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ORIGINAL ARTICLE OPEN ACCESS

Marker Effects for Fusarium Head Blight Resistance and the Dwarfing Gene *Rht-B1* in Cultivated Emmer Wheat (*Triticum turgidum* ssp. *dicoccum*) Revealed by Association Mapping

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Received: 5 June 2024 | **Revised:** 4 October 2024 | **Accepted:** 1 November 2024

Funding: This work was funded by the "Ministerium für ländlichen Raum und Verbraucherschutz" (MLR, Stuttgart) within the special program "Stärkung der biologischen Vielfalt des Landes Baden-Württemberg" (Az: 23-8872).

Keywords: FHB | gene bank | genetic resources | GWAS | plant height | selection | *Triticum turgidum*

ABSTRACT

Fusarium head blight (FHB) is a disease that affects all cereals worldwide. This includes emmer wheat (*Triticum turgidum* ssp. *dicoccum*), the ancestor of durum and bread wheat. We screened 143 cultivated emmer genotypes from a breeding program and gene bank collections for FHB severity at 2 locations over 2 years. Due to the high negative correlation between FHB severity and heading date (HD) ($r = -0.65$, $p < 0.001$), plot-level FHB scores were corrected for HD before further analysis (FHB_{corr}). Genetic variation for FHB severity was high, ranging from 2.15 to 8.33 on a 1–9 scale. Twelve genotypes carried the semi-dwarfing *Rht-B1b* marker allele, which reduced plant height by 32 cm but increased FHB severity by 20%. Genome-wide association study detected seven quantitative trait nucleotides (QTNs) for FHB_{corr} and three QTNs for plant height. The most important QTN for both traits was located on chromosome 4B, explaining 50.9% and 15.8% of the phenotypic variation in plant height and FHB_{corr}, respectively, and was localized near the semi-dwarfing *Rht-B1* locus. Three other large-effect loci for FHB_{corr} were found on chromosomes 5B and 7B. In total, 72.6% of phenotypic variation was explained by all markers. The use of *Rht-B1b* in emmer breeding has a high effect on plant height but would entail the introgression of potent FHB-resistance from either native or exotic sources.

1 | Introduction

Fusarium head blight (FHB) is caused by *Fusarium graminearum*, *F. culmorum*, and other *Fusarium* species. FHB severity can be exacerbated by simplified crop rotation, especially maize-wheat rotation combined with reduced tillage, and by high nitrogen fertilization. FHB is one of the most important diseases of cereals worldwide mainly due to its high mycotoxigenic potential. However, *Fusarium* species have also been isolated from dozens of native grasses in North America and Europe in recent decades (Inch and Gilbert 2003; Szécsi et al. 2013; Lofgren et al. 2018; Fulcher et al. 2019), indicating that the same species that infect wheat and maize are also part of the natural mycoflora. As such, the progenitors of bread wheat, such as emmer wheat (*Triticum turgidum* ssp. *dicoccum*, 2n = 4x = 28, genome AABB), may provide new genetic variation for resistance to FHB, which would then be accessible also for durum wheat improvement. This is even more probable as wild emmer wheat (*Triticum turgidum* ssp. *dicoccoides*) accessions have shown a wide range of FHB

severities (Oliver et al. 2007; Garvin, Stack, and Hansen 2009; Buerstmayr et al. 2012, 2013).

FHB resistance is quantitatively inherited and affected by environmental factors (Miedaner et al. 2001; Buerstmayr, Steiner, and Buerstmayr 2020). Extensive research has been conducted on the genetic architecture of FHB resistance in bread wheat, resulting in the identification of >550 quantitative trait loci (QTLs) associated with FHB resistance across all 21 chromosomes that could be refined to 65 metaQTL (Venske et al. 2019). Morphological traits such as plant height per se, spike architecture, heading/flowering date, and anther extrusion greatly modify FHB resistance in wheat according to the genetic background (Buerstmayr, Steiner, and Buerstmayr 2020). In a recent metaQTL analysis of wild emmer, 31 FHB resistance QTLs were extracted from three studies (Cabas-Lühmann et al. 2024). Some of the genotypes have consistently shown very low disease severity over multiple seasons but are not directly useful for breeding due to their wild character. Another study even combined QTLs from bread wheat with those from wild and cultivated emmer (Kirana et al. 2023).

Emmer was one of the earliest cultivated cereals and was first grown during the Neolithic period in the Fertile Crescent. Today, emmer is a neglected crop grown only on several thousand hectares in Germany, Austria, and Switzerland and has still some importance in India, Ethiopia and Yemen (Damania 1998). Besides the lower grain yield compared with bread wheat, emmer grain is tightly enclosed by tough glumes (hulled), the crop is generally tall (100–150 cm) and prone to preharvest lodging (Longin et al. 2016). In a comparative analysis, bread and durum wheat had no lodging at all (score 1 on the 1–9 scale), whereas the lodging of emmer genotypes varied from score 2 to 6, although nitrogen supply was reduced in this crop (Longin et al. 2016).

The problem of lodging was largely solved in bread and durum wheat by introgressing one of the dwarfing genes *Rht-B1* on chromosome 4B or *Rht-D1* on chromosome 4D as a follow up to the 'Green Revolution' (Gale and Youssefian 1985). The dwarfing allele of *Rht-B1* has also been introgressed into some elite emmer wheat lines by the senior author of this paper, C.F.H. Longin. For both dwarfing genes, it is known that they increase FHB severity in bread wheat (Buerstmayr and Buerstmayr 2016; He et al. 2016; Thambugala et al. 2020; Akohoue et al. 2022) and for *Rht-B1b* in durum wheat (Buerstmayr et al. 2012; Miedaner et al. 2017). However, whether this also applies to the progenitor of durum wheat, emmer, needs to be addressed.

We screened 143 cultivated winter emmer genotypes for FHB severity, plant height, and heading date in four environments and analysed them by DArTSeq markers. We aimed to (1) evaluate the genetic variation in FHB severity present in this comprehensive set of cultivated emmer, (2) identify FHB resistance QTNs in the emmer genome across environments via a genome-wide associations study (GWAS), and (3) assess the effects of *Rht-B1b* introgression on FHB severity and plant height by phenotypic and molecular means.

2 | Materials and Methods

2.1 | Plant Material and Field Trials

The germplasm utilized in this study consisted of 143 genotypes of winter emmer, two bread wheat (Julius, Genius), two spelt wheat (Franckenkorn, Zollernspelz), two winter durum (Wintergold, Sambadur), and one einkorn genotype (Terzino). For emmer, nine genotypes were cultivars, 37 originate from the Hohenheim breeding program of CFHL, and 97 genotypes were from gene banks (Table S1). From the latter, 61 were from the German gene bank in Gatersleben (Leibniz Institute of Plant Genetics and Crop Plant Research, IPK, 06466 Seeland, OT Gatersleben, Germany), the remainder from gene banks in Suisse (Agroscope, Changins, Cereal Gene-Bank, Rte de Duillier 60, 1260 Nyon, Switzerland), France (INRAe = INRAE Small grain cereals Biological Resources Centre in Clermont-Ferrand, France), and the Czech Republic (Výzkumný ústav rostlinné výroby, v.v.i. Praha (VÚRV) Crop Research Institute, Gene Bank Drnovská 509 161 06 Praha, Czech Republic).

The short emmer genotypes with the dwarfing allele of *Rht-B1* were developed by the senior author by crossing emmer with

TABLE 1 | Evaluation of disease resistances and agronomic traits; art. = artificial infection, nat. = natural infection.

Trait, abbreviation	Environment[a] (total number)	Rating scale/measurement
Fusarium head blight (FHB)	HOH2020 (art.), HOH2021 (art.), TUL2021 (art.), ROS2020 (nat.) (4)	1–9
Heading date (HD)	HOH2020, HOH2021, TUL2021, ROS2020 (4)	Days in year (from Jan. 1)
Plant height (PH)	HOH2020, HOH2021, TUL2021, ROS2020 (4)	cm
Lodging (LOD)	HOH2019, HOH2020, SCH2019, TUL2020, OLI2020 (5)	1–9
Anther extrusion (AEX)	HOH2019, HOH2-2019, HOH2020 (3)	1–9
Grain yield (GY)	HOH2019-LD, HOH2020-LD, SCH2019-LD, RAS2019-LD, OLI2020-LD (5)	dt/ha

[a]HOH = Hohenheim, TUL = Tulln, ROS = Rosenthal, OLI = Oberer Lindenhof, SCH = Schwäbisch Hall, RAS = Rastatt; the number following the abbreviated name of location denotes the year; LD = data from large-drilled plots.

durum genotypes carrying the dwarfing allele (Table S2). After one or two backcrosses, the progeny were selfed till the F5 generation. In each generation, progeny were selected for emmer-like appearance and short stature visually.

We tested all genotypes in two growing seasons 2020 and 2021 and two locations resulting in four environments (location × year combinations)—in Germany: Stuttgart-Hohenheim (HOH2020), Rosenthal near Peine (ROS2020), Stuttgart-Hohenheim (HOH2021), and in Austria: Tulln near Vienna (TUL2021) (Table 1). Trials were laid out in an alpha-lattice design with two replicates as double rows in plots of 1 m^2 size, except the trial at TUL2021, which was laid in a row-column design with two replicates as double rows in plots of 1 m length with 17 cm spacing. Seeds were sown using a commercial planter.

At HOH2020 and HOH2021, artificial inoculation was performed with *Fusarium culmorum* isolate FC46 at a concentration of 1.5×10^4 spores mL^{-1}. At TUL 2021, artificial inoculation was performed with the *Fusarium culmorum* isolate Fc91015 at a conidial concentration of 2.5×10^4 spores mL^{-1}. In ROS 2020, natural occurrence of FHB was observed. Approximately 100 $mL \cdot m^{-2}$ of the diluted inoculum was applied using an adapted agricultural sprayer (Hege 75, Waldenbuch, Germany). Inoculations were repeated three times at intervals of 3 to 4 days to inoculate each genotype at least once during mid-anthesis. The first inoculations were done when early cultivars started flowering. Rating scale was adjusted to the 1–9 scheme of the plant breeders and the Federal Plant Variety Office as follows: 1 = 0%, 2 = > 0%–12.5%, 3 = > 12.5%–25.0%, 4 = > 25.0%–37.5%, 5 = > 37.5%–50.0%, 6 = > 50.0%–62.5%, 7 = > 62.5%–75.0%, 8 = > 75.0%–87.5%, 9 = > 87.5%–100% (Moll, Flath, and Tessenow 2010). The first rating started at the onset of symptom development about 15 to 20 days after inoculation and was repeated in intervals of 3 to 5 days until the first signs of ripening. Each time, we assessed the percentage of visually infected spikelets per plot. This scoring approach at different epidemic stages evaluates the combination of both resistances to FHB, Type I (resistance against initial infection, incidence) and Type II (resistance to pathogen spread in infected tissue, symptom development) in one number. A definition of these types can be found in Schroeder and Christensen (1963).

Phenotypic traits such as plant height (PH) in cm, heading date (HD) in day of the year starting from January 1st, and FHB severity (1 = low to 9 = high susceptibility) were recorded in four environments. Lodging was evaluated using a 1–9 scale with 1 = no lodging, all culms are upright, 3 = Inclination of all culms by approximately 30° from the vertical or stronger lodging in nests on approximately ¼ of the plot, 5 = Inclination of all culms by approximately 45° or stronger lodging in nests on ½ of the plot, 7 = Inclination of all culms by approximately 60° or total lodging on ¾ of the plot, 9 = Total lodging (Bundessortenamt 2016). Anther extrusion was visually evaluated in three environments when about 50% of a plot was flowering on a linear 1–9 scale: 1 = no anthers extruded, 3 = up to 25% anthers extruded, 5 = up to 50% anthers extruded, 7 = up to 75% of anthers extruded, 9 = maximum anther extrusion (Boeven et al. 2016).

Yield data were available from noninoculated trials at five environments in Germany: HOH2019-LD and HOH2020-LD, Oberer Lindenhof near Reutlingen in 2020 (OLI2020-LD) and in 2019 additionally from Schwäbisch Hall (SCH2019-LD) and Rastatt (RAS219-LD, Table 1). 'LD' indicates that these results are from large-drilled plots of about 5 m^2 size grown in an augmented design. Herbicides, growth regulator and fungicides were used as locally recommended. Nitrogen fertilization was 65% lower than in bread wheat. Sowing was done mechanically in all trials.

2.2 | Phenotypic Data Analysis

Because of different experimental designs used at different environments, phenotypic analysis was performed as a two-stage procedure. Non-emmer wheat species (bread wheat, spelt wheat, durum wheat, einkorn) were excluded from the data analyses.

2.2.1 | First Stage

First, evaluation of the single locations was performed. The mixed models given in Equations (1a) and (1b) were used for alpha design and row-column design, respectively:

$$y_{ijk} = u + g_i + rep_j + b_{jk} + e_{ijk}, \quad (1a)$$

where y_{ijk} is the phenotypic observation for the ith genotype in the jth replicate in the kth incomplete block, u is the general mean, g_i the genotypic effect of the ith genotype, rep_j the effect of the jth replicate, b_{jk} is the effect of the kth incomplete block of the jth replicate, and e_{ijk} is the residual.

$$y_{ijrc} = u + g_i + rep_j + row_{jr} + col_{jc} + e_{ijrc}, \quad (1b)$$

where y_{ijrc} is the phenotypic observation for the ith genotype in the rth row and cth column nested within jth complete replicate, u is the general mean, g_i the genotypic effect of the ith genotype, rep_j the effect of the jth complete replicate, row_{jr} is the effect of the rth row within jth replicate, col_{jc} is the effect of the cth column within jth replicate, and e_{ijrc} is the residual. To estimate variance components, all effects in model 1a and all effects in model 1b except rep_j were assumed as random effects. The genotype main effect g_i in model 1a and additionally only for model 1b complete rep_j were assumed as fixed effect to obtain best linear unbiased estimates (BLUEs) ($\overline{y}_i$) and their approximated variance–covariance matrix ($\widehat{V}$). In both models, the random effects were assumed independently distributed.

Notably, before fitting the model 1a or 1b, FHB scores were corrected for HD (FHB_{corr}) due to a reasonably strong negative correlation between these two traits. In a first step the genetic variance for these traits, and the genetic correlation between them were estimated by fitting a bivariate linear mixed model to uncorrected FHB scores and HD. The bivariate model used in this study is identical to the model used by Rapp et al. (2018), except that the location effect was not included because the model was fitted to the data from individual locations. All effects were assumed to be random with an approximate bivariate normal

distribution. With the obtained estimates, the FHB_{corr} for each plot was computed as

$$FHB_{corr} = y_{FHB} - \rho\frac{\sigma_{FHB}}{\sigma_{HD}}y_{HD},$$

where y_{FHB} and y_{HD} are the observed plot values that were scaled using 'scale' function in base R, whereas σ_{FHB} and σ_{HD} are the square root of the genetic variation for FHB and HD, respectively. ρ is the correlation between FHB and HD. The plot-level data for FHB_{corr} were then forwarded to model 1a and 1b.

2.2.2 | Estimates for the Second Stage of a Two-Stage Analysis

The genotype main effect g_i in model 1a and additionally only for model 1b complete rep_j were assumed as fixed effect to obtain best linear unbiased estimates (BLUEs) ($\bar{y}_i$) and their approximated variance–covariance matrix ($\hat{V}$). Thus, separate analyses and separate effect estimates were obtained for each environment. Finally, estimates forwarded to the second stage were indexed by environment k ($\bar{y}_{ik}$, $\hat{V}_k$).

A weighting method in the context of two-stage analysis can be useful to approximate the variance–covariance structure of adjusted means and hence slightly improve the analysis (Möhring and Piepho 2009). We used Smith's weights (Damesa et al. 2017; Smith, Cullis, and Gilmour 2001; Smith, Cullis, and Thompson 2005) obtained as the diagonal elements of the inverse of $\hat{V}_k$, which is the variance–covariance matrix of adjusted means of the genotypes from first stage.

2.2.3 | Second Stage

The following mixed model was implemented in the second stage:

$$\bar{y}_{ik} = \mu + g_i + env_k + g_i{:}\,env_k + \bar{e}_{ik}, \quad (2)$$

where $\bar{y}_{ik}$ is the BLUE of the ith genotype in the kth environment obtained in the first stage, μ is the general mean, g_i is the main effect of the ith genotype, env_k is the main effect of the kth environment, $g_i{:}\,env_k$ is the genotype-by-environment interaction, and $\bar{e}_{ik}$ is the error of the mean $\bar{y}_{ik}$ obtained in the first stage.

In model 2, for estimating BLUEs, all effects except g_i were assumed as random and for obtaining variance components, all effects were assumed as random. Variance components were estimated using the restricted maximum likelihood (REML) method assuming a random model (Cochran and Cox 1957). A likelihood ratio test with model comparisons was performed to check for significance of the variance components (Stram and Lee 1994).

Broad sense heritability (h^2) across the series of trials was estimated as given in Equation (3):

$$h^2 = 1 - \frac{\vartheta}{2\sigma_G^2}, \quad (3)$$

where ϑ is the mean variance of a difference of two best linear unbiased predictors (BLUPs) and σ_G^2 the genotypic variance (Piepho and Möhring 2007). Pearson's correlation coefficients (r_p) were estimated among BLUEs of the examined traits. All analyses were performed utilizing the statistical software R (R Core Team 2022) and the software ASReml-R V4.0 (Butler et al. 2017).

2.3 | Genotypic and Molecular Analysis

2.3.1 | Molecular Markers

The diversity panel containing 143 emmer genotypes was genotyped by genotyping-by-sequencing (GBS) at Diversity Arrays Technology (Yarraluma, Australia) (Li et al. 2015). The dominant silico-DArT (diversity array technology) markers and the co-dominant single nucleotide polymorphism (SNP) markers were denoted by their clone ID with a suffix 'D' or 'S' corresponding to the marker type—DArTs or SNPs, respectively. Markers with more than 20% missing data across the diversity panel or a minor allele frequency (MAF) lower than 5% were removed from the initial marker set using PLINK (Purcell et al. 2007). Separately for DArTs and SNPs, the missing values were imputed using LinkImpute, a software package based on a k-nearest neighbour genotype imputation method, LD-kNNi (Money et al. 2015). The PLINK and LinkImpute were executed using statistical software R (R Core Team 2022). The imputation accuracy was 97% and 95% for DArTs and SNPs, respectively. The accuracy of imputation is the proportion of masked known genotypes (default = 10,000) that were correctly imputed (Money et al. 2015). Both types of markers were combined into one dataset. Markers with MAF lower than 5% were discarded again after the imputation, resulting in 67,605 markers. After the second round of filtering post imputation, 35,747 markers mapped to known positions on the reference genome assembly of *Triticum dicoccoides* - Wild Emmer Wheat Zavitan WEWSeq v2.1 (Zhu et al. 2019). The distribution of DArTSeq markers across 14 chromosome of cultivated emmer wheat is visualized in Figure S1.

2.3.2 | Population Structure

Relationships among the 143 genotypes were analysed by implementing principal coordinate analysis based on the Rogers distance (Rogers 1972), which was computed using genome-wide markers in R package 'poppr'. The function *cmdscale* of base R was used to calculate principal coordinates based on Rogers distance (Figure S2). The grouping into two clusters has been considered in this study by fitting the first and second principal coordinates as covariate variables in the model.

2.3.3 | Association Mapping

Association mapping was conducted by using 'Bayesian-information and Linkage-disequilibrium Iteratively Nested Keyway (BLINK)' method (Huang et al. 2019) implemented in the GAPIT R package (Lipka et al. 2012; Wang and Zhang 2021). BLINK is a statistically powerful and computationally efficient algorithm, which produces fewer false

positives and identifies more true positives than the most recently developed GWAS method, FarmCPU (Liu et al. 2016). In addition, BLINK does not require quantitative trait nucleotides (QTNs) to be evenly distributed throughout the genome, whereas FarmCPU does; thus, BLINK eliminates the unrealistic assumption (Huang et al. 2019). The first two principal coordinates were fitted as covariate variables to reduce the false positives due to population stratification. For association mapping, we used BLUEs of traits calculated across all environments, and 35,747 markers with known map coordinates on WEWSeq v2.1.

A p value < 0.05 corrected according to Hochberg and Benjamini was considered as the significance threshold and was used to identify significant marker-trait associations (MTAs). The sequences of the significant MTAs for FHB_{corr} are provided in Table S3. The proportion of phenotypic variance explained by the QTNs was estimated by fitting the significant markers in linear models jointly in the order of strength of their association (lower the p value, higher the strength of association) (Würschum, Langer, and Longin 2015). For additive genetic model, marker information was coded as 0, 1, 2, where 0 and 2 are the two homozygous (DArTs and SNPs) and 1 the heterozygous genotypes (SNPs). The total proportion of explained phenotypic variance (R^2) was calculated as

$$R^2_{adj} = R^2_{adj} \times 100,$$

where R^2_{adj} is the adjusted coefficient of determination of the linear model (Würschum et al. 2016). The phenotypic variance explained by individual significant markers (R^2_m) was calculated as

$$R^2_m = \left(\frac{SS_m}{ss_{total}} \right) \times 100,$$

where SS_m is the sum of squares for the marker m and ss_{total} is total sum of squares of all markers fitted in a linear model. The allele substitution effect of each significant marker was derived as the regression coefficient from the linear model with only the respective marker under consideration.

To visualize the differences in disease severity between different groups of genotypes based on the allelic state of a given molecular marker, we produced box plots. Due to the unequal number of genotypes in different groups, we opted to use the ad-hoc method of notches, which displays the confidence interval around the median. If the notches of two boxes do not overlap, there is a strong evidence (95% confidence) that their medians differ (Chambers et al. 1985).

TABLE 2 | Means for Fusarium head blight (FHB) in the individual environments.

Environment[a]	FHB (1–9)
HOH2020	5.17
HOH2021	4.47
TUL2021	6.58
ROS2020	3.54

[a]HOH = Hohenheim, TUL = Tulln, ROS = Rosenthal; the number following the abbreviated name of location denotes the year.

3 | Results

In the individual environments, mean FHB ratings ranging from 3.5 to 6.6 were found (Table 2). There was wide variation in FHB severity across environments with scores ranging

TABLE 3 | Summary of the phenotypic analysis.

Parameter	FHB (1–9)[a]	FHB_{corr}[b]	Heading date (day in year)	Plant height (cm)
# Environments	*4*	*4*	*4*	*4*
Means				
Minimum	2.15	−1.27	151.50	88.54
Mean	4.83	−0.009	157.63	141.10
Maximum	8.33	1.14	169.19	164.25
$LSD_{5\%}$	1.87	0.88	2.68	10.92
Variance components				
Genotype (G)	2.28***	0.21***	5.87***	219.96***
G × environment	1.02***	0.20***	2.22***	26.85***
Error	1.40	0.38	2.38	54.59
Heritability	0.84	0.68	0.87	0.94

Note: LSD least significant difference at $p \leq 0.05$. Min, mean, and max values are based on 143 emmer genotypes, whereas LSD, variance components, and heritability are based on data of all 150 genotypes including seven checks from other wheat species (Julius, Genius, Franckenkorn, Zollernspelz, Wintergold, Sambadur, and Terzino).

[a]1 = not visually diseased, 9 = fully diseased.

[b]Low value = not/less visually diseased, high value = more/fully diseased.

***Significant at $p \leq 0.001$.

from 2.15 to 8.33, and high heritability ($h^2 = 0.84$) across environments (Table 3). Both genotype and genotype × environment interaction had significant effects on FHB. Heading date and plant height also showed significant genetic variation and high heritabilities. Because of the significant correlation between heading date and FHB severity ($r = -0.66$, $p < 0.001$, Figure S3), all FHB data were corrected (FHB_{corr}) by fitting a bivariate linear mixed model to FHB scores and HD. As expected, this correction method removed the correlation between HD and FHB_{corr} being not significant any more ($r = -0.09$). However, the heritability of FHB_{corr} was also reduced ($h^2 = 0.68$). Lodging and anther extrusion had heritabilities of 0.73 and 0.81, respectively (Table S1).

The principal component analysis explained 48% of variation by the first two components (Figure S2) and revealed two major groups. Most of the emmer genotypes were distinct only in PC2, whereas a minority from the gene banks formed an outgroup. This consisted of 24 genotypes whereof 20 genotypes originated from the German gene bank in Gatersleben. This grouping has been considered in this study by fitting the first and second principal coordinates as covariate variables in the model.

Twelve QTNs were detected for HD with one major QTN on chromosome 7B ($R^2 = 34.6\%$) and 11 minor QTNs each explaining 0.08 to 8.02% phenotypic variance (Table S4). None of these QTNs co-localized with FHB_{corr} QTNs.

Plant height and FHB_{corr} were normally distributed with a slight negative skew (Figure 1). We found a small but significant negative correlation between both traits ($r = -0.29$, $p < 0.001$, Figure S3). GWAS detected three QTNs for plant height and seven QTNs for FHB severity (FHB_{corr}) using BLUEs calculated across all environments (Table 4). One major QTN for plant height was found on chromosome 4B that accounted for 51% of the phenotypic variance (Table 4, Figure 1). The other two QTNs for plant height had only minor effects. For FHB_{corr}, a major QTN explaining 16% of the phenotypic variance was also found on chromosome 4B, 2.3 Mbp away from the plant height locus. The plant height and FHB QTNs on chromosome 4B were most likely linked to *Rht-B1*. One QTN on chromosome 5B and two QTNs on chromosome 7B had also large effects on FHB severity. Three other QTNs on chromosome 1B and chromosome 3B were classified as minor. The seven QTNs cumulatively explained a high proportion of the phenotypic variance in FHB_{corr} ($R^2 = 72.6\%$).

When the emmer genotypes were grouped according to the *Rht-B1* marker on chromosome 4B, those genotypes expressing the semi-dwarf allele were more susceptible to FHB_{corr} than the genotypes with the wild-type allele (Table 5). Heading date was not affected by the semi-dwarfing locus, but plant height was reduced by approximately 32 cm. The scatter plot between plant height and FHB_{corr} exhibited the separation between short and tall genotypes (Figure 2). The *Rht B1b* semi-dwarfing allele clearly reduced plant height and increased FHB_{corr}, in comparison to the wild-type allele (Figure 2, Table 5). The QTNs on chromosomes 5B and 7B with the highest effect on FHB_{corr} already show a considerable reduction individually, which is exceeded by their combined effect (Figure 3).

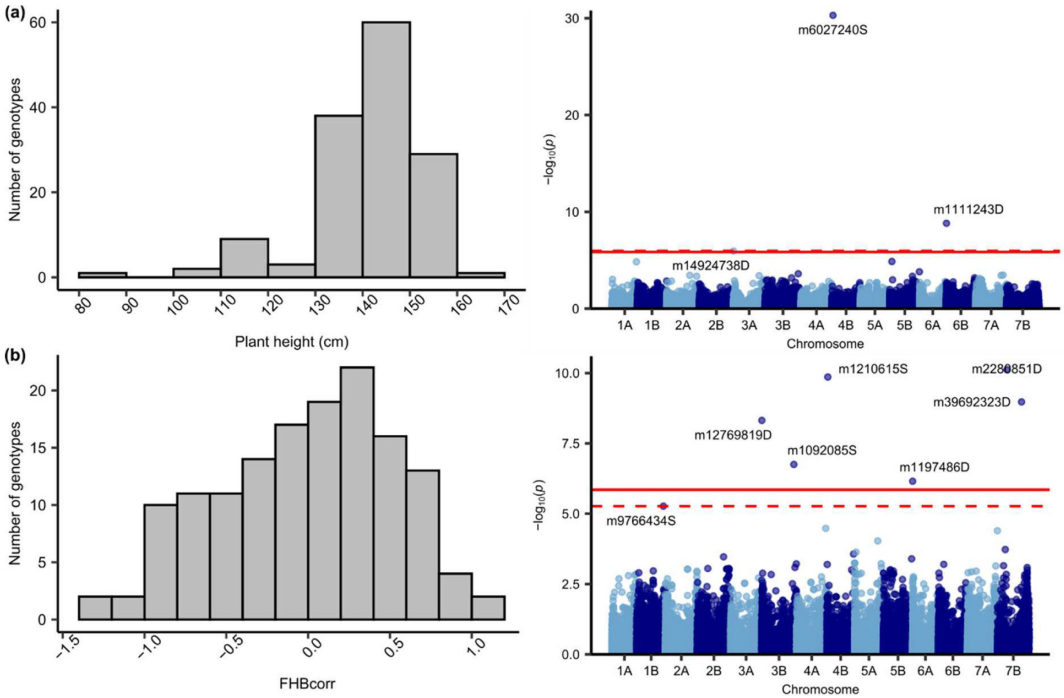

FIGURE 1 | Histogram of phenotypic BLUEs and Manhattan plot from GWAS for (a) plant height and (b) Fusarium head blight corrected for heading date (FHB_{corr}); the solid red horizontal line indicates Bonferroni-corrected *p* value threshold of 0.05, the dashed red horizontal line corresponds to the highest Hochberg and Benjamini-corrected *p* value threshold of 0.05. [Color figure can be viewed at wileyonlinelibrary.com]

TABLE 4 | Marker trait associations (MTAs) identified using BLINK model for Fusarium head blight resistance corrected for heading date (FHB_{corr}) and plant height of 143 emmer genotypes; in bold are markers with moderate to large effects ($R^2 > 14\%$).

Marker	Chr.	Pos. (bp)	MAF	*p*	HB. *p* value	R^2 (%)	α effect
Plant height							
m14924738D	3A	491,795	0.385	1.11E−06	1.32E−02	0.83	0.57
m6027240S(*Rht B1*)	4B	25,529,149	0.094	5.03E−31	1.80E−26	50.92	−14.78
m1111243D	6B	13,987,339	0.112	1.52E−09	2.72E−05	3.88	−11.68
Total						54.68	
FHB_{corr}							
m9766434S	1B	660,152,935	0.056	5.39E−06	2.75E−02	5.79	−0.198
m12769819D	3B	6,182,540	0.161	4.80E−09	4.29E−05	2.20	0.058
m1092085S	3B	799,018,429	0.448	1.77E−07	1.27E−03	4.20	0.063
m1210615S (*Rht B1*)	4B	23,238,409	0.213	1.38E−10	2.46E−06	15.79	0.325
m1197486D	5B	724,789,282	0.371	7.00E−07	4.17E−03	5.30	−0.341
m2280851D	7B	231,376,758	0.301	7.56E−11	2.46E−06	26.53	0.303
m39692323D	7B	592,507,932	0.315	1.06E−09	1.26E−05	14.14	−0.364
Total						72.59	

Note: Markers were referenced to chromosomes (Chr.) and positions (Pos.) of the reference genome assembly of *Triticum dicoccoides*—Wild Emmer Wheat Zavitan WEWSeq v2.1 from Zhu et al. (2019) in base pairs (bp); *p* value < 0.05 original, HB. *p* value = *p* value corrected according to Hochberg and Benjamini, R^2 = proportion of explained phenotypic variance.

TABLE 5 | Adjusted means of the allelic states of marker m6027240S linked with plant height QTL on chromosome 4B for Fusarium head blight severity (FHB), FHB corrected for heading date (FHB_{corr}), heading date (HD), and plant height (PH); *N* = number of genotypes in the respective allele state.

Allele	*N*	FHB (1–9)	FHB_{corr}	HD (day in year)	PH (cm)
Tall (AA)	129	4.76	−0.04	157.67	143.8
Dwarf (GG)	12	5.70	0.42	157.34	111.6
Difference	—	0.94	0.46	−0.33	−32.2

4 | Discussion

4.1 | Genetic Variation for FHB Resistance in Cultivated Emmer

This study found a large genetic variation for FHB resistance in cultivated emmer wheat with scores ranging from 2.15 to 8.33 on the 1–9 scale (1 = fully resistant, 9 = fully susceptible). Complete resistance was not expected, given the quantitative inheritance of FHB resistance reported in other wheat species. This finding is remarkable because the emmer genotypes investigated in this study have not been previously selected for FHB resistance. The diverse origins of these genotypes may have played a role in their variable resistance, as they were sourced from various institutions and private breeders (Table S1). The majority of the most resistant cultivars were obtained from the gene banks, illustrating the importance of plant genetic resources for resistance breeding. The most resistant genotypes were all tall, with plant heights ranging from 136 to 159 cm (Table S5). Reduced height in emmer is an important consideration for improved production. However, the shortest genotype with 89 cm was also among the most susceptible genotypes (score 7.58, E-12051-236-332/5-379/2/3-469, Table S5). This emmer collection included genotypes with both superior FHB performance and grain yield. For example, emmer 9.040/99 not only showed good FHB resistance but also had a grain yield comparable to the best emmer standards. The bread and durum wheats tested together with the emmer genotypes were highly susceptible to FHB demonstrating the high disease pressure obtained in our study. Only the einkorn cultivar Terzino had a comparably resistant phenotype than the best emmer.

4.2 | Genetic Architecture of FHB Resistance in Cultivated Emmer

Seven QTNs for FHB resistance were detected in this study using BLUEs calculated across four environments. We found of a major FHB QTN on chromosome 4B at 23.2 Mbp. This QTN

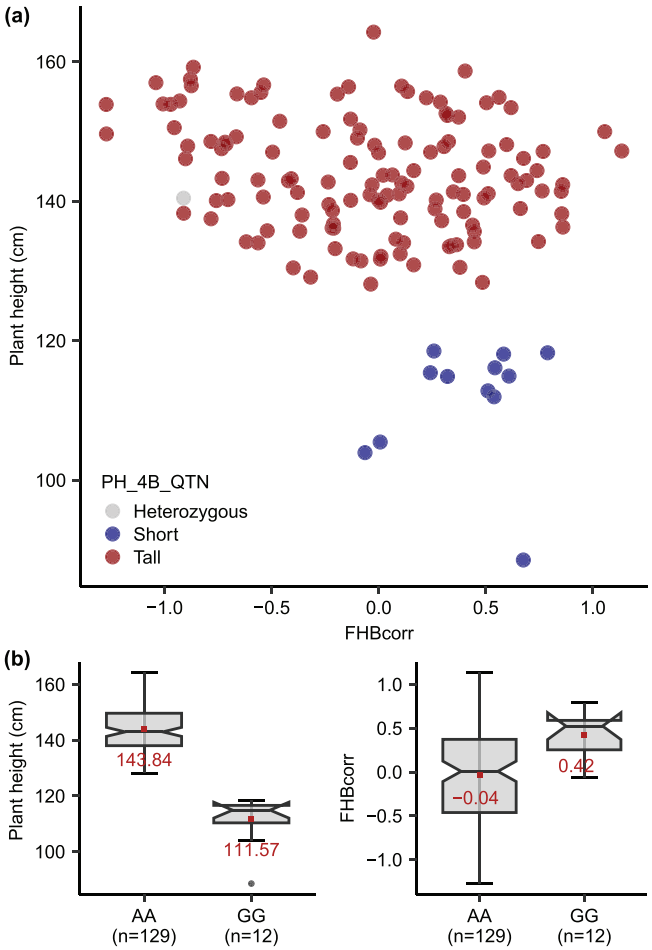

FIGURE 2 | Effect of the major QTL on chromosome 4B (m6027240S; 25.5 Mbp) on plant height and Fusarium head blight corrected for heading date (FHB_{corr}) (a) Scatter plot between FHB_{corr} and plant height for the genotypes with the tall and the short allele, respectively; (b) boxplots showing comparisons for plant height and FHB_{corr} for the two alleles (AA = tall, GG = dwarf); *n* in brackets denotes the number of genotypes in a group, the red numbers/squares denote the mean values. [Color figure can be viewed at wileyonlinelibrary.com]

localizes to a similar genomic region as the dwarfing gene *Rht-B1* that was located nearly in the same position (23.4 Mbp) by Alahmad et al. (2023). The nearest major QTN for plant height explaining 50.9% of the phenotypic variance was located at 25.5 Mbp (Table 4). This QTN is 4.5 Mbp away from the *Rht-B1* gene on chromosome 4B according to the reference genome assembly (NCBI gene symbol = LOC119294785, https://www.ncbi.nlm.nih.gov/gene/119294785#reference-sequences). As such, we associate the markers m6027240S (plant height) and m1210615S (FHB_{corr}) with the *Rht-B1* locus.

Three other QTNs for FHB_{corr} with major effects were detected on chromosomes 5B and 7B in emmer. Also in other tetraploid wheat, a QTL on chromosome 7B was found (Buerstmayr et al. 2012). In the *Triticum dicoccum* line Td161, an FHB resistance QTL on 7BS was identified overlapping with a QTL for flowering time. As, however, in this older study no physical localization could be given, the comparison is difficult. Also Buerstmayr, Ban, and Anderson (2009) and Venske et al. (2019) found a few FHB resistance QTL in bread wheat on chromosome 7B. In Schmolke et al. (2005) the 7B QTL from bread wheat overlapped with HD. So it is interesting, that we detected two loci on chromosome 7B although we corrected the data for HD. Indeed, the HD QTL on this chromosome is at 147 Mbp, a distance of 84.3 Mbp to the next FHBcorr QTN. These major QTN can thus be used for further improvement, although they do not contribute to shorter genotypes.

Three QTNs for FHB_{corr} with minor effects on chromosomes 1B and 3B were additionally detected in this study. The physical proximity of the 3B locus at 6.2 Mbp to *Fhb1*, which Ruan et al. (2020) placed between 7.6 and 13.9 Mbp, does not suggest any significant relationship between this QTN in emmer and *Fhb1* from the Chinese bread wheat Sumai 3. Steiner et al. (2019) also identified a major QTL in durum wheat at the same chromosomal interval as reported for *Fhb1*, although haplotype analysis highlighted the distinctness of both QTLs. Given that Venske et al. (2019) had already identified over 550 QTLs in bread wheat, detecting new FHB resistance loci is rather improbable. Similarly, Prat et al. (2014) emphasized in their review that most reported QTLs in tetraploid wheat have previously been identified in hexaploid wheat, further demonstrating the common genetic basis of FHB resistance.

In a recent meta-QTL study for FHB resistance in wild emmer, Cabas-Lühmann et al. (2024) analysed 31 QTLs and revealed that the majority of them were located on chromosomes 2A, 6B, and 3A. On chromosome 3A, a large QTL was derived from three independent wild emmer origins. Interestingly, none of these 'hotspots' were detected in our cultivated emmer population. This, and the high number of QTLs detected in only three studies, shows that (wild) emmer has high genetic diversity for FHB resistance, similar to bread or durum wheat.

4.3 | Effect of the Semi-Dwarfing *Rht-B1* Gene on FHB Resistance

The introgression of *Rht-B1b* into cultivated emmer reduced plant height by an average of about 32 cm (Table 5). This reduction effectively addresses lodging problems in this crop. In fact, the 12 emmer genotypes with the semi-dwarf *Rht-B1* allele ranged for lodging from 0.5 to 2.7 (on the 1–9 scale), whereas the range of emmer genotypes with the wild-type allele was 1.2 to 6.9 (Figure S4). However, it also led to a significant increase in susceptibility to FHB. This trade-off has been previously reported in bread wheat (Miedaner and Voss 2008; Buerstmayr and Buerstmayr 2016; Thambugala et al. 2020) and durum wheat (Buerstmayr et al. 2012; Miedaner et al. 2017). Our results are the first to demonstrate this in cultivated emmer wheat. The effect of the *Rht-B1* marker on FHB severity was rather small in our study explaining 15.8% of the phenotypic variance, likely due to the low frequency of the short allele (12 genotypes with the short allele vs. 129 with the tall allele, Figure 2). Similarly, Haile et al. (2023) identified a marker co-localized with *Rht-B1* in durum wheat that was significantly associated with plant height and explained a high amount of the phenotypic variation for this trait ($R^2 = 49.3\%$), but only 4.1% and 5.7% of the phenotypic variance for FHB severity and incidence, respectively.

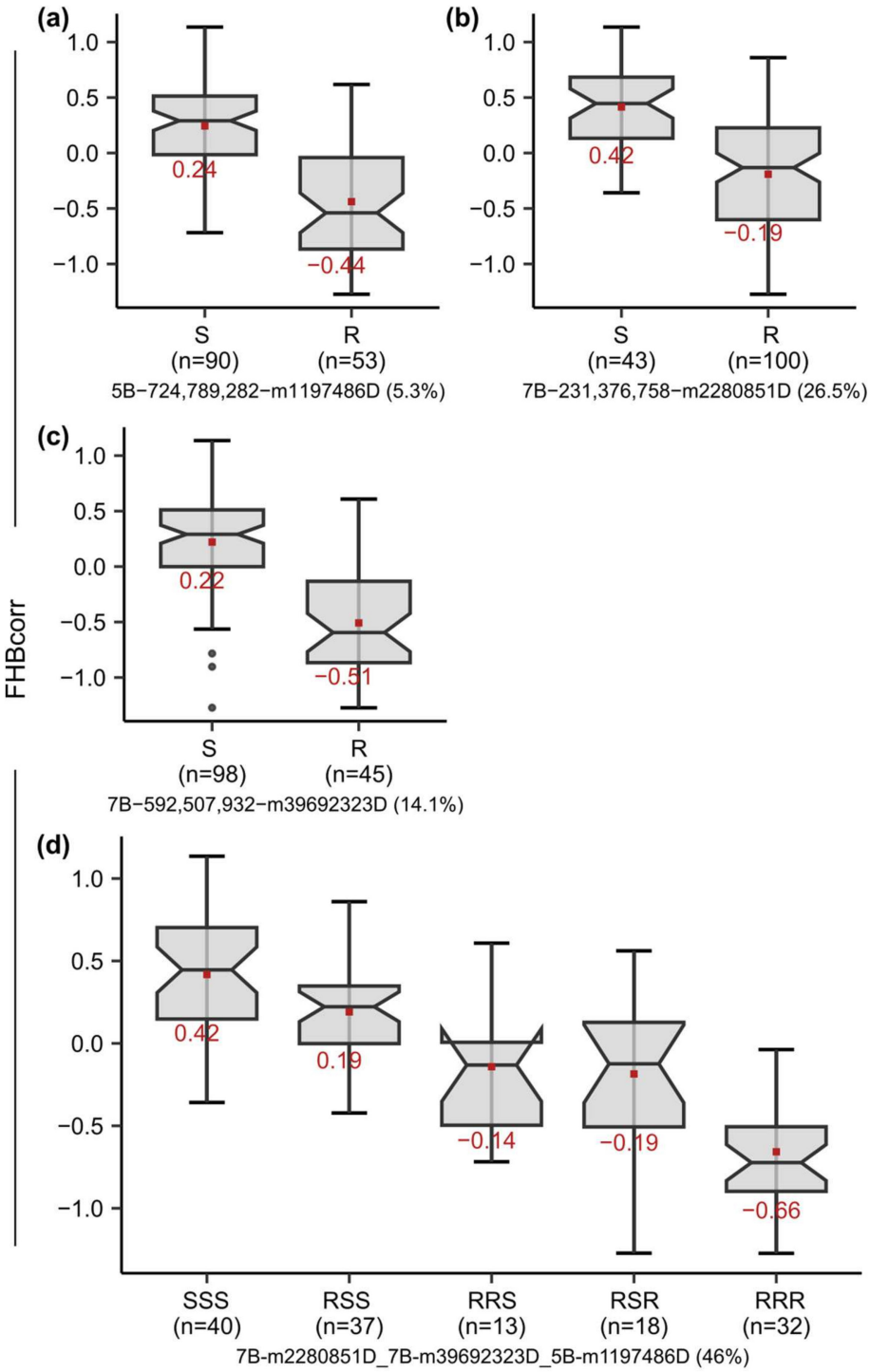

FIGURE 3 | Boxplots showing comparisons for Fusarium head blight resistance corrected for heading date (FHB_{corr}) between genotypes having different alleles (R = resistant, S = susceptible) for the following markers with large effects: (a) m1197486D on chromosome 5B, (b) m2280851D and (c) m39692323D, both on chromosome 7B, (d) various combinations of (a)–(c), only haplotypes with $n > 5$ are shown, the red number/squares denote the mean values. [Color figure can be viewed at wileyonlinelibrary.com]

4.4 | Consequences for Breeding Short, FHB-Resistant Emmer Wheat

The trade-off between FHB resistance and plant height poses a challenge for breeders. In this study, none of the genotypes carrying the height-reducing allele *Rht-B1b* were among the 20 most resistant genotypes. All of them were moderately to highly susceptible, with FHB severity ranging from 3.96 to 7.81 (Table S5). Therefore, special efforts are needed to breed short, FHB-resistant emmer varieties.

To achieve this, *Rht-B1b* could be back-crossed into the most resistant emmer genotypes detected here. Alternatively, the genotypic background of semi-dwarfing emmer could be enriched by potent

native FHB-resistance QTLs by marker-assisted or genomic selection within the adapted gene pool. This was successfully demonstrated in bread wheat (Akohoue et al. 2022); however, a large population size is necessary. A more straightforward approach for counterbalancing the negative effect of *Rht-B1b* on FHB resistance would be to introgress the two potent QTNs on chromosome 7B (Table 4) in short emmer wheat. One mechanism by which *Rht-B1b* increases FHB severity is by increasing anther retention (refer to review of Buerstmayr, Steiner, and Buerstmayr 2020). Selecting for higher anther extrusion in short wheat breeding material can counterbalance the negative effects of dwarfing genes to some extent (Michel, Steiner, and Buerstmayr 2024). Also in this study, a moderate negative correlation between anther extrusion and FHB severity was found ($r = -0.506$, $p < 0.001$, Figure S5). However, the correlation was not as close as previously reported from bread wheat (Buerstmayr and Buerstmayr 2016).

Alternatively, major QTLs from non-adapted sources, like the repeatedly detected QTL on chromosome 3A from wild emmer (Cabas-Lühmann et al. 2024) or *Fhb1* on chromosome 3BS and *Fhb5* on chromosome 5A from Chinese wheat Sumai 3 could be introgressed. In a study with European bread wheat, both foreign QTLs significantly counterbalanced the negative effect of *Rht-D1b* (Miedaner et al. 2019). *Fhb5* alone was able to compensate for the full FHB-increasing effect of the semi-dwarfing gene. The combined effect of both QTLs resulted in an additive reduction of FHB. Similar results were achieved by Lu et al. (2011) in bread wheat. The same could be expected for *Rht-B1b* in emmer.

Another alternative could be the use of semi-dwarfing *Rht* alleles that do not affect FHB resistance, like *Rht24b* on chromosome 6A from bread wheat (Herter et al. 2018; Miedaner et al. 2022). However, the effect of this gene on plant height reduction is approximately only about half as much as that of *Rht-D1b*, which might not be efficient for the long-strawed emmer. This could be a stimulus to analyse the effect of other alternative *Rht* genes on FHB resistance although the emmer community is not in favour of crossbreeding with bread wheat.

In conclusion, the use of semidwarfing genes in emmer will reduce lodging problems but hinder genetic gain for FHB resistance in the future as recently shown in bread and durum wheat (refer to review of Miedaner, Flamm, and Oberforster 2024). To achieve a short FHB resistant emmer genotype, rigorous selection for FHB will be necessary, preferably with artificial infection and accompanied by genome-assisted breeding. The first promising genotypes have been detected in this study.

Author Contributions

TM conceived and wrote the paper; MA analysed data from field trials and performed all statistical analyses; LM, BS, and HB helped with data acquisition at IFA Tulln; CFHL provided plant materials, secured funding, and planned the field experiments. All authors participated in revising the paper and approved it.

Acknowledgements

We highly appreciated the help of Dr. Philipp Boeven from LIMAGRAIN GmbH, Edemissen, Germany, for conducting our experiment at their breeding station in Rosenthal. We thank the technical teams of all stations for their valuable support. This work was funded by the "Ministerium für ländlichen Raum und Verbraucherschutz" (MLR, Stuttgart) within the special program "Stärkung der biologischen Vielfalt des Landes Baden-Württemberg" (Az: 23-8872). Open Access funding enabled and organized by Projekt DEAL.

Conflicts of Interest

TM is member of the editorial board of this journal. All other authors have no conflict of interest to declare.

Data Availability Statement

All relevant data are part of this paper including the supporting information.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.