS was smaller than that of the QTL on chromosome 1BS through all five tested years.

**Discussion**

For practical breeding, the identification and utilization of FHB resistance in advanced wheat lines are important to achieve improved FHB management. Several QTLs for FHB resistance have been reported from Asian FHB-resistant spring wheat gene pools (Anderson et al. 2001, Bai et al. 1999, Buerstmayr et al. 2002, Waldron et al. 1999). Nevertheless, the use of those FHB-resistant sources sometimes corrupts important traits like winter hardiness and bread-making quality. Thus, the exploration, validation and use of new FHB-resistant sources from elite cultivars are valuable for practical breeding.

In this study, Yumechikara showed stable resistance compared to Kitahonami by serial 5-year field trials, and a significant FHB-resistant QTL was identified on the distal region of chromosome 1BS from the resistant parent Yumechikara. Our results indicated that the expression of...
the FHB-resistance QTL is rather stable under varied climate conditions. Out of 52 studies reporting a QTL for FHB resistance in wheat, nine studies reported QTLs on chromosome 1B (Buerstmayr et al. 2009), including T1BL.1RS translocation derived from the rye chromosome. Since both parents Yumechikara and Kitahonami do not possess T1BL.1RS translocation (data not shown), we propose that the QTL of Yumechikara is a novel FHB-resistance QTL in winter wheat. From the consensus map of wheat (Somers et al. 2004), it is apparent that the Glu-B3 gene is located 5 cM from the distal end of the FHB-resistance QTL has been mapped on the long arm of wheat chromosome 1B (Qfh.s.lfl-1BL) in European winter wheat Cansas (Häberle et al. 2009), the CIMMYT breeding line CM-82036 (Buerstmayr et al. 2002), Chinese land race Wangshuibai (Zhou et al. 2004), the CIMMYT cultivar Seri 28 (Mardi et al. 2006), and the Swiss cultivar Arina (Semagn et al. 2007), whereas the rest of the QTLs are mapped on the rye chromosome of T1BL.1RS translocation (Ittu et al. 2000, Schmolke et al. 2005, Shen et al. 2003, Zhang et al. 2004).
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Fig. 2. Maps of quantitative trait loci (QTL) for FHB resistance constructed from doubled haploid lines derived from ‘Yumechikara/Kitahonami’ based on the 5-year field experiments.

Table 3. Flanking markers, logarithm of the odds (LOD), coefficients of determination (R²), and additive effect of the significant QTL regions detected by the composite interval mapping based on the 5-year field experiments.

| Years | Resistance donor | Locus Flanking markers | Yumechikara 1BS | Kitahonami 3BS | Threshold of LOD scores | LOD | R² (%) | Additive effect |
|-------|------------------|------------------------|-----------------|---------------|-------------------------|-----|--------|----------------|
| 2008  | Yumechikara      | Glu-B3-Xbarc32         | 8.71            | 1.76          | 3.61                    | 8.73| 28.0   | -0.454          |
|       | Kitahonami       | Xgwm389-Xwmc754        | 1.07            | 0.197         |                         | 7.27| 3.72   |                 |
| 2009  | Yumechikara      | Glu-B3-Xbarc32         | 7.27            | 3.72          | 3.73                    | 7.27| 12.4   | -0.327          |
|       | Kitahonami       | Xgwm389-Xwmc754        | 3.72            | 0.228         |                         | 7.27| 12.4   |                 |
| 2010  | Yumechikara      | Glu-B3-Xbarc32         | 8.73            | 0.95          | 3.03                    | 8.73| 3.0    | -0.386          |
|       | Kitahonami       | Xgwm389-Xwmc754        | 1.07            | 0.122         |                         | 7.27| 3.0    |                 |
| 2011  | Yumechikara      | Glu-B3-Xbarc32         | 8.69            | 2.95          | 3.39                    | 8.69| 2.95   | -0.480          |
|       | Kitahonami       | Xgwm389-Xwmc754        | 2.95            | 0.279         |                         | 8.69| 2.95   |                 |
| 2012  | Yumechikara      | Glu-B3-Xbarc32         | 11.15           | 4.69          | 2.7                     | 11.15| 4.69  | -0.429          |
|       | Kitahonami       | Xgwm389-Xwmc754        | 4.69            | 0.271         |                         | 11.15| 4.69  |                 |
| Combined mean | Yumechikara | Glu-B3-Xbarc32         | 13.67           | 4.69          | 3.79                    | 13.67| 4.69  | -0.396          |
|       | Kitahonami       | Xgwm389-Xwmc754        | 4.69            | 0.215         |                         | 13.67| 4.69  |                 |

Fig. 3. Effects of the four different combinations of QTLs for the FHB disease index in the doubled haploid lines derived from ‘Yumechikara/Kitahonami’ based on the 5-year field experiments. YK carries the resistance alleles at QTLs on 1BS and 3BS. YY carries a resistance allele at QTL-1BS. KK carries a resistance allele at QTL-3BS. KY carries none of the resistance QTLs. The lines in which all genotypes are the same between flanking markers (GluB3-barc32 for 1BS and gwm384-wmc754 for 3BS) were selected.

chromosome 1BS, and there are no previous reports of an FHB-resistance QTL on such a distal end region of wheat chromosome 1BS.

Wheat glume color is controlled by the three homoeologous major genes Rg-A1, Rg-B1, and Rg-D1 located on chromosomes 1A, 1B, and 1D, respectively (Khlestkina et al. 2006). Yumechikara has the Rg-B1b allele on chromosome 1B that shows red glume color, whereas Kitahonami has Rg-B1a with white glume color. The peak of the FHB resistance QTL from Yumechikara included the flanking
region of the glume color gene \( Rg-B1 \) (Fig. 2), indicating that the glume color should be a useful phenotypic marker to screen FHB-resistant lines. In the present study, the FHB resistance was scored after two weeks of flowering when the glume coloration is still in the beginning stage, and thus the FHB disease index scoring was not influenced by the glume color.

On the distal region of the short arm of chromosome 1B, the low-molecular-weight glutenin subunit gene \( Glu-B3 \) was also located close to the peak of the FHB-resistance QTL. Based on our analysis of the \( Glu-B3 \) alleles for the pedigree lines of Yumechikara (KS 831957/Satsukei 159//Kitanokari), we speculate that the source of the FHB-resistance QTL is the former major cultivar Takunekomugi, a parental line of Satsukei 159, which is derived from the native winter wheat gene pools, because only Takunekomugi and its descendants including Yumechikara possess the \( Glu-B3ab \) \((b^*)\) allele.

It has been reported that cosegregation of the wheat glume color gene \( Rg-B1 \) and the low-molecular-weight glutenin subunit gene \( Glu-B3 \) can be applied as a useful ‘field-marker’ for desired gluten strength (Fujii et al. 2011). Since Yumechikara is characterized as extra-strong-quality wheat that possesses a combination of the high-molecular-weight gluten subunit allele \( Glu-D1d \) and low-molecular-weight glutenin subunit allele \( Glu-B3ab \) \((b^*)\) (Ito et al. 2015), this close linkage between the FHB-resistance QTL, \( Glu-B3 \) and \( Rg-B1 \) brings an additional value of simultaneous screening for both quality and FHB resistance improvement.

The FHB-resistance QTL from the susceptible parent Kitahonami was mapped on the short arm of chromosome 3B, the nearby \( Fhb1 \) region between \( Xgwm389 \) and \( Xwmc754 \). The effect of combinations of the two FHB-resistance QTLs from both Yumechikara and Kitahonami were validated among the DH lines in which all genotypes are the same between flanking markers \((Glub3-barc32\) for 1BS and \( gwm384-wmc754\) for 3BS) (Fig. 3). The results indicated an obvious additive effect by the combination of two FHB-resistance QTLs derived from both parental cultivars Yumechikara and Kitahonami (YK type) with the lowest FHB disease index, whereas the DH lines with two FHB-susceptible QTLs from both parents (the KY type) showed the most severe FHB disease index (Fig. 3). The DH lines with each parental-type QTLs (YY and KK types) showed FHB disease index values that were intermediate and similar to their respective parents. In this study, most of the DH lines showed higher FHB severity compare to Yumechikara (Fig. 1), and minor QTLs that did not show significant LOD scores in all field trials were detected on chromosomes 2A, 6D and 7D from Yumechikara. We speculate that the reason for the higher FHB severity in DH lines compare to resistant parent Yumechikara is the absence of those minor QTLs. However, the number of markers in the minor QTLs is still small and further analysis is necessary to elucidate the location and interactions between them.

Among the five tested years, the effect of FHB-resistance QTLs was the least in 2010, when the mean temperature between June and August in Hokkaido was the highest since the start of recorded observations in 1946, and the mean number of days from heading to maturation was shortest at only 37 days, which brought about a historically low yield (Nishio et al. 2013). In 2010, both parents showed a higher FHB disease index than the other years, which might be one of the reasons for the lower effects of the FHB resistance QTL (Fig. 1). The correlation coefficient between the mean temperature from heading to maturing and the mean FHB disease index was not significant (data not shown); nevertheless, the unusual high temperature during the FHB disease occurrence in 2010 might have contributed to the lower effect of the FHB-resistance QTLs.

In this study, we clarified a novel FHB-resistance QTL on chromosome 1BS that is closely linked to both the glume color gene \( Rg-B1 \) and the low-molecular-weight glutenin subunit gene \( Glu-B3 \), and a minor to moderate FHB-resistance QTL on chromosome 3BS. To our knowledge, there have been no reports on the relationship between FHB resistance and glume color in wheat, and the QTL could be a potentially new FHB-resistance source. Further study is necessary to confirm whether the glume color may have a certain effect on \( Fusarium \) infection into the spike.

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