INTRODUCTION

Winter rye was grown in Northeastern Europe on 3.2 million hectares in 2017 (Food and Agriculture Organization of the United Nations (FAO), 2019). Germany, Poland, Russia, Finno-Scandinavia, Belarus and Ukraine together contribute 74% of the worldwide harvest (FAO, 2019). Rye grain is traditionally used for bread making, but also as home-grown feed and as a substrate for bioethanol and biogas production (Miedaner & Laidig, 2019). Rye is an allogamous and diploid (RR, 2n = 2x = 14) small-grain cereal crop belonging to the

Abstract

Rye is a multi-purpose cereal crop grown in Central and Eastern Europe as well as in Western Canada. Fusarium head blight (FHB) is one of the diseases that have a severe negative impact on rye, but knowledge about FHB resistance at the genomic level is totally missing in rye. The objective of this study was to elucidate the genetic architecture of FHB resistance in winter rye using genome-wide association (GWA) mapping complemented by genomic prediction (GP) in comparison with marker-assisted selection (MAS). Additionally, plant height and heading stage were analysed. A panel of 465 S1-inbred lines of winter rye was phenotyped in three environments (location-year combinations) for FHB resistance by inoculation with Fusarium culmorum and genotyped with a 15k SNP array. Significant genotypic variation and high heritabilities were found for FHB resistance, heading stage and plant height. FHB did not correlate with heading stage, but was moderately correlated with plant height (r = −.52, p < .001) caused by some susceptible short inbred lines. The GWA scan identified 15 QTL for FHB resistance that jointly explained 74% of the genotypic variance. In addition, we detected 11 QTL for heading stage and 8 QTL for plant height, explaining 26% and 14% of the genotypic variance, respectively. A genome-wide prediction approach resulted in 44% higher prediction abilities than marker-assisted selection for FHB resistance. In conclusion, genomic approaches appear promising to improve and accelerate breeding for complex traits in winter rye.

KEYWORDS
agronomic traits, Fusarium resistance, genomic prediction, GWAS, QTL, rye
Triticeae and is reported to have a large genome of approximately 7.9 Gbp (Bartoš et al., 2008). It is the paternal donor for triticale (Ammar, Mergoum, & Rajaram, 2004) and has been used to improve important agronomic traits of wheat (Kim, Johnson, Baenziger, Lukaszewski, & Gaines, 2004; Schlegel & Korzun, 1997; Zhou et al., 2007). Hybrid rye is the most common cultivar type in Central Europe, commercially available in Germany, Austria, Denmark, Sweden, Poland, Belarus and Russia. Hybrid breeding is based on the development of inbred lines from two heterotic pools that are subsequently selected for line per se and testcross performance (Miedaner & Laidig, 2019). Hybrid cultivars cover about 80% of the total rye acreage in Germany and yield 15%–20% more grain than population cultivars, the alternative type of cultivars (Laidig et al., 2017).

Generally, rye has been reported to be more resistant to biotic (Arseniuk, Foremska, Góral, & Chetkovski, 1999; Gaikpa, Lieberherr, Maurer, Longin, & Miedaner, 2019; Miedaner, Reinbrecht, Lauber, Schollenberger, & Geiger, 2001) and abiotic (Bartoš et al., 2008; Myśków, Góral ska, Lenarczyk, Czyczlyo-Mysza, & Stojalowski, 2018; Villareal, Bañuelos, Mujejeb-Kazi, & Rajaram, 1998) stress factors compared to wheat and triticale. However, rye can be infected with several diseases including Fusarium head blight (FHB), reducing grain size and grain yield and contaminating the grains with mycotoxins, like deoxynivalenol (DON) and zearalenone (ZON) (Miedaner & Geiger, 1996). These mycotoxins pose health threats to humans and animals (Pierron, Alassane-Kpembi, & Oswald, 2016) and are therefore strictly regulated in the European Union. For rye and bread wheat, the same limits apply, being 1.25 and 0.1 mg/kg for DON and ZON, respectively, in unprocessed lots for human consumption (The Commission of the European Communities, 2006). In bread, the maximum allowed levels are 0.5 mg DON/kg and 0.05 mg ZON/kg. For feed, different guidance values are recommended, of which the lowest is for pigs with 0.9 mg DON/kg. In naturally infected rye grains from Denmark, F. graminearum, F. culmorum, F. avenaceum and F. poae dominated among the Fusarium species (Nielsen et al., 2011). Integration of resistant cultivars into other disease management practices such as crop rotation and good soil tillage is an efficient, cost-effective and ecologically safe method of reducing the impact of FHB in cereals. Hence, breeding for FHB resistance in rye is crucial given its use as bread cereal with 22% of the harvest and for feed with 64% of the harvest in Germany (BLE, 2018).

FHB resistance in rye is quantitatively inherited and mainly governed by additive gene action, similar to the other cereal species, with a large genotypic variation in breeding populations (Miedaner, Borchardt, & Geiger, 1993; Miedaner & Geiger, 1996). Highly resistant material, however, can rarely be found in existing nurseries. Genotypic correlation coefficients between FHB symptoms and DON showed a tight association (r = .8–.9), allowing an indirect selection for a reduced DON content by selecting for high FHB resistance (Miedaner, Wortmann, & Geiger, 2003). Genotype-by-environment (G × E) interaction played a major role, illustrating the necessity of selecting in several environments (location × year combinations).

The genetic architecture of FHB resistance has been investigated in bread wheat, durum wheat and to some extent in triticale, but no information is available for rye. In bread wheat (2n = 6x = 42, genome composition AABBD), about 550 QTL located on all chromosomes were reported for FHB resistance that could be reduced to 65 meta-QTL (Venske et al., 2019). Some major QTL, that is Fhb1, Fhb5 and Fhb6 from Chinese wheat, have higher effects on FHB resistance (Bai, Su, & Cai, 2018), and attempts are being made to introduce Fhb1 into European durum wheat breeding programmes (Prat et al., 2017). For triticale, several minor QTL for FHB resistance were reported on rye chromosomes (Dhariwal et al., 2018; Galiano-Carneiro, Boeven, Maurer, Würschum, & Miedaner, 2019; Kalih, Maurer, & Miedaner, 2015).

In rye, genomics is still lagging behind other small-grain cereals. A few previous studies reported QTL for agronomic traits such as plant height (PH), flowering time, yield-related and quality traits (Falke, Wilde, Wortmann, Geiger, & Miedaner, 2009; Hackauf et al., 2017; Miedaner et al., 2012, 2018), frost tolerance (Li et al., 2011) as well as drought tolerance (Myśków et al., 2018). However, no QTL or genome-wide association study (GWAS) has been reported in rye for FHB resistance. Because FHB resistance is generally caused by many QTL with minor effects, genomic selection (GS) might be more appropriate for improving the trait. GS utilizes genome-wide marker data to predict the genotypic values of individuals to be selected, thus reducing phenotyping once the marker effects have been estimated (Edwards et al., 2019). GS methods were applied in rye for kernel weight and quality traits in two introgression libraries (Mahone et al., 2015) and two bi-parental populations (Schulthess et al., 2016; Wang et al., 2014) as well as diverse breeding material (Bernal-Vasquez et al., 2014). A comprehensive genetic map based on a large SNP array is meanwhile available for rye (Bauer et al., 2017). For FHB resistance, GS yielded cross-validated prediction accuracies of 0.59 to 0.95 in bread wheat (Mirdita et al., 2015; Rutkoski et al., 2012), durum wheat (Miedaner, Herter, Ebmeyer, Kollers, & Korzun, 2019; Miedaner et al., 2017) and triticale (Galiano-Carneiro et al., 2019). Therefore, it is worthwhile to assess the prospects of GS in winter rye as the only out-crossing small-grain cereal.

Our objectives were to (a) assess the genetic variation for FHB resistance and associated traits in rye, (b) identify QTL for FHB resistance by GWA mapping and estimate their effects, (c) investigate their co-localization with QTL for heading stage and plant height, and (iv) compare the potential of marker-assisted selection and genomic prediction (GP) to improve breeding for FHB resistance in winter rye. For this, a large population of 465 rye inbred lines was analysed by inoculation with F. culmorum.

2 | MATERIALS AND METHODS

2.1 | Plant materials and field experiments

A panel of 465 rye (Secale cereale L.) S1 lines from the company HYBRO Saatzucht GmbH & Co. KG were used for the study. The lines descended from the ‘Carsten’ heterotic pool that is used as pollinator pool and were made up of 372 lines that were selected...
for FHB resistance in a recurrent selection (RS) programme across five cycles. The RS procedure was a typical S₅-line testing (Lynch & Walsh, 1998) with a three-year cycle: (a) selfing and testcrossing of non-inbred materials, (b) multi-environment selection of FHB rating for inbred line and testcross performance, respectively, with a weighted index of 3:1 and (c) recombination of the superior lines. To widen genetic variation, 93 lines unselected for FHB resistance were added to the last RS cycle that was analysed here. All lines were evaluated in three environments (location × year combinations) at the experimental stations of the University of Hohenheim, Germany, in Hohenheim (HOH) near Stuttgart in 2017 and 2018, and of the company HYBRO Saatzucht GmbH & Co. KG in Wulfsode (WUL) near Wriedel, Lower Saxony in 2017. Entries were mechanically sown in single-row observation plots 0.8–1.2 m long at a sowing density of 270 kernels/m². In HOH17 and WUL17, the experimental design used was α-lattice design with two replicates. Each replicate consisted of 54 incomplete blocks and 10 genotypes per block. To fill up the field design, standard lines were used. For the field trial in HOH18, a row–column partially replicated design was used because less seeds were available for some genotypes. The number of rows and columns was 40 and 23, respectively. Eighty-five per cent (85%) of the genotypes were replicated. All genotypes were treated with standard agronomic practices as described by Gaikpa et al. (2019). Genotypes were inoculated with one Fusarium culmorum isolate (FC46) at a concentration of 7.5 × 10⁵ spores/ml using a tractor-driven sprayer. The inoculation began at the onset of flowering of early genotypes and was repeated for 4–5 times at 2–3 days intervals to ensure that all entries were inoculated at least once at mid-anthesis.

The traits recorded included FHB severity, heading stage (HS) and plant height (PH). On plot basis, FHB severity was visually rated using a scale of 0%–100% of infected spikelets per genotype, starting from the onset of FHB symptoms differentiation (Miedaner et al., 2001). Two successive ratings were taken in WUL17, five ratings in HOH17 and four ratings in HOH18. HS was rated on a 1–9 scale, where 1 = the ear/head of the crop still remain in the leaf sheath and 9 = ear stalk is at least 10 cm long under the ear or above the leaf sheath. Plant height (cm) was measured from the ground level to the tip of the heads after full flowering using a metre rule.

### 2.2 Phenotypic data analysis

Two, four and five FHB ratings from WUL17, HOH18 and HOH17, respectively, were averaged to get mean FHB severity for each genotype per environment and used for all analyses. Adjusted means and variance components of each trait were calculated based on best linear unbiased estimation (BLUE) and best linear unbiased prediction (BLUP), respectively. ASReml package (Butler, 2009) within the statistical software R (R Core Team, 2018) was used for all phenotypic analyses. Because of the different field designs used in 2017 and 2018, a two-step analysis was done to get the adjusted means and variance components. At the first step, means for each trait in WUL17 and HOH17 were estimated separately using the model:

\[ Y_{ij} = \mu + G_i + R_j + B_{jk} + e_{ijkl}, \]

where \( Y_{ij} \) = the observed phenotypic mean for genotype \( i \) in replicate \( j \) and block \( k \), \( \mu \) = general mean, \( G_i \) = effect of the \( i \)th genotype, \( R_j \) = effect of the \( j \)th replicate, \( B_{jk} \) = effect of the \( k \)th block in the \( j \)th replicate and \( e_{ijkl} \) = residual error.

For HOH18, means were estimated using the model:

\[ Y_{ijkl} = \mu + G_i + R_j + W_k + C_l + e_{ijkl}, \]

where \( Y_{ijkl} \) = the observed phenotypic mean for genotype \( i \) in replicate \( j \), row \( k \) and column \( l \), \( \mu \) = general mean, \( G_i \) = effect of the \( i \)th genotype, \( R_j \) = effect of the \( j \)th replicate, \( W_k \) = effect of the \( k \)th row and \( C_l \) = effect of the \( l \)th column and \( e_{ijkl} \) = residual error.

Row, column and genotype were treated as fixed effects and blocks and replicates considered as random effects. Adjusted entry means and corresponding standard errors of genotypes from each environment were analysed in the second step to obtain genotypic means across environments by using the following mixed model:

\[ Y_{ij} = \mu + G_i + E_j + G\times E_{ij} + e_{ij}, \]

where \( Y_{ij} \) = the observed phenotypic mean for genotype \( i \) in environment \( j \), \( \mu \) = general mean, \( G_i \) = effect of the \( i \)th genotype, \( E_j \) = effect of the \( j \)th environment, \( G\times E_{ij} \) = effect of genotype–environment interaction and \( e_{ij} \) = residual error.

A weighting factor of one divided by the squared standard error of each mean from the first step was used, so the residual variance was set to one, according to method 3 proposed by Möhring and Piepho (2009). BLUEs were estimated across environments assuming fixed effects for the genotypes and environments. Variance components were determined by the restricted maximum likelihood (REML) method. Genotype, environment and genotype–environment interaction were treated as random. Significance of variance components was determined using the likelihood ratio test.

Broad sense (entry-mean) heritability (\( H^2 \)) was estimated based on the generalized method proposed by Cullis, Smith, and Coombes (2006) as follows:

\[ H^2 = 1 - \frac{\overline{\text{vBLUP}}}{2\sigma^2_y}, \]

where \( \overline{\text{vBLUP}} \) is the squared average standard error of difference of the BLUPs and \( \sigma^2_y \) = genotypic variance. Phenotypic association between FHB and heading ratings as well as FHB and plant height were estimated by Pearson correlation tests using the "cor.test" function in the R statistical software (R Core Team, 2018).
2.3 | Molecular data analysis

Fresh leaves were collected from the 465 rye lines at four-leaf stage, and genotyping was performed by a commercial laboratory by Illumina Technology (Illumina, San Diego) with a 15 k in-house single nucleotide polymorphism (SNP) chip yielding 8,942 polymorphic SNPs. We checked the quality of the markers using the "check.marker ( )" function in the R package GenABEL (Aulchenko et al., 2018) and removed SNPs which showed more than 20% missing genotypes or had a minor allele frequency <5% from further analyses. In the end, 7,728 SNPs were available for the genome-wide association mapping across the 465 genotypes. Only 2,719 SNPs in our marker data overlapped with the markers in an already published linkage map (Bauer et al., 2017). To increase the number of mapped markers for our study, we established a consensus map including the 2,719 already mapped SNPs from Bauer et al. (2017) and unpublished maps created from nine bi-parental rye populations in our working group with MergeMap (Wu et al., 2008). All markers were used for the analysis. To perform genome-wide prediction, missing values in the marker data were imputed using the software Beagle version 5.0 (Browning et al., 2018).

A genome-wide association scan was performed to analyse marker–trait associations for FHB resistance, heading stage and plant height using the R package GenABEL (Aulchenko et al., 2018). Principal component analysis based on the distance matrix of genomic kinship showed two major clusters in the association panel (Figure 1). Therefore, both the genomic kinship matrix (K) and the genomic kinship (K) explained by the identified QTL was estimated as:

$$
\rho_G = \frac{R^2_{adj}}{H^2},
$$

where $H^2$ is the heritability of the trait, and $R^2_{adj}$ is the adjusted $R^2$ (Utz et al., 2000). The adjusted $R^2$ was obtained by fitting all significant SNPs simultaneously in a linear model in a decreasing order of the strength of their association with the trait, that is they were fitted beginning with the SNP that had the lowest P-value (Würschum et al., 2015). The linear model can be represented as:

$$
Y = m_i + m_j + m_k + m_\ldots + e,
$$

where $y$ is the calculated phenotypic mean, $y_i$, $y_j$, and $y_k$ are the marker effects where the P-value of $m_i < m_j < m_k$ (i.e. in a decreasing order of the strength of their association with $y$), and $e$ is the residual error.

The $\rho_G$ of individual QTL was estimated by using the sums of squares obtained from the analysis of variance of the linear model including the significant SNPs (Würschum et al., 2015), that is:

$$
\rho_G = (SS_m/SS_{total}) / h^2 \times 100%.
$$

FIGURE 1 Principal component (PC) analysis of 465 S_{4} lines of winter rye with the first and second PCs and the percentage of variation explained in brackets. Green filled circles = rye lines selected for Fusarium head blight (FHB) severity (n = 372), black filled rectangles = rye lines unselected for FHB severity (n = 93) by applying the function "r2fast( )" which was based on a slightly modified code of Hao, Di, and Cawley (2007). A pair of SNPs having $r^2$ values >.60 were considered as being in LD. In addition, we used the LD values to correct all significantly associated SNPs for collinearity, that is to determine which of the significant markers likely identify the same putative QTL. The total proportion of genotypic variance ($\nu_G$) explained by the identified QTL was estimated as:
where $SS_m$ refers to the sums of squares of the individual SNP and $SS_{\text{total}}$ refers to the total sums of squares. In addition, we calculated the additive effect ($\beta$-effect) of each significant SNP by fitting only one SNP at a time in a linear model. Furthermore, a genomic prediction (GP) approach was applied to exploit the additive effects of small-effect QTL which cannot be identified in the GWA mapping. The GP was conducted by ridge regression-BLUP (RR-BLUP) with the R package "rrBLUP" (Endelman, 2011; Endelman & Jannink, 2012) using imputed SNPs, including both mapped and unmapped markers. A weighted ridge regression-BLUP (wRR-BLUP) was also performed, where the significant SNPs from the GWA mapping, explaining more than 5% of the genotypic variance, were treated as fixed effect in the GP model (Spindel et al., 2016; Zhao, Mette, Gowda, Longin, & Reif, 2014). Additionally, we compared the predictive ability of MAS and GP. For each trait, the significant SNPs explaining >5% of the genotypic variance in the GWA mapping were used for MAS and all genome-wide SNPs were used for GP (Miedaner et al., 2017). Fivefold cross-validation was done for both MAS and GP by dividing the 465 lines into two sets, (a) estimation set consisting of 80% of the genotypes and (b) prediction set consisting of the remaining 20% of the genotypes (Liu et al., 2013; Würschum, Abel, & Zhao, 2014; Würschum & Kraft, 2014). Resampling of the lines was repeated 1,000 times. The predictive ability was calculated as the correlation coefficient between the predicted and observed trait values of 20% of the genotypes based on the effect estimates from the 80% of the genotypes.

For MAS, we used the model:

$$Y = X_\beta + e$$

and for the wRR-BLUP approach the model:

$$Y = X_\beta + Zu + e,$$

where $Y$ = the vector of phenotypic observation, $\beta$ = the vector of fixed marker effects, $u$ = the vector of random marker effects, $X$ and $Z$ = the design matrices coded as −1, 0, 1 relating to $\beta$ and $u$, respectively, in $Y$ and $e$ = the residual error. For RR-BLUP, the same model of wRR-BLUP was used by omitting the factor $X_\beta$. We assumed additive effects of markers.

3 | RESULTS

3.1 | Phenotypic variation among rye genotypes

The *F. culmorum* isolate FC46 caused FHB infection in all three environments with a slightly higher infection level occurring in HOH18 (Table 1). Heading was earlier in WUL17 compared to HOH18 and HOH18. The highest mean plant height was observed in WUL17. In the combined analysis across environments, wide ranges of mean FHB severity, heading stage and plant height were observed among the rye lines. Both the genotypic variance and the genotype-by-environment interaction variance were significantly different from zero for all recorded traits ($p \leq .001$). Broad sense heritabilities were high throughout, being 0.80 for FHB severity and heading stage and 0.89 for plant height. The correlation between FHB severity and heading stage was low and not significant ($r = -0.05$). Between FHB and plant height, a moderate correlation ($r = -.52$, $p \leq .001$) was found that was mainly triggered by some very short susceptible lines (Figure 1). For the selected lines, the correlation was considerably lower, although significant ($r = -0.22$, $p \leq .001$). They were, on average, 13.52 cm taller than the non-selected lines, but also 11.9% more FHB resistant (Table S2). Heading stage, by contrast, showed no substantial difference between the selected and the unselected subpopulations (5.37 vs. 4.95, Table S2).

3.2 | Genome-wide association mapping and genomics-assisted selection

Principal coordinate (PC) analysis showed two major population substructures reflecting the genetic background of the lines used in this study (Figure 1). The first and second PC explained 60.6% and 15.2% of the variation, respectively. The larger group comprised of 372 S1 lines selected for FHB resistance in a recurrent selection breeding programme across five cycles, and the smaller group comprised of 93 S1 lines not previously selected for FHB resistance. As a result, we used the first PC and the K matrix to correct for population substructure and familial relatedness, respectively.

The GWA scan revealed ten SNP–trait associations for FHB severity on chromosomes 1R, 3R, 5R and 6R that exceeded the Bonferroni-corrected significance threshold with a P-value of 8.13E-06 (Figure 2). At the exploratory threshold ($p < .0001$), significant associations for FHB severity were found on all chromosomes except for chromosome 7R (Table 2, Figure 2). In total, 15 putative QTL were identified with this threshold for FHB severity which jointly explained 74% of the genotypic variance. Each QTL explained between 0.22% and 33.12% of the genotypic variance, five explaining more than 5% $\rho_G$ (Table 2). The SNP Contig1930 significantly associated with the major QTL on chromosome 1R, explaining about 33% of the genotypic variance for FHB severity, was not co-localizing with any of the other significant SNPs at the selected LD threshold ($r^2 > .60$). Additive effects of the FHB QTL ranged from -4.41 to 7.76. The FHB resistance QTL, that explained more than 5% of the genotypic variation, had additive effects except for one locus (isotig15981) that was dominant for the resistant allele and another (isotig14873) that was dominant for the susceptible allele. Generally, the heterozygotes showed intermediate resistance to FHB (Figure 3).

Three SNP–trait associations were identified for heading stage (1R, 2R, 5R) at the Bonferroni-corrected significance threshold (p-value = 8.13E-06, Figure S1). At the exploratory significant threshold, we found significantly associated SNPs on all chromosomes except chromosome 6R (Table 2, Figure S1). Overall, 11 QTL were detected for heading stage and jointly explained 26% of the genotypic variances, with the $\rho_G$ of single QTL ranging between 0.01% and 12.02%. Two SNPs explained more than 5% $\rho_G$ for this trait.
Additive effects of the QTL ranged from −0.62 to 0.44 for heading stage. SNPs isotig12834 and isotig32608 showed an additive and a dominance effect, respectively, for heading time.

For plant height, one significant SNP was found at the Bonferroni-corrected threshold on chromosome 2R. Three significant associations exceeding the exploratory threshold were identified on chromosomes 2R, 3R and 7R, with several significantly associated markers located on chromosome 3R (Table 2, Figure S1). In total, 8 putative QTL were identified for plant height with the exploratory significance threshold. These QTL jointly explained 14% of the genotypic variance and individually between 0.06% and 5.42% (Table 2). Only two SNPs explained slightly more than 5% \( \sigma^2_a \). Additive effects of the QTL for plant height ranged from −2.40 to 3.01. Both loci, isotig24773 and isotig23589, showed dominant allelic effects for mean plant height (Figure 3).

No common QTL were found for FHB severity and heading stage. The significantly associated SNP isotig18865 on chromosome 3R was common to heading stage and plant height. There was a high LD between SNP isotig 15,081 (FHB QTL) and SNP isotig24773 (plant height QTL) on chromosome 3R (\( r^2 = 0.84 \)).

Because of the apparent presence of additively inherited minor-effect QTL contributing to the total genotypic variance of FHB severity, heading stage and plant height, we compared the potential of MAS and GS in a fivefold cross-validation procedure (Figure 4). GS was clearly superior over MAS for all three traits. For FHB severity, the prediction ability of MAS approach was 44% less than the prediction ability of the two genomic prediction approaches. Similarly, genome-wide predictions were 42% and 63% higher than MAS for heading stage and plant height, respectively (Figure 4). A weighted GS approach, incorporating the identified medium- to large-effect QTL as fixed effects, did not yield a higher mean prediction ability than the non-weighted GS approach. Cross-validated prediction ability of the RR-BLUP procedure was 0.86 illustrated by a narrow correlation between observed and predicted FHB severities (Figure S2).

### Discussion

Knowledge about the genetic architecture of FHB resistance is vital for genomics-assisted resistance breeding, but to date nothing...
is known about QTL that confer this resistance in rye. The aim of this study was therefore to (a) perform the first GWA mapping to discover QTL that control FHB resistance in rye and (b) evaluate the potential of genomics-assisted selection for FHB resistance breeding.

4.1 Construction of the GWA population

For European wheat, many QTL with small effects were reported to govern FHB resistance, as known for other quantitative traits (Lynch & Walsh, 1998). In the cross-pollinating rye, we expect many alleles
per locus (Newell & Butler, 2013). Therefore, a bi-parental mapping population exploring only the effects of the two parental alleles of the population is not adequate to identify QTL for FHB. Moreover, performing a GWAS in unselected populations may not be able to identify effectively QTL present at low frequency. Therefore, we followed here the strategy to enrich our GWA population for FHB resistance by several cycles of recurrent selection. To demonstrate the genetic progress, we added 93 unselected S$_1$ lines to the 372 selected lines. On average, the selected lines showed an 11.9% higher resistance against FHB than the unselected lines (Table S2). Thus, this strategy can be expected to improve the chances of identifying QTL and superior combinations of resistance QTL alleles. Because all tested lines belonged to the pollinator gene pool, our population can serve as a training population for this heterotic group. Whether it can also be used for the opposite pool requires further research.

### 4.2 Phenotypic variation for FHB resistance and agronomic traits

The rye lines analysed in this study showed a high variation for FHB resistance, heading stage and plant height (Table 1). The different genetic background of the genotypes, with selected and unselected lines, might have contributed to the wide range of FHB severity.
observed in our study. It should be noted, however, that we used only elite lines from a commercial hybrid rye breeding programme without introgression of genetic resources or foreign material. There was a clear molecular distinction between the selected and the unselected lines, although they belong to the same heterotic group (Figure 1), showing the benefits of recurrent selection in combining favourable alleles within breeding populations. Although the main selection trait was FHB resistance, an indirect selection for plant height might have occurred as illustrated by the moderate negative association between plant height and FHB resistance ($r = -0.52, p \leq .001$; Figure 5). Several very short and susceptible unselected lines, however, mainly caused this correlation. If all lines <110 cm were omitted, the correlation was much lower ($r = -0.27, p \leq .001$). Obviously, in the long-strawed rye the correlation between FHB severity and plant height occurs only when short entries are included. Heading stage varied among the genotypes but did not correlate with FHB severity. This result corresponds to a previous report in triticale (Miedaner, Kalih, Großmann, & Maurer, 2016) and can partly be attributed to the synchronization of the plant developmental stage with the date of inoculation, which ensured that each genotype was inoculated at the optimal growth stage (mid flowering), thus reducing the confounding effects of heading stage on disease severity. The finding here indicated that selection for FHB resistance did not affect the maturity period of the rye genotypes, hence simultaneous selection for high FHB resistance and earliness should be possible in this breeding material.

Entry-mean heritabilities were high for all traits, which is in part attributable to the high genotypic variation present in this panel, and similar to the values reported in previous studies in rye (Gaikpa et al., 2019; Hackauf et al., 2017; Miedaner et al., 2012; Wang et al., 2014). Genotype-by-environment interaction variances were significant for all traits, reflecting the importance of phenotyping across several environments for quantitative traits (Fowler, N’Diaye, Laudenci-Chingcuanco, & Pozniak, 2016; Prat et al., 2017; Würschum et al., 2015).

### 4.3 GWA mapping identified QTL for FHB resistance in rye

Generally, cross-pollinating, highly heterozygous rye cultivars are more resistant to FHB than self-pollinating homogeneous triticale, durum wheat and bread wheat cultivars (Arseniuk et al., 1999; Gaikpa et al., 2019; Langevin, Eudes, & Comeau, 2004). Therefore, analysis of the genetic mechanisms underlying the increased resistance in rye is of vital importance. For the first time, we performed GWAS to elucidate the genomic basis of FHB resistance in winter rye and identified 15 genomic regions that are associated with FHB resistance (Table 2). These 15 QTL jointly explained a rather high proportion of the genotypic variance (74%), which may in part be due to the accumulation of these QTL in the recurrent selection.

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**FIGURE 4** Box plots showing the comparison of prediction abilities of marker-assisted selection (MAS), ridge regression-BLUP (RR-BLUP), and weighted ridge regression-BLUP (wRR-BLUP) for Fusarium head blight (FHB) severity, heading stage (HS) and plant height (PH) in winter rye.

**FIGURE 5** Association between Fusarium head blight severity (%) and plant height (cm) for 372 selected (green circles) and 93 unselected (black triangles) S$_1$ rye lines.
4.4 | QTL for heading stage and plant height in rye

Heading stage and plant height are important agronomic traits which might confer passive resistance to FHB in small-grain cereals (Mesterházy, 1995). Therefore, we took these traits into account in our GWAS to investigate their possible co-localization with FHB resistance QTL.

For heading stage, we found 11 QTL, but none of these QTL co-localized with the QTL for FHB severity, which is in line with the lack of phenotypic correlation between these traits. Here, only two of the significantly associated SNPs explained more than 5% of the $\rho_G$, illustrating the high genetic complexity of the trait. We observed both additive and dominance effects of the QTL alleles for heading stage. Recently, Hackauf et al. (2017) reported 7 QTL that jointly explained 85% of $\rho_G$ for heading time across 272 F$_2$ : 3 rye lines derived from a bi-parental population not preselected for FHB resistance. In the present study, by contrast, we used material that was purposefully selected for FHB resistance, but not directly selected for heading stage, which might partly account for the lower $\rho_G$ explained by the 11 QTL for heading stage compared to the QTL identified for FHB resistance.

In triticale, Kalih et al. (2015) reported QTL on chromosomes 4R, 5R, 6R, 7R, explaining 4.55% - 39.82% of $\rho_G$ for heading across four populations in triticale. Similarly, we identified significant marker-trait associations for heading stage on all chromosomes except chromosome 6R. Interestingly, the SNP with the highest genotypic effect in our study (isotig12834) is located on chromosome 5R where two large-effect QTL ($\rho_G = 15.5, 39.8$) were previously reported in triticale (Kalih et al., 2015).

The proportion of genotypic variance jointly explained by the 8 QTL for plant height was lower than the variation explained for FHB resistance and heading stage. Similar to heading stage, two QTL explained more than 5% of the $\rho_G$ for plant height. Non-additive genetic factors, such as QTL × QTL and QTL × environment interactions, might partly account for the remaining unexplained total genotypic variance. Plant height was controlled by dominant alleles for the two most prominent QTL (Figure 3). SNP isotig15081 on chromosome 3, which was significantly associated with FHB resistance, was in LD with the SNP isotig24773 associated with plant height on chromosome 3 ($r^2 = .84$). Interestingly, these SNPs had medium effects on their respective traits and may partly explain the negative phenotypic correlation observed between FHB severity and plant height. No major dwarfing gene segregated in this population as shown by our GWAS results for plant height, where only minor QTL were found (Table 2). Generally, major genes controlling plant height are not routinely used in rye breeding to date and the height seems to be controlled by a plethora of minor QTL as reported previously in rye (Miedaner et al., 2018; Miedaner, Müller, Piepho, & Falke, 2011) and triticale (Galiano-Carneiro et al., 2019; Kalih et al., 2015).

4.5 | The potential of marker-assisted and genomic prediction in winter rye

For all traits analysed, prediction abilities of both RR-BLUP and wRR-BLUP were by far higher than predictions based on marker-assisted selection (MAS, Figure 4) that considers only QTL with medium to major effects. Overall, the mean prediction abilities of MAS ranged from 29% to 48%, while the mean prediction abilities of the genome-wide approach ranged from 72% to 86% for the three traits (Figure 4). This implies that improvement of FHB resistance, heading stage and plant height by MAS will be slower compared to genomic prediction approaches. This result is in accordance with previous studies reporting higher prediction abilities for genomic prediction than for MAS in triticale (Galiano-Carneiro et al., 2019), bread wheat (Mirdita et al., 2015; Rutkoski et al., 2012) and durum wheat (Miedaner et al., 2019, 2017). However, in the present study, we observed a higher prediction ability for FHB resistance than reported in the earlier studies and even slightly higher than for heading stage and plant height. This is likely due to the continuous selection for FHB resistance, resulting in increased resistance allele frequencies for the QTL underlying this trait (Figure 2). Thus, recurrent selection breeding schemes assisted by genomic prediction appear promising to improve rye resistance against FHB. It is worth to note that our prediction accuracies might be overestimated to some extent, because the training (80% of the lines) and prediction (20%) set were from the same population and have been tested in the same environments.
5 | CONCLUSIONS

Resistance towards FHB might become a trait of increasing importance in hybrid rye breeding. The observed phenotypic variation in elite germplasm is an important and promising prerequisite to gain breeding progress with respect to FHB resistance in rye breeding programmes. There is great potential to improve FHB resistance by genome-based approaches. For the first time, GWA mapping identified several significant marker–trait associations for FHB severity in winter rye of which two can be classified as major QTL. These are candidates for further analyses of FHB resistance to increase our understanding in resistance mechanisms in rye. No co-localization of QTL for FHB and plant height or rather heading stage was observed, which mirrors the moderate correlation between FHB and plant height. Genomic prediction yielded similar high prediction abilities with and without weighted data. Genomics-assisted recurrent selection appears as a promising tool to accelerate breeding for complex disease resistances in rye. These results encourage further research to study FHB resistance in rye hybrids. One opportunity in rye breeding is the reduced level of mycotoxins in the harvest compared to wheat that should facilitate the increase of rye productivity and consumer protection.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

AUTHOR CONTRIBUTIONS

T. Miedaner and F.J. Fromme designed the research and coordinated the experiments, S. Koch and D.S. Gaikpa did the trial organization and phenotyping at Hohenheim in 2017 and 2018, respectively. D.S. Gaikpa performed all statistical analyses and wrote the manuscript. T. Würschum helped with statistical advice, edited and improved the manuscript together with T. Miedaner. D. Siekmann revised the manuscript critically. All authors read and approved the final version of the manuscript.

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