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ORIGINAL PAPER

Genetic mapping of QTL for resistance to Fusarium head blight spread (type 2 resistance) in a *Triticum dicoccoides* × *Triticum durum* backcross-derived population

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Abstract Improvement of resistance to Fusarium head blight (FHB) is a continuous challenge for durum wheat breeders, particularly due to the limited genetic variation within this crop species. We accordingly generated a backcross-derived mapping population using the type 2 FHB resistant *Triticum dicoccoides* line Mt. Gerizim #36 as donor and the modern Austrian *T. durum* cultivar Helidur as recipient; 103 $BC_1F_{6:7}$ lines were phenotyped for type 2 FHB resistance using single-spikelet inoculations and genotyped with 421 DNA markers (SSR and AFLP). QTL mapping revealed two highly significant QTL, mapping to chromosomes 3A and 6B, respectively. For both QTL the *T. dicoccoides* allele improved type 2 FHB resistance. Recombinant lines with both favorable alleles fixed conferred high resistance to FHB similar to that observed in the *T. dicoccoides* parent. The results appear directly applicable for durum wheat resistance breeding.

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Introduction

Fusarium head blight (FHB), caused by different *Fusarium* species, affects small grains throughout the world (McMullen et al. 1997). FHB causes severe yield and quality losses, but the primary food safety issue is the contamination of the crops with mycotoxins (Dexter and Nowicki 2003; de Nijs et al. 1996; Beardall and Miller 1994). This is particularly relevant for durum wheat (*Triticum durum* Desf.), because it is used almost exclusively for human nutrition. Durum wheat was reported early to appear more susceptible than bread wheat (Atanasoff 1920), and durum wheat cultivars are still generally considered susceptible to FHB. Almost no variation in resistance to FHB has been found within *T. durum,* with most lines being susceptible, even among large germplasm collections of several thousand lines (Stack et al. 2002; Elias et al. 2005). Recently, five *T. durum* lines from a Tunisian source with moderate FHB resistance were identified by single-floret inoculation with Fusarium spore suspensions (Huhn et al. 2012) and four Syrian durum landraces showed stable resistance after spray inoculation (Talas et al. 2011). With the aim of expanding the resistance resources for durum breeders, related tetraploid species have been screened. Lines with moderate to good resistance to FHB were found in *T. dicoccoides, T. dicoccum,* and *T. carthlicum* (Buerstmayr et al. 2003a; Oliver et al. 2007, 2008).

Inheritance of resistance to FHB in wheat is of a quantitative, polygenic nature (Bai and Shaner 1994). Genotype-by-environment interactions and the complex nature of FHB resistance make breeding for improved resistance challenging. Different types of resistance have been described (Schroeder and Christensen 1963; Mesterhazy 1995), of which resistance to initial infection (type 1) assessed by spray inoculation and resistance to fungal

spread within the spike (type 2) assessed by single-floret inoculation are frequently evaluated in QTL mapping. Schroeder and Christensen (1963) observed that type 1 and 2 resistance varied independently among cultivars. In several studies, different QTL and/or dissimilar estimates of QTL effects for type 1 or 2 resistance have been observed in the same plant material (Buerstmayr et al. 2002, 2003b, 2009; Lin et al. 2004, 2006; Chen et al. 2006). Type 2 resistance is generally considered to be less influenced by environmental variation than type 1, and many researchers (Bai and Shaner 1996; Schroeder and Christensen 1963; Wang and Miller 1988) have concluded that resistance to fungal spread within the spike provides one of the most reliable estimates of FHB resistance. This reliability may rest on the direct placement of inocula into the florets, reducing escapes from infection. Moreover type 2 resistance evaluations are usually performed in greenhouse experiments, with better-controlled environments and thus less influence from genotype-by-environment interactions.

Although numerous mapping studies have been conducted in bread wheat, resulting in more than 100 reported QTL for FHB resistance, relatively little research has been done to evaluate resistance to FHB in tetraploid wheat (reviewed by Buerstmayr et al. 2009). To date, QTL mapping analysis has characterized the genetics of only six tetraploid resistance sources, comprising three different *T. dicoccoides* lines or accessions (Otto et al. 2002; Stack and Faris 2006; Kumar et al. 2007; Gladysz et al. 2007), one *T. carthlicum* cultivar (Somers et al. 2006) and two *T. dicoccum* lines (Buerstmayr et al. 2012; Ruan et al. 2012). An association-mapping study examined resistance in a breeding population derived from Tunisian by USA *T. durum* crosses (Ghavami et al. 2011). The so far published studies in tetraploid wheat resulted in reports of QTL on chromosome 3A (Otto et al. 2002; Gladysz et al. 2007), 4A (Gladysz et al. 2007), 6A (Buerstmayr et al. 2012), 7AL (Kumar et al. 2007; Ruan et al. 2012), 2BL (Somers et al. 2006; Gladysz et al. 2007), 3B (Buerstmayr et al. 2012; Ruan et al. 2012), 4B (Gladysz et al. 2007; Buerstmayr et al. 2012), 5BL (Ghavami et al. 2011), 6BS (Stack and Faris 2006; Somers et al. 2006; Buerstmayr et al. 2012), and 7B (Buerstmayr et al. 2012). QTL on 3A, derived from either *T. dicoccoides* accession Israel A (Otto et al. 2002) or *T. dicoccoides* line Mt. Hermon #22 (Gladysz et al. 2007), mapped close to marker *Xgwm2*. QTL on 6BS, derived from *T. carthlicum* cultivar Blackbird (Somers et al. 2006) or from *T. dicoccum* line 161 (Buerstmayr et al. 2012) coincided with *Fhb2* (Cuthbert et al. 2007), and QTL on 4B derived from *T. diccocum*-161 co-mapped with the plant height allele *Rht-B1a* (Buerstmayr et al. 2012).

Negative associations between plant height and FHB severity were frequently apparent in field trials conducted by spray inoculation—shorter lines tended to be more diseased—and QTL for FHB severity and plant height co-localized several times (Buerstmayr et al. 2011, 2012; Draeger et al. 2007; Gervais et al. 2003; Handa et al. 2008; Klahr et al. 2007; Paillard et al. 2004; Schmolke et al. 2005, 2008; Srinivasachary et al. 2008). For example, in a *T. dicoccum* (line 161) × *T. durum* (cultivar Helidur) population a strong association of QTL for FHB severity with the plant height gene *Rht-B1* was detected (Buerstmayr et al. 2012). Such a relationship was not found in studies where type 2 resistance with single-floret inoculations was tested.

Mt. Gerizim #36 and Mt. Hermon #22 were the lines with the lowest FHB disease ratings among a set of 151 *T. dicoccoides* genotypes tested by Buerstmayr et al. (2003a). The objectives of the present study were to characterize resistance to fungal spread of Mt. Gerizim #36 in a backcross population using Mt. Gerizim #36 as donor parent and the Austrian *T. durum* cultivar Helidur as recurrent parent and to evaluate the relationship between type 2 FHB resistance and plant height in this population.

Materials and methods

Plant material

A population of 103 BC_1F_6 lines was developed from a cross between the *T. dicoccoides* line Mt. Gerizim #36 and the Austrian *T. durum* cultivar Helidur. F_1 plants were backcrossed as female to Helidur. The resulting 103 BC_1F_1 plants were advanced by single-seed-descent to the BC_1F_6 generation. Seed from each BC_1F_6 plant was bulked, to yield $BC_1F_{6:7}$ lines, which formed the plant material for genotyping and phenotyping. The resistance donor parent Mt. Gerizim #36 was identified as moderately type 2 FHB resistant by Buerstmayr et al. (2003a). Mt. Gerizim #36 originates in a collection of the Institute of Evolution, University of Haifa, Israel. It is a hulled wheat with brittle rachis, has a short and awned spike phenotype and tough glumes, and is tall and sensitive to lodging. The Austrian *T. durum* cultivar Helidur with pedigree Pandur/CPB132/3/Valdur//Pandur/Valgerado was used as recurrent parent. Helidur is susceptible to FHB and has a dense-spike phenotype and long awns. Helidur carries the semi-dwarfing allele *Rht-B1b*.

FHB resistance evaluation

The 103 $BC_1F_{6:7}$ lines, both parents and several control lines were tested for type 2 resistance using single-floret inoculations. Four independent inoculation experiments were conducted in the greenhouse: winter 2007/08, spring 2008, winter 2008/09, and spring 2009; hereafter abbreviated as GW07, GS08, GW08, and GS09, respectively.

Experiments were arranged in a randomized complete block design with two replicates in GW07 and GS09, and three replicates in GS08 and GW08. Replicates were intentionally planted several days apart, resulting in a few days difference in anthesis between the replicates.

Seeds were germinated in multi-trays and seedlings (1–2 leaf stage) were vernalized at 4 °C for 4–6 weeks. Six to eight vernalized seedlings were planted in 4 l pots (16 cm diameter, 20 cm height) filled with a standard potting mix consisting of 70 % recycled compost, 28 % peat, and 2 % silica sand in the greenhouse experiments. Temperature regime was 14 °C/10 °C day/night with a 12 h photoperiod for the first 30 days and then increased to 20–22 °C/18 °C day/night with a 16 h photoperiod. For fungal disease control, greenhouse cabins were treated with vaporized sulphur twice per week until BBCH stage 37. For insect control, plants were treated with the insecticide 1-(6-chlor-3-pyridinylmethyl)-*N*-nitroimidazolidin-2-ylidenamin (brand name Confidor® WG70, Bayer Crop Science, Germany) 0.1 g/l in aqueous solution at the late tillering stage (BBCH 29).

A macro conidial suspension of *F. graminearum* single-spore isolate 'IFA-104', prepared as described by Buerstmayr et al. (2002), was used for the greenhouse experiments. Conidia aliquots were stored at −30 °C. Prior to inoculation, frozen aliquots were thawed at 37 °C and diluted to a concentration of 50,000 conidia/ml with distilled water. Individual florets of the spikes were inoculated slightly above the center of the spike at mid-anthesis. In experiment GW07 a 10 µl droplet of conidia suspension was directly pipetted into two florets, avoiding any injury of the spike. In experiments GS08, GW08, and GS09, a micropipette equipped with an injection needle was punched through the outer glumes, and 10 µl inoculum suspension was injected into one spikelet per spike. On average eight spikes per genotype were inoculated in each replicate. After inoculation, spikes were covered overnight with translucent polyethylene bags to maintain high humidity.

The number of FHB symptomatic or bleached spikelets was counted as infected spikelets (NIS), and the total number of spikelets per spike (TNS) was counted at day 21 post inoculation, and the percentage of infected spikelets per spike (PIS) was calculated. PIS was used as measure for FHB spread within the spike (type 2 resistance sensu Schroeder and Christensen 1963).

Plant height was measured in experiments GS08, GW08, and GS09.

Molecular marker analysis

Genomic DNA was extracted from fresh leaves of 10 pooled plants of each $BC_1F_{6:7}$ line and of the parental lines using a simplified CTAB-based procedure modified from Saghai Maroof et al. (1984). All lines were genotyped with 116 simple sequence repeat (SSR) markers, composed of 42 BARC markers (Song et al. 2005), 66 GWM markers (Roeder et al. 1998), seven WMC markers (Somers et al. 2004), one CFA marker (Sourdille et al. 2001), and allele specific markers discriminating *Rht-B1a/Rht-B1b* (Ellis et al. 2002). SSR marker analysis was done as described by Steiner et al. (2004). Lines were additionally screened with 59 amplified fragment length polymorphism (AFLP) primer combinations. AFLP marker analysis (Vos et al. 1995) was performed using *Mse*I/*Sse8387*I restriction enzymes as described by Hartl et al. (1999) and Buerstmayr et al. (2002). AFLP markers were abbreviated according to the standard list of AFLP primer nomenclature (http://wheat.pw.usda.gov/ggpages/keygeneAFLPs.html) followed by a number that refers to a specific polymorphic band.

Statistical analysis

Phenotypic data

Means of the phenotypic traits within replicates and within experiments were calculated and used for statistical analysis. Pearson correlation coefficients were calculated for PIS between experiments and between means over all experiments for PIS, NIS, TNS, and plant height. The effects of replicates within experiments, experiment, genotype, and genotype-by-experiment interaction were estimated using the general linear model (GLM) procedure, with all effects fixed. Broad-sense heritability was estimated from variance components with the equation $H^2 = \sigma_G^2/(\sigma_G^2 + \sigma_{G\times E}^2/e + \sigma_\varepsilon^2/en)$, where σ_G^2 = genotypic variance, $\sigma_{G\times E}^2$ = genotype-by-experiment interaction variance, σ_ε^2 = error variance, e = number of experiments, and n = number of replicates (Nyquist 1991). For the estimation of variance components and broad-sense heritability all effects were considered random. ANOVA and correlation analyses were calculated in SAS/STAT version 9.2 (SAS Institute Inc 2008).

Marker data

Conformity of marker segregation with expected ratios was determined by Chi square tests. Map construction was done with CarthaGene 1.2-LKH (de Givry et al. 2005) specifying a BC_1F_6 genetic model. Distances between markers in cM were calculated based on the Kosambi mapping function. SSR markers, particularly their map information from GrainGenes (http://wheat.pw.usda.gov/ggpages/maps.shtml), were used as reference points to assign linkage groups to specific chromosomes.

QTL analysis

QTL analysis was done with QGene (version 4.3.10, Nelson 1997). QTL were identified using simple interval mapping (SIM, Haley and Knott 1992) and composite interval mapping (CIM, Zeng 1994) as implemented in QGene. The support interval of a QTL was declared as the map distance with a LOD decrease of 2 from the maximum LOD position. The percentage of phenotypic variance explained by a QTL and its additive effect were calculated. The critical LOD values at a type I error rate of α 0.1 < LOD, α 0.05 < LOD, and α 0.01 < LOD were determined by 1,000 permutations. We fitted a linear model of all significant QTL simultaneously using the GLM procedure of SAS/STAT to estimate the total percentage of phenotypic variance explained by QTL. Linkage groups and LOD profiles were drawn with MapChart 2.2 (Voorrips 2002).

Results

Trait variation and correlation

The backcross-derived mapping population allowed reducing the frequency of *T. dicoccoides*-like plant types, and most of the lines had a durum wheat-like phenotype. The frequency distributions of the lines for PIS and for NIS are depicted in Fig. 1. The histogram for NIS shows a bimodal distribution with a peak at five and a second peak at eight infected spikelets—these peaks correspond to 30 and 60 % infected spikelets for PIS, respectively. The *T. durum* parent Helidur developed on average three times more diseased spikelets (NIS 7.6) than the *T. dicoccoides* parent Mt. Gerizim #36 (NIS 2.7) across all experiments (Table 1). The hexaploid control line CM-82036 (highly type 1 and 2 resistant) showed consistently only one infected spikelet per spike, resulting in 6 % PIS, and the cultivar Frontana (type 1 resistant, type 2 susceptible) had an average of 54 % (means across experiments). Transgressive segregation towards susceptibility was apparent in all experiments. About 20 % of the lines reached significantly higher infection severity for means across all experiments (Fig. 1; Table 1) compared to the susceptible parent Helidur. Means of the parents, means and ranges of the population, least significant differences and broad-sense heritability for PIS, NIS, TNS, and plant height are summarized in Table 1. Correlation coefficients for PIS between greenhouse experiments ranged from $r = 0.40$ to 0.71 ($p < 0.0001$). TNS was positively correlated with NIS ($r = 0.31$, $p = 0.0015$), but TNS was not correlated with PIS. There was a significant positive correlation between plant height and PIS ($r = 0.29$, $p = 0.0025$) for means across all experiments, but in individual experiments, this association reached significance only in experiment GW08, with taller plants showing more disease than shorter plants. For detailed information on correlations between experiments and between traits refer to the electronic supplementary material (S1). Analysis of variance for PIS

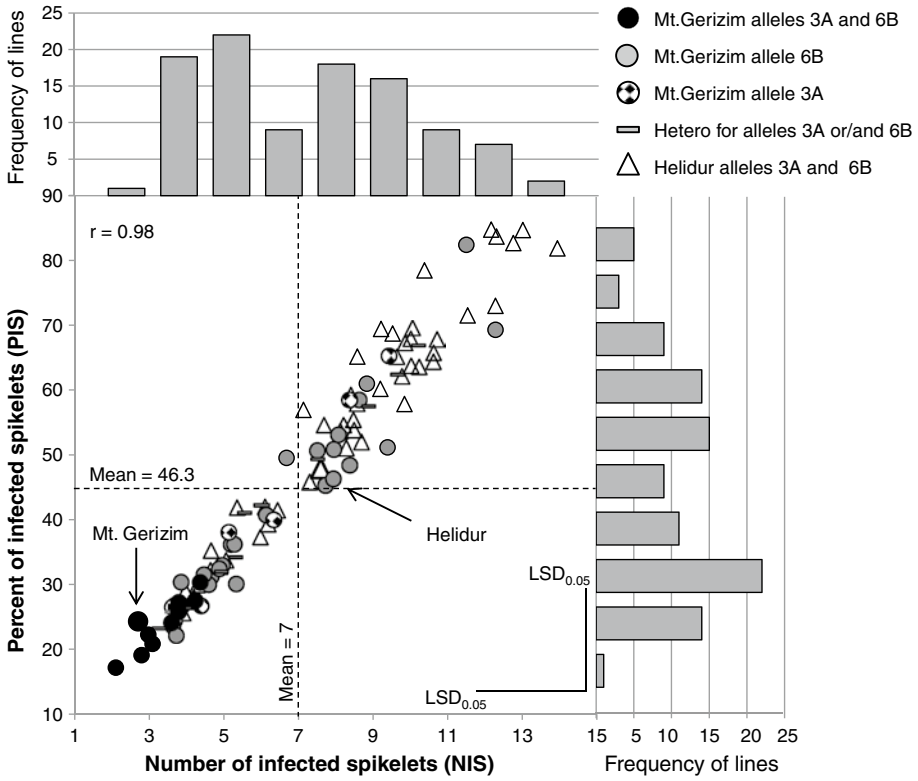

Fig. 1 Scatterplots of overall means for number of infected spikelets (NIS) against percentage of infected spikelets (PIS) with marginal histograms of their frequency distribution. Allele status in the QTL regions on 3B and 6B are represented by different symbols. *Arrows* indicate means for parents

Table 1 Means of parents and population, minimum and maximum values of the BC_1F_6 population, least significant differences at $\alpha < 0.05$ ($LSD_{0.05}$) and broad-sense heritability (H^2) or repeatability of the analyzed traits: percentage of infected spikelets per spike (PIS), number of infected spikelets per spike (NIS), total number of spikelets per spike (TNS) and plant height

	Mt. Gerizim #36	Helidur	Mean	Max	Min	H^2	$LSD_{0.05}$
PIS							
Overall mean[a]	24.3	47.9	46.3	84.7	17.2	0.84	15.5
GW07	29.4	49.1	36.5	93.4	6.1	0.60[b]	18.1
GS08	35.0	56.8	58.9	100.0	18.3	0.77[b]	14.6
GW08	11.1	35.0	45.9	96.3	6.5	0.79[b]	16.7
GS09	21.6	50.8	43.9	90.4	10.8	0.78[b]	12.0
NIS[a]	2.7	7.6	7.0	13.9	2.1	0.84	2.5
TNS[a]	11.3	15.8	15.1	18.3	12.4	0.88	0.8
Plant height (cm)[a]	90.8	71.6	86.4	128.9	58.3	0.94	17.8

[a] Means averaged over all experiments

[b] Repeatability

revealed highly significant effects for all sources of variance (electronic supplementary material S2). The magnitude of genotype-by-experiment interaction was low compared to the effect of genotype, resulting in a high broad-sense heritability coefficient $H^2 = 0.84$.

Map construction

SSR and AFLP genotyping resulted in 421 distinct polymorphic markers. These markers were used for map construction and fell into 38 linkage groups, of which 15 could be assigned to genome A and 14 to genome B, while nine groups could not be unambiguously assigned to a chromosome. Total genome length was 1,808 cM, partitioned into 845 cM for genome A, 772 cM for genome B, and 191 cM unassigned. Average distance between markers was 4.3 cM. Small segregation distortion at $p < 0.05$ was observed over 82 and 29 cM towards the Helidur and *T. dicoccoides* parents, respectively. No segregation distortion was evident at any of the QTL regions described below.

QTL analysis

FHB spread within the spike—type 2 resistance

Using SIM and CIM fitting, two QTL for PIS were identified, mapping to chromosomes 3A and 6B. The allele of the *T. dicoccoides* parent Mt. Gerizim #36 improved resistance in both cases. Positions of these QTL and estimates of QTL effects for individual experiments and for means over experiments are listed in Table 2, and LOD profiles are shown in Fig. 2. The QTL on 3A reached LOD > 3 in experiment GS08, GW07, and GW08. The support interval of this QTL stretched over a distance of 30 cM by SIM, and was narrowed to 12 cM by CIM. QTL on 6B exceeded LOD 3 in experiments GS08, GS09, and GW08, but was not detected in experiment GW07. The support interval spanned a distance of 13 cM by SIM and CIM. Both QTL, on 3A as well as on 6B, exceeded the critical LOD value of $\alpha < 0.01$ estimated by permutation tests for means averaged over experiments.

Whereas QTL on 3A and 6B explained 17 and 19 %, respectively, of the phenotypic variance there were still individual lines fixed for the favorable allele on either 3A or 6B that appeared FHB susceptible (Fig. 1). QTL on 3A and 6B together explained 29 % of the phenotypic variance. Nine lines were homozygous for the resistance-improving alleles at both QTL and these nine lines expressed resistance levels similar to that of the resistance donor Mt. Gerizim #36. But there were a few lines with moderate resistance scores that carried none of the positive alleles on 3A and 6B (Fig. 1).

Weak but not significant associations between *Rht-B1* and PIS were apparent in experiments GW07 and GW08 (results not shown), in which the semi-dwarf allele *Rht-B1b* was associated with improved resistance to fungal spread.

Plant height

The *Rht-B1* locus had a pronounced effect on plant height and accounted for 82 % of phenotypic variance. The mutant allele for reduced height was contributed by the *T. durum* parent Helidur. Plants homozygous for the *Rht-B1b* allele were on average 36 cm shorter than lines homozygous for the *Rht-B1a* wild-type allele (Table 3).

Discussion

In this study we analyzed resistance to Fusarium spread within the spike (type 2 resistance) in four greenhouse experiments. Correlations between the individual greenhouse experiments were moderate. The high broad-sense

Table 2 Locations and estimates of QTL for FHB spread measured by percentage of infected spikelets (PIS)

	Chromosome 3A				Chromosome 6B			
Closest marker	*Xs13m26_1*				*Xs13m24_2*			
Flanking markers	*Xgwm779–Xgwm1121*				*Xs23m17_5–Xgwm626*			
	Add[a]	% PV[b]	LOD[c]		Add[a]	% PV[b]	LOD[c]	
Simple interval mapping								
GS08	9.5	16	**4.0**	**	9.6	20	**5.0**	***
GS09	7.3	12	2.7		10.4	22	**5.4**	***
GW07	10.2	14	**3.3**		4.2	3	0.6	
GW08	13.3	17	**4.2**	**	9.9	14	**3.4**	*
Overall mean	9.2	17	**4.2**	**	8.5	19	**4.5**	***
Composite interval mapping								
Overall mean	8.0	22	**5.6**	***	7.3	22	**5.5**	***

LOD values > 3 are shown in bold

* α 0.1 < LOD; ** α 0.05 < LOD; *** α 0.01 < LOD

[a] Positive additive effects denote PIS-reducing effect of the Mt. Gerizim allele

[b] Percentage of phenotypic variance explained by the QTL

[c] Significance thresholds were estimated by permutation tests (number of iterations = 1,000)

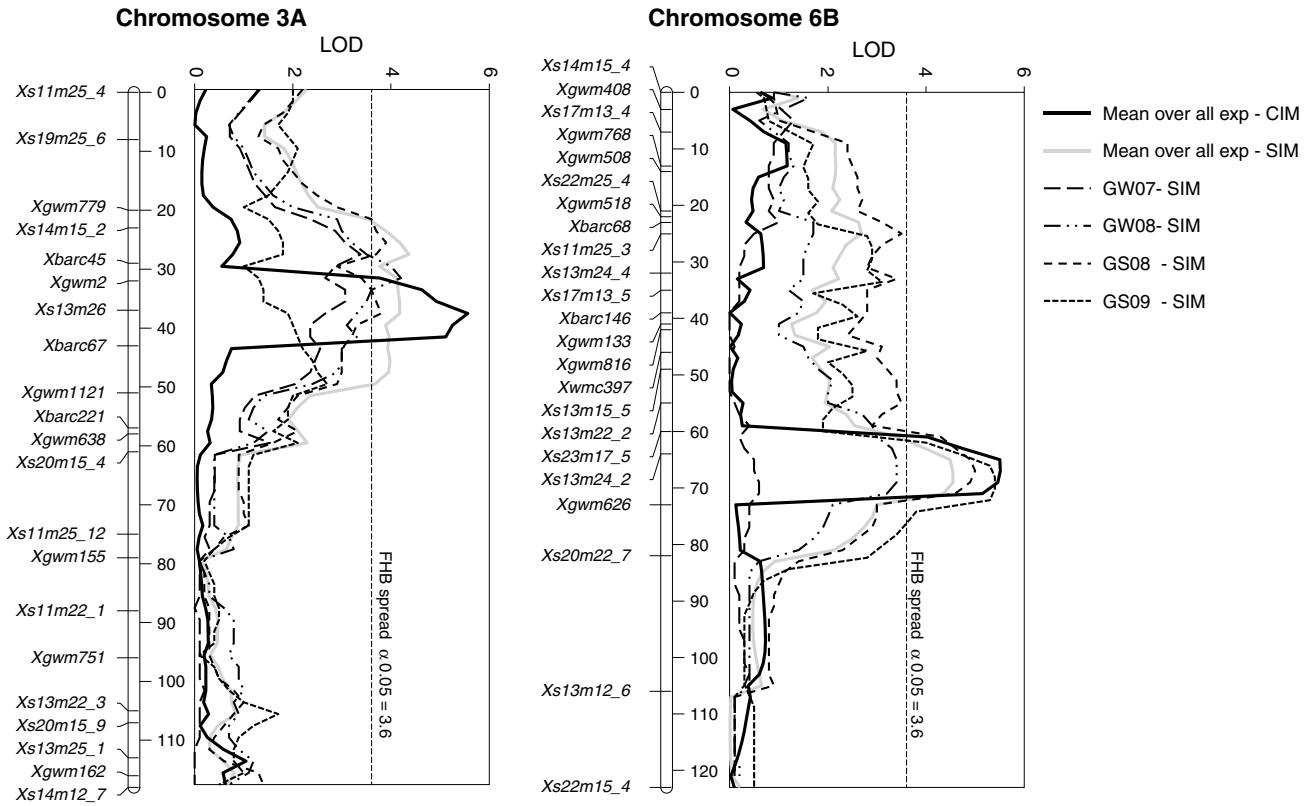

Fig. 2 Linkage maps of chromosome 3A and 6B with LOD profiles for percentage of infected spikelets (PIS). Individual experiments were calculated by simple interval mapping (SIM); overall means were calculated by SIM and CIM (composite interval mapping)

Table 3 Location and estimates of QTL for plant height by SIM

Trait	Chromosome	Closest marker	Add[a]	%PV[b]	LOD[c]	
Plant height	4B	*Rht-B1*	−18.0	82.1	38.1	***

*** α 0.01 < LOD

[a] Positive additive effects denote trait-reducing effect of the Mt. Gerizim allele

[b] Percentage of phenotypic variance explained by the QTL

[c] Significance thresholds were estimated by permutation tests (number of iterations = 1,000)

heritability estimate for means across experiments confirms that a large proportion of the observed variation was due to genetic variation in the population.

QTL for FHB resistance

Two genomic regions, one on chromosome 3A and a second on 6B, were found associated with type 2 resistance to FHB. The QTL on 3A was coincident with a QTL previously detected in two unrelated mapping populations, which used either the *T. dicoccoides* accession Israel A (Chen et al. 2007; Otto et al. 2002) or the *T. dicoccoides* line Mt. Hermon #22 (Gladysz et al. 2007) as resistance donors. Either study identified marker *Xgwm2* as closest to the QTL peak, similar to the finding in our study using the *T. dicoccoides* line Mt. Gerizim #36. It appears likely that these three *T. dicoccoides* lines carry the same resistance allele at the 3A QTL.

The QTL on 6B was detected in all experiments but GW07. The inoculation technique used in GW07 differed from the other experiments, in that the inoculum was applied without physically injuring the florets. This resulted in a comparatively low infection level (see Table 1). But given that this technique was applied in only one experiment, no final conclusion can be drawn. The *Fhb2* QTL on chromosome 6B has been finely mapped near *Xgwm133* in bread wheat (Cuthbert et al. 2007) and repeatedly detected in multiple independent studies (Buerstmayr et al. 2009). The QTL was also found associated with FHB resistance in two tetraploid studies (Somers et al. 2006; Buerstmayr et al. 2012). In the present study the QTL position on 6B was placed in a 15 cM interval and reached its highest LOD near *Xgwm626*. *Xgwm133* mapped 28 cM proximal to *Xgwm626* in our map and was not near the peak of the QTL for type 2 FHB resistance. We accordingly conclude that the 6B QTL of this study is not allelic to *Fhb2*.

Several lines in the mapping population were more diseased than the susceptible parent Helidur. This indicates that Helidur may possess small effect resistance alleles which remained non-discovered in this study. Given that the mapping population was relatively small (103 lines), the results on the detected QTL effects should be interpreted with caution and certainly need further validation. Despite that, the excellent performance of lines with both favorable alleles fixed provides evidence that resistance expression is more stable when more than one resistance QTL are combined. Potentially, improved and stable resistance can be achieved through pyramiding two or more resistance QTL, as examples in bread wheat have shown (e.g., Agostinelli et al. 2012). Still, the lines with improved type 2 resistance of this study need to be field evaluated using spray inoculations in order to assess their overall resistance performance under natural conditions.

Plant height and its association with FHB spread

The *Rht-B1b* allele contributed by the *T. durum* parent Helidur reduced plant height considerably. Its effect on plant height was similar to those observed in three separate backcross populations obtained from crosses of the resistance donor *T. dicoccum*-161 to Helidur, Floradur or DS-131621 (Buerstmayr et al. 2012). Results of several studies have suggested that type 2 resistance is less dependent on plant height than type 1 resistance (Steiner et al. 2004; Srinivasachary et al. 2008, 2009; Somers et al. 2003). Moreover, Yan et al. (2011) reported a positive influence on type 2 resistance for five of ten different *Rht* genes. Isolines carrying the *Rht-B1b* allele expressed a small but significantly improved type 2 resistance in his study. Similarly, Srinivasachary et al. (2009) found a positive influence of the *Rht-B1b* semi-dwarf allele on type 2 resistance. These findings are in agreement with our observations in this study: we observed a weak dependence between plant height and FHB spread, with shorter lines developing lower type 2 FHB severity. Contrasting results were obtained in several other studies (Hilton et al. 1999; Xue et al. 2010; McCartney et al. 2007; Buerstmayr et al. 2012). These populations were evaluated for FHB incidence (type 1 resistance) and/or FHB severity in the field after spreading infected plant residuals onto the soil and/or after spray inoculation. In all of these experiments, taller plants developed lower FHB symptoms, and QTL for FHB severity or FHB incidence coincided with the *Rht-B1* gene. This is supported by a QTL meta-analysis which reported overlapping QTL for plant height and FHB severity after spray inoculation near *Rht-B1* (Mao et al. 2010). Miedaner and Voss (2008) tested near isogenic lines (NILs) carrying different *Rht* genes using spray inoculation in field tests. They observed an increase of FHB severity for NILs carrying *Rht-B1b,* but not significantly different to the tall wild-type lines. In summary we conclude that the semi-dwarf allele *Rht-B1b* does not interfere with type 2 FHB resistance when tested under controlled greenhouse conditions, but that it can, depending on the genetic background, increase overall *FHB* severity under high infection pressure and open field conditions (Srinivasachary et al. 2009).

Conclusions

Wild emmer wheat is an important genetic resource for past and future wheat improvement (Nevo et al. 2002). Selected wild emmer lines such as Mt. Hermon #22 (Gladysz et al. 2007) and Mt. Gerizim #36 (this study) can be used as sources for improving FHB resistance particularly for durum wheat breeding. FHB resistance in tetraploid wheat is inherited in a quantitative manner as in bread wheat, and several QTL have been genetically mapped and are therefore amenable for marker-assisted breeding. Combining two or more QTL via marker-assisted backcrossing is suggested as a promising breeding strategy leading to novel cultivars with enhanced FHB resistance and reduced risk of Fusarium mycotoxin contamination.

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