Identification and mapping of QTL for resistance to Fusarium head blight in a durum wheat mapping population

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Abstract

Fusarium head blight (FHB) is a serious disease of durum wheat (*Triticum turgidum* subsp. *durum*) worldwide. Most cultivated durum varieties are susceptible to FHB, and durum breeding programs have been challenged by limited sources of FHB resistance. Great efforts have been devoted to identifying and introgressing FHB resistance from tetraploid relatives of durum, such as cultivated emmer wheat (*T. turgidum* subsp. *dicoccum*), into adapted durum varieties. However, the quantitative trait loci (QTL) controlling the FHB resistance in cultivated emmer and its introgression lines have not been well characterized. In this study, we aimed to identify and map FHB resistance QTL in a population of 186 recombinant inbred lines (RILs) derived from a cross between durum cultivar 'Joppa' and the introgression line LPA-4, which carries FHB resistance from a cultivated emmer line (PI 254188). The population was genotyped using a genotyping-by-sequencing (GBS) approach for discovery of single nucleotide polymorphism (SNP) markers and phenotyped for FHB disease severity and Fusarium damaged kernel (FDK) in multiple greenhouse and field experiments. A genetic map with a total length of 1137.62 cM was generated with 757 unique SNP markers. QTL analysis identified six LPA-4-derived QTL for FHB resistance on chromosome arms 1BL, 3AS, 3AL, 5BS, 5BL, and 6BL and one Joppa-derived QTL on chromosome 7BL. Additionally, two LPA-4-derived QTL for resistance to FDK were detected on chromosomes 6AL and 6BS, respectively. This research provides new insights into FHB resistance in tetraploid emmer wheat and will facilitate the development of resistant durum cultivars in FHB resistance breeding programs.

Key message

Six QTL associated with FHB resistance were identified in a durum introgression line derived from cultivated emmer (PI 254188), while a QTL was detected in durum cultivar Joppa.

Introduction

Fusarium head blight (FHB), also known as scab, is a major disease of both common wheat (*Triticum aestivum* L., 2n = 6x = 42, AABBDD) and durum wheat (*T. turgidum* L. subsp. *durum* Desf., 2n = 4x = 28, AABB) worldwide (Chen et al. 2007). The disease is primarily caused by the fungus *Fusarium graminearum* Schwabe, which attacks cereal heads during flowering time and leads to direct crop losses due to scabby grains produced (Bai and Shaner 2004; McMullen et al. 2012). The fungus can downgrade grain quality by accumulating mycotoxins, especially deoxynivalenol (DON), in infected kernels and pose serious health concerns for both humans and livestock (Desjardins 2006; McMullen et al. 2012). The use of host genetic resistance through planting FHB-resistant cultivars is a key component in managing the disease and minimizing the crop losses worldwide (Bai and Shaner 2004). However, resistance to FHB in wheat is very complex, quantitatively inherited, and controlled by many genes (Buerstmayr et al. 2009). Most efforts have focused on characterizing resistance to FHB in common wheat. This has led to the identification of numerous quantitative trait loci (QTL) and resistance sources that are now widely used in common wheat breeding programs (Buerstmayr et al. 2012, 2013; Bai et al. 2018; Viviani et al. 2025).

In contrast, progress in breeding FHB resistant durum wheat has been limited, and most currently commercially grown cultivars remain highly susceptible to FHB (Stack et al. 2002; Zhang et al. 2014; Steiner et al. 2019). This is largely due to the narrow genetic variation for FHB resistance and the lack of reliable resistance sources in the base germplasm collections of durum wheat (Zhang et al. 2014; Steiner et al. 2019). Several studies have extensively screened thousands of durum accessions from worldwide collections for FHB resistance, but only a few lines with a moderate level of resistance have been identified (Elias et al. 2005; Talas et al. 2011; Huhn et al. 2012). Attempts to identify QTL for FHB resistance using adapted durum germplasm have led to the discovery of several minor effect QTL on different chromosomes (Ghavami et al. 2011; Huhn et al. 2012; Giancaspro et al. 2016; Prat et al. 2017; Zhao et al. 2018; Sari et al. 2018; Pirseyedi et al. 2019; Steiner et al. 2019). In parallel, researchers continue to uncover novel alleles associated with FHB resistance by exploring tetraploid relatives of durum wheat (Oliver et al. 2007; Prat et al. 2014). Although incorporating alleles from tetraploid species into modern durum cultivars is challenging due to their exotic genetic background (Otto et al. 2002), it remains a promising option in durum breeding programs (Oliver et al. 2007).

Tetraploid relatives of durum wheat harbor valuable genetic diversity for various traits, including disease resistance, and can be readily utilized in durum breeding programs due to their shared ploidy level and genetic constitution (Gladysz et al. 2007; Kumar et al. 2007; Zhang et al. 2014). To date, accessions of wild emmer wheat (*T. turgidum* subsp. *dicoccoides*) (Miller et al. 1998; Otto et al. 2002; Buerstmayr et al. 2003; Chen et al. 2007; Oliver et al. 2007; Kumar et al. 2007; Gladysz et al. 2007; Buerstmayr et al. 2013), cultivated emmer wheat (*T. turgidum* subsp. *dicoccum*) (Oliver et al. 2008; Buerstmayr et al. 2012; Ruan et al. 2012; Zhang et al. 2014) and Persian wheat (*T. turgidum* subsp. *carthlicum*) (Somers et al. 2006; Oliver et al. 2008; Sari et al. 2018) have been studied to identify sources of resistance to FHB. Several QTL for FHB resistance derived from wild emmer have been mapped to chromosomes 3A (Otto et al. 2002; Chen et al. 2007; Gladysz et al. 2007; Buerstmayr et al. 2013; Soresi et al. 2017), 4A (Gladysz et al. 2007), 6B (Buerstmayr et al. 2013) and 7A (Kumar et al. 2007) in different mapping populations. Different QTL for FHB resistance derived from Persian wheat (cv. Blackbird) have been mapped to chromosomes 1A, 2A, 3A and 6B (Somers et al. 2006; Sari et al. 2018). Cultivated emmer accessions have shown FHB resistance in multiple independent evaluations, and some of them have been used to develop durum germplasm resistant to FHB (Xu et al. 2007). Evaluations of populations derived from crosses between cultivated emmer and three Austrian durum lines have identified QTL for FHB resistance on chromosomes 3B, 4B, 6A, 6B and 7B (Buerstmayr et al. 2012). Ruan et al. (2012) developed a mapping population from a cross between a cultivated emmer line (BGRC3487/2) and an advanced durum breeding line (DT735), and identified eleven emmer-derived QTL for FHB resistance on chromosomes 2A, 3B, 5A, 5B, 7A and 7B. Zhang et al. (2014) crossed the cultivated emmer accession PI 41025 with the FHB-susceptible durum cultivar ‘Ben’ (PI 596557) and identified two emmer wheat-derived QTL on chromosomes 3A and 5A.

In this study, we developed a recombinant inbred line (RIL) (F2:8) mapping population derived from a cross between the durum cultivar ‘Joppa’ and a durum introgression line LPA-4 derived from the pedigree of ‘Lebsock’/PI 254188/‘Alkabo’. The objectives of our research were to identify and map QTL for FHB

resistance in the mapping population, presumably having FHB resistance originated from the cultivated emmer accession PI 254188, and to identify the DNA markers associated with the QTL for use in durum FHB resistance breeding programs.

Materials and Methods

Plant materials

A mapping population (designated as 10FARJA) consisting of 186 recombinant inbred lines (RILs) ($F_{2:8}$) was developed from the cross between the durum cultivar 'Joppa' (PI 673106) and the durum introgression line LPA-4 by single seed descent method. Joppa was originally developed from the cross Maier/D97643 by the North Dakota Agricultural Experiment Station, which shows high grain yield potential, excellent end use quality and less susceptibility to FHB compared to previously released durum wheat varieties at NDSU (Elias and Manthey 2016). LPA-4 is a durum introgression line (BC_1F_8) derived from a cross between the durum cultivar 'Lebsock' and the emmer accession PI 254188 followed by one backcross to the durum cultivar 'Alkabo' and eight generations of selfing and selection. LPA-4 showed a moderate level of FHB resistance, which is likely inherited from the emmer wheat accession PI 254188 (Xu et al. 2007). The wheat genotypes Wheaton (FHB-susceptible), Alsen (FHB-resistant), and ND2710 (FHB-resistant) were used as controls in FHB evaluation experiments.

FHB inoculation and phenotyping

The mapping population, along with the parents and controls, was evaluated for type II FHB resistance (resistance to the disease spread within the infected spikes) in both greenhouse and field conditions from 2021 to 2023. FHB inoculations were carried out in four greenhouse experiments (2021-GH, 2022-GH, 2022-GH2, 2023-GH) and four field experiments in FHB nurseries located in Fargo (2021-FAR and 2022-FAR) and Langdon (2021-LNG and 2022-LNG), North Dakota.

In the greenhouse experiments, plants were grown in 6-inch clay pots, with four seeds sown per pot and three replicates (pots) per genotype. Plants were kept under a 14-h photoperiod using artificial light (600-W high-pressure sodium lamps; P.L. Light Systems Inc, Beamsville, Canada) and maintained at a temperature of 24–26°C. To support normal plant growth, a small amount (~ 20 g/L) of slow-release granular NPK fertilizer (20:20:20) was added to the pots upon emergence of the seedlings. To promote tillering, plants were supplemented three times with a water-soluble, instant NPK (20:20:20) fertilizer, applied every other week. FHB inoculations were carried out using the single floret inoculation method at anthesis (Stack et al. 2002). Four aggressive field strains of *F. graminearum* (Puri and Zhong 2010; Poursafar et al. unpublished data) were cultured in CMC medium (15 g CarboxyMethyl-Cellulose (C-4888, Sigma, USA), 1.0 g NH_4NO_3, 1.0 g KH_2PO_4 monobasic, 0.5 g $MgSO_4$.$7H_2O$ and 1.0 g Yeast extract in a final volume of 1 L distilled water), and used for inoculum preparation. The macroconidia harvested from the four strains were then equally mixed and diluted to prepare a spore suspension with a concentration

of 100,000 spores/ml. Ten microliters of the spore suspension were injected into the middle spikelet of eight to ten spikes per pot at anthesis with a fine syringe needle. Inoculated spikes were covered with transparent plastic bags sprayed with distilled water for 48 h to maintain high humidity and facilitate initial fungal infection.

In the field experiments, each wheat line was sown in three replicate hill plots, with 8 to 10 seeds planted per plot. The same inoculation method used in the greenhouse experiments was applied to inoculate 10 to 15 spikes per plot at anthesis, defined as when 50% of the spikes in the plot were flowering. The nurseries were misted for 12 hours daily, using a cycle of 5 minutes of misting every 60 minutes from 4:00 p.m. to 4:00 a.m. This misting schedule began on the first day of inoculation and continued until 14 days after the last flowering wheat materials were inoculated in the nursery (Zhao et al. 2018). For both greenhouse and field experiments, the percentage of symptomatic spikelets was recorded for each spike at 21 days post-inoculation (dpi) using the visual disease rating scale (0–100%) described by Stack and McMullen (1995). The overall mean FHB disease severity for each replicate was calculated by averaging the scores of all inoculated spikes. All experiments were arranged in a randomized complete block design with three replicates.

Evaluation of Fusarium damaged kernels (FDKs)

To evaluate the percentage of FDKs for each genotype, inoculated and uninoculated spikes (controls) from two FHB inoculation experiments in greenhouse (2021-GH2 and 2023-GH) were harvested and threshed using an Agriculex SPT-1 belt seed threshing machine. The number of grains harvested from uninoculated spikes was used to determine the average number of seeds per spike produced by each genotype. Grains harvested from inoculated spikes of each genotype were visually assessed, and scabby kernels were removed. The average number of healthy grains per spike harvested from inoculated plants was then used to calculate the percentage of FDKs for each genotype using the formula:

FDK (%) = [{1 − (average number of healthy grains per spike harvested from inoculated spikes/ average number of grains per spike from uninoculated spikes)} × 100].

Days to flowering (DTF)

The number of days from planting to flowering (DTF) was recorded for each of the RIL mapping population and parental lines in four independent field experiments conducted at the Fargo and Langdon nurseries in 2021 and 2022. For each genotype, mean DTF was used, defined as the average number of days from planting to the time when 50% of the spikes in a hill plot were at anthesis.

Genotyping by sequencing (GBS)

Leaf tissues collected from seedlings of each RIL genotype and parents were used for DNA extraction. The plant tissues were collected into 96-deep well plates, frozen in liquid nitrogen and ground using a Tissue Lyser (Qiagen, USA). The genomic DNA was extracted using the Wizard® Magnetic 96 DNA Plant System kit (Promega) following the manufacturers' instructions. The quality of the extracted DNA was checked on 1% agarose gel. DNA concentration was measured using the Quant-iT PicoGreen dsDNA Assay Kit (Thermo Fisher Scientific™, USA) and diluted to ~ 20 ng/µL for GBS-library preparation. The library preparation and genotyping by sequencing (GBS) was done in the USDA-ARS Central Small Grain Genotyping Lab in Manhattan, KS, according to Poland et al. (2012). Briefly, the extracted genomic DNA samples of each recombinant inbred lines and parents were digested using *PstI* (CTGCAG) and *MspI* (CCGG) restriction enzymes, and the resultant DNA fragments were ligated with forward and Y-reverse adaptors. Equal volumes of the products after ligation from each DNA sample were pooled, amplified by PCR and size-selected for 200-300bp fragments. Libraries were quantified using KAPA Library Quantification Kit (Roche, USA) and sequenced in a NextSeq2000 sequencer (Illumina, San Diego, 5200 Illumina Way, USA). Single nucleotide polymorphisms (SNPs) calling was carried out using the sequencing data with the pipeline described by Bradbury et al. (2007) based on the durum cultivar 'Svevo' reference genome sequence (Maccaferri et al. 2019). Raw SNP calls were filtered and reordered using two parameters: heterozygosity less than 10% and a minor allele frequency (MAF) greater than 20%. SNPs with less than 30% missing data (< 30%) were kept for genetic linkage analysis and QTL mapping.

Linkage map construction and QTL mapping

A Chi-square test was performed on SNP markers that segregated in the mapping population. Markers showing a distorted segregation were eliminated from the analysis. When multiple markers co-segregated in the mapping population, only a single representative marker was retained to build the genetic map. Genetic linkage analysis was done in MapDisto program (Lorieux 2012) with a logarithm of the odds (LOD) value threshold of 3.0 and a maximum recombination fraction (theta/r) of 0.3. The Kosambi mapping function was used to convert recombination frequencies to genetic distances. The initial order of markers within the linkage group was determined using the 'order a linkage group' command. The best marker order was determined by applying 'check inversion' and 'ripple order' commands. The resultant linkage maps were visualized with MapChart version 2.1 (Voorrips 2002). QTL mapping was performed using composite interval mapping (CIM) (Zeng 1994) in QGene v. 4.3.10 program (Joehanes and Nelson 2008). QTL with LOD scores equal or greater than 3.0 were considered significant. The significant threshold for each identified QTL was also confirmed by a permutation test with 1,000 random shuffles of phenotypic data relative to genotypes. The percentage of phenotypic variation explained (PVE) by each QTL was determined based on the coefficient of determination (R^2) value. The additive effect of each QTL was estimated to determine the average phenotypic change associated with the substitution of one parental allele for the other, with positive and negative values defined according to the parental origin of the trait-increasing allele. The flanking markers and the peak marker for each identified QTL were determined.

Statistical analysis

All statistical analyses were done in SAS software version 9.4 (SAS Institute, 2011). Analysis of variance (ANOVA) and homogeneity of variances (Levene's test) was performed using the GLM procedure. Shapiro–Wilk normality test of phenotypic data was done through the UNIVARIATE procedure. Correlations among traits were tested using Pearson correlation coefficients and the CORR procedure. The interactions between genotypes and environment were calculated using the GLM procedure with genotypes as fixed and environment as random effect variables. The analysis of variance for FHB severity in RILs due to genotype, environment, and genotype-by-environment interactions for the QTL detected in at least two environments was performed using GLM procedure. Broad-sense heritability (H^2) for each trait was estimated based on variance components obtained from the ANOVA. Heritability in a single environment and multiple environments, was calculated using the equation $H^2 = \sigma^2_G / (\sigma^2_G + \sigma^2_E / r)$ and $H^2 = \sigma^2_G / (\sigma^2_G + (\sigma^2_{GE} / (y) + \sigma^2_E / (y \times r)))$, respectively. In both equations, σ^2_G is the genetic variance, σ^2_{GE} is the genotype and environment (G×E) interaction variance, σ^2_E is the residual (error) variance, y is the number of environments and r is the number of replications.

Results

FHB disease severity, FDKs and DTF

FHB disease severities collected from four greenhouse experiments (2021-GH1, 2021-GH2, 2022-GH and 2023-GH) and four field experiments (2021-FAR, 2021-LNG, 2022-FAR and 2022-LNG) showed highly significant differences among the recombinant inbred lines (RILs) in the mapping population (Fig. 1). Normality tests across all evaluations indicated that the FHB disease severity data were not normally distributed in the population (Table 1). Levene's test for homogeneity of variances in FHB severity was significant among experiments under both greenhouse ($P < 0.0001$, df = 3) and field ($P < 0.0001$, df = 3) conditions, indicating that variances between experiments were heterogeneous. Some RILs exhibited a lower or higher disease severity than either parent, indicating transgressive segregation in the mapping population (Fig. 1). FHB disease severities across all experiments ranged from 14.6 to 96.52% for Joppa and 9.25 to 35.1% for LPA-4. In all experiments, except for one field experiment in Langdon (2021-LNG), the LPA-4 parent consistently showed lower disease severity than Joppa although some experiments had missing values. The average disease severity of Joppa, LPA-4, and the mapping population across all FHB evaluations was 46.37, 20.11, and 33.93%, respectively (Table 1).

Table 1
The summary of FHB disease severity, Fusarium damaged kernels (FDKs), and number of days from planting to flowering (DTF) of the parents and the mapping population (10FARJA) across all experiments. The broad-sense heritability (H^2) was calculated for FHB severity, FDKs and DTF.

	Parents		**Population**				
	Joppa	**LPA-4**	**Mean**	**Min**	**Max**	**H^2**	**Normality test (Pr < W)**
FHB severity (%)							
2021-GH1	60.44	24.63	43.59	15.46	81.65	0.68	< 0.001
2021-GH2	33.94	9.25	24.07	8	59	0.58	< 0.0001
2022-GH	41.82	**M**	34.52	7.91	85.37	0.75	< 0.0001
2023-GH	96.52	11.67	48.07	7.66	97.5	0.84	< 0.0001
2021-FAR	30.89	17.94	27.62	12.02	76.74	0.47	< 0.0001
2021-LNG	14.6	19.48	20.83	9.78	39.89	0.25	< 0.0001
2022-FAR	**M**	22.73	29.58	12.88	72.01	0.54	< 0.0001
2022-LNG	**M**	35.1	43.17	21.04	71.84	0.58	< 0.01
Mean	46.37	20.11	33.93	11.32	73.0	0.75	
FDKs (%)							
2021-GH2	**M**	7.92	34.65	2.31	83.93	0.45	ns
2023-GH	**M**	**M**	56	14	95	0.77	< 0.01
Mean	**M**	7.92	45.32	8.15	89.46	0.38	
DTF (day)							
2021-FAR	48	49	48	44	58	0.18	-
2021-LNG	48	48	49	43	54	0.33	-
2022-FAR	**M**	57	57	53	63	-	-
2022-LNG	**M**	59	58	56	62	0.41	-
Mean	48	53	53	49	59	0.44	

FDK: Fusarium-damaged kernel, DTF: Days to flowering, GH: greenhouse condition, FAR: Fargo FHB nursery, LNG: Langdon FHB nursery, M: Missing value, ns: non-significant, dash (-): not performed analysis.

The mapping population was also evaluated for FDKs in the two greenhouse experiments, 2021-GH2 and 2023-GH. The average percentage of FDKs was 34.65% and 56% in 2021-GH2 and 2023-GH, respectively, with an overall mean of 45.32% (Table 1; Fig. 2). The normality test showed a normal distribution for data obtained from 2021-GH2, but not from 2023-GH (Table 1). The Pearson correlation coefficient indicated moderate to strong positive correlations between mean FHB severity and FDK percentage in the 2021-GH2 ($r = 0.50$, $p < 0.0001$) and 2023-GH ($r = 0.78$, $p < 0.0001$) experiments, as well as between the overall mean FHB severity and FDKs across the two experiments ($r = 0.69$, $p < 0.0001$) and across all nine evaluations ($r = 0.59$, $p < 0.0001$).

The overall average number of days from planting to flowering (DTF) across four field experiments in the mapping population was 53 days (Table 1). In 2021 (2021-FAR and 2021-LNG), the average DTF for Joppa, LPA-4 and the mapping population was 48, 48.5, and 48.5 days, respectively. In 2022 (2022-FAR and 2022-LNG), the average DTF for LPA-4 and the mapping population was 58 and 57.5, while data for Joppa was missing. The relationship between DTF and FHB severity varied across different locations. In the Fargo (FAR) experiments, a non-significant and very weak positive correlation ($r = 0.05$; $p = 0.54$) was observed between DTF and FHB severity in 2021 (2021-FAR), while the 2022-FAR experiment showed a non-significant and very weak negative correlation ($r = -0.03$; $p = 0.72$). In the Langdon (LNG) experiments, a weak but statistically significant positive correlation ($r = 0.18$; $p = 0.01$) was detected in 2021 (2021-LNG), whereas a weak but significant negative correlation ($r = -0.21$; $p = 0.004$) was observed in 2022-LNG.

Broad sense heritability (H^2) estimates for FHB severity were calculated for each of the eight single environments (defined by year-location combinations) and multiple environments. Broad sense heritability values ranged from 0.25 to 0.84 ($0.25 < H^2 < 0.84$) for individual environments and 0.75 for multiple environments, indicating variable levels of genetic control across environments. Broad sense heritability for mean FDKs and days to flowering was low to moderate (Table 1). The analysis of variances revealed a significant effect for genotype (G), environment (E), and the interactions between genotype and environment (G × E) for disease severity in greenhouse and field experiments, and for FDKs in greenhouse experiments (Table 2).

Table 2
Analysis of variance for FHB disease severity and Fusarium damaged kernels (FDKs) in greenhouse (GH) and field experiments for the mapping population (10FARJA) derived from the Joppa × LPA-4 cross.

Source of variation	df	Type III SS	MS	F value	p-value
FHB severity (GH)					
Genotype (G)	185	243688.0314	1317.2326	7.44	< .0001
Environment (E)	3	142551.7006	47517.2335	268.51	< .0001
G × E	555	203216.6398	366.1561	2.07	< .0001
FHB severity (Field)					
Genotype (G)	185	79313.8982	428.7238	3.31	< .0001
Environment (E)	3	134703.3307	44901.1102	346.60	< .0001
G × E	555	104131.2105	187.6238	1.45	< .0001
FDKs (GH)					
Genotype (G)	185	119630	646.651270	3.33	< .0001
Environment (E)	1	83088	83088	428.16	< .0001
G × E	172	73651	428.200793	2.21	< .0001

SNP marker data and linkage analysis

Genotyping by sequencing (GBS) and subsequent SNP calling resulted in a total number of 34,470 SNP markers across the A and B genomes. After excluding SNP markers with more than 30% (> 30%) missing data and those showing a distorted segregation, a total of 4,765 SNPs was left. Out of these markers, 2,267 and 2,498 SNPs were from the A and B genomes, respectively. A total of 757 unique SNP markers were used to construct the genetic linkage map, which spanned 1,137.62 cM, with 496.68 and 640.94 cM corresponding to the A and B genomes, respectively. The linkage maps of the A genome (496.68 cM/354 unique markers) and B genome (640.94 cM/403 unique markers) had marker densities of 1.40 cM and 1.59 cM per marker, respectively. The average marker density of the total genetic linkage map was 1.50 cM per marker. The linkage analysis resulted in 44 linkage groups of varying lengths. Linkage groups shorter than 10 cM were excluded from the study, and 25 linkage maps were retained for QTL analysis (Supplementary Table 1).

QTL analysis

Based on Levene's test, which is less sensitive to non-normal distribution, the variances of FHB disease severities among the experiments were heterogeneous. Therefore, QTL analysis was conducted separately using the mean FHB severity from each individual environment (year-location combination). The QTL analysis detected six LPA-4-derived QTL and one Joppa-derived QTL for FHB resistance on

different chromosomes (Table 3). The permutation test revealed the LOD threshold for determining the statistical significance and validity of the identified QTL at α = 0.05 and 0.01 significance levels (Supplementary Table 2). The LPA-4-derived QTL were mapped to chromosome arms 1BL (designated as *Qfhb.ndwp-1BL*), 3AS (*Qfhb.ndwp-3AS*), 3AL (*Qfhb.ndwp-3AL*), 5BS (*Qfhb.ndwp-5BS*), 5BL (*Qfhb.ndwp-5BL*), and 6BL (*Qfhb.ndwp-6BL*), respectively. The Joppa-derived QTL was mapped to chromosome arm 7BL (*Qfhb.ndwp-7BL*) (Table 3).

Table 3
Quantitative trait loci (QTL) identified for resistance to FHB resistance and Fusarium damaged kernels (FDKs), and days to flowering (DTF) in the mapping population. The chromosomes, contributing parents, marker intervals, and peak markers of each QTL are summarized.

QTL	Chromosome	Contributor	Marker interval	Peak marker
FHB resistance				
Qfhb.ndwp-1BL	1BL	LPA-4	*S1B_18549019–S1B_497215081*	*S1B_281688689*
Qfhb.ndwp-3AS	3AS	LPA-4	*S3A_19789522–S3A_587030530*	*S3A_180254505*
Qfhb.ndwp-3AL	3AL	LPA-4	*S3A_733364211–S3A_715467866*	*S3A_726760921*
Qfhb.ndwp-5BS	5BS	LPA-4	*S5B_43013098–S5B_456513899*	*S5B_77950808*
Qfhb.ndwp-5BL	5BL	LPA-4	*S5B_674872055–S5B_680171147*	*S5B_680171147*
Qfhb.ndwp-6BL	6BL	LPA-4	*S6B_453373096–S6B_636109619*	*S6B_632614688*
Qfhb.ndwp-7BL	7BL	Joppa	-	*S7B_594649651*
FDK resistance				
Qfhb.ndwp-6AL	6AL	LPA-4	*S6A_106485028–S6A_526968289*	*S6A_526968289*
Qfhb.ndwp-6BS	6BS	LPA-4	*S6B_577313027–S6B453291443*	*S6B_169052291*
DTF				
Qdtf.ndwp-2AL	2AL	Joppa	*S2A_199490989–S2A_78492640*	*S2A_124355541*
Qdtf.ndwp-4AL	4AL	Joppa	*S4A_625919649–S4A_640185346*	*S4A_636321625*

Qfhb.ndwp-1BL (peak marker: *S1B_281688689*) was detected in one greenhouse experiment (2022-GH) and one field experiment (2022-FAR), explaining up to 14.1% of the phenotypic variation. Within the same marker interval (*S1B_18549019-S1B_497215081*) on chromosome 1B, three additional significant peaks (α = 0.05) were detected in one greenhouse experiment (2023-GH) and two field experiments (2021-FAR and 2021-LNG), accounting for up to 7.6% of the phenotypic variation (Fig. 3a; Supplementary Table 3). The QTL *Qfhb.ndwp-3AS*, spanned from *S3A_19789522* marker to *S3A_587030530* marker (peak marker *S3A_180254505*), was detected in two greenhouse experiments (2021-GH1 and 2023-GH) and one field experiment (2021-LNG) with LOD values of 8.98, 3.80 and 3.74 (significant at α = 0.01), respectively, and explained up to 20% of phenotypic variation (Fig. 3b; Supplementary Table 3). Another QTL, *Qfhb.ndwp-3AL* (peak marker: *S3A_726760921*), on the long arm of chromosome 3A, was detected in a field experiment (2022-FAR) with LOD value of 5.42 and explained 12.6% of the phenotypic variation (Fig. 3b; Supplementary Table 3).

Qfhb.ndwp-5BS (Peak marker: *S5B_77950808*) was detected in three greenhouse experiments (2021-GH1, 2022-GH and 2023-GH) with LOD values of 3.38, 4.01 and 6.03 and accounted for 8, 9.5 and 13.9% of the phenotypic variation, respectively (Fig. 3c; Supplementary Table 3). *Qfhb.ndwp-5BL* was mapped to a small genetic region between markers *S5B_674872055* and *S5B_680171147* on chromosome 5B and was detected only in one field experiment (2022-FAR), accounting for 8% of the phenotypic variation (Fig. 3d; Supplementary Table 3).

Qfhb.ndwp-6BL spanned 32.1 cM between marker intervals *S6B_453373096* and *S6B_636109619* on chromosome 6B (Fig. 3e). This QTL was detected (peak marker: *S6B_632614688*) in one field experiment (2022-FAR) and explained 10.6% of the phenotypic variation (Fig. 3e; Supplementary Table 3). However, three other significant peaks at *S6B_416372129* (α = 0.05, LOD = 3.5), *S6B_169052291* (α = 0.05, LOD = 3.27), and *S6B_519134460* (α = 0.01, LOD = 3.18) were detected in two greenhouse (2023-GH and 2022-GH) and one field experiments (2021-FAR), respectively, and explained up to 8.8% of the phenotypic variation (Fig. 3e). One minor Joppa-derived QTL on chromosome 7BL (peak marker: *S7B_594649651*) was only detected in one field experiment in Langdon (2022-LNG) with an LOD value of 3.00 and explained 7.2% of the phenotypic variation (Fig. 3f; Supplementary Table 3).

Three QTL for FHB resistance (*Qfhb.ndwp-1BL*, *Qfhb.ndwp-3AS* and *Qfhb.ndwp-5BS*) identified in at least two environments, were selected for variance analysis. The genotypes at the peak markers of each QTL, along with the FHB severity data of RILs carrying the markers, were used to perform a variance analysis and detect the possible interactions among the FHB resistance QTL, as well as between the QTL and the environment. The results revealed that the variance of FHB severity due to marker genotype was highly significant ($p < 0.0001$), confirming the results of QTL analysis (Table 4). The variance of FHB severity due to environment was highly significant ($p < 0.0001$), which indicates the significant effects of environment on FHB severity (Table 4). The interactions between environment and each individual QTL, *Qfhb.ndwp-1BL* (peak marker: *S1B_281688689*), *Qfhb.ndwp-3AS* (*S3A_180254505*) and *Qfhb.ndwp-5BS* (*S5B_77950808*), were significant, suggesting that the effect of these QTL was affected by environment (Table 4). The interaction between the three QTL as well as the interaction between the three QTL and

environment was not significant, indicating that the effects of individual QTL were consistent across environments and that the QTL acted independently without influencing each other's effects (Table 4).

Table 4
Analysis of variance for FHB severity in RILs due to genotype, environment, and genotype-by-environment interactions for the three consistently detected QTL in the mapping population.

Source of variance	**df**	**p-value**
Qfhb.ndwp-1BL (*S1B_281688689*)	1	< .0001
Qfhb.ndwp-3AS (*S3A_180254505*)	1	< .0001
Qfhb.ndwp-5BS (*S5B_77950808*)	1	< .0001
Environment	7	< .0001
Qfhb.ndwp-1BL × environment	7	< .0001
Qfhb.ndwp-3AS × environment	7	< .0001
Qfhb.ndwp-5BS × environment	7	< .0001
Environment	7	< .0001
Qfhb.ndwp-1BL × *Qfhb.ndwp-3AS* × *Qfhb.ndwp-5BS*	2	ns
Qfhb.ndwp-1BL × *Qfhb.ndwp-3AS* × *Qfhb.ndwp-5BS* × environment	14	ns
ns: non-significant		

The QTL analysis on FDK data collected from two greenhouse experiments (2021-GH2 and 2023-GH) detected two minor LPA-4-derived QTL for resistance to FDK on the long arm of chromosome 6A (*Qfhb.ndwp-6AL*, peak marker: *S6A_526968289*) and short arm of chromosome 6B (*Qfhb.ndwp-6BS*, peak marker: *S6B_169052291*) (Fig. 3e, g). The LOD values of *Qfhb.ndwp-6AL* and *Qfhb.ndwp-6BS* were 4.17 and 3.1 and explained 10 and 7.4% of phenotypic variation, respectively. These two QTL were only detected in the 2023-GH experiment, but not in the 2021-GH2. The QTL detected on short arm of chromosome 6B overlapped with the region harboring QTL for FHB resistance (Fig. 3e).

The DTF was recorded for the mapping population in four different field experiments (2021-FAR, 2022-FAR, 2021-LNG, 2022-LNG) (Table 1). QTL analysis revealed two Joppa-derived QTL for DTF on the long arm of chromosomes 2A (*Qdtf.ndwp-2AL*, Supplementary Fig. 1a) and 4AL (*Qdtf.ndwp-4AL*) (Supplementary Fig. 1a) only in the Langdon plantings (2021-LNG and 2022-LNG) (Table 3; Supplementary Table 2). *Qdtf.ndwp-2AL* (peak marker *S2A_124355541*) and *Qdtf.ndwp-4AL* (peak marker *S4A_636321625*) explained 9 and 8% of the phenotypic variation, respectively.

Discussion

Durum wheat cultivars are generally more susceptible to FHB than hexaploid common wheat varieties, and limited sources of resistance are available in the primary durum gene pool. Tetraploid relatives of durum wheat have shown promise as sources of FHB resistance, which can be exploited to improve elite durum cultivars (Stack et al. 2002; Buerstmayr et al. 2003, 2013; Xu et al. 2007; Oliver et al. 2007, 2008; Ruan et al. 2012; Kirana et al. 2023). The cultivated emmer accession PI 254188 has been found to have some level of resistance to FHB (Xu et al. 2007), but limited information is available on its QTL conferring FHB resistance. In this study, we developed a RIL mapping population derived from the cross between the durum introgression line LPA-4 ('Lebsock'/PI 254188/'Alkabo') and durum cultivar Joppa, and identified QTL associated with FHB resistance derived from both parents.

The RILs ($F_{2:8}$) within the mapping population showed considerable variation in FHB severity with a transgressive segregation across different experiments as previously shown by several other studies (Ghavami et al. 2011; Zhao et al. 2018; Pirseyedi et al. 2019). Joppa has been shown to be moderately susceptible to FHB and only carries a minor QTL for FHB resistance (Zhao et al. 2018), suggesting that the resistance observed in the population was mostly contributed by the LPA-4 parent, presumably inherited from PI 254188. This is confirmed by our QTL analysis, which identified six LPA-4-derived QTL (*Qfhb.ndwp-1BL, Qfhb.ndwp-3AS, Qfhb.ndwp-3AL, Qfhb.ndwp-5BS, Qfhb.ndwp-5BL* and *Qfhb.ndwp-6BL*) and one Joppa-derived QTL (*Qfhb.ndwp-7BL*) associated with resistance to FHB. Three QTL, *Qfhb.ndwp-3AL, Qfhb.ndwp-5BL* and *Qfhb.ndwp-7BL*, were detected only in a single field experiment, while the other three QTL were identified in at least two experiments. Notably, most of the QTL identified in this study were mapped to different B-genome chromosomes, as previously reported by Pirseyedi et al. (2019), highlighting the importance of genome B in FHB resistance architecture. Given that the introgression line LPA-4 is derived from a pedigree involving the cultivated emmer accession PI 254188, the identification of multiple QTL for FHB resistance highlights the value of tetraploid durum relatives as sources of resistance, consistent with findings from previous studies (Viviani et al. 2025).

We detected a minor QTL for FHB resistance on chromosome 7B in Joppa, confirming the existence of minor QTL for FHB resistance in North Dakota elite durum cultivars (Ghavami et al. 2011; Zhang et al. 2014; Zhao et al. 2018; Pirseyedi et al. 2019). This QTL was detected only in one of the field experiments and is the second QTL detected for resistance to FHB in Joppa. There are also reports of the existence of QTL associated with FHB severity and FDKs on the short arm chromosome 7B in durum wheat (Buerstmayr et al. 2012; Sari et al. 2018; Pirseyedi et al. 2019; Ruan et al. 2020; Haile et al. 2023). Since the QTL identified in our study is on the long arm of chromosome 7B, it is likely different from the previously identified QTL for FHB resistance. Another minor QTL for FHB resistance in Joppa has been previously identified on chromosome 2A (Zhao et al. 2018). However, this QTL was not detected in Joppa in the current study. In a separate study, a major FHB resistance QTL was identified on chromosome 2A of durum variety Divide, another North Dakota elite durum cultivar, in a mapping population derived from the cross between Divide and PI 254188 (Leng et al. Unpublished data). Notably, the LPA-4 parent used in our study contains two durum varieties ('Lebsock' and 'Alkabo') in its pedigree. It is therefore possible that both Joppa and LPA-4 parents carry the same QTL at this location. It is also likely that the presence of strong resistance alleles inherited from LPA-4 may have masked the

phenotypic effects of the minor QTL contributed by Joppa, making it undetectable in our mapping population.

Of the six QTL identified in the LPA-4, four QTL, including *Qfhb.ndwp-3AS, Qfhb.ndwp-1BL, Qfhb.ndwp-3AL*, and *Qfhb.ndwp-5BS*, conferred large effects on FHB resistance, respectively. The QTL *Qfhb.ndwp-3AS* (marker intervals *S3A_19789522–S3A_587030530*) and *Qfhb.ndwp-3AL* (marker interval *S3A_733364211* and *S3A_714567866*) were detected on chromosome 3A. Based on the physical position of the peak markers of each QTL in the reference genome sequence of the durum cv. 'Svevo' (Maccaferri et al. 2019), these QTL were mapped to the short and long arms of chromosome 3A, respectively. The major QTL *Qfhb.ndwp-3AS* has repeatedly been reported for FHB resistance on chromosome 3A in tetraploid wheat species (Otto et al. 2002; Chen et al. 2007; Gladysz et al. 2007; Buerstmayr et al. 2012; Zhang et al. 2014; Sari et al. 2018). This QTL was originally mapped close to the *Xgwm2* locus, which has been confirmed to be located on short arm of chromosome 3A (Otto et al. 2002; Chen et al. 2007; Zhu et al. 2016). More DNA markers have been identified in the QTL region on 3AS with *Xwgc501* as one of the closest markers to the *Xgwm2* locus (Zhu et al. 2016). The Blast search of the primers for the marker *Xwgc501* in the reference genome of durum revealed an approximate physical position of 54 Mbp on chromosome 3A. However, our identified 3AS QTL was located around 180 Mbp. This large difference in the QTL physical positions suggests that those QTL are probably not the same.

Zhang et al. (2014) identified 3A QTL for FHB resistance near the SSR marker *Xgwm5* in cultivated emmer accession PI 41025. Based on the DNA marker associations, they suggested that the identified QTL is probably different from the QTL reported by Otto et al. (2002) and Chen et al. (2007). This report further supports our findings on the existence of different QTL for FHB resistance on chromosome 3A in durum tetraploid relatives.

The marker interval for QTL on 3AS identified in our study spanned a large genomic region which contains another significant peak (*S3A_482261119*) at 58.4 cM and physical position of around 482 Mbp located on the long arm of chromosome 3A. Giancaspro et al. (2016) reported a QTL on chromosome 3AL for type I FHB resistance from bread wheat close to the markers *IWB37509* using the inclusive composite interval mapping (ICIM) method. Based on the position of the marker *IWB37509* (~ 555 Mbp), these QTL are likely located in the same region. Additionally, Giancaspro et al. (2016) used multiple trait multiple interval mapping (MTMIM) method and identified the marker *IWB8288* (physical position ~ 717 Mbp) close to QTL peak. Based on the physical position of the peak marker (726,760,921 bp) of another QTL on chromosome 3A (*Qfhb.ndwp-3AL*) identified in our study, it is possible that they are the same or closely linked QTL within the same genomic region.

The QTL *Qfhb.ndwp-5BS* was detected in three greenhouse experiments within the marker intervals *S5B_43013098* and *S5B_456513899*. The peak marker (*S5B_77950808*) was located on the short arm of chromosome 5B. We detected another QTL, *Qfhb.ndwp-5BL* (marker interval *S5B_674872055–S5B_680171147*) on long arm chromosome 5B, which was detected only in one field experiment. Ruan et al. (2012) identified three QTL, *QFhb.usw-5B1*, *QFhb.usw-5B2* and *QFhb.usw-5B3*, for FHB severity, FHB

incidence and FHB index on chromosome 5B, respectively, in tetraploid wheat. QTL *QFhb.usw-5B1* was detected from the cultivated emmer landrace BGRC3487. The closest marker for QTL *QFhb.usw-5B1* was *barc59* which is in ~ 661 Mbp on the short arm of chromosome 5B. Based on the position of the peak marker of the QTL *Qfhb.ndwp-5BL* identified in our study (~ 674 Mbp), the two QTL are likely in the same region. *QFhb.usw-5B2* derived from a durum breeding line DT735 was associated with FHB incidence. The closest markers for the QTL were *barc4*, which is located in ~ 69 Mbp on the short arm of chromosome 5B. Based on the physical position of the peak marker (~ 77 Mbp) of the QTL *Qfhb.ndwp-5BS* identified in our study, the two QTL are likely located in the same genomic region and are probably the same or closely linked QTL.

Ghavami et al. (2011) used populations resulting from the cross between the Tunisian line Tun34 and the durum cultivar 'Lebsock' and reported a major QTL for FHB resistance on 5BL derived from Lebsock. Since LPA-4 has Lebsock in the pedigree, it is also possible that the QTL on chromosome 5BL detected in our study was derived from Lebsock. Moreover, Pirseyedi et al. (2019) reported a QTL on chromosome 5B derived from the durum cultivar Ben (recurrent parent) in a population from a cross with Tunisian line Tun108. The LPA-4 parent has two durum cultivars ('Lebsock' and 'Alkabo') in the pedigree, and these findings altogether suggest that the two 5B QTL identified in our study were also likely derived either from Lebsock or Alkabo.

The QTL *Qfhb.ndwp-1BL* was mapped to marker interval *S1B_18549019* and *S1B_497215081* and detected in one greenhouse and one field experiment. Based on the position of the peak marker (*S1B_281688689*), it was mapped to the long arm of chromosome 1B. However, within the same marker interval on chromosome 1B, three additional significant peaks were detected. Several other studies have reported QTL for FHB resistance at different locations of chromosome 1B (Sari et al. 2018; Ruan et al. 2012, 2020; Kirana et al. 2023; Haile et al. 2023). Giancaspro et al. (2016) reported a QTL on long arm of chromosome 1B close to marker *IWB65943* for type II FHB resistance. Additionally, the QTL on chromosome 1B was found to be associated with resistance to FDK (Pirseyedi et al. 2019). More studies are needed to determine whether all these QTL are in the same region. We detected a QTL (peak marker *S6B_632614688*) on chromosome 6B, which spanned 32.1 cM between the markers *S6B_453373096* and *S6B_636109619*. This QTL was detected in only one experiment. However, three additional significant peaks within the interval but at different physical positions were detected. Previous studies have reported QTL for FHB resistance on different locations of chromosome 6B in tetraploid wheat (Somers et al. 2006; Buerstmayr et al. 2012, 2013; Giancaspro et al. 2016; Sari et al. 2018; Steiner et al. 2019; Ruan et al. 2020; Haile et al. 2023). Therefore, additional information is needed to elucidate the QTL associated with FHB resistance on chromosome 6B.

We harvested kernels from the inoculated plants in two greenhouse experiments (2022-GH and 2023-GH) for FDK analysis. The QTL analysis revealed two QTL for resistance to FDK on the long and short arms of chromosomes 6A (*Qfhb.ndwp-6AL*, peak marker *S6A_526968289*) and 6B (*Qfhb.ndwp-6BS*, peak marker: *S6B_169052291)*, respectively. These two QTL were only detected in the 2023-GH experiments. Multiple QTL associated with FDK have been previously reported on different wheat chromosomes

(Pirseyedi et al. 2019; Berraies et al. 2024). It has been found that there are associations between QTL identified for FHB and FDK resistance (or other FHB-associated traits) and the same QTL can contribute to both types of FHB resistance (Chu et al. 2011; Zhao et al. 2018). In our study, *Qfhb.ndwp-6BS* detected for resistance to FDK overlapped with one of the significant peaks in QTL region identified for FHB resistance on chromosome 6B. The overlapping regions suggest that the QTL *Qfhb.ndwp-6BS* may contribute to resistance against both FHB severity and FDK in tetraploid wheat.

Resistance to FHB in wheat has been found to be associated with plant morphological and developmental traits such as flowering date and plant height (Buerstmayr et al. 2009; Kirana et al. 2023). In this study, we identified two Joppa-derived QTL for flowering date on chromosomes 2A and 4A in two field experiments. Previous studies have indicated associations between flowering time and FHB development in wheat plants (Gervais et al. 2003; Steiner et al. 2004, Holzapfel et al. 2008; Buerstmayr et al. 2009, 2012). However, our results showed that there was not a consistent relationship between DTF and FHB severity in different environments. A weak positive correlation was found between DTF and FHB severity in the 2021 field experiment, whereas a weak negative correlation was observed in the 2022 field experiment. These contrasting results suggest that the association between flowering time and FHB resistance is likely influenced by other factors such as environmental conditions. This is consistent with the findings of Buerstmayr et al. (2012), who found inconsistent relationships between DTF and FHB severity in different experiments.

Conclusion

In the current study, we evaluated a RIL mapping population derived from the cross between Joppa (a durum cultivar) and LPA-4 (a durum introgression line) for resistance to Fusarium head blight across multiple greenhouse and field experiments. We identified six FHB resistance QTL derived from the LPA-4 parent on chromosomes 1B, 3A, 5B and 6B as well as two QTL associated with FDK on chromosomes 6A and 6B. Additionally, we identified a minor Joppa-derived QTL for FHB resistance on chromosome 7B. Two Joppa-derived QTL for flowering time were detected on chromosomes 2A and 4A. Overall, this research provides new insights into FHB resistance in durum tetraploid relatives and will facilitate the introgression of the identified FHB-resistance QTL into durum breeding programs.

Declarations

Author contribution statement

AP: data curation, formal analysis, investigation, methodology, visualization, writing original draft, review and editing. **YL, SS, AR, OA, AB, YX, PA, CC:** formal analysis, investigation, methodology, review and editing. **GB, SSX** and **ZL:** funding acquisition, supervision, review and editing. **SZ:** conceptualization, data curation, funding acquisition, project administration, resources, supervision, visualization, writing original draft, review and editing.

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Compliance with ethical standards

Conflict of interest

The authors claim that there is no conflict of interest.

Ethical standards

The authors state that all experiments in the study comply with ethical standards in the USA.

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Figures

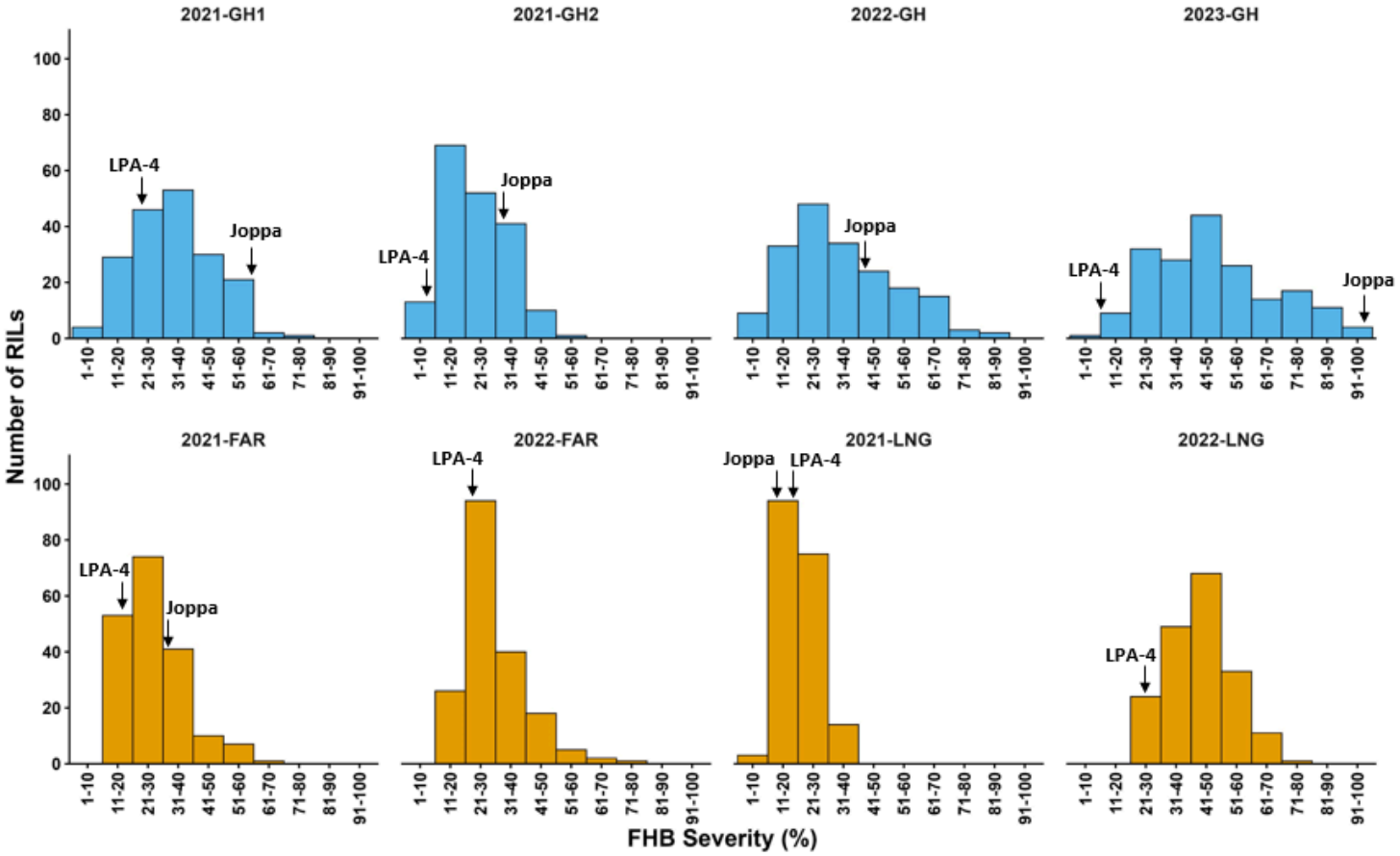

Figure 1

Distribution of Fusarium head blight (FHB) disease severity (average percentage of the infected spikes) in the mapping population (10FARJA) consisting of 186 recombinant inbred lines (RILs) derived from the cross between 'Joppa' and LPA-4 ('Lebsock'/PI 254188 /'Alkabo'). The population was evaluated for FHB resistance in four greenhouse (blue) and four field (orange) experiments. 2021-GH1, 2021GH2, 2022-GH, and 2023-GH represent the FHB evaluations conducted in greenhouse from 2021 to 2023. 2021-FAR, 2021-LNG, 2022-FAR and 2022-LNG indicate the FHB inoculations performed in field experiments located in Fargo and Langdon nurseries, North Dakota, from 2021 to 2023. The disease severity of the parents (Joppa and LPA-4) is indicated by arrows. LPA-4 and Joppa have missing values in the 2022-GH and 2022 field (FAR and LNG) experiments.

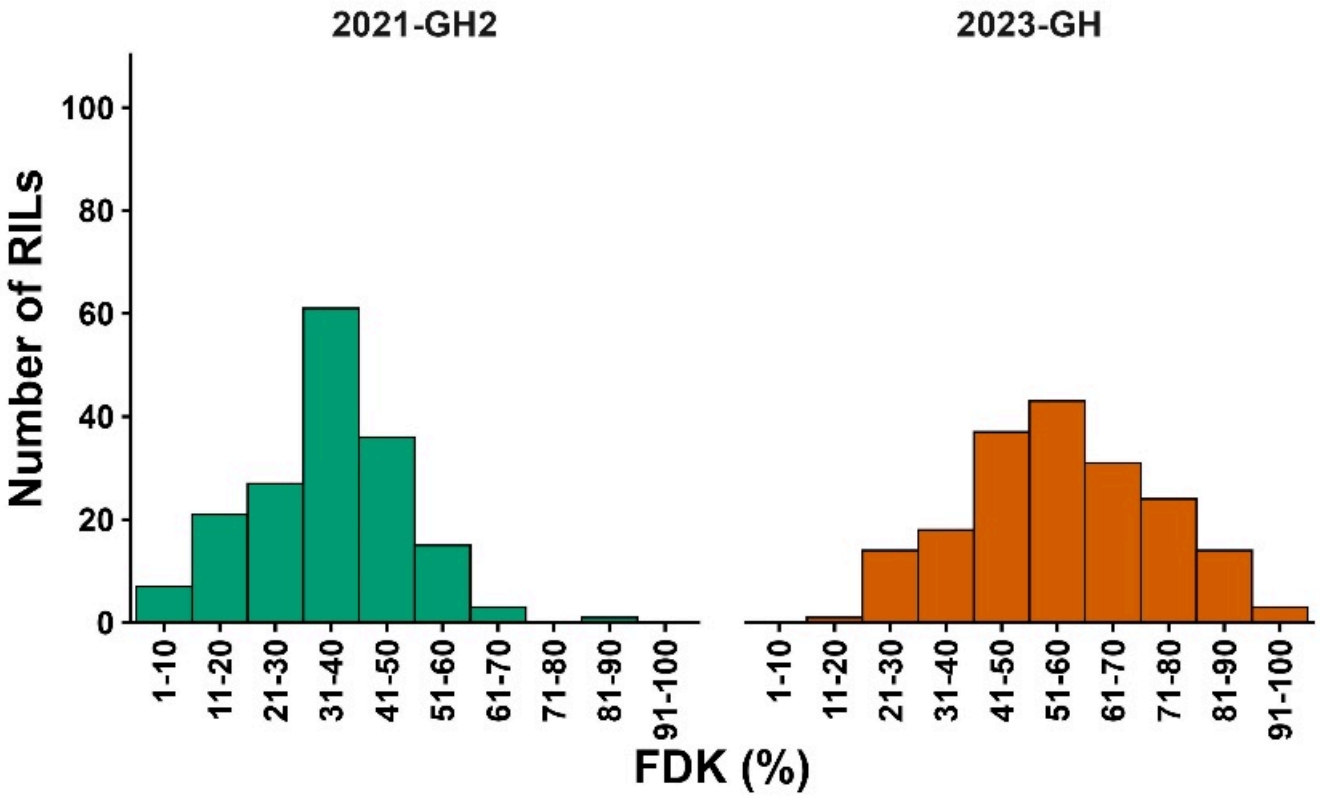

Figure 2

Distribution of the average percentage of Fusarium damaged kernels (FDKs) in the mapping population (10FARJA) consisting of 186 recombinant inbred lines (RILs) derived from the cross between 'Joppa' and LPA-4 ('Lebsock'/PI 254188 /'Alkabo'). The population was evaluated for resistance to FDKs in two greenhouse experiments, 2021-GH2 (green) and 2023-GH (brown).

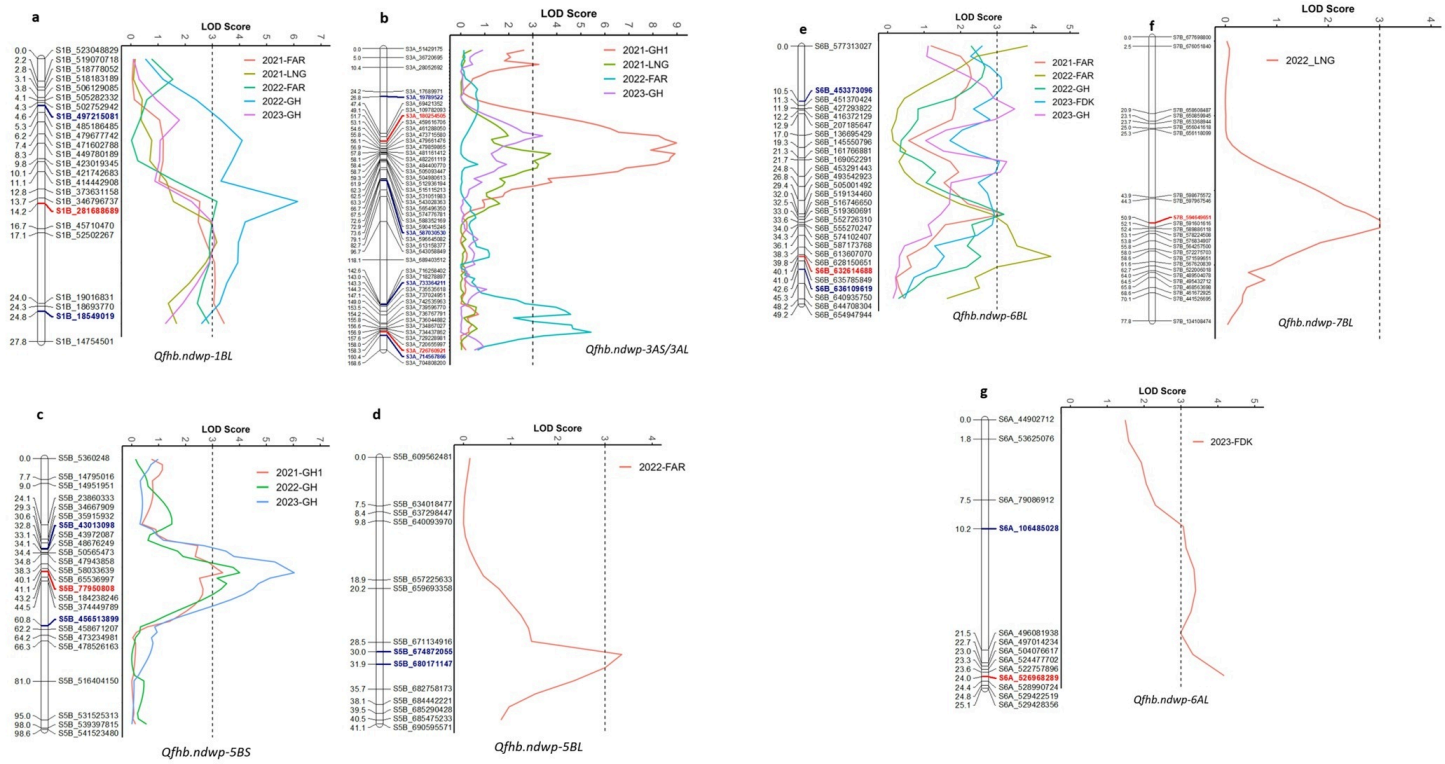

Figure 3

Regions of linkage maps for chromosomes 1B (a), 3A (b), 5B (c, d), 6B (e), 7B (f) and 6A (g) harboring QTL for resistance to FHB and FDKs in the 10FARJA population. All QTL for FHB resistance, except for QTL on chromosome 7B, were derived from LPA-4 parent. The QTL detected for resistance to FDKs on chromosome 6B overlapped with the region harboring QTL for FHB resistance (e). The genetic distance (centimorgan, cM) between SNP marker loci and the physical positions of the SNP markers are shown on the left and right sides of the linkage maps, respectively. The LOD significance threshold 3.0 is represented by a vertical dashed line. Peak markers or the markers closely linked to QTL, and the flanking markers of QTL are in bold red and blue colors, respectively. 2021-GH1, 2022-GH, and 2023-GH represent the FHB evaluations conducted in the greenhouse during 2021-2023. 2021-FAR, 2021-LNG, 2022-FAR and 2022-LNG indicate the FHB inoculations performed in field experiments located in Fargo and Langdon nurseries, North Dakota, during 2021–2023.

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