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vinay kumar reddy nannuru

Norwegian University of Life Sciences Faculty of Veterinary and Biosciences: Norges miljø- og biovitenskapelige universitet Veterinaerhøgskolen <https://orcid.org/0000-0002-5776-8235>

Susanne S. Windju

Graminor AS

Tatiana Belova

UiO Det medisinske fakultet: Universitetet i Oslo Det medisinske fakultet

Jon Arne Dieseth

Graminor AS

Muath Alsheikh

Graminor AS

Yanhong Dong

University of Minnesota

Curt A. McCartney

Morden Research and Development Centre

Maria Antonia Henriques

Morden Research and Development Centre

Hermann Buerstmayr

BOKU: Universität für Bodenkultur Wien

Sebastian Michel

BOKU: Universität für Bodenkultur Wien

Theodorus H.E. Meuwissen

Norwegian University of Life Sciences Faculty of Veterinary and Biosciences: Norges miljø- og biovitenskapelige universitet Veterinaerhøgskolen

Morten Lillemo (✉ morten.lillemo@nmbu.no)

Norwegian University of Life Sciences Faculty of Veterinary and Biosciences: Norges miljø- og biovitenskapelige universitet Veterinaerhøgskolen <https://orcid.org/0000-0002-8594-8794>

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Genetic Architecture of Fusarium Head Blight Disease Resistance and Associated Traits in Nordic Spring Wheat

Vinay Kumar Reddy Nannuru¹, Susanne S. Windju², Tatiana Belova³, Jon Arne Dieseth², Muath Alsheikh², Yanhong Dong⁴, Curt A. McCartney⁵, Maria Antonia Henriques⁵, Hermann Buerstmayr⁶, Sebastian Michel⁶, Theodorus H.E. Meuwissen⁷, Morten Lillemo^{1*}

¹ Department of Plant Sciences, Norwegian University of Life Sciences, Ås, 1432 Norway

² Graminor AS, Ridabu, 2322 Norway

³ Centre for Molecular Medicine Norway, Faculty of Medicine, University of Oslo, Blindern, 0318 Norway

⁴ Department of Plant Pathology, University of Minnesota, St. Paul, Minnesota, 55108 USA

⁵ Morden Research and Development Centre, Agriculture and Agri-Food Canada, Morden, Manitoba, Canada

⁶ Institute of Biotechnology in Plant Production, Department of Agrobiotechnology, University of Natural Resources and Life Sciences Vienna, Tulln, 3430 Austria

⁷ Department of Animal and Aquacultural Sciences, Norwegian University of Life Sciences, Ås, 1432 Norway

* Corresponding author: morten.lillemo@nmbu.no; Orcid-ID – 0000-0002-8594-8794

Abstract

Key message – This study identified a significant number of QTL that are associated with FHB disease resistance in NMBU spring wheat panel by conducting genome-wide association study.

Fusarium head blight (FHB) is a widely known devastating disease of wheat caused by *Fusarium graminearum* and other *Fusarium* species. FHB resistance is quantitative, highly complex and divided into several resistance types. Quantitative trait loci (QTL) that are effective against several of the resistance types give valuable contributions to resistance breeding. A spring wheat panel of 300 cultivars and breeding lines of Nordic and exotic origins was tested in artificially inoculated field trials and subjected to visual FHB assessment in the years 2013 to 2015, 2019 and 2020. Deoxynivalenol (DON) content was measured on harvested grain samples and anther extrusion (AE) was assessed in separate trials. Principal component analysis based on 35K and 25K SNP arrays revealed the existence of two subgroups, dividing the panel into European and exotic lines. We employed a genome-wide association study to detect QTL associated with FHB traits and identify marker-trait associations (MTAs) that consistently influenced FHB resistance. Several MTAs were found to be significant for the FHB resistance. Of importance, haplotype analysis revealed a highly significant and consisted QTL on 7A, *Qfhb.nmbu.7A.2*, which was further validated on an independent set of breeding lines. Breeder-friendly KASP markers were developed for this QTL that can be used in marker-assisted selection. The lines in the wheat panel harboured from zero to five resistance alleles, and allele stacking showed that resistance can be significantly increased by combining several

of these resistance alleles. This information enhances breeders' possibilities for genomic prediction and to breed cultivars with improved FHB resistance.

Keywords:

Wheat, fusarium head blight, disease resistance, genome-wide association study, quantitative trait loci

Statements and Declarations:

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Conflicts of interest

The authors declare no conflict of interest.

Availability of data and other material

All the data from this study i.e., both phenotypic and genotypic datasets are included with manuscript submission and other relevant information.

Code Availability

The scripts and software used for data analyses in this study are available upon request.

Author contributions

VKRN SW TB JAD MA YD CM MAH HB SM TM ML.

Funding acquisition, project administration and management, MA, ML;

Data curation, VKRN, TB, ML;

Data analysis and visualization, VKRN;

Field testing at Morden in Canada, MAH, CM;

Field testing in Norway, SW, ML, JAD;

Field testing at Tulln in Austria, SM, HB;

DON analysis, YD;

Draft writing, SW, VKRN;

Draft writing-review and editing, VKRN, SW, TB, SM, TM, ML;

Supervision, TM, ML.

Abbreviations:

AE	Anther Extrusion
DH	Days to Heading
DA	Days to Anthesis
DON	Deoxynivalenol
DONcPHDH	DON corrected for Plant Height and Days to Heading
FHB	Fusarium Head Blight
FHBcPHDH	FHB disease severity corrected for Plant Height and Days to Heading
Mbp.	Mega base pairs
MTA	Marker-Trait Associations
PCA	Principal Component Analysis
PH	Plant Height
tDON	Transformed DON content

Introduction

Wheat (*Triticum aestivum* L.) is one of the most widely cultivated crops with around 214 million hectares and a global production of 734 million tonnes in 2018 (FAOSTAT 2018). It is also the most cultivated cereal crop in Europe (FAOSTAT 2018). To meet the increasing demand for food and feed, breeders are continuously developing novel, more efficient germplasm with improved yield, quality, and disease resistance. To lower the risk of crop losses from plant diseases and to reduce the dependency on pesticides and their environmental impact, new cultivars are needed that combine excellent disease resistance with productivity and end-use quality. Fusarium head blight (FHB) is a devastating fungal disease affecting the wheat production worldwide. FHB can cause severe yield losses due to failed kernel development or because infected kernels are shrivelled, discoloured and low in test weight (McMullen et al. 2012). *Fusarium graminearum* which produces the mycotoxin deoxynivalenol (DON) is found to be the most causal agent of FHB in wheat (McMullen et al. 1997; Goswami and Kistler 2004; Hofgaard et al. 2016). Mycotoxins, such as DON may cause severe problems and is a threat to both animals and humans, reaching from feed refusal and poor weight gain in animals to immunological problems in humans (McMullen et al. 2012). Threshold levels of DON concentration set by the European Union range between 200 µg/kg for processed cereal based foods and baby foods for infants and young children to 750 µg/kg for cereals intended for direct human consumption and cereal flour. For unprocessed cereals the threshold has been set to 1250 µg/kg (European Commission 2006).

Breeding for disease resistance is the most cost-effective method to control this disease (Buerstmayr et al. 2002). To develop resistant cultivars, proper understanding of resistance mechanisms is required. Resistance to FHB has been divided into active and passive resistance mechanisms. The active mechanisms are further divided into five different types; Type I: Resistance to invasion (initial infection), Type II: Resistance to pathogen spread in infected tissue, Type III: Resistance to toxin accumulation (Miller et al. 1985), Type IV: Resistance to kernel infection, and Type V: Tolerance (Schroeder and Christensen 1963; Miller et al. 1985; David Miller and Arnison 1986; Mesterhazy 1989, 1995; Mesterházy et al. 1999; Buerstmayr and Lemmens 2015). The passive resistance mechanisms are divided into four different types: Type I: plant height, Type II: presence/absence of awns, where the presence of awns increases the disease severity and otherwise, Type III: spikelet density within the head, Type IV: escape, flowering in boot stage and the ability of florets to extrude anthers (Mesterhazy 1995). The different resistance mechanisms are quantitative (complex) in nature and highly influenced by the environment, making breeding for resistance by traditional ways difficult. In addition, due to incomplete understanding of factors that influence the disease development and difficulty in efficient application, use of fungicides for controlling FHB is limited (McMullen et al. 1997; Goswami and Kistler 2004).

New marker technologies, enabling quantitative trait loci (QTL) detection, association mapping and subsequently marker detection, have the potential to increase the efficacy of resistance breeding for FHB and in addition dissect and enhance the understanding of the genetic basis of the complex resistance mechanisms.

Many QTL mapping studies for FHB resistance in wheat have been performed over the past two decades, and with the single nucleotide polymorphism (SNP) marker technology, more markers are available, and it is possible to map resistance QTL more precisely in linkage maps and physical maps. These studies have been performed using different bi-parental populations and have revealed chromosomal regions harbouring FHB resistance loci. More than a decade ago, Buerstmayr et al. (2009) and Liu et al (2009) compared and assembled the information from several of these studies in maps displaying interesting chromosomal regions harbouring FHB resistance QTL, which can be further tested and potentially utilised in resistance breeding. The possibility to map the resistance QTL also led scientists more precisely to get a closer understanding of genes and mechanisms underlying the resistance trait. The most researched QTL for FHB resistance in wheat is *Fhb1* on chromosome arm 3BS, which is derived from the Chinese wheat cultivar Sumai 3 (Waldron et al. 1999) and has been detected in many mapping studies (Buerstmayr et al. 2009; Liu et al. 2009). It confers mainly Type II resistance (Bai et al. 2018) and can reduce disease severities by 20–50% (Bernardo et al. 2012; Jin et al. 2013). The gene was recently cloned in two independent studies and shown to encode a histidine-rich calcium-binding protein (Li et al. 2019; Su et al. 2019).

In recent years, genome-wide association studies (GWAS) for detection of FHB resistant QTL have been conducted. The benefit of these studies is the ability to capture historic recombination events and utilize collections with a wide genetic background. This increases the possibility for breeders to detect interesting QTL for resistance directly in breeding relevant germplasm and enable a targeted incorporation of resistance QTL into their breeding programs. Some of these studies have demonstrated both plant height (PH) and anther extrusion (AE) to be negatively correlated to FHB. Skinnes et al. (2010) detected a consistent and negative correlation between AE and FHB disease severity, and AE and DON content in the Arina x NK93604 mapping population. A study by Lu et al. (2013) performed on the mapping population Shanghai3/Catbird x Naxos confirmed the negative correlation between AE and FHB, and the QTL analysis further confirmed the relationship; eight out of ten AE QTL detected in the study coincided with FHB severity. Kubo et al. (2013) demonstrated that partially extruded anthers were a good source for FHB infection, while rapid extrusion and ejection of the anthers contributed to the avoidance of infection by FHB. A meta-analysis performed by Mao et al. (2010) confirmed a negative association between PH and FHB, where coincident QTL for PH and FHB were detected on chromosomes 2D, 3A, 4B, 4D and 7A.

QTL for both FHB disease severity and DON content can serve as valuable sources for disease resistance breeding in wheat. SNP markers closely linked to the resistance QTL could be further tested and used in resistance-breeding for FHB and DON resistance.

The aim of this study was to identify QTL for both FHB disease severity and DON in a diverse panel of 300 Nordic spring wheat lines by GWAS, to study the consistency of these QTL across environments and their association with AE. Then, to further validate these QTL to observe the potential enhancement of genomic prediction models for FHB disease resistance.

Material and Methods

Plant material

The germplasm used in the study was a collection of 300 hexaploid spring wheat accessions including 186 lines from Norway, 40 lines from Sweden, 37 from CIMMYT and some additional accessions from Australia, Brazil, Canada, China, Czech Republic, Denmark, Finland, France, Germany, Netherlands, Poland, Russia, Slovakia, South Africa, Switzerland, United Kingdom and USA. This panel is hereafter referred to as the NMBU spring wheat panel (Online resource 1).

As a validation panel, an independent set of 358 new breeding lines from the commercial spring wheat breeding program of Graminor was used to validate QTL identified in the NMBU spring wheat panel. This set of lines is hereafter referred to as the validation panel (Online resource 1).

Fusarium field design, inoculation, and scoring

Field trials in Norway

The NMBU spring wheat panel was planted in α -lattice designs with two replicates at two locations in Norway: Vollebekk research farm at the Norwegian University of Life Sciences, Ås (59°N, 90 m above sea level) in 2013, 2014, 2015, 2019, and Staur research farm close to Hamar (60°N, 153 m above sea level) in 2014 and 2015. To ensure a high Fusarium disease pressure in these trials, spawn inoculation of the fields was performed. Grain spawn, oat kernels infected with *F. graminearum*, was prepared, and distributed in the field based on a modified protocol from Dr. Bernd Rodemann, Julius Kuhn Institute, Braunschweig using a mixture of four isolates as described by Lu et al. (2013) and Tekle et al. (2018). Mist irrigation was applied every 10-15 minutes per hour from 19:00 to 23:00 every evening in the period starting from spawn inoculation at the booting stage to 3-4 weeks after flowering to ensure high disease pressure.

FHB disease assessments were performed at the beginning of maturity, when the stems of the plants in the individual plots just started to turn yellow, but the heads were still green. At Staur research farm five random heads at three different positions in each plot were evaluated. At Vollebekk research station ten random heads at two different positions in each plot were evaluated. The evaluation was performed visually by counting the number of *Fusarium*-infected spikelets and dividing this by the total number of spikelets giving a percentage of infected spikelets in each plot. The field plots were harvested with a plot combine, and the level of DON in each sample was evaluated by GC-MS at the University of Minnesota mycotoxin diagnostic lab (Mirocha et al. 1998). Days to heading (DH) was scored in the same field as the Fusarium disease evaluation in every testing environment at the time when 50% of the heads in the plot had emerged. Plant height (PH) was measured in centimeter in the fusarium nurseries at Vollebekk (in 2013, 2014, 2015, and 2019) and Staur (in 2014 and 2015).

Anther extrusion (AE) was evaluated at Vollebekk in both the greenhouse (2013) and small field plots (2013) and in hill plots (2014, 2019 and 2020), and at Staur in hill plots (2014 and 2015) in different nurseries adjacent to the fusarium disease assessment field, avoiding the confounding effects of mist irrigation on the AE assessment. AE was assessed visually by a scale from 0-9, where 0 represented no anther extrusion and 9 full anther extrusion as described by Skinnnes et al. (2010).

Field trial in Canada

A field trial was also conducted in Canada at Morden, Manitoba in the year 2019. The lines were planted in a single 1 m long row using a six-row cassette Row XL, Wintersteiger planter. *F. graminearum* corn kernel inoculum was used. The inoculum was prepared using four *F. graminearum* isolates (HSW-15-39 (3-ADON), HSW-15-87 (3-ADON), HSW-15-27 (15-ADON), and HSW-15-57 (15-ADON) from Dr. Henriquez's culture collection, modifying the protocol of Gilbert and Woods (2006). Each isolate was inoculated in individual pans containing sterile corn and incubated for one month. The inoculum was dispersed at a rate of 8 g per row at 4-5 leaf stage. The inoculum application was followed by irrigation three times a week (Monday, Wednesday, and Friday) using Cadman Irrigations travellers with Briggs booms. Infected rows were rated around 21 days after anthesis for incidence and severity. The incidence was calculated using a scale from 0-100% (percent of heads with infection per plot), while the severity (0-100 % scale) is the average amount of infection on infected heads per plot. The FHB index is the product of Incidence $\times$ Severity divided by 100. Plant height was measured in cm from ground to top of heads excluding the awns. After harvest (using low wind speed on the combine to retain lightweight Fusarium-damaged kernels (FDK), samples were cleaned and a minimum of 10 g of well-mixed seed were ground to flour. From each replicate, 1,000 g of flour was used for DON (ppm) analysis using ELISA tests.

Field trial in Austria

A subset of 200 lines from the NMBU spring wheat panel was tested in a field trial conducted at the experimental station of the Department of Agrobiotechnology, Tulln in 2020 (9°N, 177 m above sea level). The trial was laid out as a randomized complete block design and inoculated with the DON-producing *Fusarium culmorum* isolate Fc91015. Spray inoculations of each individual plot were performed with a backpack sprayer when 50% of the plants within this plot reached the flowering stage. FHB assessments and plant height was measured in a similar manner as it was in the other field trials. Anther retention was recorded with a scale of 0-20, where four central florets on five normal main spikes per plot were manually opened and the number of florets with at least one anther inside or trapped between palea and lemma were counted. This was converted to anther extrusion with the following formula: Anther Extrusion = $[1 - (\text{Anther Retention } 0-20)/20] \times 9$. Days to anthesis (DA) was recorded in this field trial, and was used instead of DH, to correct the FHB and DON measurements for confounding effects of earliness.

Field trial of the validation panel

The validation panel of new breeding lines was evaluated in a spawn-inoculated disease nursery with two replicates at Vollebekk research station in 2020, following the same methodology as described for the NMBU spring wheat panel above. Data was obtained for the traits DH, PH,

FHB and DON. In addition, AE was assessed in an adjacent hill plot nursery with two replicates following the same methodology as described above for the NMBU spring wheat panel.

The validation panel was also evaluated with two replicates at the experimental station of the Department of Agrobiotechnology, Tulln in 2021 in Austria. Data was obtained for the traits DA, PH, FHB and Anther Retention. However, data for DON was not obtained from this trial. Anther Retention was converted to Anther Extrusion with the formula mentioned above.

Phenotypic statistical analysis

The raw data from the DON level assessment was transformed by $\log_{10}$ (DON level +1) to approximately obtain normally distributed values that were used throughout the entire analyses. Least square means of FHB disease severity, DON, DH, PH and AE for single environments (=trials), and across different environment for each trait were calculated using the “lme4” package (Bates et al. 2014) and “lmerTEST” (Kuznetsova et al. 2017) in R (Team 2021) using the following models:

$$P_{ikn} = \mu + g_i + B_k + R_n + R: B_{kn} + e_{ikn} \quad (1)$$

and

$$P_{ijkn} = \mu + g_i + E_j + g \times E_{ij} + B_k + R_n + R: B_{kn} + e_{ijkn} \quad (2)$$

From the above models, model 1 was used to calculate LSmeans of single environments for each trait, where P_{ikn} is the phenotype (trait value) of the i^{th} variety in the n^{th} replicate in the k^{th} block. μ is the general mean, g_i is the fixed effect of the i^{th} variety, B_k is the random effect of k^{th} block, R_n is the random effect of n^{th} replicate, $R: B_{kn}$ is the random effect of the k^{th} block within n^{th} replicate, and e_{ikn} is the error term. And model 2 was used to calculate LSmeans across the different environments tested for each trait, where P_{ijkn} is the phenotype (trait value) of the i^{th} variety in the n^{th} replicate in the k^{th} block in j^{th} environment. μ is the general mean, g_i is the fixed effect of the i^{th} variety, E_j is the random effect of the j^{th} environment, $g \times E_{ij}$ is the random effect of the i^{th} variety grown under j^{th} environment (interaction), B_k is the random effect of k^{th} block, R_n is the random effect of n^{th} replicate, $R: B_{kn}$ is the random effect of the k^{th} block within n^{th} replicate, and e_{ijkn} is the error term

FHB scorings and DON content can be confounded with variation in PH and DH, to adjust for these confounding effects regressions of FHB and DON on PH and DH was performed and the residuals used for the further analysis, while in the case of the trials at Morden and Tulln DA was used instead of DH. The residuals are FHB corrected for PH and DH/DA (FHBcPHDH), and DON corrected for PH and DH/DA (DONcPHDH).

Pearson correlations between the traits were calculated by Pearson method (Benesty et al. 2009) in R. Principal component analysis was performed on the available phenotypic data of FHBcPHDH, DONcPHDH and AE from all the tested environments to determine the principal coordinate values (PC scores) for each of the above-mentioned traits using the R-package “Factoextra” (Kassambara and Mundt 2017).

Broad-sense heritabilities were calculated for the across environment means of FHB, DON and AE based on the variance component estimates (Genetic Variance and Residual Variance) obtained from the R-package “Heritability” (Kruijer et al. 2015) in the complete panel and in the European panel using the formula:

$$H^2 = \frac{\sigma_g^2}{\sigma_g^2 + \sigma_E^2}$$

In the above formula, σ_g^2 is genetic variance, and σ_E^2 is the error variance.

Genotypic data

Seedlings of the NMBU spring wheat panel were grown in the greenhouse and genomic DNA was extracted from fresh young leaves using the DNeasy plant DNA extraction kit (Qiagen). The lines were genotyped with the 35K Axiom® array (Allen et al. 2017), Trait Genetics Illumina 25K SNP Chip, and in addition, some KASP and SSR markers for key agronomic and disease resistance traits (Rasheed et al. 2016) were also included in our study. The SSR markers were converted to biallelic state. Both the KASP and SSR markers were initially assigned to fictive chromosome in 35K and 25K genotypic datasets and later the markers which showed significant association with the traits were approximately placed close to the relative chromosomal position on the consensus map, based on physical map positions obtained from the wheat reference genome IWGSC RefSeq v1.0 (Appels et al. 2018) and LD with other significant markers. “MapChart 2.32.” (Voorrips 2002) was used for graphical representation QTL positions with their intervals.

Markers were filtered based on 10% missing data and minor allele frequency of $\geq 5\%$ in the lines. Heterozygous genotypes were regarded as missing data. Positional information was assigned using the consensus 35K SNP map (Allen et al. 2017) and Trait Genetics Illumina 25K SNP Chip. After filtering and removing redundant markers, in total 14713 and 21652 markers remained for the association mapping in 35K and 25K genotypic datasets (Online resource 1).

The validation panel was genotyped using Trait Genetics Illumina 25K SNP Chip. After genotyping, the markers were filtered based on the criteria mentioned above. For the present study, marker data was available for only 131 of the lines in the validation panel. The most significant SNP markers of the *Qfhb.nmbu.7A.2* region were converted to KASP markers, which were used for validation purpose in the study. Primer sequences were designed using the online PolyMarker tool (<http://www.polymarker.info/>) (Ramirez-Gonzalez et al. 2015) or

downloaded directly from CerealsDB
(<https://www.cerealsdb.uk.net/cerealgenomics/CerealsDB/>).

Linkage Disequilibrium

Calculation of the linkage disequilibrium (LD) over the entire genome was performed using the software TASSEL 5 (Bradbury et al. 2007). The LD was calculated with every mapped marker with allele frequencies > 0.05, over the 21 chromosomes, in a sliding window approach, with 1000 as the window size. Squared allele frequency correlation r^2 (Hill and Weir 1988) was used to calculate pair-wise LD. The p-values were calculated using Fisher's test in TASSEL, and a threshold of $p < 0.001$ was used for detecting significant LD between markers. The average genome-wide LD decay was visualized by plotting all intra-chromosomal r^2 values of all chromosomes against genetic distance in cM between the marker pairs. To summarize the relationship between the LD decay and genetic distance, a non-linear model described by Marroni et al. (2011) was used. For further information on the method of LD calculation, refer to Ruud et al. (2019).

Genome-Wide Association Mapping

Association analysis was performed on the traits AE, FHB corrected for PH and DH (FHBcPHDH) and DON corrected for PH and DH (DONcPHDH) for all the tested environments, and across-environment means and PC-scores of each trait using the "GAPIT" R package (Lipka et al. 2012). In our study, we tested several methods for GWAS, such as Mixed Linear Model, Compressed MLM, Enriched Compressed MLM and FarmCPU. Each model is briefly explained below.

MLM is a widely used single-locus model, which uses kinship matrix and population structure or principal components for controlling spurious associations (Yu et al. 2006). Compressed MLM (CMLM) by Zhang et al. (2010) is another single-locus model that clusters individuals into certain groups based on relatedness and uses genetic values as random effects, which improves statistical power compared to MLM. Enriched CMLM is an improvement of CLMM that examines alternatives to calculating kinship between groups as the average of pair-wise individual kinships (Li et al. 2014). This model uses algorithms and calculates kinship, therefore classifies, and chooses the best combination for the analysis. Altogether these three single-locus models improve statistical power and reduce spurious marker-trait associations.

FarmCPU is a multi-locus model, and a modified MLM method (Liu et al. 2016). This method works iteratively with a Fixed Effect Model (FEM) and Random Effect Model (REM). Testing markers each at a time and associated markers which are estimated in REM are used as covariates in FEM. REM avoids the over-fitting problem present in FEM. In this model kinship is defined with the help of associated markers estimated by REM. At each iteration p -values of testing markers and associated markers are combined.

Association mapping was performed separately for the total population of 300 spring wheat lines and for the European sub-population consisting of 237 lines using both genotype data sets (35K and 25K).

Since FHB resistance is a highly quantitative trait with mostly small-effect loci (Bai et al. 2018), the Bonferroni correction or False Discovery Rate (FDR) would be too strict criteria for identifying QTL (Haikka et al. 2020). However, in this study we decided to use FDR at a rate of 5% for across environment means of each trait. And the QTL were considered significant at $FDR < 5\%$. In addition, we evaluated how often these significant QTL were detected with a less stringent threshold of $-\log_{10}(p) > 3.0$ in the different environments. Those markers which were consistently detected above this threshold in two or more tested environments for a particular QTL region were considered as robust associated markers and later used for haplotype analysis and allele stacking. Quantile-Quantile (QQ) plots were inspected to identify the level at which the observed p-values started to deviate from the expected values under the null hypothesis, and for the presence of spurious associations.

Haplotype analysis and allele stacking

QTL that were consistently detected in different environments were further investigated to build haplotypes of common QTL for the traits FHBcPHDH, DONcPHDH and AE. Associated markers in those consistent QTL regions were used to construct haplotypes provided they were significant, consistent across the environments and having good LD with the highest significant marker in the QTL region. These haplotypes were tested with across environment means of corrected FHB disease severity, corrected DON content and AE from the complete panel to assess which haplotype contributes to resistance. For this purpose, pair-wise comparisons by Mann-Whitney-Wilcoxon Test (Mann and Whitney 1947) were conducted in the European and complete panel. Haplotype analysis was also carried out in the validation panel of new breeding lines from Graminor to validate the effect in an independent data set for which the haplotypes were constructed by using the same QTL detected in the NMBU spring wheat panel (25K).

Additionally, a validation was done on the haplotypes constructed based on KASP markers from the QTL *Qfhb.nmbu.7A.2*. These haplotypes were tested with across environment means of corrected FHB disease severity, corrected DON content and AE from the complete panel to assess which haplotype contributes to resistance.

For allele stacking, markers which were highly significant in different QTL regions were chosen for FHBcPHDH, DONcPHDH and AE. The resistant allele was determined based on the predicted allele effect of the markers associated with the QTL. Then, the lines were grouped based on the number of resistance alleles they contained. Mann-Whitney-Wilcoxon Test (Mann and Whitney 1947) was used to compare the significance of differences between the groups.

R-studio (Team 2021) was used to visualize the graphs using the following packages: “ggplot2” (Wickham et al. 2016) and “ggpubr” (Kassambara and Kassambara 2020)

Results

Phenotypic data analysis

Across environment means of FHB severity, DON, AE, PH, and DH showed continuous variation that resembled normal distributions (Figure 1). Considerable variation was observed within the complete panel and the European panel for all the traits in all the tested environments (Supplementary figure 1).

The FHB severity was positively correlated with DON with high significance ($r=0.66$, $p<0.0001$), and negatively correlated with PH ($r=-0.43$, $p<0.0001$) and AE ($r=-0.48$, $p<0.0001$). DON was positively correlated with DH ($r=0.38$, $p<0.0001$), and negatively correlated with PH ($r=-0.31$, $p<0.0001$) and AE ($r=-0.47$, $p<0.0001$) (Table 1).

Estimated heritabilities (H^2) of across environment means of FHB, DON and AE were 0.50, 0.69 and 0.74 in the complete panel and 0.34, 0.55 and 0.72 for the European panel respectively.

Linkage disequilibrium and Population structure

The estimated r^2 for half decay was 0.22 for the NMBU spring wheat panel and the estimated genome-wide half decay distance was 1 cM (the markers within the half decay distance of 1 cM were considered to be located in the same QTL on the genetic map).

Principal component analysis of the marker data confirmed the presence of two groups in the NMBU spring wheat panel, with principal component 1 (PC1) clearly separating the European lines from those originating outside Europe (Figure 2). For this reason, association analysis was done separately for the complete set of lines and the European sub-population.

Genome-Wide Association Mapping

Various GWAS models were tested on the available data, from which FarmCPU was chosen for its proven efficiency over other models in several recent studies (Liu et al. 2016; Kaler et al. 2020). Also, FarmCPU based QQ-plots showed more significant p-values compared to other models (Figure 3).

Association mapping detected significant marker-trait associations on several chromosomes for each trait. However, these significant MTAs varied from one environment location by year combination) to another. The most important and robust MTAs among the different environments and among the three traits FHBcPHDH, DONcPHDH and AE amounted to thirteen QTL in total. These robust QTL were significant across different tested environments and traits. They are located on the following chromosomes (the number following the chromosome number denotes different QTL region on the same chromosome, physical positions in parentheses) : *Qfhb.nmbu.1A.1* (9 – 46 Mbp), *Qfhb.nmbu.1A.2* (520 – 590 Mbp), *Qfhb.nmbu.3A.1* (683 – 738 Mbp), *Qfhb.nmbu.3B.1* (7 – 9.77 Mbp), *Qfhb.nmbu.4A.1* (537 – 607 Mbp), *Qfhb.nmbu.4A.2* (721 – 743 Mbp), *Qfhb.nmbu.4B.1* (527 – 609 Mbp), *Qfhb.nmbu.5A.1* (480 – 552 Mbp), *Qfhb.nmbu.6A.1* (608 – 609 Mbp), *Qfhb.nmbu.6B.1* (630 –

688 Mbp), *Qfhb.nmbu.7A.1* (120 – 129 Mbp) and *Qfhb.nmbu.7A.2* (670 – 710 Mbp) (Table 2). All those detected MTAs on specified chromosomes were found using genotyping with 35K and 25K SNP chips. And most of these were detected both in complete and European panel, except for two QTL regions which were specific only to the complete panel *Qfhb.nmbu.1A.2* (520 – 590 Mbp) and *Qfhb.nmbu.4A.1* (537 – 607 Mbp), and one QTL region specific to the European panel on chromosome 4B, QTL *Qfhb.nmbu.4B.1* (527 – 609 Mbp) (Table 2). In addition, a significant MTA was detected at the *Fhb1* locus on chromosome 3B, *Qfhb.nmbu.3B.1* (7 – 9 Mbp). This was found significant in two environments, along with the across environment means and PC scores for DONcPHDH. This MTA was only detected in one environment (Tulln, Austria in 2020) for FHBcPHDH and none for AE (Table 2). However, this MTA was only specifically detected in the complete panel but not in the European panel.

Since the detected significant MTAs were in the near similar QTL regions (physical positions) for both the genotypic datasets, we will hereafter present further analysis using the 25K data, as it is the latest data and will be used for further validations on a new set of germplasm in this study. The detailed overview of identified significant MTAs for each trait in all the tested environments, across-year means and PC scores, and in both the cases of 25K and 35K genotypic data are reported in Online Resource 3; Supplementary Tables 6 to 17.

Graphical representation of QTL locations and Manhattan plots from association mapping are shown in Online Resource 2 (Supplementary figure 3, 4, 5, 6, and 7).

Haplotype Analysis

Seven QTL regions were selected for haplotype analysis and the associated markers in those selected regions were used to construct haplotypes based on the criteria mentioned in the methods section. Marker information of haplotypes and number of haplotypes are shown in Online Resource 3; Supplementary Table 12 and the number of haplotype alleles varied among the selected QTL regions ranging from three to six.

For corrected FHB disease severity, only the haplotypes from the QTL region on chromosomes *Qfhb.nmbu.5A.1* (480 – 552 Mbp) and *Qfhb.nmbu.7A.2* (670 – 710 Mbp) showed significant effects on the disease resistance, whereas significant haplotype effects were shown for DON content in all the tested QTL regions. Haplotype effects and significant differences between the haplotypes for *Qfhb.nmbu.7A.2* (670 – 710 Mbp) are shown in Figure 4. Haplotypes effects of all other tested QTL regions are shown in Online Resource 2 (Supplementary figure 8, 9, 10, 11, and 12)

The same haplotypes from the QTL regions (Online Resource 3; Supplementary table 12) were tested on the validation panel consisting of new breeding lines tested in two different environments (Vollebekk in 2020 and Tulln in 2021). Here, only the QTL region on chromosome 7A (*Qfhb.nmbu.7A.2*) showed significant haplotype effects. For corrected FHB disease severity and AE, the resistant haplotype was significantly different from other haplotypes. A clear trend in the same direction was also shown for DON content in Vollebekk 2020, although not statistically significant due to the low statistical power. Data for DON is not

available from the location Tulln, 2021. Only two lines in the validation panel showed the resistant haplotype, and these lines share a similar pedigree with lines having the resistant haplotype on chromosomal region 7A (*Qfhb.nmbu.7A.2*) in the NMBU spring wheat panel (Figure 5). The most significant SNP markers in this region (*BS00098483_51*, *AX-95248570* and *Kukri_c57593_79*) were converted to breeder-friendly KASP markers and tested on the NMBU spring wheat panel (Online Resource 2; Supplementary figure 14). The KASP markers produced similar genotype calls as the original SNP marker data, primer sequences used for these KASP markers are available in Online Resource 3 (Supplementary table 14). A few anomalies were noted, which could be caused using new DNA samples for the KASP genotyping. Nevertheless, the same significant haplotype effects were seen in the KASP validation of complete panel and European panel as compared to the SNP chip data (Online Resource 2; Supplementary figure 13)

Allele Stacking

The effect of stacking different alleles was examined on corrected FHB disease severity, corrected DON content and AE (Figure 6) in both the complete and the European panel. In the complete panel the groups ranged from zero to five resistant alleles and the markers selected for the allele stacking are shown in Online Resource 3 (Supplementary table 13). In the European panel this number was reduced due to fewer lines and reduced genetic diversity, ranging from one to five resistant alleles. Overall, the lines carrying a greater number of resistant alleles (i.e., four or more) were more resistant than the groups carrying fewer resistant alleles, and significant effects of allele stacking was observed for all three traits (Figure 6).

Discussion:

Although similar QTL were detected by all the tested models in our study, the FarmCPU model detected fewer QTL than the other models. Since FarmCPU is known to have a better control of false positives and higher power to detect true QTL compared to the other models (Liu et al. 2016; Kaler et al. 2020) we decided to base our analysis on the FarmCPU results. Overall, we detected thirteen significant QTL regions in this study. Five of them were of major importance based on different factors such as consistency across the environments and traits and considering the LD between the significant markers in the respective QTL region. Significant markers from these five QTL regions were tested for their effect on FHB disease resistance traits with the help of haplotype and allele stacking analyses. From these analyses, we were able to identify the resistant haplotypes and the lines contributing the resistance. These results were further validated by performing haplotype analysis on an independent germplasm (i.e., Validation panel), which found a QTL region *Qfhb.nmbu.7A.2* (670 – 710 Mbp) on 7A to be important for improving FHB disease resistance in future breeding efforts.

Heritability

Heritabilities for FHB and DON were low when compared to AE and were further reduced in the individual analyses of the European panel, which highlights the contribution of genetic diversity in resistance by the exotic lines. The association mapping for FHB revealed several QTL over all chromosomes, but only some of those were consistent in two or more environments. For DON, the disease assessment was performed measuring the content by GC-MS, which is not subjected to the same rate of error as the visual scoring of FHB in the field. The association mapping for DON also revealed several QTL in each environment, but some of the QTL were consistent in two or more environments. These findings suggest that G x E interactions play a significant role in the NMBU spring wheat panel tested in our study. Therefore, the QTL that were significantly detected in two or more environments for FHB and DON should be emphasized for resistance breeding in the future.

Trait correlations

Days to heading has been reported to be negatively correlated with FHB (Emrich et al. 2008), while in our study the correlation between DH and FHB was very low and negative and not significant. Correlations between DH and FHB may have confounding effect on the association mapping results, because weather conditions (humidity and temperature) during anthesis can have a huge impact on the success of FHB infection. Hence, days to heading (or anthesis) might affect the disease scores when the germplasm differ in earliness and correlations can be positive or negative, depending on weather conditions in different years. It is therefore important to correct for this factor in individual nurseries. Since we observed a positive and significant correlation between DON and DH, we decided to correct both DON and FHB for DH in the association analyses to avoid any confounding effects of this trait.

Also, PH has been reported to be associated with FHB (Mao et al. 2010; Lu et al. 2013; Kubo et al. 2013). The correlation analyses in the present study further confirm these reports as both

FHB and DON were negatively correlated to PH. In wheat production, taller plants may lodge when fertilized and may also make use of modern machinery more difficult, the preferred plants are therefore the shorter ones. To avoid the confounding effect of PH, FHB and DON were also corrected for PH.

Correlation of AE with FHB and DON were found to be negative and highly significant in this study. High anther extrusion has been proposed as a valuable passive resistance trait to avoid FHB infection. Many studies (Skinnes et al. 2010); Lu et al. 2013; Kubo et al. 2013; Buerstmayr and Buerstmayr 2015; He et al. 2016a, b) have reported the correlation of AE with FHB and DON and detected lower FHB and DON infections in lines with high AE, and suggested high AE to contribute to Type I resistance. These studies clearly suggested that lines with low FHB could be achieved by selecting genotypes that displayed high anther extrusion but also pointed out that lines that shed anthers well still got infected by FHB. All these findings were further confirmed by Lu et al. (2013), which reported AE to be positively correlated with Type I resistance to FHB in a biparental mapping population and also detected several AE QTL to coincide with FHB QTL. In the association mapping of the present study, we considered only the significant QTL consistent across FHB, DON, and AE to be of foremost importance and used them for validation by haplotype analysis. These findings further validate the correlation between AE and FHB, and DON. Therefore, searching for genotypes with high AE, could possibly be a valuable contribution to the resistance breeding for FHB. AE is easy to score in the field and shows higher heritability than FHB and DON as demonstrated by the calculated heritabilities in this study. Fusarium resistance breeding can be enhanced by screening lines for high AE before the more laborious and costly FHB testing (Skinnes et al. 2008).

Several studies have reported correlation between FHB and DON in wheat both in segregating material and in collections of varieties with different resistance level (Bai et al. 2001; Miedaner et al. 2003; Snijders 2004; Ji et al. 2015; Hofgaard et al. 2016). The correlation between FHB and DON in the present study was also very significant and positive with a correlation coefficient of 0.66. Reports of the correlation between FHB and DON suggests a complicated relationship, Bai et al. (2001) performed thus a study with 116 cultivars and breeding lines of wheat to get further insight into this relationship. The results from their study show that cultivars moderately resistant and moderately susceptible to FHB usually had higher DON levels than resistant cultivars, but that there also were exceptions, especially for cultivars with a moderate Type II resistance. In the present study, we assume to have assessed a combination of both Type I and Type II FHB resistance in the field, because the field scoring was performed at a late stage in the development of the plants when both the initial infection (Type I) and spread (Type II) had occurred.

Comparison with reported or confirmed QTL from previous studies

Fhb1 is a well-characterized QTL descending from the Asian cultivar Sumai-3, and has been found in numerous QTL studies (Anderson et al. 2001; Liu et al. 2006; Buerstmayr et al. 2009) and is well-known for conferring Type II resistance and to a lesser extent Type I resistance (Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2002, 2003; Cuthbert et al. 2006). Diagnostic markers (*wgrb619_730* and *wgrb619_1450*) obtained from the study by Li et al.

(2019) for *Fhb1* were included in the genotypic data at a putative region on chromosome 3B.1 in our study, while the physical position was determined based on reference maps (Liu et al. 2006; Pumphrey et al. 2007). These diagnostic SNP markers showed MTAs with highly significant *p*-values in two tested environments and across years for the trait DONcPHDH, whereas it was only significant in one environment for FHBcPHDH and not detected in any environment for AE. These significant marker-trait associations were only detected in the complete panel but not in the European panel suggesting that resistance source for *Fhb1* was found among the non-European lines. Our study demonstrates that *Fhb1* is effective in providing Type III resistance, which is in line previous studies (Lemmens et al. 2005; Jiang et al. 2007a; Kluger et al. 2015). Its low allele frequency and lower heritability of FHB compared to DON likely contributed to the lack of consistent detection of an *Fhb1* effect for combined Type I and Type II resistance (FHB visual assessment) in the present study. However, *Fhb1* is an important QTL to be considered for practical resistance breeding.

The *Qfhb.nmbu.1A.1* QTL (Table 2) was located in the same region as the major QTL previously reported by Buerstmayr et al. (2009); Liu et al. (2009); and Venske et al. (2019) at the distal end of 1AS. Other studies have located FHB resistance QTL in a similar region: An FHB severity QTL from the Chinese wheat line CJ9306 was mapped to position 27.2 Mb (Jiang et al. 2007a, b) and a GWAS identified an FHB incidence QTL from Chinese elite germplasm in the same region (Zhu et al. 2020), while another recent study (Sari et al. 2018) identified an important FHB QTL for incidence and severity in the region of 1AS in *T. turgidum* ssp. *carthlicum* cv. Blackbird that agrees well with the 1A.1 QTL found in the present study.

Our study revealed *Qfhb.nmbu.7A.2* (Table 2) as a promising QTL for FHB resistance breeding, which we validated on new breeding lines and developed KASP assays for. The resistance haplotype at this locus explained a significant proportion of both Type I + Type II (FHB severity) and Type III (DON resistance) FHB disease resistance. A similar QTL was reported by Ruan et al. (2020) located at 671 Mb, which showed a large effect on all the examined FHB resistance related traits. Some previous studies also identified a major QTL for Type II resistance based on point inoculation in the vicinity of the *Qfhb.nmbu.7A.2* region through the physical mapping of the SSR markers *gwm276* and *gwm262* to positions of 642.9 and 681.4 Mb (Semagn et al. 2007; Buerstmayr et al. 2009). Two further studies reported FHB resistance QTL in the same chromosomal area: Wu et al. (2019) identified a QTL affecting DON accumulation in the same region of an elite Chinese germplasm and Sari et al. (2018) reported QTL for FHB severity and incidence in the same region from the durum wheat inbred line DT696.

Additionally, several recent studies identified and reported QTL which are in line with the results from our current study. Venske et al. (2019) published a QTLome meta-analysis of FHB resistance, which reported validated QTL from various studies (Liu et al. 2009; Ruan et al. 2020) that are in the same chromosomal region as the QTL detected in our study such as *Qfhb.nmbu.1A.1*, *Qfhb.nmbu.5A.1* and *Qfhb.nmbu.7A.2* (Table 2). Some other QTL from our study were tested for prediction of candidate FHB-response genes based on transcriptomic and proteomic data by Zheng et al. (2020). They identified and reported seventeen putative candidate genes, which are within the QTL base pair intervals of *Qfhb.nmbu.1A.2*, *Qfhb.nmbu.4A.1*, and *Qfhb.nmbu.5A.1* of our study (Table 2).

Prospects for resistance and genomic breeding of European and Nordic wheat germplasm

Many FHB resistance QTL are derived from Asian sources (Buerstmayr et al. 2009; Steiner et al. 2017). This is one of the reasons why we performed association mapping on two different sets of the wheat panel in the present study, aside from the apparent existence of two sub-populations in the NMBU spring wheat panel. This was done to enable the detection of resistance sources within adapted material (European and Nordic lines) that might not appear when performing association analysis on the complete panel. Using this approach, it was also possible to detect QTL in the exotic material that are not yet integrated into the European and Nordic breeding material. These QTL will be interesting for breeders to evaluate and possibly utilise in resistance breeding. Being strongly influenced by the environment, QTL for FHB and DON resistance need to show a consistent effect across several environments to be interesting for breeding and valuable for developing new more resistant varieties. Studying the association results for each environment many QTL turned up as significant, but when comparing every environment and their significant QTL, many of these QTL were only significant in one or two environments. Hence, we focused on consistent QTL that were significantly detected in two or more environments and choose two different significance thresholds: one for the across environment LSmeans and one for individual environment data. QTL were regarded as significant in across-environment data based on the FDR and examined for their consistency over individual environments with a more lenient significance threshold. By this criterion it was possible to assess the consistency of QTL effects across environments. It was also observed that only one QTL region was specifically significant in the European panel, while two other QTL regions were specific to the complete panel (Table 2). The QTL that are specific to complete panel should be of importance for improving the resistance in the European and Nordic material because they lack the resistant alleles from these significant QTL regions.

The QTL region (*Qfhb.nmbu.7A.2*) on chromosome 7A has an importance in the contribution of FHB disease resistance, which was evident from haplotype analysis of associated markers in this region both in NMBU spring wheat panel and the validation panel consisting of new breeding lines. The resistant haplotype of this QTL region was significantly more resistant from other haplotypes, showing the lowest corrected FHB disease severity and corrected DON content (Figure 4). There were five European lines with this resistant haplotype, and they are progeny of the Chinese resistant cultivar Ning 8343. Interestingly, two lines in the validation panel shared the same haplotypes and similar pedigree with the lines that showed the resistant haplotype in NMBU spring wheat panel.

Allele stacking demonstrated higher DON and FHB resistance levels among the lines that carried combinations of multiple favorable alleles from the QTL detected in our study. Most of the lines with a higher number of resistant alleles contributing to disease resistance originated from CIMMYT (Gondo-1 and lines derived from crosses with Cartbird) and China (Sumai 3, Ning 8343 and CJ9306). This list also includes some adapted lines from Norway and Sweden, including the lines whose resistance source originated from the Chinese cultivar Ning8343 (Runar//Ning8343/Brakar). This signifies the importance of the exotic resistance sources for contributing FHB resistance. Hence, making use of these resistance sources and fixing the resistant alleles in new breeding lines can improve the disease resistance considerably.

Conclusion and outlook

We have identified thirteen QTL regions for FHB resistance traits in this GWAS, all of which may have potential for a further use in resistance breeding. Most importantly the five QTL regions on chromosomes 1A, 3A, 5A, 6A and 7A were most consistent among all the detected marker-trait associations based on the criteria discussed above. The QTL *Qfhb.nmbu.7A.2* was additionally validated on an independent set of breeding lines, where lines carrying the resistant haplotype shared a similar pedigree with the resistant lines in the NMBU spring wheat panel. Moreover, breeder friendly KASP assays were developed and validated for this QTL. The resistance sources and QTL identified in this study will facilitate a further genetic improvement of FHB resistance in Nordic and European wheat germplasm by genomic breeding strategies, which enable an accelerated stacking of multiple resistance alleles to develop new improved cultivars.

Tables and Figures:

Table 1. Pearson`s correlation coefficients between FHB severity, DON content, anther extrusion (AE), plant height (PH) and days to heading (DH) for across environment means (LSmeans) of the NMBU spring wheat panel.

	AE mean	DH mean	FHB mean	PH mean
DH mean	0.054	-		
FHB mean	-0.48***	-0.013	-	
PH mean	0.16**	0.0013	-0.43***	-
DON mean	-0.47***	0.38***	0.66***	-0.31***

***P < 0.0001, **P < 0.001, *P < 0.05

733 **Table 2.** Overview of the most significant and consistent QTL detected in the present GWAS study, and comparison with previously
734 published FHB resistance QTL based on positions on the wheat reference genome assembly (RefSeq v1.0; International Wheat Genome
735 Sequencing Consortium (IWGSC) et al. 2018)
736
737

QTL name	Physical map position (Start – End, Mbp)	-log(p) value	NMBU Spring Wheat Panel	Published References
<i>Qfhb.nmbu.1A.1</i>	9 - 46	3.05 - 13.27	Complete and European panels	Jiang et al, 2007a and 2007b; Buerstmayr et al, 2009; Liu et al, 2009; Sari et al, 2018; Venske et al, 2019; Zhu et al, 2020
<i>Qfhb.nmbu.1A.2</i>	520 - 590	3.04 - 8.16	Complete panel	Venske et al, 2019; Zheng et al, 2020
<i>Qfhb.nmbu.3A.1</i>	683 - 738	3.09 - 12.27	Complete and European panels	Venske et al, 2019
<i>Qfhb.nmbu.3B.1</i>	7 - 9	3.49 - 12.86	Complete panel	Anderson et al, 2001; Liu et al, 2006; Buerstmayr et al. 2009
<i>Qfhb.nmbu.4A.1</i>	537 - 607	3.20 - 9.21	Complete panel	Zheng et al, 2020
<i>Qfhb.nmbu.4A.2</i>	721 - 743	3.09 - 8.40	Complete and European panels	In this present study
<i>Qfhb.nmbu.4B.1</i>	527 - 609	3.02 - 6.41	European panel	In this present study
<i>Qfhb.nmbu.5A.1</i>	480 - 552	3.0 - 12.49	Complete and European panels	Venske et al, 2019; Zheng et al, 2020
<i>Qfhb.nmbu.6A.1</i>	450 - 531	3.06 - 6.66	Complete and European panels	In this study
<i>Qfhb.nmbu.6A.2</i>	608 - 609	3.09 - 5.73	Complete and European panels	Ruan et al, 2020
<i>Qfhb.nmbu.6B.1</i>	630 - 688	3.03 - 6.55	Complete and European panels	In this present study
<i>Qfhb.nmbu.7A.1</i>	120 - 129	3.05 - 3.86	Complete and European panels	In this present study
<i>Qfhb.nmbu.7A.2</i>	670 - 710	3.50 - 25.45	Complete and European panels	Semagn et al, 2007; Buerstmayr et al, 2009; Sari et al, 2018; Wu et al, 2019; Ruan et al, 2020

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Figure 1. Histogram distributions based on the mean frequency data over all testing environments for AE, DH, FHB, PH and tDON for the NMBU spring wheat panel.

Figure 2. Principal Component Analysis based on the 25K data showing the population structure of NMBU spring-wheat panel, which is divided mainly into two groups – European and Others (lines from outside the Europe such as from CIMMYT, China, USA etc.).

Figure 3. QQ-plots of FHBcPHDHmean, DONcPHDHmean and AEmean for different GWAS models - GLM, CMLM, ECMLM and FarmCPU.

Figure 4: Boxplot showing the haplotype effect of QTL on chromosome 7A based on the a) mean AE, b) mean corrected DON content, c) mean corrected FHB disease severity of complete panel, and d) mean AE, e) mean corrected DON content, f) mean corrected FHB disease severity of European panel. Wilcoxon method is used for pair-wise comparisons (***P < 0.0001, **P < 0.001, *P < 0.05, ns > 0.05). Non-significant comparisons are not shown in this figure.

Figure 5: Boxplot showing the haplotype effect of QTL on chromosome 7A based on the a) AE, b) corrected DON content, c) corrected FHB disease severity of validation panel at Vollebakk in 2020 and d) AE, e) corrected FHB disease severity of validation panel at Tulln in 2021. Wilcoxon method is used for pair-wise comparisons (***P < 0.0001, **P < 0.001, *P < 0.05, ns > 0.05). Non-significant comparisons are not shown in this figure.

Figure 6: Boxplot showing the effect of number of stacked resistant alleles based on the a) mean AE, b) mean corrected DON content, c) mean corrected FHB disease severity of complete panel, and d) mean AE, e) mean corrected DON content, f) mean corrected FHB disease severity of European panel. Wilcoxon method is used for pair-wise comparisons (***P < 0.0001, **P < 0.001, *P < 0.05, ns > 0.05). Non-significant comparisons are not shown in this figure.

Supplementary Information:

Online Resource 1. Phenotypic data and Genotypic data used in the study. Phenotypic data - Information of NMBU spring wheat panel including genotype ID, names, country of origin. and the raw and corrected data for each genotype in various environments for each trait for NMBU spring wheat panel and Validation panel. Genotypic data – 35K and 25K data for 14713 and 21653 SNPs, respectively, of 296 lines for NMBU spring wheat panel including SNP ID, chromosome, physical position, - and 25K data for 19679 SNPs of 131 lines of the validation panel, including SNP ID, chromosome, physical position.

Online Resource 2. Supplementary figures including the frequency distributions of all the traits tested in different environments, correlations between the traits, Manhattan plots of GWAS with supporting QQ-plots, boxplots showing the haplotype effects of QTL validation, KASP validation and cluster plots of KASP markers.

782 **Online Resource 3.** Supplementary tables containing the information of MTAs including
783 marker name, chromosome number, physical position, p-value, $-\log_{10}(\text{p-value})$, FDR-adjusted
784 value, starting position and ending position on chromosomes in bp, information of selected
785 significant markers used for allele stacking and haplotype analysis and information about KASP
786 marker primer sequences.

References:

- Allen AM, Winfield MO, BurrIDGE AJ, et al (2017) Characterization of a Wheat Breeders' Array suitable for high-throughput SNP genotyping of global accessions of hexaploid bread wheat (*Triticum aestivum*). Plant Biotechnol J 15:390–401. <https://doi.org/10.1111/pbi.12635>
- Anderson JA, Stack R, Liu S, et al (2001) DNA markers for Fusarium head blight resistance QTLs in two wheat populations. Theor Appl Genet 102:1164–1168. <https://doi.org/10.1007/s001220000509>
- Appels R, Eversole K, Stein N, et al (2018) Shifting the limits in wheat research and breeding using a fully annotated reference genome. Science 361:6403. <https://www.science.org/doi/10.1126/science.aar7191>
- Bai G, Su Z, Cai J (2018) Wheat resistance to Fusarium head blight. Can J Plant Pathol 40:336–346. <https://doi.org/10.1080/07060661.2018.1476411>
- Bai G-H, Plattner R, Desjardins A, Kolb F (2001) Resistance to Fusarium head blight and deoxynivalenol accumulation in wheat. Plant Breed 120:1–6. <https://doi.org/10.1046/j.1439-0523.2001.00562.x>
- Bates, Douglas, Martin Mächler, Ben Bolker, and Steve Walker. 2015. “Fitting Linear Mixed-Effects Models Using Lme4”. Journal of Statistical Software 67 (1):1-48. <https://doi.org/10.18637/jss.v067.i01>
- Benesty J., Chen J., Huang Y., Cohen I. (2009) Pearson Correlation Coefficient. In: Noise Reduction in Speech Processing. Springer Topics in Signal Processing, vol 2. Springer, Berlin, Heidelberg. https://doi.org/10.1007/978-3-642-00296-0_5
- Bernardo AN, Ma H, Zhang D, Bai G (2012) Single nucleotide polymorphism in wheat chromosome region harboring Fhb1 for Fusarium head blight resistance. Mol Breed 29:477–488. <https://doi.org/10.1007/s11032-011-9565-y>
- Bradbury PJ, Zhang Z, Kroon DE, et al (2007) TASSEL: software for association mapping of complex traits in diverse samples. Bioinformatics 23:2633–2635. <https://doi.org/10.1093/bioinformatics/btm308>
- Buerstmayr H, Ban T, Anderson JA (2009) QTL mapping and marker-assisted selection for Fusarium head blight resistance in wheat: a review. Plant Breed 128:1–26. <https://doi.org/10.1111/j.1439-0523.2008.01550.x>
- Buerstmayr H, Lemmens M (2015) Breeding healthy cereals: genetic improvement of Fusarium resistance and consequences for mycotoxins. World Mycotoxin J 8:591–602. <https://doi.org/10.3920/WMJ2015.1889>

- 823 Buerstmayr H, Lemmens M, Hartl L, et al (2002) Molecular mapping of QTLs for Fusarium
824 head blight resistance in spring wheat. I. Resistance to fungal spread (Type II
825 resistance). *Theor Appl Genet* 104:84–91. <https://doi.org/10.1007/s001220200009>
- 826 Buerstmayr H, Steiner B, Hartl L, et al (2003) Molecular mapping of QTLs for Fusarium head
827 blight resistance in spring wheat. II. Resistance to fungal penetration and spread.
828 *Theor Appl Genet* 107:503–508. <https://doi.org/10.1007/s00122-003-1272-6>
- 829 Buerstmayr M, Buerstmayr H (2015) Comparative mapping of quantitative trait loci for
830 Fusarium head blight resistance and anther retention in the winter wheat population
831 Capo × Arina. *Theor Appl Genet* 128:1519–1530. <https://doi.org/10.1007/s00122-015-2527-8>
- 833 Cuthbert PA, Somers DJ, Thomas J, et al (2006) Fine mapping Fhb1, a major gene controlling
834 fusarium head blight resistance in bread wheat (*Triticum aestivum* L.). *Theor Appl*
835 *Genet* 112:1465–1472. <https://doi.org/10.1007/s00122-006-0249-7>
- 836 David Miller J, Arnison PG (1986) Degradation of deoxynivalenol by suspension cultures of
837 the Fusarium head blight resistant wheat cultivar Frontana. *Can J Plant Pathol* 8:147–
838 150. <https://doi.org/10.1080/07060668609501818>
- 839 Emrich K, Wilde F, Miedaner T, Piepho HP (2008) REML approach for adjusting the
840 Fusarium head blight rating to a phenological date in inoculated selection experiments
841 of wheat. *Theor Appl Genet* 117:65–73. <https://doi.org/10.1007/s00122-008-0753-z>
- 842 European Commission (2006) Commission Regulation (EC) No 1881/2006 of 19 December
843 2006 setting maximum levels for certain contaminants in foodstuffs. *J Eur Union*
844 364:5
- 845 FAOSTAT 2018 FAOSTAT (Food and Agriculture Organization of the United Nations) 2018
- 846 Goswami RS, Kistler HC (2004) Heading for disaster: Fusarium graminearum on cereal
847 crops: Fusarium graminearum on cereal crops. *Mol Plant Pathol* 5:515–525.
848 <https://doi.org/10.1111/j.1364-3703.2004.00252.x>
- 849 Haikka H, Manninen O, Hautsalo J, et al (2020) Genome-wide Association Study and
850 Genomic Prediction for Fusarium graminearum Resistance Traits in Nordic Oat
851 (*Avena sativa* L.). *Agronomy* 10:174. <https://doi.org/10.3390/agronomy10020174>
- 852 He X, Lillemo M, Shi J, et al (2016a) QTL characterization of Fusarium head blight
853 resistance in CIMMYT bread wheat line Soru# 1. *PloS One* 11:e0158052.
854 <https://doi.org/10.1371/journal.pone.0158052>
- 855 He X, Singh PK, Dreisigacker S, et al (2016b) Dwarfing genes Rht-B1b and Rht-D1b are
856 associated with both type I FHB susceptibility and low anther extrusion in two bread
857 wheat populations. *PloS One* 11:e0162499.
858 <https://doi.org/10.1371/journal.pone.0162499>

859

860 Hill W, Weir B (1988) Variances and covariances of squared linkage disequilibria in finite
861 populations. *Theor Popul Biol* 33:54–78. [https://doi.org/10.1016/0040-](https://doi.org/10.1016/0040-5809(88)90004-4)
862 5809(88)90004-4

863 Hofgaard IS, Aamot HU, Torp T, et al (2016) Associations between *Fusarium* species and
864 mycotoxins in oats and spring wheat from farmers' fields in Norway over a six-year
865 period. *World Mycotoxin J* 9:365–378. <https://doi.org/10.3920/WMJ2015.2003>

866 Ji F, Wu J, Zhao H, et al (2015) Relationship of deoxynivalenol content in grain, chaff, and
867 straw with *Fusarium* head blight severity in wheat varieties with various levels of
868 resistance. *Toxins* 7:728–742. <https://doi.org/10.3390/toxins7030728>

869 Jiang G-L, Dong Y, Shi J, Ward RW (2007a) QTL analysis of resistance to *Fusarium* head
870 blight in the novel wheat germplasm CJ 9306. II. Resistance to deoxynivalenol
871 accumulation and grain yield loss. *Theor Appl Genet* 115:1043–1052.
872 <https://doi.org/10.1007/s00122-007-0630-1>

873 Jiang G-L, Shi J, Ward RW (2007b) QTL analysis of resistance to *Fusarium* head blight in the
874 novel wheat germplasm CJ 9306. I. Resistance to fungal spread. *Theor Appl Genet*
875 116:3–13. <https://doi.org/10.1007/s00122-007-0641-y>

876 Jin F, Zhang D, Bockus W, et al (2013) *Fusarium* Head Blight Resistance in U.S. Winter
877 Wheat Cultivars and Elite Breeding Lines. *Crop Sci* 53:2006–2013.
878 <https://doi.org/10.2135/cropsci2012.09.0531>

879 Kaler AS, Gillman JD, Beissinger T, Purcell LC (2020) Comparing Different Statistical
880 Models and Multiple Testing Corrections for Association Mapping in Soybean and
881 Maize. *Front Plant Sci* 10:1794. <https://doi.org/10.3389/fpls.2019.01794>

882 Kassambara A, Kassambara MA (2020) Package 'ggpubr'

883 Kassambara A, Mundt F (2017) Package 'factoextra.' *Extr Vis Results Multivar Data Anal*
884 76:

885 Kluger B, Bueschl C, Lemmens M, et al (2015) Biotransformation of the Mycotoxin
886 Deoxynivalenol in *Fusarium* Resistant and Susceptible Near Isogenic Wheat Lines.
887 *PLOS ONE* 10:e0119656. <https://doi.org/10.1371/journal.pone.0119656>

888 Kruijer W, Boer MP, Malosetti M, et al (2015) Marker-Based Estimation of Heritability in
889 Immortal Populations. *Genetics* 199:379–398.
890 <https://doi.org/10.1534/genetics.114.167916>

891 Kubo K, Fujita M, Kawada N, et al (2013) Minor Differences in Anther Extrusion Affect
892 Resistance to *Fusarium* Head Blight in Wheat. *J Phytopathol* 161:308–314.
893 <https://doi.org/10.1111/jph.12060>

894 Kuznetsova A, Brockhoff PB, Christensen RH (2017) lmerTest package: tests in linear mixed
895 effects models. *J Stat Softw* 82:1–26

896 Lemmens M, Scholz U, Berthiller F, et al (2005) The Ability to Detoxify the Mycotoxin
897 Deoxynivalenol Colocalizes With a Major Quantitative Trait Locus for *Fusarium*

898 Head Blight Resistance in Wheat. *Mol Plant-Microbe Interactions*® 18:1318–1324.
899 <https://doi.org/10.1094/MPMI-18-1318>

900 Li G, Zhou J, Jia H, et al (2019) Mutation of a histidine-rich calcium-binding-protein gene in
901 wheat confers resistance to Fusarium head blight. *Nat Genet* 51:1106–1112.
902 <https://doi.org/10.1038/s41588-019-0426-7>

903 Li M, Liu X, Bradbury P, et al (2014) Enrichment of statistical power for genome-wide
904 association studies. *BMC Biol* 12:73. <https://doi.org/10.1186/s12915-014-0073-5>

905 Lipka AE, Tian F, Wang Q, et al (2012) GAPIT: genome association and prediction
906 integrated tool. *Bioinformatics* 28:2397–2399

907 Liu S, Hall MD, Griffey CA, McKendry AL (2009) Meta-Analysis of QTL Associated with
908 Fusarium Head Blight Resistance in Wheat. *Crop Sci* 49:1955–1968.
909 <https://doi.org/10.2135/cropsci2009.03.0115>

910 Liu S, Zhang X, Pumphrey MO, et al (2006) Complex microcolinearity among wheat, rice,
911 and barley revealed by fine mapping of the genomic region harboring a major QTL for
912 resistance to Fusarium head blight in wheat. *Funct Integr Genomics* 6:83–89.
913 <https://doi.org/10.1007/s10142-005-0007-y>

914 Liu X, Huang M, Fan B, et al (2016) Iterative Usage of Fixed and Random Effect Models for
915 Powerful and Efficient Genome-Wide Association Studies. *PLOS Genet* 12:e1005767.
916 <https://doi.org/10.1371/journal.pgen.1005767>

917 Lu Q, Lillemo M, Skinnes H, et al (2013) Anther extrusion and plant height are associated
918 with Type I resistance to Fusarium head blight in bread wheat line ‘Shanghai-
919 3/Catbird.’ *Theor Appl Genet* 126:317–334. [https://doi.org/10.1007/s00122-012-1981-](https://doi.org/10.1007/s00122-012-1981-9)
920 9

921 Mann HB, Whitney DR (1947) On a test of whether one of two random variables is
922 stochastically larger than the other. *Ann Math Stat* 50–60

923 Mao S-L, Wei Y-M, Cao W, et al (2010) Confirmation of the relationship between plant
924 height and Fusarium head blight resistance in wheat (*Triticum aestivum* L.) by QTL
925 meta-analysis. *Euphytica* 174:343–356. <https://doi.org/10.1007/s10681-010-0128-9>

926 Marroni F, Pinosio S, Zaina G, et al (2011) Nucleotide diversity and linkage disequilibrium in
927 *Populus nigra* cinnamyl alcohol dehydrogenase (CAD4) gene. *Tree Genet Genomes*
928 7:1011–1023. <https://doi.org/10.1007/s11295-011-0391-5>

929 McMullen M, Bergstrom G, De Wolf E, et al (2012) A unified effort to fight an enemy of
930 wheat and barley: Fusarium head blight. *Plant Dis* 96:1712–1728.
931 <https://doi.org/10.1094/PDIS-03-12-0291-FE>

932 McMullen M, Jones R, Gallenberg D (1997) Scab of Wheat and Barley: A Re-emerging
933 Disease of Devastating Impact. *Plant Dis* 81:1340–1348.
934 <https://doi.org/10.1094/PDIS.1997.81.12.1340>

- 935 Mesterhazy A (1989) Progress in breeding of wheat and corn genotypes not susceptible to
936 infection by *Fusaria*. In: *Fusarium*. Elsevier, pp 357–386.
937 <https://doi.org/10.1016/B978-0-444-87468-9.50026-2>
- 938 Mesterhazy A (1995) Types and components of resistance to *Fusarium* head blight of wheat.
939 *Plant Breed* 114:377–386. <https://doi.org/10.1111/j.1439-0523.1995.tb00816.x>
- 940 Mesterházy Á, Bartók T, Mirocha CG, Komoróczy R (1999) Nature of wheat resistance to
941 *Fusarium* head blight and the role of deoxynivalenol for breeding. *Plant Breed*
942 118:97–110. <https://doi.org/10.1046/j.1439-0523.1999.118002097.x>
- 943 Miedaner T, Schneider B, Geiger HH (2003) Deoxynivalenol (DON) Content and *Fusarium*
944 Head Blight Resistance in Segregating Populations of Winter Rye and Winter Wheat.
945 *CROP Sci* 43:8. <https://doi.org/10.2135/cropsci2003.5190>
- 946 Miller J, Young J, Sampson D (1985) Deoxynivalenol and *Fusarium* head blight resistance in
947 spring cereals. *J Phytopathol* 113:359–367. <https://doi.org/10.1111/j.1439-0434.1985.tb04837.x>
- 949 Mirocha CJ, Kolaczowski E, Xie W, et al (1998) Analysis of Deoxynivalenol and Its
950 Derivatives (Batch and Single Kernel) Using Gas Chromatography/Mass
951 Spectrometry. *J Agric Food Chem* 46:1414–1418. <https://doi.org/10.1021/jf970857o>
- 952 Pumphrey MO, Bernardo R, Anderson JA (2007) Validating the *Fhb1* QTL for *Fusarium*
953 Head Blight Resistance in Near-Isogenic Wheat Lines Developed from Breeding
954 Populations. *Crop Sci* 47:200–206. <https://doi.org/10.2135/cropsci2006.03.0206>
- 955 Ramirez-Gonzalez RH, Uauy C, Caccamo M (2015) PolyMarker: A fast polyploid primer
956 design pipeline: Fig. 1. *Bioinformatics* 31:2038–2039.
957 <https://doi.org/10.1093/bioinformatics/btv069>
- 958 Rasheed A, Wen W, Gao F, et al (2016) Development and validation of KASP assays for
959 genes underpinning key economic traits in bread wheat. *Theor Appl Genet* 129:1843–
960 1860. <https://doi.org/10.1007/s00122-016-2743-x>
- 961 Ruan Y, Zhang W, Knox RE, et al (2020) Characterization of the Genetic Architecture for
962 *Fusarium* Head Blight Resistance in Durum Wheat: The Complex Association of
963 Resistance, Flowering Time, and Height Genes. *Front Plant Sci* 11:592064.
964 <https://doi.org/10.3389/fpls.2020.592064>
- 965 Ruud AK, Dieseth JA, Ficke A, et al (2019) Genome-wide association mapping of resistance
966 to *Septoria nodorum* leaf blotch in a nordic spring wheat collection. *Plant Genome* 12.
967 <https://doi.org/10.3835/plantgenome2018.12.0105>
- 968 Sari E, Berraies S, Knox RE, et al (2018) High density genetic mapping of *Fusarium* head
969 blight resistance QTL in tetraploid wheat. *PLOS ONE* 13:e0204362.
970 <https://doi.org/10.1371/journal.pone.0204362>
- 971 Schroeder H, Christensen J (1963) Factors affecting resistance of wheat to scab caused by
972 *Gibberella zeae*. *Phytopathology* 53:831–838

973 Semagn K, Skinnes H, Bjørnstad Å, et al (2007) Quantitative Trait Loci Controlling Fusarium
 974 Head Blight Resistance and Low Deoxynivalenol Content in Hexaploid Wheat
 975 Population from ‘Arina’ and NK93604. *Crop Sci* 47:294–303.
 976 <https://doi.org/10.2135/cropsci2006.02.0095>

977 Skinnes H, Semagn K, Tarkegne Y, et al (2010) The inheritance of anther extrusion in
 978 hexaploid wheat and its relationship to Fusarium head blight resistance and
 979 deoxynivalenol content. *Plant Breed* 129:149–155. <https://doi.org/10.1111/j.1439-0523.2009.01731.x>

981 Skinnes H, Tarkegne Y, Dieseth J, Bjørnstad Å (2008) Associations between anther extrusion
 982 and Fusarium Head Blight in European wheat. *Cereal Res Commun* 36:223–231.
 983 <https://doi.org/10.1556/CRC.36.2008.Suppl.B.19>

984 Snijders CHA (2004) Resistance in wheat to Fusarium infection and trichothecene formation.
 985 *Toxicol Lett* 153:37–46. <https://doi.org/10.1016/j.toxlet.2004.04.044>

986 Steiner B, Buerstmayr M, Michel S, et al (2017) Breeding strategies and advances in line
 987 selection for Fusarium head blight resistance in wheat. *Trop Plant Pathol* 42:165–174.
 988 <https://doi.org/10.1007/s40858-017-0127-7>

989 Su Z, Bernardo A, Tian B, et al (2019) A deletion mutation in TaHRC confers Fhb1 resistance
 990 to Fusarium head blight in wheat. *Nat Genet* 51:1099–1105.
 991 <https://doi.org/10.1038/s41588-019-0425-8>

992 Team RC (2021) R: A language and environment for statistical computing (R Version 4.0. 3,
 993 R Foundation for Statistical Computing, Vienna, Austria, 2020)

994 Tekle S, Lillemo M, Skinnes H, et al (2018) Screening of Oat Accessions for Fusarium Head
 995 Blight Resistance Using Spawn-Inoculated Field Experiments. *Crop Sci* 58:143–151.
 996 <https://doi.org/10.2135/cropsci2017.04.0264>

997 Venske E, dos Santos RS, Farias D da R, et al (2019) Meta-Analysis of the QTLome of
 998 Fusarium Head Blight Resistance in Bread Wheat: Refining the Current Puzzle. *Front*
 999 *Plant Sci* 10:727. <https://doi.org/10.3389/fpls.2019.00727>

1000 Voorrips RE (2002) MapChart: Software for the Graphical Presentation of Linkage Maps and
 1001 QTLs. *J Hered* 93:77–78. <https://doi.org/10.1093/jhered/93.1.77>

1002 Waldron BL, Moreno-Sevilla B, Anderson JA, et al (1999) RFLP Mapping of QTL for
 1003 Fusarium Head Blight Resistance in Wheat. *Crop Sci* 39:805–811.
 1004 <https://doi.org/10.2135/cropsci1999.0011183X003900030032x>

1005 Wickham H, Chang W, Wickham MH (2016) Package ‘ggplot2.’ Create Elegant Data Vis
 1006 Using Gramm Graph Version 2:1–189

1007 Wu L, Zhang Y, He Y, et al (2019) Genome-Wide Association Mapping of Resistance to
 1008 Fusarium Head Blight Spread and Deoxynivalenol Accumulation in Chinese Elite
 1009 Wheat Germplasm. *Phytopathology®* 109:1208–1216.
 1010 <https://doi.org/10.1094/PHYTO-12-18-0484-R>

1011 Yu J, Pressoir G, Briggs WH, et al (2006) A unified mixed-model method for association
 1012 mapping that accounts for multiple levels of relatedness. *Nat Genet* 38:203–208.
 1013 <https://doi.org/10.1038/ng1702>

1014 Zhang Z, Ersoz E, Lai C-Q, et al (2010) Mixed linear model approach adapted for genome-
 1015 wide association studies. *Nat Genet* 42:355–360. <https://doi.org/10.1038/ng.546>

1016 Zheng T, Hua C, Li L, et al (2020) Integration of meta-QTL discovery with omics: Towards a
 1017 molecular breeding platform for improving wheat resistance to Fusarium head blight.
 1018 *Crop J* S221451412030177X. <https://doi.org/10.1016/j.cj.2020.10.006>

1019 Zhu Z, Chen L, Zhang W, et al (2020) Genome-Wide Association Analysis of Fusarium Head
 1020 Blight Resistance in Chinese Elite Wheat Lines. *Front Plant Sci* 11:206.
 1021 <https://doi.org/10.3389/fpls.2020.00206>

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Figures

Figure 1

Histogram distributions based on the mean frequency data over all testing environments for AE, DH, FHB, PH and tDON for the NMBU spring wheat panel.

Figure 2

Principal Component Analysis based on the 25K data showing the population structure of NMBU spring-wheat panel, which is divided mainly into two groups – European and Others (lines from outside the Europe such as from CIMMYT, China, USA etc.).

Figure 3

QQ-plots of FHBcPHDHmean, DONcPHDHmean and AEmean for different GWAS models - GLM, CMLM, ECMLM and FarmCPU.

Figure 4

Boxplot showing the haplotype effect of QTL on chromosome 7A based on the a) mean AE, b) mean corrected DON content, c) mean corrected FHB disease severity of complete panel, and d) mean AE, e) mean corrected DON content, f) mean corrected FHB disease severity of European panel. Wilcoxon method is used for pair-wise comparisons ($***P < 0.0001$, $**P < 0.001$, $*P < 0.05$, $ns > 0.05$). Non-significant comparisons are not shown in this figure.

Figure 5

Boxplot showing the haplotype effect of QTL on chromosome 7A based on the a) AE, b) corrected DON content, c) corrected FHB disease severity of validation panel at Vollebekk in 2020 and d) AE, e) corrected FHB disease severity of validation panel at Tulln in 2021. Wilcoxon method is used for pair-wise comparisons ($***P < 0.0001$, $**P < 0.001$, $*P < 0.05$, $ns > 0.05$). Non-significant comparisons are not shown in this figure.

Figure 6

Boxplot showing the effect of number of stacked resistant alleles based on the a) mean AE, b) mean corrected DON content, c) mean corrected FHB disease severity of complete panel, and d) mean AE, e) mean corrected DON content, f) mean corrected FHB disease severity of European panel. Wilcoxon method is used for pair-wise comparisons (** $P < 0.0001$, ** $P < 0.001$, * $P < 0.05$, ns > 0.05). Non-significant comparisons are not shown in this figure.

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