Genome-Wide Association Studies Combined with Genomic Selection as a Tool to Increase *Fusarium* Head Blight Resistance in Wheat and its Wild Relatives

By

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Abstract

Fusarium head blight (FHB) is a devastating wheat (*Triticum aestivum* L.) disease worldwide. Presently, there is insufficient FHB resistance in the Canadian wheat germplasm. Genome-wide association study (GWAS) and genomic selection (GS) can be utilized to identify sources of resistance that could benefit wheat breeding. To define the genetic architecture of FHB resistance, association panels from a spring and a winter collection were evaluated using the Wheat Illumina Infinium 90K array. A total of 206 accessions from the spring panel and 73 from the winter panel were evaluated in field trials for 3-4 years at two locations, namely Morden (Manitoba) and Ottawa (Ontario). These accessions were phenotyped for FHB incidence (INC), severity (SEV), visual rating index (VRI), and deoxynivalenol (DON) content. Significant ($p < 0.05$) differences among genotypes for all traits were found. Genetic characterization using the wheat 90K array identified a set of 20,501 single nucleotide polymorphisms (SNPs). The probe sequences (~100 bp) of these SNPs were mapped to the Chinese Spring reference genome v2.0 to identify 13,760 SNPs in the spring panel, and 10,421 SNPs in the winter panel covering all 21 wheat chromosomes. GWAS was performed to identify novel FHB resistance loci for INC, SEV, VRI and DON content for the spring and the combined panels separately using these 13,760 SNPs and for the winter panel using 10,421 SNPs. A total of 107, 157, 174 unique quantitative trait loci (QTNs) were identified for the four traits using two single-locus and seven multi-locus GWAS models for the spring, winter, and combined panels, respectively. These QTNs represent a valuable genetic resource for the improvement of FHB resistance in commercially grown wheat cultivars. In addition, these GWAS-defined QTNs were further used for GS to determine the breeding value (BV) of individuals as outlined below.

In order to understand the role of the model and that of the marker type and density in trait prediction modelling, a GS study was conducted. GS is considered as an important tool for increasing genetic gain for economically important traits such as FHB resistance. GS uses genome-wide molecular markers to develop statistical models that predict genomic estimated breeding values (GEBVs) of an individual. Our results support genomic prediction (GP) as an alternative to phenotypic selection to predict the BVs of individuals for this trait. GS accounts for minor effect QTNs, which is beneficial when breeding for quantitative traits. Moderate to high GP accuracies can be achieved for FHB resistance-related traits when implemented in a breeding

program. The correlation between the estimate of the missing phenotypic value and the observed phenotype is known as predictive ability (*r*). Overall, the predictive ability increased significantly using a QTN-based GP approach for FHB traits in wheat and its wild relatives. DON content had the highest predictive ability among all FHB traits, and that was in the winter panel, highlighting the importance of objectively measured traits in breeding for disease resistant genotypes. Interestingly, the winter panel contained several wild relative species that may harbor genes of interest to prevent the accumulation of mycotoxins in the grain.

This study showed the usability of genomic prediction by improving the predictive ability of the FHB traits, which can be applied in early generation selection to accelerate the improvement of FHB resistance in wheat. The results show that GS can be successfully implemented in wheat breeding programs over multiple breeding cycles and can be effective for economically important traits. It is anticipated that GS will play a substantial role in the future of wheat breeding.

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Dedications

To my dearest sister Sajana Bartaula. You are our everything.

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Acronyms

AAFC	Agriculture and Agri-Food Canada
BLUP	Best linear unbiased predictors
CS	Chinese Spring
DNA	Deoxyribonucleic acid
DON	Deoxynivalenol
FHB	*Fusarium* head blight
Gb	Giga bases
GEBV	Genomic estimated breeding value
GLM	Generalized linear model
GP	Genomic prediction
GS	Genomic selection
GWAS	Genome-wide association study/ies
INC	Incidence
IWGSC	The International Wheat Genome Sequencing Consortium
LD	Linkage disequilibrium
MAF	Minor allele frequency
MAS	Marker-assisted selection
MLM	Mixed linear model
mrMLM	Multi-locus random-SNP-effect mixed linear model
MTA	Marker-trait association
ORDC	Ottawa Research and Development Centre
PS	Phenotypic selection
QTL/s	Quantitative trait locus/loci
QTN	Quantitative trait nucleotide
SEV	Severity
SHW	Synthetic hexaploid wheat
SNP	Single nucleotide polymorphism
TE	Transposable element
VRI	Visual rating index

General Introduction

1.1 Wheat Crop

Wheat (*Triticum aestivum* L.) is a staple crop cultivated on approximately 200 million hectares worldwide. Wheat alone delivers one-fifth of the total caloric demand and proteins of the world's population [1]. Canada is the world's sixth largest producer and the third largest exporter of wheat, producing an average of 32 million tonnes annually, of which approximately 15 million tonnes are exported (http://www.fao.org/faostat/en/#data/QC). Breeding programs around the world aim to develop the best performing wheat varieties, i.e., those that offer higher yield, disease, and insect resistance, and the ability to better withstand environmental conditions while meeting the strict quality standards required to address market needs (https://westerngrains.com/special-initiatives/the-harvest-canadian-national-wheat-improvement-program/). To accomplish this lofty goal, a wide range of genetic resources are utilized by breeding programs to improve wheat and develop superior varieties.

1.2 Wheat Evolution and Classification

Cultivated wheat evolved through three major steps: natural crossings of wild species, domestication and human selection [2]. Archaeobotanical and molecular genetic evidence suggest that wheat was domesticated in the Near East, a region referred to as the Fertile Crescent [3]. Somewhere around 0.5M years ago, initial hybridizations between the grass species *T. urartu* (A genome donor) and a B genome donor species closely related to *Ae. speltoides* gave rise to the tetraploid species *T. turgidum* (AABB). More recent hybridizations, approximately 8,000-10,000 years ago, between domesticated emmer wheat (*T. turgidum L.* ssp. *dicoccum*, 2n=4x=28; AABB genome) and Tausch's goatgrass (*Ae. tauschii* Coss., 2n=2x=14; DD genome) gave rise to *T. aestivum* (bread wheat; 2n=6x=42; AABBDD genome; **Figure 1.1**) [4-6]. Hexaploid wheat is thus the result of allopolyploidization events involving species from the *Aegilops* and *Triticum* plant genera [7].

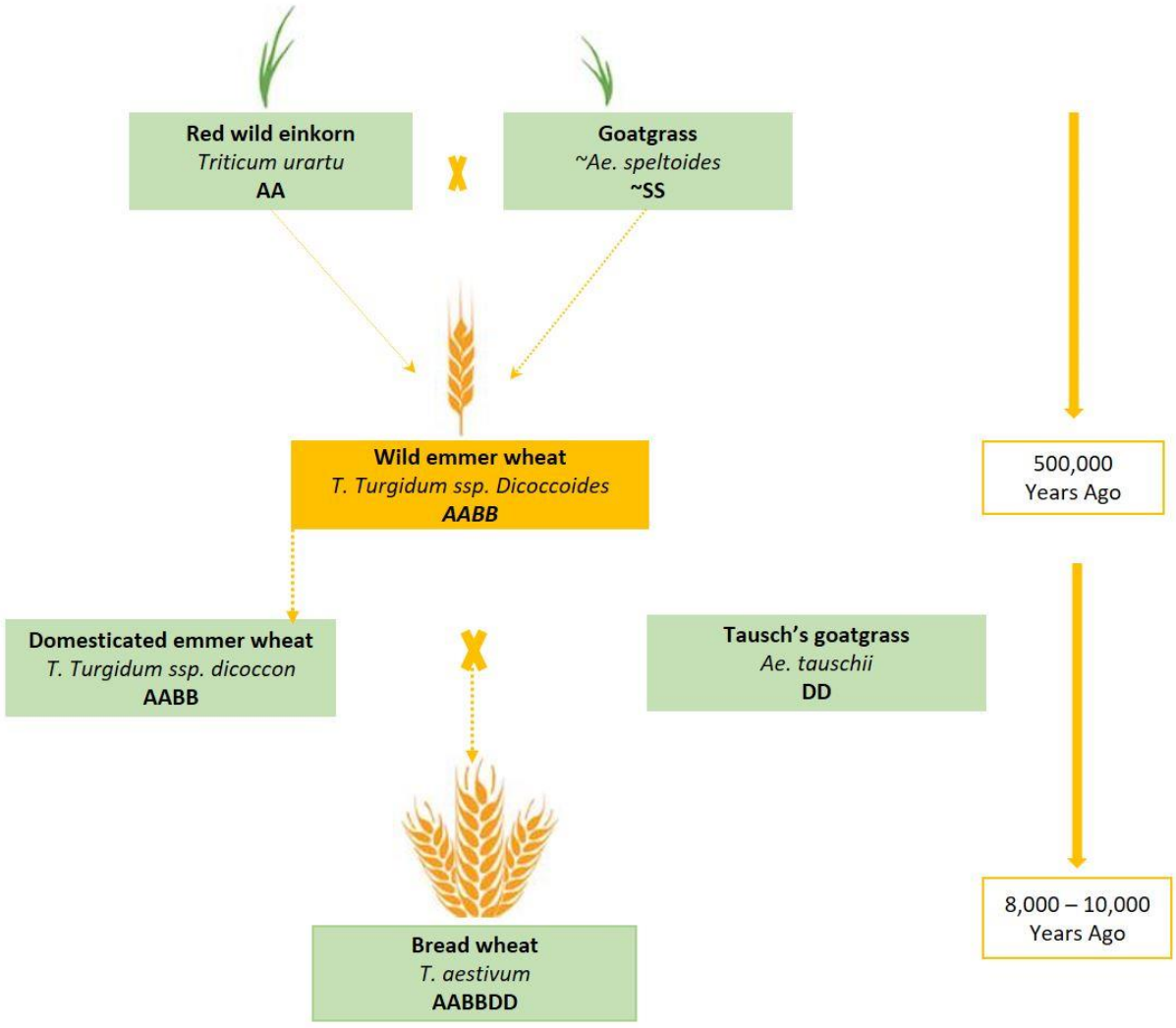

Figure 1.1 Evolution of cultivated wheat. The red wild einkorn wheat diploid species *Triticum urartu* (2n = 2x = 14, AA) naturally hybridized to a species closely related to *Aegilops speltoides* (SS) that was the donor of the B genome (2n = 2x = 14, BB). The subsequent chromosome doubling gave rise to the wild emmer wheat species *T. turgidum* ssp. *dicoccoides* (2n = 4x = 28, AABB) which was later domesticated to produce the species *T. turgidum* ssp. *dicoccum*. The subsequent natural pollination of the tetraploid species *T. turgidum* ssp. *dicoccum* by *Ae. tauschii* (2n = 2x = 14, DD) gave rise to *Triticum aestivum* (2n = 6x = 42, AABBDD), today's modern common wheat species, including bread wheat [8].

Historically, it is claimed that wheat was the earliest field crop used for human food processing. *Triticum monococcum* (einkorn) and *Triticum turgidum* ssp. *dicoccum* (emmer) were the earliest wheat species to be thus used around 12,000 – 17,000 years ago in the Near East [9-11].

Wheat belongs to the *Triticum* genus of the Triticeae family [12]. *Triticum* species are either diploid with 14 chromosomes, tetraploid with 28 chromosomes or hexaploid with 42 chromosomes. Bread wheat (*T. aestivum*) is a hexaploid species that is comprised of three genomes: A, B and D. Diploid species *T. urartu* and *T. monococcum* contain the AA and A^mA^m genomes, respectively, whereas *T. turgidum* and *T. aestivum* contain the AABB and AABBDD genomes, respectively [9, 10].

Spring and winter wheats are the two primary forms of wheat. This classification is based on their requirements for vernalisation in order to flower, a prerequisite that dictate their seeding and harvesting times. Winter wheat varieties generally yield more and have a lower protein content than spring wheat varieties [13]. Species of wheat display a wide range of variations, explaining their equally wide agroecological adaptation [14]. Among them, common wheat (*T. aestivum* L.) is by far the most significant. It is widely grown on the Canadian prairies and in many other parts of the world. Common wheat, which accounts for 95% of all consumed wheat, is widely used for the production of baked products and noodles. The remaining 5% is mostly *T. turgidum* ssp. *durum* which is used to make pasta, couscous and semolina flour. The latter is cultivated in hot and arid environments to achieve the high protein content and quality required for making pasta. It is widely cultivated in Minnesota, North and South Dakota and in the southern parts of Saskatchewan and Alberta [13, 14].

1.3 The Wheat Genome

Each of the three sub-genomes of wheat has its own homologous chromosomes. The A, B, and D sub-genomes have seven pairs of chromosomes. Wheat consists of both homologous and homoeologous chromosomes (**Figure 1.2)**. Allopolyploidization brings homoeologous chromosomes from separate parental species together in the same genome [15]. Homoeologous chromosomes have a somewhat conserved gene content and structural organization, but their repetitive DNA, which is primarily transposable elements (TEs), differ. The International Wheat Genome Sequencing Consortium (IWGSC) has released a completely annotated reference genome for the bread wheat variety Chinese Spring, which contains 107,891 projected high-confidence protein coding genes in a 14.5 Gb assembly [16]. The hexaploid genome is estimated to comprise nearly 17 billion nucleotides of which more than 85% is repetitive [16]. Major

classes of TEs account for the majority of the repetitive DNA, with 3,968,974 copies of TEs belonging to 505 families. The B sub-genome is the largest with an assembly size of 5.2 Gb, followed by the A at 4.9 Gb and the D at 3.9 Gb, although the distribution of high-confidence genes across all sub-genomes is similar with 35,345, 35,643 and 34,212, respectively [16].

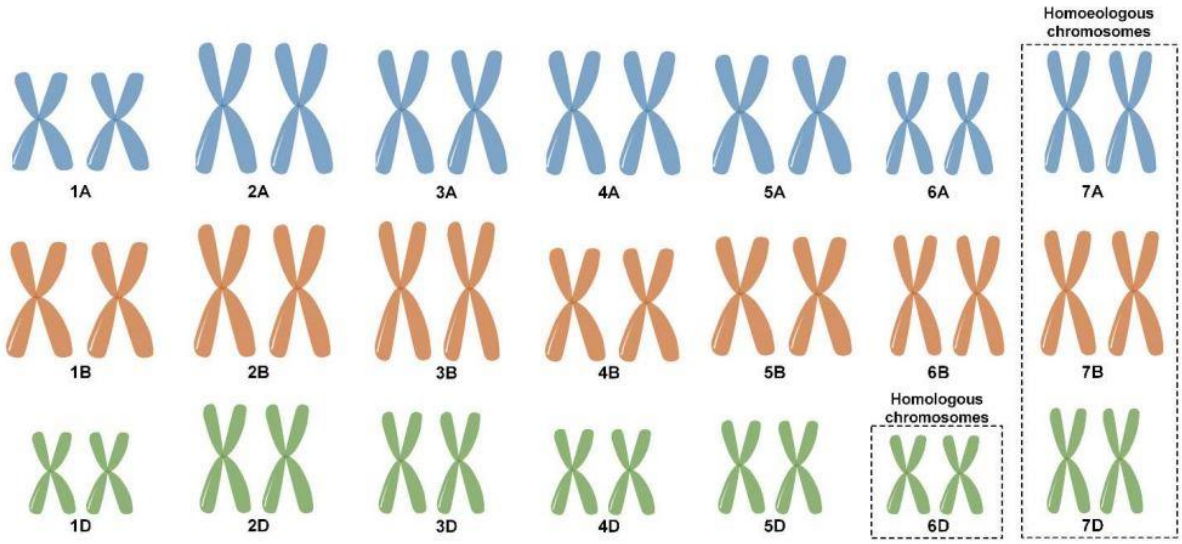

Figure 1.2 Structural organization of the hexaploid bread wheat chromosomes illustrating homologous chromosome pairs and homoeologous chromosomes across the three genomes. The figure is reproduced with permission from Fizza Fatima © [17].

1.4 Genetic Diversity Bottlenecks

The genetic diversity of the gene pools available to a species constitutes potential for trait improvement through breeding [18]. Diversity bottlenecks caused by speciation through polyploidization, domestication and, natural and artificial selections can be observed in today's bread wheat germplasm which shows a narrow genetic diversity. The bottleneck of speciation is referred to as the founder effect because only a few individuals participated in the original rare crossing events [19]. As such, the initial tetraploids and hexaploids comprise a limited portion of the genetic diversity of their diploid progenitors. Subsequent domestication, natural selection, and artificial (breeding) selection have further contributed to the erosion of the genetic diversity in bread wheat varieties. It is however possible to recover some of this lost genetic diversity. One

way is through the use of synthetic hexaploid wheats (SHWs) that are reconstituted by crossing its progenitors: *T. turgidum* ssp. L. and *Ae. tauschii* Coss.

The low genetic diversity of hexaploid wheat is particularly acute in the D genome because it was the last one incorporated and, as such, it had less time to undergo mutations and rearrangements [20-22]. The production of SHWs using diverse *Ae. tauschii* has the potential to broaden the genetic diversity of the D genome of bread wheat [23-25]. Indeed, the *Ae. tauschii* gene pool is more diverse than that contained within the D genome of bread wheat [26] and, this greater diversity is reflected in the SHWs produced to date [27]. SHWs have been capitalized upon to improve biotic and abiotic stress tolerance and other agronomic traits [28, 29]. However, to date, SHWs have been produced mostly through random crossing schemes between tetraploid and diploid progenitor species without *a priori* knowledge of their combining abilities or the combining abilities of the resulting SHWs with modern varieties. This gap in knowledge has impeded their uptake in breeding programs because most SHWs fell short of breeders' expectations. The project underlying this thesis aims in part to address this knowledge gap, to eventually develop a solid scientific approach to better designing primary SHW crosses and subsequent backcrosses to elite varieties.

1.5 Genome-wide Association Study/ies (GWAS)

Genome-wide association study (GWAS) is a methodology that test hundreds of thousands of genetic DNA markers across genomes to identify those that are statistically associated with a specific trait, e.g. disease resistance. To perform this type of study, at least two sets of input data are required: a genome-wide marker set constitutes the genotyping data and trait measurements make up the phenotyping data. For the genotypic data, GWAS is typically performed using genome-wide single nucleotide polymorphism (SNP) markers; insertion/deletion and structural variants such as microsatellites can also be used to identify associations [30]. In agriculture, phenotypic data are measurable traits that could be controlled by one or a few genes, such as the reaction type observed following inoculation by a single rust isolate, or by many genes, such as yield and yield components. Polygenic traits can be controlled by one or a few major genes in combination with a large number of minor genes or sometimes exclusively by minor genes.

Phenotypic data can be quantitative or even qualitative measurements of traits ranging from agronomic traits to biotic and abiotic stress tolerance traits for example.

Genome-assisted breeding has become a viable tool for improving selection efficiency because the cost of genotyping continually decreases. However, in order to implement genome-assisted breeding methods, one must identify the polymorphism(s) associated with the traits of interest. GWAS originated in human genetics [31-33], and has progressively found its way into plant studies [34, 35]. It is a method designed to find associations between genotype and phenotype based on statistical models; such associations are often referred to as marker-trait associations. The models consider population structure and the linkage disequilibrium (LD) between the genetic markers and the phenotype to identify quantitative trait loci (QTLs) or quantitative trait nucleotides (QTNs) of large and small effects [36]. GWAS uses genome-wide markers to estimate marker effects and test marker significance on phenotype. Compared with linkage mapping, higher allelic diversity at the corresponding loci may be accounted in GWAS, and ancestral recombination events in a population or species can be exploited [37, 38]. As a result, GWAS has become a widely used method for identifying genetic variations or QTNs linked to traits in crops.

In recent studies, both single and multi-locus GWAS models were used in crops such as rice [39], cotton [40] and others. The single-locus models GLM (Generalized linear model) [41] and MLM (Mixed linear model) [41] implemented in the rMVP package [42] can be used to determine marker-trait associations. To reduce the false positive rate, the thresholds of significance for GLM and MLM are determined by a critical *P* value subjected to the Bonferroni correction ($\alpha = 0.05/n$) where n is the number of markers. The main difference between single and multi-locus models is that single-locus models test the association between each marker and the trait in a consecutive fashion whereas all multi-locus methods involve two-step algorithms. During the first step, a single-locus GWAS method is applied to scan the entire genome, and putative QTNs are detected according to a less stringent critical value, such as $P < 0.005$ or $P < 1/n$, where n is the number of markers. During the second step, all selected putative QTNs are examined by a multi-locus model to determine the significant QTNs based on a log of the odds (LOD) score. The multi-locus models do not rely on the stringent Bonferroni correction and thus

more marker-trait associations may be identified [43]. FarmCPU is the only multi-locus model to use the Bonferroni correction to determine marker-trait associations [44].

Recently, several multi-locus GWAS models have been released, such as mrMLM [45], FASTmrMLM [46], FASTmrEMMA [47], pKWmEB [48], pLARmEB [45], and ISIS EM-BLASSO [49] implemented in the R package mrMLM [47]. The default threshold of significance for the mrMLM package models is a LOD score of 3.0 [50].

Population structure refers to the composition of a population as defined by its genetic background driven by the combined effects of evolutionary process such as genetic drift, mutation, and natural selection. Population structure analysis is a prerequisite to GWAS to alleviate spurious associations between markers and traits that could increase the rate of false positives [51]. However, the rate of false positives can be reduced by using population structure as a covariate. Several methods can be used to determine population structure. These include the separation of breeding lines based on their origins or traits, inclusion of kinship or genotype matrices, inclusion of marker fixed effects, or use of principle components in an analysis based on subpopulations [52-54]. GWAS models used without correction for population structure would results in a high rate of false positives because these models cannot correct for the linkage disequilibrium caused by the inherent nature of the population. False positives are therefore reduced by accounting for population structure as a covariate in the models [55].

1.6 Genomic Selection (GS)

Genomic selection (GS) is a form of marker-assisted selection (MAS) that is based on the global analysis of a large number of genome-wide markers as opposed to one or a few markers linked to qualitative or quantitative trait loci [24]. Contrary to GWAS, GS has emerged as a promising technique to develop statistical models using all the molecular markers information and phenotype data of a training population to predict genomic estimated breeding values (GEBVs) in test individuals for which only genotypic information is available [56, 57]. GS enables the selection of superior individuals based on their GEBVs, which take into account the complex genetic nature of traits including the genotype by environment (GxE) interactions involved in the observed phenotype(s) of the trait(s) [58, 59]. The principle of GS is straightforward. GS

requires the development of training (calibration) sets consisting of individuals that have been both phenotyped and genotyped, followed by model calibration. The models are subsequently used to predict the breeding value (BV) of individuals from testing sets that have been genotyped but not phenotyped [60]. GS is a relatively recent technique in plant breeding that uses a genome-wide set of genotypic information together to predict the phenotypic traits of individuals. This method assumes that a quantitative trait is determined by an infinite number of unlinked and non-epistatic loci, each one with a very small effect, that satisfies normality and linearity [61]. To determine the phenotypic trait values with genome-wide markers, GS uses both parametric and non-parametric statistical models. These models use genome-wide markers to predict the BV, also called GEBV, of phenotypic traits as a means to improve selection efficiency and precision in breeding programs [56]. The use of the genomic prediction (GP) approaches in the selection process is referred to as GS. Phenotyping for agriculturally-important quantitative traits can be expensive because accuracy depends heavily on labor-intensive field trials that must be conducted at multiple sites to properly evaluate GxE. Recent sequencing technologies have enabled the fast generation of high throughput genotypic information at relatively low costs, thereby facilitating the implementation of GWAS and GS to assist breeding efforts [62, 63].

Genomic selection requires two populations: a training population (TRN) and a testing population (TSN). The TSN is the breeding population for selection whose outcomes are predicted using models obtained from the genotypic and phenotypic data of the TRN. GS uses genomic prediction models based on genome-wide markers and measured phenotypic traits of the TRN to predict the GEBVs of the TSN solely based on genotypic data. These GEBVs are used for selecting the lines to include in the next breeding cycle [64]. The accuracy of prediction depends upon the GEBVs calculated through specially-designed genomic models for the traits, the precision with which they were evaluated on the TRN and the relationship between the TRN and TSN. Other factors affecting GEBVs include G*E interactions, phenotype of both TSN and TRN, selection of genetic markers, and variation in the advance lines [65]. Choosing the best statistical model is also an important consideration of the study. The statistical problem resides in attempting to estimate many QTN effects from a limited number of phenotypes because it over-saturate ordinary least-square models, thereby preventing the capture of small effect markers. The GS solution is to treat the predictors as random effects which allows to estimate all marker

effects and avoids the problem of fixed effects in an ordinary least-square models where set thresholds would exclude small effect QTNs and where the accuracy would decline upon saturation of the model that would occur when there is a large number of significant markers. Mixed linear models use the effects of all markers as random effects. Current methods like Bayesian estimation and machine learning used in this study are useful because there is no test of significance and many QTL effects, set as random effects, can be estimated simultaneously. Model evaluation can be done via two main approaches such as cross-validation and Pearson correlation (*r*). In this study, the prediction ability was estimated for genomic prediction models and a five-fold cross-validation scheme with 50 replications was implemented to evaluate the accuracy for different models. For each replication, the dataset was randomly divided into five subsets and 4/5 partitions were used as the training population to fit the models using both phenotype and genotype information while the remaining 1/5 partition was used as the validation set from which GEBVs were obtained from the models based only on the genotypes. In practice, the accuracy is estimated by means of cross-validation methods. The predictive ability of the models are evaluated by estimating the correlation between the measured phenotypes and the GEBVs [66].

The model used in this study falls under two categories: parametric and non-parametric. The basic idea behind the parametric method is that there is a set of fixed parameters to determine a probability model. Parametric methods assume normality of the population, or if not then we can easily approximate it using a normal distribution [67]. The parametric models include GBLUP, RRBLUP, Bayesian models, such as Bayes A, Bayes B, Bayes C, and Bayes LASSO, that use prior distribution for estimating the markers' effects using the computationally intensive Markov Chain Monte Carlo (MCMC) method. All these parametric models assume *a priori* relationship between markers and traits, thus ignoring the higher-order interactions during model training. This method only account for additive effect and the linear relationship between genotype and phenotype [67]. In non-parametric methods, there is no assumption for the population. In fact, the methods do not depend on the population *per se*. Here no fixed set of parameters nor distribution (normal distribution, etc.) are used [67]. Non-parametric methods are also referred to as distribution-free methods. The non-parametric models RF, SVR, BRR and BL were used in this study.

GS has been applied in plant and animal breeding to increase selection efficiency, reduce cost, and shorten breeding cycles. GS has been intensively studied in major crop for key traits such as yield, disease resistance and quality [68-70]. The positive impact can be seen in the rates of genetic gain through improved accuracy of predicted BVs and/or through shorter breeding cycles [71-75]. GS offers a significant opportunity to enhance crop production through the shortening of the breeding cycles that is accomplished by bypassing phenotyping and either selecting superior germplasm or removing inferior germplasm strictly based on their predicted BVs. GS takes advantage of all markers and the reduction in phenotyping of the progeny. Thus GS saves not only time but also major resources needed for phenotyping. The latter must be weighed against the cost of genotyping which, because of advances in high throughput sequencing, tend to be lower than phenotyping [76, 77]. The cost and genetic gain benefits are the most important considerations for GS application in crop improvement. GS can be applied to both simple as well complex traits, however, while it is better suited than other methods for complex traits, a high GP ability is more difficult to achieve in the latter case [57].

Markers are critical factors in GP accuracy because they are the fundamental input with which BVs are predicted [62]. With the development of many types of molecular markers, marker-assisted breeding strategies have been broadly deployed over the last decade [78]. Large sets of high-density genome-wide markers can be readily generated at low costs due to advances in next-generation sequencing technologies. Traditionally, QTL mapping was performed using biparental mapping populations consisting of recombinant inbred lines (RILs), F_2- or backcross-derived lines. All individuals of the mapping populations were genotyped and phenotyped for a trait of interest. QTL mapping was performed based on linkage maps, i.e., genetic recombination between markers. QTLs explaining the variation for the trait(s) of interest were thus identified and their contribution to the genetic variance was quantified [79]. One of the issues of this approach is that the resolution was often low because of the limited recombination observed in these generally small biparental populations. GWAS' main advantages are the higher polymorphism and the fact that it takes advantage of the historical recombination of the panel which is greater than that of the biparental populations [80].

Contrary to QTL mapping and GWAS, GS generally uses genome-wide molecular markers to perform the GP of the individuals. In most studies, GS models are constructed using genome-

wide random SNPs. However, GS models have also been built using QTL markers as an alternative to random SNPs [81]. One study suggested that GP models built using QTLs consistently outperformed models derived from random SNPs. In this case, the comprehensive set of QTLs had been identified using multiple single- and multi-locus GWAS models [81]. A reduction in background noise or a reduced multicollinearity due to the removal of unrelated markers were deemed to be the most likely factors contributing to the higher predictive ability in GS models based on pre-identified QTLs [81]. The comparatively lower predictive ability observed using genome-wide SNPs could also be attributed to errors introduced in the data set through the imputation of missing SNP data [82]. Briefly, QTL-based genomic prediction outperformed high density genome-wide markers, thereby producing higher predictive ability [83]. GS has been demonstrated to enhance predictive ability for crops like wheat and maize [84-88]. GS is considered an effective breeding tool for complex traits such as FHB resistance [79, 89-92].

1.7 Genomic Selection (GS) and the Breeder's Equation

The breeder's equation ($R = ir\sigma_A$) has been used to demonstrate how genetic gain (R) increases in response to selection intensity (i), the square root of the additive genetic variation (σ_A), and the accuracy of selection, which reflects the narrow sense heritability in phenotypic selection (r) [93]. Plant breeders must improve at least one of the three components of the equation within a specific length of time (t) in a breeding cycle or year ($R = ir\sigma_A /t$) in order to enhance the genetic gain [94, 95]. While phenotypic selection can be efficient for highly heritable traits, selection for low heritability traits are often relegated to the later stages of a breeding program, particularly with inbred crops [96]. This is because the lines need to become more homozygous to increase the accuracy of selection. When the breeding lines are highly homozygous (F_5 or F_6), they can be harvested in bulk and evaluated in replicated field trials. Increment in genetic gain can be hampered by such limitations imposed by phenotypic selection methods. Optimizing selection accuracy through the use of GS is one strategy to maximise genetic gain within a given breeding cycle. The Pearson correlation between the GEBVs and the true BVs or phenotypic values is the best technique to measure predictive accuracy in GS [97].

1.8 *Fusarium* Head Blight (FHB)

Fusarium head blight (FHB), caused by the biotrophic fungus *Fusarium graminearum* Schwabe [98], is a devastating disease in both wheat and barley (*Hordeum vulgare* L.). Infection results in grain yield losses, deterioration of grain quality, and contamination of grains with mycotoxins such as deoxynivalenol (DON) that can render the wheat unfit for human and animal consumption [99, 100]. Four types of resistance (Type I-IV) have been described for FHB in wheat [101]. Type I refers to the resistance to initial infection which is phenotyped as incidence (INC). To date, no QTL has been cloned for this type of resistance. Type II represents the resistance to spread within the wheat head which refers to the severity (SEV) phenotypic score. Some of the visual symptoms are tan or light brown lesions encompassing one or more spikelets. Some diseased spikelets may have a dark brown discoloration at the base and an orange fungal mass along the lower portion of the glume. Type III refers to the ability of the grain to prevent DON accumulation. Type IV is the resistance to *Fusarium* damaged kernels (FDK) [101-104]. INC and SEV, which are visually recorded in inoculated field trials called FHB nurseries, are used to calculate the visual rating index (VRI) using the following equation: INC x SEV/100. As such, VRI reflects a combination of Type I and Type II resistance. The percentage of FDK is obtained upon visual inspection of the harvested grains. Kernels damaged by *Fusarium* infection can become discolored, wrinkled, small and light. FDK is reported as the percentage of the grains displaying these visual characteristics. DON content, however, is a more expensive and time-consuming phenotype to obtain because it requires ground seed sample preparation and wet chemistry analytical methods [105]. Management strategies for this disease include cultural, chemical and genetic control practices. Disease control in wheat requires good agronomic practices whether conventional or direct seeding systems are used. However, the most sustainable way to control the disease is to grow resistant or tolerant varieties.

1.8.1 Breeding for FHB Resistance

Due to a scarcity of FHB resistant germplasm and the poor agronomic characteristics of the little that exists, breeding for commercial wheat cultivars with high levels of FHB resistance combined to all other required agronomic characteristics and mandatory seed quality requirements is difficult [106]. Breeding for FHB resistance in wheat is complicated because of

the polygenic nature of the resistance mechanisms, the influence of the environment on the establishment of the disease and its interaction with the genotype(s) (GxE), as well as by the complex disease assessment process [106]. Multiple genes regulate wheat's resistance to *Fusarium*. Several researchers theorized that FHB resistance is governed by minor genes [107] while others have argued that it is governed by a few major genes [108, 109].

Although additive gene effects dictate resistance in some cultivars, non-additive gene effects such as dominance and epistasis may also play a role in FHB resistance [110]. Sumai3 (Funo-germplasm and Taiwanxiaomai-landraces), a spring wheat cultivar created in China, has been utilized as a resistant parent in wheat breeding across the world [104]. Another prominent resistance source utilized in contemporary FHB breeding is Ning 7840 (Abpoba/Anhui11 x Sumai3), which is a Sumai3-derivative [111-113]. Ning 7840 has the same degree of FHB resistance as Sumai3, but it has a higher yield potential and better resistance to other wheat diseases including leaf, stem and stripe rusts, as well as powdery mildew [114]. Sumai3 and Ning7840 carry FHB resistance genes on chromosomes 1B, 2A, 2B, 3B, 5A, 6B, 6D and 7D. Resistance genes from wheat cultivar Wangshuibai were found on chromosomes 4A, 5A, 7A, 7B and 4D [115]. In addition to this, FHB resistant wheat cultivars used in breeding programs include the Brazilian wheat cultivars Frontana and Encruzilhada, the American winter wheat cultivars Ernie and Freedom, and the Japanese cultivars Shinchunaga and Nobeokabouzu komugi [114, 116, 117]. In addition, cultivars Chokwang from Korea and Fundulea 201R from Romania both exhibit resistance that is distinct from that of Sumai3 and its derivatives [114]. These examples clearly illustrate the complex polygenic nature of FHB resistance in wheat.

1.9 Scope and Purpose of this Study

Recent sequencing technologies have enabled the relatively inexpensive and fast generation of high throughput genotypic information, thereby facilitating the implementation of GP and GS to assist breeding efforts. GS takes advantage of all markers to reduce phenotyping of the progeny. Thus, GS saves not only time but also major resources usually allocated to phenotyping.

This study aims to build GS models using both GWAS-based markers and random-SNP markers to predict the BV of individuals. This approach can be used by the breeders to enhance

genetic gain through improved accuracy of GEBVs. The outcomes of this research will guide breeders in their selection of superior individuals to advance in the trials and/or to use as parents.

1.9.1 Hypotheses

1. Genomic regions associated with *Fusarium* head blight (FHB)-associated traits can be identified in diverse panels of wheat and wild relatives using genome-wide association study (GWAS) models.

2. Genomic Selection models built using trait-specific markers have higher predictive ability that those produced using random genome-wide markers.

1.9.2 Specific Objectives

The specific objectives of this study include:

1. To perform association analyses for FHB-related traits using multiple genome-wide association study (GWAS) models;
2. To identify quantitative trait nucleotides (QTNs) associated with the genetic architecture of FHB-related traits;
3. To identify candidate genes for the traits harbored at the QTN loci identified;
4. To perform genomic prediction and compare genomic prediction models based on trait-defined QTN datasets and genome-wide SNP datasets.

2.0 Genome-wide Association Analysis for *Fusarium* Head Blight Resistance in Wheat and Wild Relatives

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2.1 Abstract

Fusarium Head Blight (FHB) is one of the most common diseases impacting wheat production. The identification of resistant germplasm and the identification of quantitative trait nucleotide (QTNs) associated with FHB resistance are critical to successfully breed for improved resistance. Here a genome-wide association study (GWAS) was conducted on 279 accessions of wheat and its wild relatives to identify quantitative trait nucleotides (QTNs) linked with FHB resistance. This germplasm was divided into a spring and a winter diversity panel that included *Triticum* and *Aegilops* species. FHB incidence (INC), severity (SEV), visual rating index (VRI) and deoxynivalenol (DON) content were evaluated in the field for four years in Ottawa, Ontario and in Morden, Manitoba, Canada. After filtering, 20,501 single nucleotide polymorphisms (SNPs) were obtained through hybridization of the Illumina wheat 90K array. The probe sequences of these SNPs were mapped to the Chinese Spring reference genome sequence v2.0, and, 13,760 and 10,421 of the 20,501 SNPs were uniquely positioned onto the 21 wheat chromosomes on the spring and winter panel, respectively. Marker-trait association was performed using two single-locus and seven multi-locus GWAS models. A total of 336 unique QTNs were detected for the four FHB-related traits. Among them, 49 were deemed to be stable across datasets and have large effects (R^2), explaining ~5-42% of the variance. The 2-Mb regions that underpinned the QTNs were searched for candidate genes. From candidate gene analysis, 145 QTNs were identified flanking with 362 RGAs within their 2-Mb window flanking the QTNs. A total of eight QTNs located within the RGAs and 43 QTNs co-located with RGAs within 100 Kb up- and downstream of the associated QTN, harbored genes predicted to play roles in modulating the associated trait or traits. The remaining major QTNs were considered as potential novel FHB resistance gene loci. The four pleiotropic and other major QTNs identified herein are likely the most valuable for marker-assisted selection.

2.2 Introduction

Fusarium head blight (FHB) is a devasting fungal disease of wheat (*Triticum aestivum* L.) and other small grain cereals, affecting both grain yield and quality. In addition, *Fusarium* fungi produce secondary metabolites called mycotoxins in the kernel, resulting in wheat unfit for consumption [118]. To protect the consumers from these mycotoxins, many countries have

established maximum levels of mycotoxins in wheat flour. Several *Fusarium* species including *Fusarium graminearum*, *F. avenaceum*, and *F. culmorum* can infect wheat depending on the climatic conditions [119]. However, *F. graminearum* is the predominent causal agent of FHB in the USA, Canada, China, Europe, and other countries. In wheat, four main types of resistance have been defined for FHB. Type I is the resistance to initial infection as measured by incidence (INC). To date, no quantitative trait loci (QTLs) have been cloned for Type I resistance. Type II, the resistance to fungal spread within the wheat head, is measured as severity (SEV). Type III refers to the resistance to deoxynivalenol (DON) accumulation. Type IV, the resistance to kernel infection, is measured as the percentage of FHB damaged kernels (FDK) [101]. SEV and INC are evaluated in the field in inoculated FHB nurseries. FDK is measured on harvested and threshed grain samples. DON measurement requires ground grain samples and is achieved through laboratory analysis [120].

The most convenient and efficient way to control FHB is to breed for stable resistant cultivars [121]. However, breeding for FHB resistance is challenging due to the complex nature and challenges associated with its phenotyping, the costly artificial inoculation, the disease establishment which is highly dependant on the environmental conditions and as a consequence of the quantitative nature of the resistance mechanisms [122]. Hundreds of QTLs controlling resistance to FHB have been mapped across all wheat chromosomes using various mapping populations and validated in independent studies. Even though several hundreds of QTLs have been identified, few have been used in the production of resistant cultivars. Out of several hundreds of QTLs, only seven QTLs have been explicitly classified as Mendelized genes for FHB resistance : *Fhb1* from Sumai 3 [123], *Fhb2* from Sumai 3 [124], *Fhb3* from *Leymus racemosus* [125], *Fhb4* from Wangshuibai [126], *Fhb5* from Wangshuibai and Sumai [127], *Fhb6* from *Elymus tsukushiensis* [128], and *Fhb7* from *Thinopyrum pont*icum [129]. The major *Fhb1* QTL, conferring Type II resistance, identified in the Chinese wheat variety Sumai-3 has been mapped on chromosome 3BS by multiple independent studies. Although resistance of the Chinese spring wheat cultivar ‘Sumai 3’ (*Fhb1* QTL) remains the only FHB QTL to have been cloned and even so, the identity of the causal gene(s) remains controversial [100, 130].

The traditional way of introgressing resistance genes is by crossing donor lines to adapted lines followed by backcrossing. Although several QTLs have been identified, the main

hindrances to their introgression into breeding programs are their small effect, differential response to genetic backgrounds and associated linkage drags. Approaches such as transformation with gene cassettes and genome editing have been proposed to bypass some of these challenges but these have yet to come to fruition [131].

Novel sources of resistance to FHB in cultivated wheat and its wild relative germplasm can be identified using genome-wide association studies (GWAS) [132]. Hybridization-based arrays are a cost-effective and fast genotyping method. In wheat, several arrays such as Illumina Wheat 9K iSelect, Wheat 15K SNP array, Illumina 90K iSelect SNP array, 55K SNP array developed from a 660K array, 35K Axiom array developed from an 820K array, and the Axiom wheat 660K SNP array have been developed and widely used in wheat GWAS [133, 134].

To date, numerous GWAS have been conducted on cultivated wheat using single-locus models but these suffer from various limitations, such as identifying loci for polygenic traits such as FHB [135]. However, multi-locus GWAS can overcome some of these limitations by performing multi-dimensional genome-wide scans to identify interactions between QTNs and to identify QTNs with small effect by increasing the detection power and reduce the rate of false positive QTNs. As these multi-locus GWAS models are more recent, limited results have been reported on their use to identify genomic regions controlling FHB resistance in cultivated wheat and its wild relatives.

Most simple GWAS analyses use a generalized linear model (GLM) where SNP and population structure are fixed [136]. The mixed linear model (MLM) considers population structure as fixed and kinship as a random effect in the model to control false-positive associations [136]. The MLM treats each individual separately in the model, making it computationally demanding. GLM and MLM are single-locus models that scan one marker at a time, iteratively repeating the process for each marker. Unlike multi-locus models, these single-locus models are not specifically designed to address traits that are controlled by a large number of QTLs. These induced false negatives through overfitting that failed to identify true associations [135]. The fixed and random circulating probability unification model FarmCPU is a novel multi-locus model controlling both false negatives and false positives while being computationally very efficient. The multi-locus GWAS models mrMLM, FASTmrMLM,

FASTmrEMMA, pKWmEB, pLARmEB and ISIS EM-BLASSO are implemented in the mrMLM R package.

This study performed GWAS for four traits, namely, INC, SEV, VRI and DON, with nine different single and multi-locus GWAS models using 279 accessions of *Triticum* and *Aegilops* belonging to a spring and a winter diversity panel. These accessions were evaluated over multiple years and locations in Canada. After performing population structure analysis and identifying marker-trait associations for these four traits, candidate gene search was performed through scanning 2-Mb regions underlying the stable major QTNs.

2.3 Material and Methods

2.3.1 Plant Material

The germplasm consisted of 279 accessions divided into a spring diversity panel of 206 accessions and a winter diversity panel of 73 accessions. This diverse collection includes diploid, tetraploid and hexaploid *Triticum* species encompassing cultivated wheat, progenitor species, synthetic hexaploid wheat (SHW) and non-domesticated wheat as well as wild relatives of the *Aegilops* genus from the primary, secondary and tertiary genepools (**Appendix 1**). The panels were comprised of 51 diploid, 110 tetraploid and 118 hexaploid accessions. The 228 accessions with the AB and ABD genomes included several subspecies of *Triticum turgidum* and *T. aestivum* as well as 33 SHWs. The remaining 51 accessions belonged to the following species and genomes: *T. urartu* (A), *T. monococcum* (A^m), *Aegilops tauschii* (D), *Ae. cylindrica* (DC), *Ae. geniculata* (MU), *Ae. peregrina* (SU) and *Ae. biuncialis* (UM). The species names and genome symbols follow the nomenclature previously described [137, 138].

2.3.2 Phenotyping of FHB traits

INC, SEV, VRI and DON were evaluated for the spring diversity panel in FHB nurseries located in Morden, Manitoba and Ottawa, Ontario, Canada. INC, SEV and VRI were collected in Morden in 2016, 2017, 2018 and 2019 and in Ottawa in 2016, 2017 and 2018, while DON was

obtained from Morden in 2017 and Ottawa in 2017 and 2018. The winter panel was evaluated in Morden in 2017, 2018 and 2019, and DON data was obtained in 2017. The location-year datasets were defined as separate environments for the spring and winter panels which were analyzed separately and as a combined panel.

In Morden and Ottawa, 65-70 seeds per accession were planted in single 1m-long rows spaced 30 cm apart. Entries were sown using a randomized completed block design with two replicates. Inoculation was initiated at the 4-5 leaf stage using a mixture of four aggressive isolates of *F. graminearum*: HSW-15-39 (3-ADON), HSW-15-87 (3-ADON), HSW-15-27 (15-ADON) and HSW-15-57 (15-ADON). Colonized corn grains were broadcasted between the rows at a rate of 8 g per row twice weekly starting when the earliest lines were at the 4-5 leaf stage. A mist irrigation system was applied three times a week to promote FHB symptom development using Cadman Irrigation travellers with Briggs booms.

FHB symptoms were scored at approximately 21 days post-anthesis. The proportion of infected spikes per row (INC) and the average of infected spikelets per head (SEV) were recorded using a 0-100 scale [139]. The VRI was calculated for each line using the following equation: INC x SEV/100. From each replicate, one gram of flour was used for DON analysis using ELISA tests [140-143].

2.3.3 Genotyping

DNA extraction, quantification, hybridization to the wheat 90K array, and genotype calling with Genome Studio software v2.0.4 (Illumina, San Diego, CA, USA) were previously described [144]. A total of 20,501 SNPs with a call rate >80%, a minor allele frequency (MAF) >5% and heterozygosity <5% were obtained. The probe sequences (~100 bps) of these SNPs were mapped to the Chinese Spring reference genome v2.0 [16] using BLAST and custom Perl scripts. Of the 20,501 SNPs, the 13,760 SNPs uniquely positioned onto the 21 wheat chromosomes were used for downstream analysis of the spring and combined diversity panel, and a subset of 10,421 SNPs (MAF<0.05) was used for the winter panel.

2.3.4 Statistical Analysis

Broad-sense heritability (H^2) and best linear unbiased prediction (BLUP) values for each trait were estimated using the R package Lme4 [145]. The R command with the mixed linear model equation, lmer(Trait ~ Location + (1| Genotype) + (1| Year) + (1| Genotype:Location) + (1| Genotype:Year) + (1| Genotype:Year:Location)) was used to calculate the BLUP values for each trait across locations and years and estimate the variance components where Location was considered as a fixed effect and Genotype and Year as random effects. H^2 was estimated as $\frac{\sigma_g^2}{\sigma_g^2+\sigma_{gl/l}^2+\sigma_{gyl/yl}^2+\sigma_e^2}$, where σ_g^2 and σ_e^2 are the genetic and error variances, respectively; σ_{gl}^2 and σ_{gyl}^2 are the interaction variances between Genotype and Location, and between Genotype, Year and Location, respectively, while *y* and *l* are the number of years and the number of locations, respectively [146]. Genomic heritability (h^2) represents the proportion of additive genetic variance component in the total phenotypic variance; it was estimated based on the 13,760 SNPs in spring panel, and 10,421 in winter panel using the R package sommer with the GBLUP model [81]. H^2 and h^2 were calculated for all three panels separately.

2.3.5 Population Structure

One of the factors affecting GWAS is population structure [147]. Here the population structure of each panel was evaluated to determine the genetic relationships between accessions using the 13,760 and 10,421 SNPs in the spring, and winter panel respectively. To find the most suitable method to measure population structure, STRUCTURE [148], PCA-based method LEA [149] and PCAdapt [150] were compared. Comparative analysis showed a clearer outcome with STRUCTURE, which calculates the number of components based on the second-order rate of change of the likelihood (ΔK). The ΔK shows a clear peak at the true value of *k* for the respective number of subpopulations. STRUCTURE provides the Q-matrix, which gives a probability that an individual belongs to a subpopulation. The number of *k* was tested for one to ten subpopulations for the spring, winter and combined panels, separately.

2.3.6 Genome-wide Association Study (GWAS)

A basic assumption that must be met to analyze the combined data of the two panels is that the phenotypic traits have the same or similar experimental errors for the accessions grown across panels. To meet this assumption, we performed additional statistical adjustments based on the average performance in different years and locations known as BLUP. As a result, BLUP estimates of the four traits across years and locations were used for GWAS. GWAS was performed for INC, SEV, VRI and DON content separately for the spring, winter and combined panels. The multi-locus models mrMLM [45], FASTmrMLM [46], FASTmrEMMA [47], pKWmEB [48], pLARmEB [45], and ISIS EM-BLASSO [49] implemented in the R package mrMLM [47], the multi-locus model FarmCPU [44], and the single-locus models GLM and MLM [41] implemented in the rMVP package [42] were used to determine marker-trait associations. The thresholds of significance for marker-trait associations for FarmCPU, GLM and MLM were determined by a critical *P* value subjected to the Bonferroni correction ($\alpha = 0.05/n$) where *n* is the number of markers. A log of odds (LOD) score of 3.0 was used for the six multi-locus models of the mrMLM package [50].

2.3.7 Resistance Gene Analogs (RGAs) and Candidate Genes

Resistance gene analogs (RGAs) are potential disease resistance genes in plants that contains specific conserved domains and motifs. To identify candidate genes co-located with the identified QTNs, identification of all potential RGAs from the Chinese Spring wheat reference genome sequence v2.0 was performed using RGAugury, a pipeline for large-scale genome-wide RGA prediction [151]. For each QTN identified, the RGAs located within a 2-Mb region (1-Mb on either sides of the QTNs) were extracted [152]. These RGAs were further investigated for their putative role in abiotic and biotic stresses, especially disease response, using the wheatmap database (https://www.wheatgmap.org/) by loading the individual candidate gene name into the database.

2.4 Results

2.4.1 Phenotypic Evaluation

Pearson's correlation coefficients (*r*) between the four FHB-related traits ranged from 0.42 to 0.94 for the spring panel (**Figure 2.1A**), 0.0086 to 0.85 for the winter panel (**Figure 2.1B**), and 0.41 to 0.92 for the combined panel (spring + winter) (**Figure 2.1C**). At a significance level of 0.001, SEV had the highest positive correlation with VRI in the spring (0.94) and combined (r = 0.92) panels, whereas correlations of the field visual ratings with DON content ranged from 0.41 to 0.49 for these panels (**Figures 2.1A and 2.1C**). All correlations were positive. In the winter panel, correlations between INC, SEV and VRI were lower than in the spring panel but were still significant and ranged from 0.73 to 0.87 but the correlations with DON were much lower ranging from 0.086 to 0.19 (**Figure 2.1B**). The heritability of the four traits measured in the three panels is summarized in **Table 2.1**. The highest broad-sense heritability (H^2) was observed for INC in all panels with a maximum of 0.67 in the combined panel. H^2 was lower for all traits in the winter panel compared to the spring panel, and was particularly low for SEV (0.10).

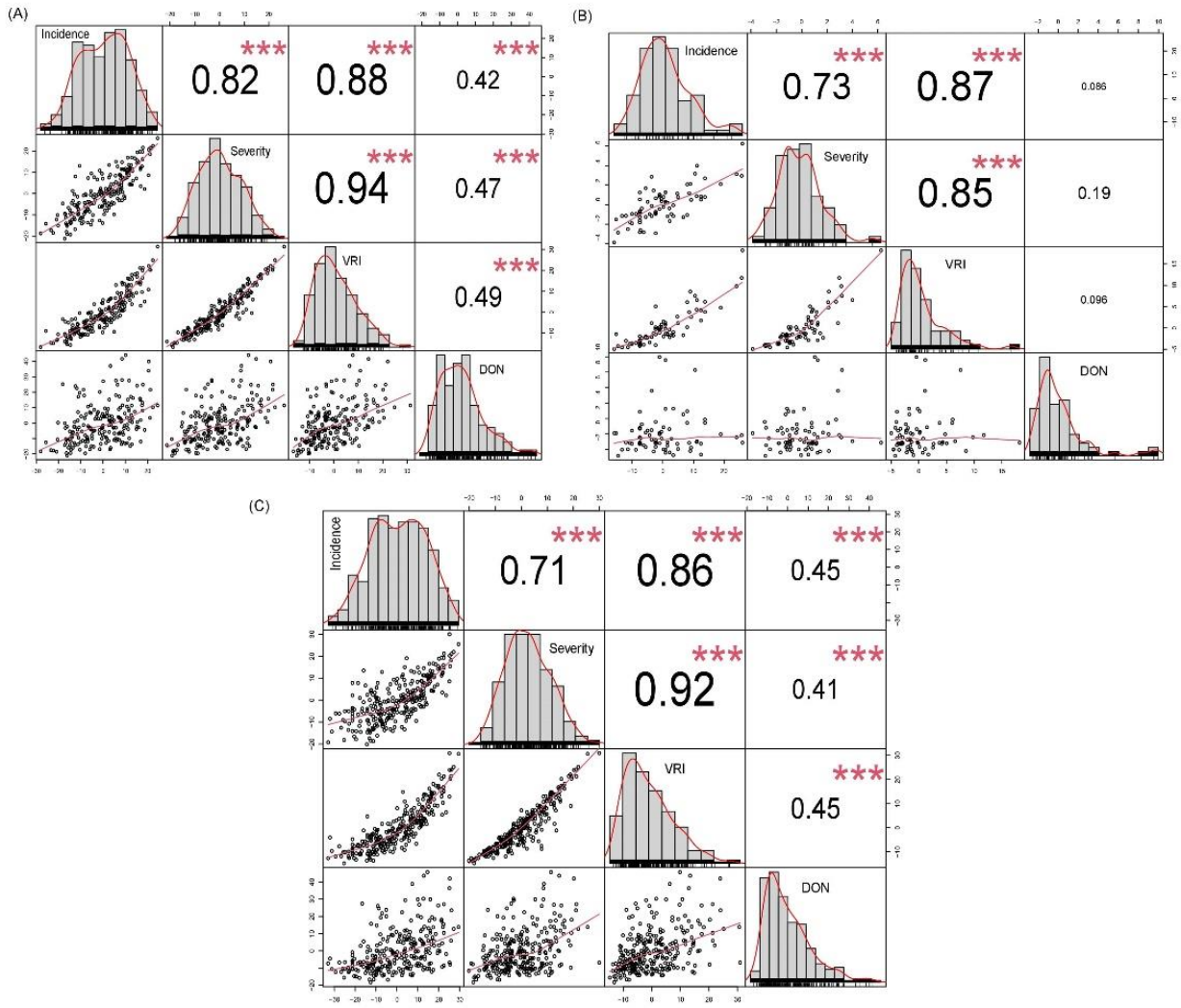

Figure 2. 1 Histograms of phenotypic data for incidence (INC), severity (SEV), visual rating index (VRI) and deoxynivalenol (DON) content and their Pearson's correlation coefficients for the following diversity panels: (**A**) spring, (**B**) winter and (**C**) combined. The lower left corners of each panel are scatter plots of the correlated traits. BLUP values across years and locations were used. *** indicate significance at the *P*-value ≤ 0.001.

Table 2.1 Broad-sense and genomic heritability of four FHB-related traits for the combined, spring and winter diversity panels.

Trait[1]	**Spring**		**Winter**		**Combined**	
	H^2	h^2	H^2	h^2	H^2	h^2
INC	0.58	0.60	0.47	0.50	0.67	0.58
SEV	0.42	0.60	0.10	0.56	0.41	0.58
VRI	0.50	0.63	0.20	0.77	0.48	0.64

Trait[1]	Spring		Winter		Combined	
	H^2	h^2	H^2	h^2	H^2	h^2
DON	0.35	0.54	0.25	0.87	0.34	0.60

[1] INC: incidence; SEV: severity; VRI: visual rating index; DON: deoxynivalenol; [2] H^2: broad-sense heritability: h^2: genomic heritability

2.4.2 Population Structure

PCAdapt, Landscape and Ecological Association (LEA) and STRUCTURE were used to analyze the population structure of the three genetic panels. Scree plots graphically represented the number of subpopulations predicted from the three methods in each panel (**Appendix 2**). PCAdapt and LEA did not provide a clear assessment of the number of populations but STRUCTURE (ΔK) showed clear peaks at the true value of k = 8 for the spring, k = 5 for the winter and k = 4 for the combined panels, respectively, which did not disagree with the PCAdapt and LEA results (**Appendix 2**). Thus, the result from STRUCTURE were used as covariate in the mixed models for GWAS.

2.4.3 QTN Identification

To understand if the 13,760 SNPs in the spring and combined panels, and 10,421 SNPs in the winter panel can explain the variation of four FHB-related traits, genomic heritability (h^2) for the four traits were estimated (**Table 2.1**). The h^2 values ranged from 0.50 to 0.87 for the traits within each panel, implying that a large portion of phenotypic variation of the four FHB-related traits can be explained by the SNPs, or some potential QTNs.

GWAS was carried out separately for all four traits and all three panels using BLUP estimates of each trait and the 13,760 and 10,421 SNPs in the spring, and winter panel respectively that were mapped to the wheat Chinese Spring reference sequence V2.0. A total of 107, 157, 174 unique QTNs in the spring, winter, and winter panels, respectively, were identified using the two single-locus and seven multi-locus models for all four FHB-related traits (**Table 2.2)**.

Table 2. 2 Number of quantitative trait nucleotides (QTNs) detected in all panels for incidence (INC), severity (SEV), visual rating index (VRI) and deoxynivalenol (DON) content using ten genome-wide association study (GWAS) models.

Trait[1]	Diversity panel		
	Spring	Winter	Combined
INC	42	5	32
SEV	21	57	18
VRI	12	77	72
DON content	32	18	52
Total unique QTNs	107	157	174

[1] INC: incidence; SEV: severity; VRI: visual rating index; DON: deoxynivalenol

Out of 107 significant QTNs in spring panel, 58 were detected by multiple models. The proportion (R^2) of variance explained by each QTN ranged from 0.008–21% (**Appendix 3**). Similarly, in the winter panel, 3 of 157 significant QTNs were detected by more than one model. Out of 7 QTNs identified for DON content in winter panel, QTN 65160_RFL_Contig5906_985 on chromosome 1A explained the highest proportion of the variance (42%), while R^2 of the remaining QTNs ranged from 0.001–38% (**Figure 2.2 & Appendix 4**). Similarly, the QTN 65160_RFL_Contig5906_985 on chromosome 1A explained the second highest proportion of the variance (38%) for VRI by the model FarmCPU (**Appendix 4**). The model with correction for the population structure was superior to other models according to the quantile-quantile (QQ) plot generated, hence the probability (*P*) values of this model were used for determining the significant SNPs (**Figure 2.3**). In the combined panel, the proportion of variance explained by each QTN ranged from 0.0005–25% (**Appendix 3**). Among those, 68 QTNs were identified by more than one model.

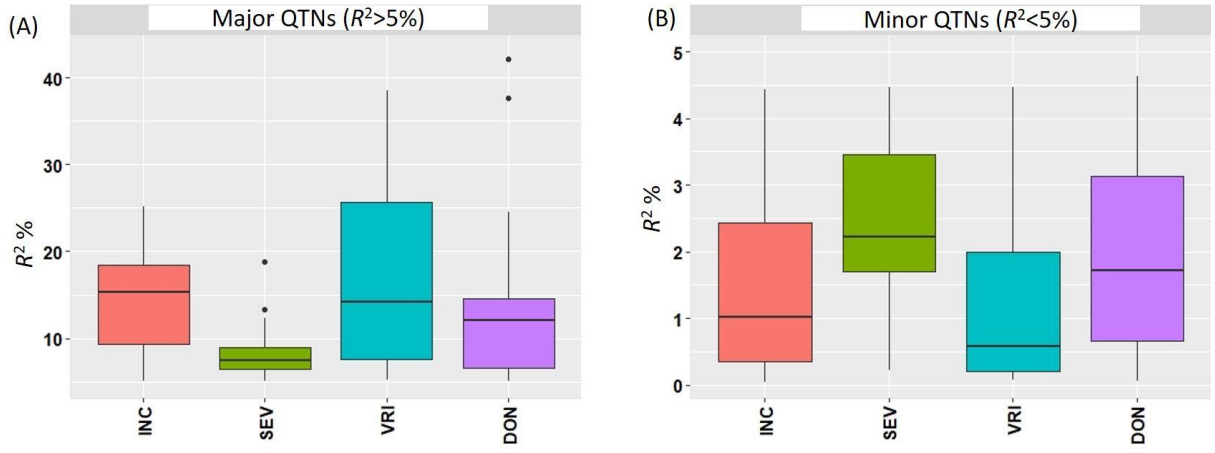

Figure 2.2 Boxplots representing the genetic variance explained (R^2) by **(A)** major and **(B)** minor quantitative trait nucleotides (QTNs) ranging from 0.001 to 42.09% for incidence (INC),

severity (SEV), visual rating index (VRI) and deoxynivalenol (DON) content detected using nine genome-wide association study (GWAS) models.

Using BLUP estimates of each trait, the single-locus general linear model (GLM) identified the most QTNs with 51-173 depending on the panel. In contrast, the multi-locus model FarmCPU identified the fewest in the combined panel (8) and the single-locus model MLM identified none in both spring and winter panels. Overall, the combined and spring panels shared 57 QTNs, whereas the combined and winter panels shared 46 (**Appendix 3**). Moreover, no QTN were shared between winter panel and spring panel as the population used in between these panel differ from each other in large extent (**Appendix 3**). In combined panel, 14 QTNs were pleiotropic with two or three traits; four of them with three traits, nine QTNs were pleotropic in spring panel; four of them with three traits whereas only 1 pleiotropic QTN was found in winter panel. Among this of these QTNs, four QTNs (12504_BS00110278_51, 13266_CAP12_c1981_78, 46344_Kukri_c53506_1141, 81112_wsnp_Ra_c34203_42948357) were identified from combined and spring panels (**Appendix 5**). QTNs identified by each model for the four traits are shown in **Appendix 6**.

2.4.4 Candidate Genes

A total of 3,903 RGAs were identified from the Chinese Spring wheat reference genome v2.0 using RGAugury. These RGAs belonged to four types of RGAs: receptor-like protein (RLP), receptor-like kinase (RLK), transmembrane coiled-coil protein (TM-CC), nucleotide-binding and leucine-rich repeat receptors (NLR) genes (including TNL, CN, NL and others), and coiled-coil (CC) domain-containing (CNL) subfamilies. Of these 3,903 RGAs, 362 were found in the 2-Mb window surrounding 145 of the QTNs identified (**Appendix 7**). Among them, eight QTNs were located within the RGAs *per se*: 79457_wsnp_Ex_rep_c68712_67571580 (R^2=17.96%), 42938_Kukri_c24194_2659 (16.76%), 56084_RAC875_c27611_467 (7.73%), 51875_Ra_c33766_656 (4.11%), 80328_wsnp_Ku_c2797_5284087 (2.57%), 18201_D_GBB4FNX01CNX9D_254 (2.43%), 40694_Kukri_c119_295 (<1%), 6908_BS00022107_51, and 40510_Kukri_c110735_106 (< 1%). In addition, 43 were located within 100 Kb of the QTNs.

We further analyzed candidate genes co-located with the 49 stable QTNs with large effect (R^2 = 5.12 – 42.09%). A total of 122 RGAs were found to be co-located with the 49 large-effect QTN within 2-Mb window flanking the QTNs. Each QTN had one ore more co-located RGAs, but only the ones closest to the QTNs were listed in **Table 2.3**. Among the 49 QTNs, three were located within the RGAs while the remaining located within 1 Mb (**Table 2.3**). Several QTNs on chromosome 1B form an LD block that can be considered as a single QTN. This 700-Kb region (462,183,400-462,883,977), where six QTNs for INC (R^2 = 11.31-19.82%) were detected, harbors candidate gene *TraesCS1B01G259000* predicted to encode a TM-CC protein (**Table 2.3**). Other disease-resistance-related genes for other QTNs identified encode NBS-LRR proteins, RPM1 proteins, TM-CC proteins, and serine/threonine kinase receptors.

The major Chr1A:16958960 QTN locus (65160_RFL_Contig5906_985) associated with DON content (R^2 = 42.09%) harbored a gene (*TraesCS1A01G031800*) predicted to encode a receptor-like kinase (RLK) (**Table 2.3**). This locus has not previously been associated with DON accumulation. Among the unique QTNs identified with all models, four were pleotropic with major QTN effects on two traits; these harbored genes predicted to encode NBS-LRR proteins, RLKs and serine/threonine-protein kinases (**Table 2.4)**.

Table 2. 3 List of 49 stable quantitative trait nucleotides (QTNs) explaining more than 5% of the phenotypic variance for four FHB resistance traits and putative co-located candidate genes.

Trait[1]	QTN	Chr[2]	Position	Gene/annotation	Functional annotation[3]	QTN to gene distance (bp)	R^2 (%)
INC	12462_BS00110052_51	1D	342788950	*TraesCS1D01G247800*	TM-CC	94401	17.44
DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G346300*	RLK	330529	10.39
DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345700*	RLK	477893	10.39
DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345500*	RLK	494538	10.39
DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345400*	RLK	505992	10.39
DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345100*	RLK	611658	10.39
DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345000*	RLK	633184	10.39
DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G344600*	RLK	771934	10.39
DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G344400*	RLK	840298	10.39
VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G486800*	RLK	169583	38.46
VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G486400*	RLK	283319	38.46
VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G486300*	RLK	335284	38.46
VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G486200*	RLK	343954	38.46
VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G485700*	RLK	765187	38.46
VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G485400*	RLK	967045	38.46
INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G457600*	RLK	216686	6.21

Trait[1]	QTN	Chr[2]	Position	Gene/annotation	Functional annotation[3]	QTN to gene distance (bp)	R^2 (%)
INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G457400*	RLK	229288	6.21
INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G457300*	RLK	252722	6.21
INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G456800*	RLK	825810	6.21
INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G456700*	RLK	863007	6.21
INC	34012_GENE-4564_387	7A	699200157	*TraesCS7A01G507700*	RLK	94366	7.08
INC	34012_GENE-4564_387	7A	699200157	*TraesCS7A01G507500*	RLK	158108	7.08
INC	34012_GENE-4564_387	7A	699200157	*TraesCS7A01G507400*	RLK	183080	7.08
INC	34012_GENE-4564_387	7A	699200157	*TraesCS7A01G507100*	RLK	314963	7.08
INC	35131_IAAV6011	1B	682639244	*TraesCS1B01G460100*	CN	200412	10.01
INC	37431_JD_c4128_277	3B	73391914	*TraesCS3B01G093800*	RLK	551594	9.07
DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G168400*	NL	2436	6.43
DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G203600*	NL	3864	6.43
DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G213400*	CN	18927	6.43
DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G194700*	CN	85659	6.43
DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G219700*	CN	95793	6.43
DON	39158_Ku_c3169_1250	6A	618329085	*TraesCS6A01G414300*	NL	414235	6.43
DON	39158_Ku_c3169_1250	6A	618329085	*TraesCS6A01G414400*	CNL	424027	6.43
DON	39158_Ku_c3169_1250	6A	618329085	*TraesCS6A01G415000*	CN	595973	6.43
VRI	39160_Ku_c31764_136	4D	48541991	*TraesCS4D01G073300*	RLK	103064	25.64
VRI	39160_Ku_c31764_136	4D	48541991	*TraesCS4D01G074100*	RLK	590673	25.64
VRI	42062_Kukri_c19017_1039	1D	487714262	*TraesCS1D01G440300*	RLP	916886	7.58
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G487300*	NL	0	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G487000*	RLK	46055	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488000*	RLK	93458	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488200*	NL	174722	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G486500*	CNL	198340	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488400*	NL	254501	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488700*	CN	317324	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488800*	CNL	343532	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488900*	RLK	372156	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G489800*	CN	626723	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G490200*	NL	683358	16.76
DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G490500*	NL	775036	16.76
INC	43566_Kukri_c2842_248	6D	331186031	*TraesCS6D01G219000*	RLK	639208	18.34
SEV	44603_Kukri_c36747_195	5D	340132967	*TraesCS5D01G230000*	RLK	35666	8.47
SEV	44603_Kukri_c36747_195	5D	340132967	*TraesCS5D01G230300*	TM-CC	154597	8.47
INC	45329_Kukri_c43306_282	7D	4394602	*TraesCS7D01G007600*	NL	289893	25.2
INC	45329_Kukri_c43306_282	7D	4394602	*TraesCS7D01G007000*	NL	426217	25.2
INC	45329_Kukri_c43306_282	7D	4394602	*TraesCS7D01G006800*	NL	467351	25.2
DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G042500*	NL	349140	16.38
DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G042600*	NL	360251	16.38
DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G042700*	NL	432113	16.38
DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G043000*	CNL	467813	16.38
DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G044100*	CNL	854485	16.38

Trait[1]	QTN	Chr[2]	Position	Gene/annotation	Functional annotation[3]	QTN to gene distance (bp)	R^2 (%)
DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G044200*	NL	861844	16.38
DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G044500*	NL	919670	16.38
VRI	47776_Kukri_c7874_1096	5A	535804823	*TraesCS5A01G323800*	RLK	377540	25.64
VRI	47776_Kukri_c7874_1096	5A	535804823	*TraesCS5A01G323900*	RLK	385996	25.64
VRI	55857_RAC875_c25839_225	6D	313735680	*TraesCS6D01G206100*	RLK	136696	25.64
VRI	55857_RAC875_c25839_225	6D	313735680	*TraesCS6D01G205900*	RLK	290151	25.64
SEV	56084_RAC875_c27611_467	2B	9233744	*TraesCS2B01G001600*	RLK	0	7.73
DON	57507_RAC875_c39339_400	3B	817272964	*TraesCS3B01G567500*	RLK	649227	5.66
VRI	59116_RAC875_c55445_92	6B	721147245	*TraesCS6B01G454800*	RLK	471266	29.01
VRI	59116_RAC875_c55445_92	6B	721147245	*TraesCS6B01G454900*	RLK	473981	29.01
DON	60140_RAC875_c6798_467	1A	538108963	*TraesCS1A01G354100*	CN	642146	5.14
DON	60140_RAC875_c6798_467	1A	538108963	*TraesCS1A01G354200*	CN	646687	5.14
VRI	60475_RAC875_c77534_451	5B	671028119	*TraesCS5B01G502500*	RLK	623556	7.58
VRI	60475_RAC875_c77534_451	5B	671028119	*TraesCS5B01G502400*	RLK	645212	7.58
INC	60785_RAC875_c8494_93	4A	500153212	*TraesCS4A01G207800*	RLK	7414	25.18
DON	610_BobWhite_c14222_571	2B	104978464	*TraesCS2B01G129700*	NL	50121	13.17
VRI	61578_RAC875_rep_c106337_522	5D	393494214	*TraesCS5D01G292500*	RLK	667086	29.01
DON	61616_RAC875_rep_c106596_127	2B	29764155	*TraesCS2B01G054500*	RLK	177271	7.15
DON	61616_RAC875_rep_c106596_127	2B	29764155	*TraesCS2B01G053500*	RLK	456094	7.15
DON	61616_RAC875_rep_c106596_127	2B	29764155	*TraesCS2B01G056500*	NL	786443	7.15
DON	61616_RAC875_rep_c106596_127	2B	29764155	*TraesCS2B01G057100*	TM-CC	975923	7.15
VRI	64957_RFL_Contig5277_888	2A	775589238	*TraesCS2A01G580900*	RLK	987841	5.76
DON	65160_RFL_Contig5906_985	1A	16958960	*TraesCS1A01G031800*	RLK	828996	42.09
VRI	69845_Tdurum_contig29607_294	6D	483997120	*TraesCS6D01G384800*	RLP	315208	25.64
INC	70065_Tdurum_contig30621_328	7A	71357685	*TraesCS7A01G111100*	CN	5258	17.12
INC	70065_Tdurum_contig30621_328	7A	71357685	*TraesCS7A01G111000*	CN	5483	17.12
INC	70065_Tdurum_contig30621_328	7A	71357685	*TraesCS7A01G111200*	CN	37773	17.12
VRI	7136_BS00022524_51	1D	10383887	*TraesCS1D01G025200*	CN	432705	13.95
VRI	7136_BS00022524_51	1D	10383887	*TraesCS1D01G026000*	CNL	697944	13.95
VRI	7136_BS00022524_51	1D	10383887	*TraesCS1D01G021200*	NL	887992	13.95
VRI	7136_BS00022524_51	1D	10383887	*TraesCS1D01G021000*	CNL	940173	13.95
VRI	71614_Tdurum_contig45504_166	6A	104735561	*TraesCS6A01G130200*	RLP	3900	25.64
VRI	71614_Tdurum_contig45504_166	6A	104735561	*TraesCS6A01G130000*	RLP	14323	25.64
VRI	71614_Tdurum_contig45504_166	6A	104735561	*TraesCS6A01G129900*	RLP	294166	25.64
DON	74007_Tdurum_contig97656_120	3B	24979593	*TraesCS3B01G040100*	CN	143317	20.19
DON	74007_Tdurum_contig97656_120	3B	24979593	*TraesCS3B01G039200*	CNL	475733	20.19
SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G401300*	RLP	30111	5.12
SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G401200*	RLP	34777	5.12
SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G401100*	RLP	84233	5.12
SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G401000*	RLP	86386	5.12
SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G400900*	RLP	113324	5.12
SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G400800*	RLP	115806	5.12
SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G400700*	RLP	237154	5.12
SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G400500*	TM-CC	316978	5.12

Trait[1]	QTN	Chr[2]	Position	Gene/annotation	Functional annotation[3]	QTN to gene distance (bp)	R^2 (%)
VRI	79013_wsnp_Ex_rep_c101269_86664147	3A	712607958	*TraesCS3A01G479400*	CN	983723	14.18
INC	79457_wsnp_Ex_rep_c68712_67571580	1B	462183400	*TraesCS1B01G259000*	TM-CC	0	17.96
VRI	80485_wsnp_Ku_c4045_7380115	1A	553440047	*TraesCS1A01G379100*	CN	106750	6.03
VRI	8117_BS00037784_51	6B	672525118	*TraesCS6B01G388800*	CN	190865	7.58
SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G060000*	RLK	15888	8.75
SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G060100*	RLK	58312	8.75
SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G060200*	RLK	69206	8.75
SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G059400*	RLP	157389	8.75
SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G061400*	RLK	427107	8.75
VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G365500*	RLK	94542	7.58
VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G365400*	RLK	137622	7.58
VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G366000*	RLK	303408	7.58
VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G366200*	RLK	308246	7.58
VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G366300*	RLK	383507	7.58
VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G366600*	RLK	656634	7.58
VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G364500*	RLK	768805	7.58
VRI	8809_BS00060541_51	6B	165407501	*TraesCS6B01G157800*	RLP	616475	8
SEV	9955_BS00067096_51	5A	36408984	*TraesCS5A01G037500*	RLP	374845	18.81

[1] INC, incidence; SEV, severity; VRI, visual rating index; DON, deoxynivalenol content; [2] Chr, chromosome; [3] Toll/interleukin-1 receptor-like domain; CNL, CN; RLK, receptor-like protein kinase; RLP, receptor-like protein; TM-CC, transmembrane coiled-coil protein; unknown/random; RNL.

Table 2. 4 Quantitative trait nucleotides (QTNs) that were pleotropic for two of the four FHB traits

Quantitative trait nucleotide (QTN)	Trait[1]	Chr[2]	Position	Gene ID	Gene annotation	QTN to gene distance (bp)	R^2 (%)
12462_BS00110052_51	INC, DON	1D	342788950	*TraesCS1D01G247800*	NBS-LRR disease resistance protein	94,401	17.44
13266_CAP12_c1981_78	INC, SEV	1B	462097527	*TraesCS1B01G259000*	Serine/threonine-protein kinase	78,817	18.43
79457_wsnp_Ex_rep_c68712_67571580	INC, VRI	1B	462183400	*TraesCS1B01G259000*	Receptor-like protein kinase	0	17.96
81493_wsnp_RFL_Contig3344_3442711	SEV, VRI	3A	37135958	*TraesCS3A01G060000*	Receptor-like protein kinase	15,888	8.75

[1] INC, incidence; SEV, severity; VRI, visual rating index; DON, deoxynivalenol content; [2] Chr, chromosome

2.5 Discussion

Fusarium head blight is a devastating disease of wheat worldwide, and cultivars with higher levels of resistance are desperately needed to prevent its incidence. The discovery of QTNs linked to resistance has the potential to assist in breeding for improved resistance. In this study, a collection of 279 accessions belonging to a spring and a winter panel was evaluated in multiple environments to further explore the genetic basis of FHB resistance in wheat and its wild relatives. Identifying FHB resistance loci in wheat accessions required multi-location and multi-year data because a large proportion of the variance for FHB-related traits is accounted by environment and genotype x environment interactions. The identification of QTNs has been made easier by recent developments in genome-wide marker development and statistical models even for traits as complex as FHB [153]. These new technological advances have facilitated the production of large-scale genome-wide markers and have consequently resulted in the proliferation of genome-wide association studies.

To date different mapping populations and mapping strategies have been used to identify QTLs for FHB resistance. Only *Fhb1-Fhb7* QTLs have been have been Mendelized through precise mapping in biparental populations out of more than 250 documented QTLs imparting FHB resistance. Among those, *Fhb1* is the only one for which causal gene(s) have been isolated [154, 155]. Differences in gene expression levels are one possible cause of variation in quantitative traits. On the basis of numerous sorts of evidence including changed expression in response to infection, several genes and gene families have been suggested as playing essential roles in plant defence [156]. Many of them play important roles in plant defense, including altered expression in response to infection. RGAs have been used to discover candidate genes for resistance in a variety of plant species [157].

The most common kind of known resistance gene encodes intracellular immune receptors with NBS-LRR domains, many of which also possess a coiled-coil (CC) N-terminal motifs. Pathogen identification and the beginning of downstream signalling cascades are crucial functions of these genes. A total of 661 to 1,560 full-length NBS-LRR genes have been reported in the wheat genome, which is more than any other plant species [158, 159]. Six of the seven QTNs on chromosome 1B are linked to a gene that encode a TM-CC protein explaining a significant proportion of the phenotypic variation for INC. 80997_wsnp_Ra_c2027_3945764

was the most prominent QTNs of this region and its favorable allele was present in some interesting germplasm, including some SHWs.

A number of QTNs present in genes coding for other known resistance proteins in wheat and other plant species were identified. The highest number of QTNs within genes encoding RLK proteins was in the winter panel for VRI. The winter panel was predominantly a collection of SHWs and wild relative species. 14615_CAP8_c2439_210 was be the most prominent VRI linked QTN discovered in the winter panel. This QTN was found on the distal end of chromosome 2D, within a RLK gene that identifies the bacterial epitope elf18, which is derived from elongation factor-Tu, known to activate the plant's defense system [160].

In this study, 362 RGAs linked to FHB resistance were identified. The QTNs co-located within the same LD block on a 700-Kb region on chromosome 1B explained 10–20% of the phenotypic variation for INC. QTNs for INC have not been reported at this locus previously. This region is therefore of particular interest because few QTLs for Type I resistance have been identified to date [161].

The predicted proteins for the 1B syntenic block include the LETM1 and the mitochondrial EF-hand domain-containing protein 1 previously shown to be associated with plant disease resistance. To date, few studies have been conducted to investigate the biological roles of the LETM1 orthologues in filamentous fungi. One study showed that the LETM1 mutation slows down the dynamic change in the mitochondria, which plays a crucial role in mitochondrial mobility [162]. In plant-pathogen interactions, reactive oxygen species (ROS) play a key role in suppressing pathogen invasion by triggering an oxidative burst in the host plant. During a successful infection, pathogens must deal with ROS generated by plants [163]. Furthermore, *F. graminearum* has developed its oxidative stress response (OSR) system to use it as a signal to initiate DON production, which is a key virulence component during infection [164, 165]. One study found that the deletion of LETM1 nearly totally abolished the production of ROS in mitochondria, and resulted in a decrease in DON biosynthesis both *in vitro* and *in planta* [162]. The mitochondria integrity and endogenous ROS generation in *Fusarium* species are critical for DON formation and pathogenicity [162, 166].

Therefore, the QTNs with major, stable and/or pleotropic effects hold potential for germplasm improvement. They can be pyramided and combined with smaller effect QTNs, a

strategy that has shown some success in improving FHB resistance in wheat [98]. The use of multiple GWAS models is an effective method for identifying QTNs and candidate genes for FHB resistance

2.6 Conclusion

In the present study, a total of 336 QTNs including 49 stable and large effect QTNs were identified for four FHB traits using nine statistical GWAS models. We identified these QTNs co-localized with 423 RGAs within the pre-defined 2-Mb window flanking the QTNs whereas the maximum distance of a QTN to a gene distance is 1 Mb. A few major QTNs co-located on co-localized with RGAs were new for the associated trait, hinting at their potential in breeding. The QTNs of large effect can be developed into diagnostic markers for marker-assisted selection. Together, the entire 336 QTN dataset have potential in genomic selection as well as was shown in other crops [167, 168].

Article

3.0 Genomic Prediction for Fusarium Head Blight Resistance in Wheat and Wild Relatives

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3.1 Abstract

Improving *Fusarium* head blight (FHB) resistance is a central goal in most wheat breeding programs. Genomic selection (GS), which uses dense genome-wide or quantitative trait nucleotide (QTN) markers to estimate breeding values for quantitative traits, is a promising strategy that could speed up gains towards FHB resistance. The objectives of this study were to compare genomic prediction models and input genotype datasets for their ability to accurately estimate the breeding values of FHB traits. The germplasm consisted of 279 accessions of *Triticum* and *Aegilops* species divided into a spring (206) and a winter (73) diversity panel that were genotyped for 13760 single nucleotide polymorphism (SNP) for spring panel and 10,421 SNPs markers for winter panel markers using the wheat 90K array and evaluated for FHB resistance in field trials at two locations for 2-3 years. Here, a genome-wide association study (GWAS) was performed for incidence (INC), severity (SEV), visual rating index (VRI) and deoxynivalenol (DON) content independently for the spring, winter and combined panels. The QTN obtained from GWAS were used to perform genomic prediction (GP) analyses. The mean predictive ability ranged from 0.50-0.83 for INC, 0.54-0.82 for SEV, 0.61-0.85 for VRI, and 0.49-0.80 for DON across all three panels. Among all models, RR-BLUP achieved the best predictive ability, whereas GBLUP, BRR, Bayes A, B & C and Bayes LASSO were slightly superior to the remaining models. For most datasets, the spring panel produced the highest accuracies, while the winter panel delivered the lowest. The results also shows that the predictive ability obtained using QTN-based GP yielded slightly higher *r* estimates for all traits compare to the random genome-wide SNP marker set for all three panels. We conclude that the use of QTN-based single and multi-locus GWAS models simplifies the selection of suitable genomic variants for GP which is a promising method for improvement of FHB resistance in wheat that should be implemented in breeding.

Keywords: wheat; *Fusarium* head blight (FHB); genome-wide association study (GWAS); genomic selection (GS); genomic prediction (GP)

3.2 Introduction

Fusarium head blight (FHB), caused by the biotrophic fungus *Fusarium graminearum* Schwabe [98], is a devastating disease in both wheat (*Triticum aestivum* L.) and barley (*Hordeum vulgare* L.). Infection results in grain yield losses, deterioration of grain quality, and contamination of grains with mycotoxins including deoxynivalenol (DON), making the wheat unfit for human and animal consumption [99, 100, 169]. Resistance to FHB can be grouped into four types (Types I-IV) in wheat [101]. Type I, called resistance to initial infection, is phenotyped as incidence (INC). To date, no gene has been cloned for this type of resistance. Type II represents the resistance to spread within the wheat head, referring to the severity (SEV) which is scored phenotypically. INC and SEV, which are visually recorded in FHB nurseries, are used to calculate a visual rating index (VRI). Type III refers to the ability of the grain to prevent DON accumulation [102-104]. The evaluation of DON content is relatively expensive and time-consuming since it requires sample preparation and wet chemistry analytical methods [105]. Finally, Type IV, resistance to kernel damage, represents the proportion of FHB damaged kernels (FDKs) in the harvested grains which is measure its visual inspection.

Breeding for FHB resistance is challenging because good phenotyping data depends on establishing a good and uniform infection at heading. This is conditioned in part by uncontrollable weather parameters, germplasm variation in flowering time and, is compounded by the quantitative nature of the disease resistance mechanisms [170]. For more than two decades, researchers have identified QTL for FHB resistance in a wide variety of germplasm and, to date, more than 250 QTL have been described [98, 171]. Several FHB resistance-associated QTL have been mapped and named, including *Fhb1* and *Fhb2* from *T. aestivum* cv. Sumai, *Fhb3* from *Leymus racemosus*, *Fhb4* and *Fhb5* from *T. aestivum* cv. Wangshuibai, *Fhb6* from *Elymus tsukushiensis*, and *Fhb7* from *Thinopyrum ponticum* [124, 128, 129, 172-176]. The major Type II QTL on chromosome 3BS, known as *Fhb1*, was identified in multiple independent studies, and it is the best characterized QTL to date [177, 178]. Also, the recent cloning of *Fhb1* provides new insights to understand the mechanisms of resistance to FHB in wheat [179]. The *Fhb1* QTL, first identified in the Chinese spring wheat accession Sumai 3, has been introduced into breeding lines worldwide [100, 180]. However, several other small effect QTL conferring

resistance to FHB have been identified on all wheat chromosomes, emphasizing the complex genetic nature of the resistance [100, 104, 130, 180].

Considering the quantitative nature of FHB resistance in wheat, the pyramiding of multiple resistance alleles can readily be hypothesized as a mean to produce resistant cultivars [181, 182]. In addition to the quantitative nature of the disease resistance, the challenges associated with high quality and consistent phenotyping hint at the potential of genomic selection (GS) as a strategy for improving FHB resistance. Genomic prediction (GP) considers all genome-wide variations including major and small-to-moderate effect QTLs, which collectively control a large proportion of the genetic variance [183]. GP relies on accurate phenotypic and genotypic information of a training population to test the models' ability to predict phenotypes strictly from genotypic data. The selected trained GP model is then used to predict test lines that have been genotyped but not phenotyped [59, 184]. GP considers the genotypic and phenotypic data of a training population to predict the breeding value, with the goal of increasing the genetic gain per unit time and cost [185]. As the occurrence of FHB depends heavily upon the environmental conditions and FHB phenotyping is expensive, previously trained GP models enhance the selection [167, 186]. GP is now an integral part of several breeding programs in Canada and in the eastern and mid-west USA [187, 188].

Rutkoski et al. [182] evaluated the potential of GP for FHB in 2,000 breeding lines using data collected from U.S. cooperative wheat nurseries by comparing the performance of ten single- and multi-trait GP models using cross-validation. This study indicated that the QTL from these datasets could be used to improve the predictive ability. Similarly, Arruda et al. [189] found moderate to high predictive ability for FHB resistance in wheat using three different GP models, five different genotyping data imputation methods, a variety of training population sizes and marker density, and the impact of genetic relationship. In addition, MAS and GP were compared for their ability to predict six traits related to FHB resistance, and the results indicated that under the same selection intensity, GP resulted in a significantly higher selection differential [187]. Therefore, GP was recommended over the traditional linkage and association mapping strategies for improvement of FHB resistance.

Genomic best linear unbiased prediction (GBLUP) is the most common GP model used in wheat breeding because of its simplicity, ability to capture additive genetic variance, and

similarity to BLUP [190, 191]. Ridge regression is used interchangeably with GBLUP; the former uses the shrinkage operator for estimating the markers' effects while assuming constant variance for each marker during effect estimation [192, 193]. Bayesian models, such as Bayes A, Bayes B, Bayes C, and Bayes LASSO, use prior distribution for estimating the markers' effects using the computationally intensive Markov Chain Monte Carlo (MCMC) method. Bayes A, Bayes B, Bayes C, and Bayes LASSO use the scaled-t, scaled-t mixture, Gaussian mixture, and double exponential distributions, respectively, for model training [194-196]. All these parametric models assume *a priori* relationship between markers and traits, thus ignoring the higher-order interactions during model training.

The rapid adoption of machine learning (ML) and deep learning (DL) in plant breeding has opened up new avenues for GP [197, 198]. Several ML and DL models, such as random forest (RF), boosting, decision tree regression, support vector machine (SVM), multi-layer perceptron (MLP), and convolutional neural network (CNN), have been used for trait prediction in wheat and other cereal crops [138, 193, 199, 200]. Here we explore the potential of traditional mixed, Bayesian and ML models for predicting FHB resistance in spring and winter wheat diversity panels. The specific objectives of this study were (1) to optimize GP models for predicting four traits associated with FHB; 2) to evaluate the predictive ability of different marker sets, and 3) to assess the performance of these models using cross-validation.

3.3 Materials and Methods

3.3.1 Genomic Prediction (GP)

To define the best marker set(s) to maximize the predictive ability of GP, the GP models for ten different datasets were compared. Marker set one was made of the QTNs identified using the default threshold values of nine GWAS models (LOD 3 or $P < 0.05/n$). Datasets two to nine were comprised of the QTNs obtained with the same models but based on different threshold levels as described in **Appendix 8**. The last datasets were the random genome-wide SNP datasets called the "All-SNPs" that included 13,760 SNPs in the spring and combined panels and 10,421 SNPs in the winter panel. First, the GBLUP model was used to compare the predictive ability of these nine GWAS-derived dataset and the All-SNPs dataset for all four FHB-related traits.

To compare the predictive ability across GP statistical models, the following ten models were evaluated for SEV, INC, VRI and DON in each panel using GWAS-derived dataset 1 (highest stringency) and the All-SNPs dataset: rrBLUP [192], GBLUP [201], RFR, BRR, BL, SVR [196], RKHS [183, 192], BayesA, BayesB, and BayesC [183] . All of these models falls into two categories: parametric (GBLUP, RRBLUP, Bayesian models, such as Bayes A, Bayes B, Bayes C, and Bayes LASSO) and non-parametric (RF, SVR, BRR, BL). The parametric models assume a *priori* relationship between markers and traits, thus ignoring the higher-order interactions during model training. This method only account for additive effect and the linear relationship between genotype and phenotype [202]. An alternative approach to the standard parametric modeling of complex interactions is provided by non-parametric methods. In non-parametric methods, there is no need to make any assumption of parameters for the given population or the population we are studying [67]. In fact, the methods don't depend on the population. Here there is no fixed set of parameters are available, and there is no distribution (normal distribution, etc.) of any kind is available for use. This is also the reason that nonparametric methods are also referred to as distribution-free methods.

The predictive ability (r) of GP models was defined as the Pearson's correlation coefficient between the mean genomic estimated breeding values (GEBVs) and the observed phenotypes. A five-fold cross-validation with 50 iterations was used to estimate the predictive ability. Each subset of the five randomly-partitioned subsets was iteratively treated as a test dataset, while the remaining four subsets were used as a training dataset. A custom genomic selection pipeline (GSPipeline) integrating the ten GP models implemented in the R packages rrBLUP [192], BGLR [195], BLR [203], randomForest [204] and sommer [205] was used for GP model construction and cross-validation. Tukey's multiple pairwise comparisons (HSD.test function) was performed to examine the statistical significance of variations in predictive ability values to compare GP models constructed from different marker sets.

3.4 Results

3.4.1 QTN Identification

For GWAS, BLUP estimates for each trait were used as phenotypic data along with the 13,760 SNPs in the spring and combined panel whereas 10,421 SNPs in the winter panel that were mapped to the wheat Chinese Spring reference sequence V2.0. GWAS was carried out separately for all four traits and all three panels. A total of 107, 157 and 174 unique QTNs were identified using the two single-locus and seven multi-locus models for INC, SEV, VRI, and DON in the spring, winter, and combined panels, respectively. The QTNs identified from each panel was further used for downstream analysis of GP.

3.4.2 Predictive Ability in Determining Significant Marker Set

Testing the ten marker datasets with GBLUP aimed to define the dataset that generated the best predictive ability which was then used to test other GP models. Marker set 1 consistently produced the highest predictive ability and thus outperformed the other nine marker sets for the four traits in all panels (**Appendix 8)**. As such, marker set 1, obtained using the most stringent GWAS thresholds of significance and corresponding to the smallest number of markers, and the AllSNPs datasets were selected to test the ten GP models. In marker set 1, the number of markers found in each panel was 107, 157 and 174 for the spring, winter and combined panels, respectively. The highest predictive ability for this marker set was 0.82±0.05 in the spring panel, while the lowest was 0.50±18 in the winter panel. Lowering the threshold criteria for the GWAS models increased the number of QTN but decreased the predictive ability of GBLUP, with the genome-wide all-SNPs set having the lowest predictive ability in two of the three panels (**Figure 3.1**).

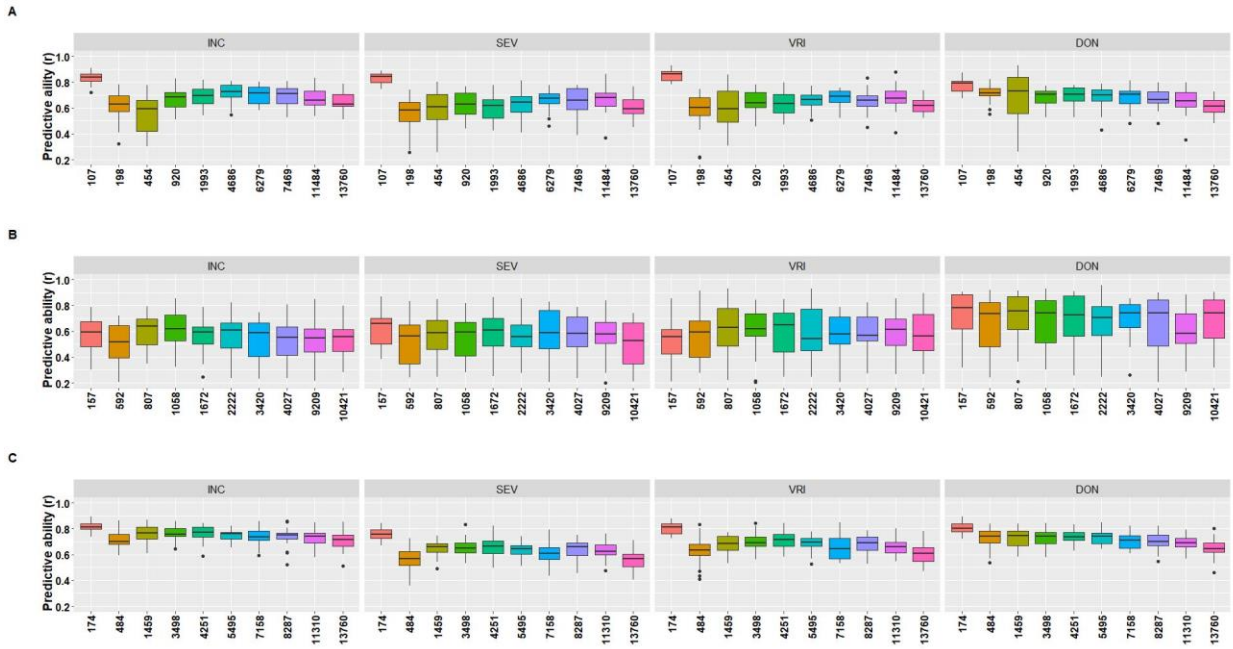

Figure 3.1 Predictive ability ($r \pm s$) for the *Fusarium* head blight traits incidence (INC), severity (SEV), visual rating index (VRI) and deoxynivalenol content (DON) obtained using GBLUP for ten marker sets, of which the first nine were defined through genome-wide association study (GWAS) analyses at a range of significance thresholds (**Appendix 8**) and the last one corresponds to the genome-wide SNPs. (A) Spring panel comprising 206 accessions, (B) Winter panel of 73 accessions, and (C) Combined panel of 279 accessions. The numbers on the x axis represent the number of quantitative trait nucleotides (QTNs) of the nine GWAS-derived datasets and last marker set represents the number of genome-wide SNPs of the All-SNPs set (far right of each graph). The first marker set of each graph represents the QTNs identified using the default threshold value of each model.

3.4.3 Predictive Ability of FHB-related Traits of Multiple Genomic Selection Models

The predictive ability for INC, SEV, VRI and DON was evaluated for all three panels independently using cross-validation. Marker sets 1 were used to estimate the *r* values of the traits using ten GS models for the spring, winter, and combined panels, respectively (**Figure 3.2**). In the combined panel, nine of the ten GS models generated high *r* value ranging from 0.80 to 0.82 for INC, while the lowest at 0.67±0.07 for SEV was obtained with GS model RFR. In the spring panel, the best predictive ability of 0.82-0.83 were obtained by seven of the ten GS models for INC and SEV, while the lowest *r* was observed for VRI (0.70±0.07), once again with RFR. Interestingly, the winter panel produced the highest *r* for DON content (0.64±0.25) and SEV (0.63±0.19), followed by INC (0.51±0.24), and VRI (0.62±0.21) with the model RKHS,

RFR, BayesA, and RFR respectively. (**Appendix 9**). The analysis of variance (ANOVA) indicated significant differences among traits, models and panels, as well as their interactions at a 0.05 probability level (**Appendix 10)**. The *r* values in the winter panel were significantly lower than the other two panels.

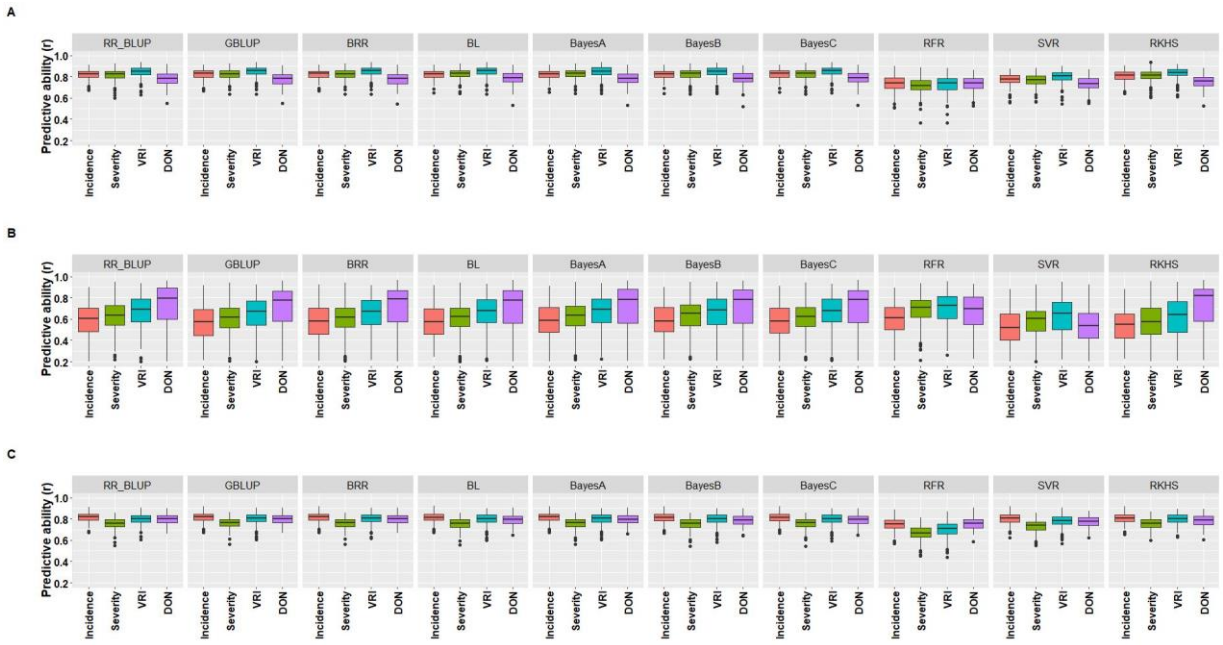

Figure 3.2 Boxplots showing single trait genomic prediction (GP) comparing the predictive ability of ten genomic selection models using the QTNs defined by dataset "1" as markers. (A) Spring panel based on 107 QTNs, (B) Winter panel based on 157 QTNs, and (C) Combined panel based on 174 QTNs.

In the combined panel, all models except RFR and SVR generated non-significantly different predictive abilities ranging from 0.77 to 0.83 for all four traits. In the spring panel, BayesC, BayesA, GBLUP, rrBLUP, BRR and BL generated the highest *r* values for INC, VRI, and DON content. The lowest *r* was observed for VRI (0.70±0.07) using model RFR. Similarly, in the winter panel, RFR, rrBLUP, BayesA, B and C achieved the highest predictive ability from 0.56 to 0.70 for INC, SEV and VRI. In case of SEV, high predictive ability was observed only with RFR (0.68±0.16) (**Figure 3.2**, **Appendix 9**).

3.4.4 Comparison Between Input Datasets: QTN vs All-SNPs

To define the marker sets that generate the best predictive ability, we compared the predictive ability of the QTN-based dataset 1 defined by GWAS to the genome-wide All-SNPs dataset for the four traits using ten GS models. With the All- SNPs dataset in the combined panel, the average *r*-value of 0.54±0.09 for SEV was obtained and the lowest predictive ability came from the SVR model (**Appendix 11**). RFR, GBLUP, BRR, BL, BayesC and RKHS produced high predictive ability (*r*) for all four traits (**Figure 3.3**). In the spring panel, the average *r*-value using the All-SNPs dataset was 0.58±0.11 for INC (**Figure 3.3**). In winter panel, the trait DON content obtained the highest predictive ability of 0.63±0.30 with the model RKHS. The average *r*-value were lower for the All-SNPs datasets than the QTN-based dataset "1" for all traits, all panels and all models (**Figure 3.3, Appendix 11**).

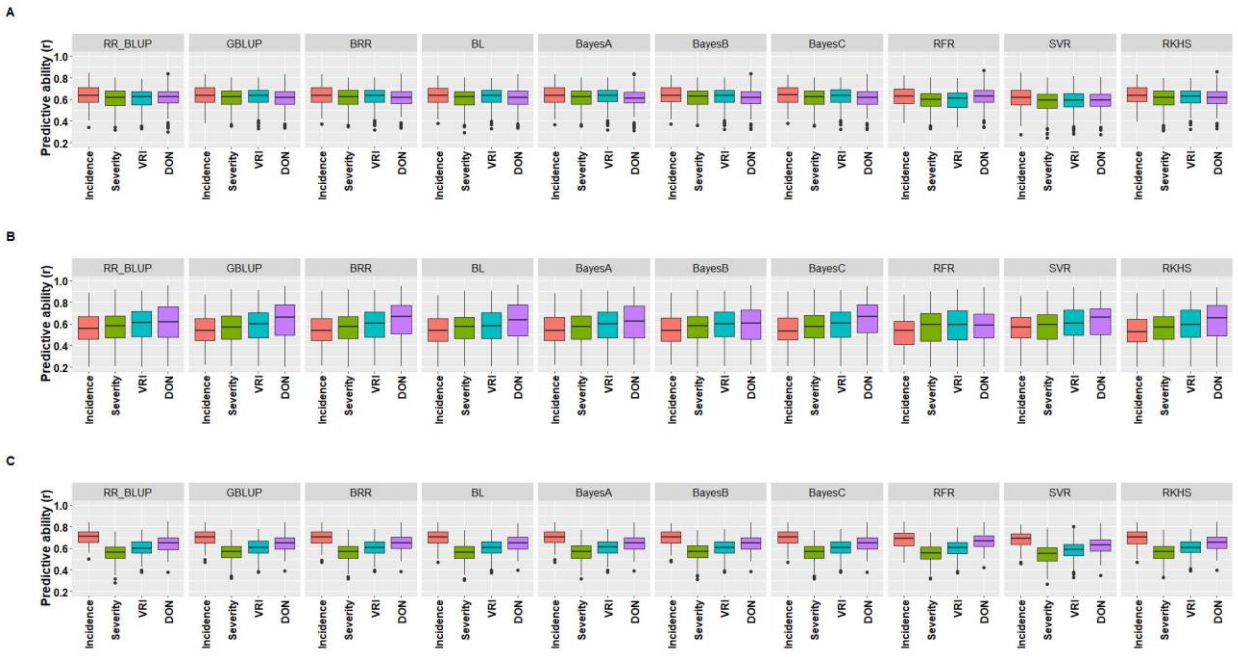

Figure 3.3 Boxplots of genomic prediction (GP) comparing the predictive ability of ten genomic selection models using the random genome-wide marker set of 13,760 SNPs for spring and combined, and 10,421 for the winter panel (All-SNPs) in the (A) spring, (B) winter, and (C) combined panels.

3.5 Discussion

Resistance to FHB is urgently needed worldwide because this widely spread disease has devastating consequences on yield, grain quality and food safety. As such, FHB is a threat to food safety and food security. Historically, phenotypic selection has been the most important method used by wheat breeders to evaluate germplasm and select high-performance individuals. However, phenotyping is expensive, sensitive to measurement errors, often focused on easily measurable traits and easily applied methods, among other drawbacks. GP is an alternative or a complementary approach to phenotypic selection that is based on genome-wide DNA markers, most often SNPs, to predict the breeding values (BVs) of individuals **[206]**. Assessment of BVs is important for selecting desirable characters and determining the magnitude and type of gene action(s) on trait expression in breeding programs. GP-based BVs can be used as a proxy to select the target trait(s). The use of GP and high-throughput phenotyping has the potential to alleviate the difficulties that come with quantifying resistance to FHB. GP can improve selection efficiency, speed up generation progress, and boost selection intensity **[207]**.

This study evaluated a collection of 279 accessions consisting of *Triticum* and *Aegilops* species that assess GP models for the FHB-associated traits INC, SEV, VRI and DON. The germplasm in this study comprised elite cultivars, breeding lines, landraces, synthetic hexaploid wheats (SHWs), progenitor species and wild relatives of the *Aegilops* genus. The germplasm mentioned above was investigated as a potential source of novel resistance **[208, 209]**. Although wild relatives lack adaptation traits, they remain an excellent source of genetic variation for many traits, as demonstrated for several diseases **[210]**. The identification of quantitative trait loci has been made easier by recent developments in technologies and other strategies. These new technological advances have facilitated the production of large-scale genome-wide markers and have consequently resulted in the proliferation of genome-wide association studies **[211]**. Previous studies using SNP marker sets demonstrated low to intermediate predictive ability **[167, 212]**. Our study used GWAS-derived marker sets as input for GS which showed significant increase in predictive ability, thereby addressing the issue of low predictability. The use of QTN-based single- and multi-locus GWAS models simplifies the selection of suitable genomic variants for modern plant molecular breeding **[213]**. Here, we conducted GWAS using an array-based SNP genotyping platform with both single and multi-locus models for all three panels. The

different panels produced different sets of QTNs. These were grouped into several marker sets based on significance thresholds that were subsequently used for GP. Although the winter panel produced the most QTNs, the result indicated that the predictive ability was highest for the spring panel for all traits except DON content. This is mainly due to the lower diversity in the spring panel which is mostly comprised of *Triticum* species compared to the winter panel which is highly diverse, encompassing several *Aegilops* and *Triticum* species.

In genomic selection, to calculate the predictive ability of the traits, markers are used as genotypic data **[214]**. We believe that genome-based methods will become the strategies of choice in predicting traits. Although obtaining marker data requires cost and time investments, obtaining high-density marker information has become increasingly cost-effective in recent years **[215]**.

GP has been used in crop breeding to improve efficiency and speed up genetic progress, thereby minimizing the cost of production. The potential application of our GP study is to improve the efficiency of breeding by performing early selection of promising lines from a large number of segregating lines. In the present study, we evaluated the performance of different panels. This strategy could be promising for selection among non-phenotyped individuals and accelerate genetic gain. These results are encouraging from a practical breeding point of view because selection based on breeding values could save time and resources associated with phenotyping and still identify the most resistant lines. Predictive abilities have been reported to vary greatly among traits and GP models **[216]**. However, consistent predictive ability can be found for seven of the ten GP models used here. The number of predictors (markers) has been reported to have significant effects on GS model performance **[217, 218]**. Therefore, we assessed the effects of the number and type of markers of the input dataset on the performance of the GS models. Overall, the QTN dataset defined by the most stringent GWAS criteria outperformed all other dataset. In flax, QTN-based GP also outperformed the All-SNPs marker set, likely as a consequence of a reduction in background noises or the removal of unrelated markers **[81]**. Therefore, methodologies aimed at identifying and removing redundant or false positives are likely to improve accuracy. Comparison between genome-wide random SNPs and QTNs identified by GWAS models shows that predictive ability significantly increased for seven traits in flax when using the latter dataset **[81]**. The reduction in background noises or lower multi-

collinearity due to the removal of unrelated markers are likely responsible for the superior performance of QTN datasets over genome-wide random SNP datasets. In this study, we consistently observed that QTN marker sets improve the predictive ability compared to the random genome-wide All-SNPs marker set for all four traits and all three diversity panels. Previous studies using SNP marker sets demonstrated low to intermediate predictive ability of 0.22-0.44 **[167, 212]**. In addition to this, the use of random marker selection as training population produced accuracies of 0.63, 0.43, and 0.42 for INC, SEV, and DON content, respectively **[219]**. Our study used GWAS-derived marker sets as input for GS which shows significant increase in predictive ability.

In GP, the heritability of a trait is an important factor **[220]**. High heritability is advantageous because it leads to higher predictive abilities **[81]**. However, GP can be a valuable strategy for low heritability traits. The heritability of the four FHB-related traits was low to moderate, suggesting that their non-genetic effects have considerable influence. FHB resistance is polygenic; hence, the moderate heritability of the trait was expected. -Furthermore, accurate phenotyping plays a critical role in identifying FHB-related QTNs. In this study, INC had relatively high $\boldsymbol{H^2}$ values, which was congruent with the higher predictive ability observed in the combined and winter panels. In the winter panel, a higher $\boldsymbol{H^2}$ was estimated for DON content, and its predictive ability was also high. A direct relationship exists between $\boldsymbol{H^2}$of the traits and predictive ability.

3.6 Conclusion

Despite the fact that some FHB-related traits have a certain degree of heritability and that some large-effect loci have been identified, the quantitative nature, the genotype by environment interactions, the phenotyping challenges and cost are all indicative of the potential efficiency of GP for breeding improvement of FHB resistance. Historically, phenotypic selection has been the most important method used by wheat breeders to evaluate germplasm and select high performance individuals. However, phenotyping is expensive, sensitive to measurement errors, often focused on easily measurable traits and easy to apply methods, among other drawbacks. Here we evaluated the potential of GP models for predicting FHB-related traits. Our results

support GP as an alternative to phenotypic selection to predict the breeding values of individuals for this trait. Our study considered various options for genomic datasets, GWAS models and GP models to improve predictive ability while proposing a strategy to reduce genotyping cost in breeding selection for these important traits. Moderate to high GP accuracies can be achieved for FHB resistance-related traits when implemented in a breeding program. The models' performances were compared and rrBLUP was found to outperform all other models for all traits. In the evaluation of GP models, we compared QTNs identified by different types of GWAS models for all three panels with the All-SNPs data-set and we demonstrated that predictive ability increased significantly using QTN-based datasets for FHB traits in wheat and relatives [81].

4.0 General Discussion

FHB response in wheat is a difficult trait for breeding because of its quantitative nature. Due to limited genetic gains from traditional breeding, the breeding for FHB resistance has been slow. Discovering and manipulating genetic resources are urgently needed worldwide in order to mount a resistance against FHB before this widely spread disease causes further devastation on grain yield, and quality. The prime goal of the breeders is to select germplasm with superior FHB resistance in order to guaranty food safety and security.

This thesis focused mainly on two approaches to improve the understanding of the genetics underlying the FHB response in wheat and its wild relatives. First, phenotypic data and genotypic data collected from two diversity panels were combined to identify the QTNs associated with FHB-related phenotypic traits linked with the candidate genes. GWAS have become an important tool to determine the genomic regions associated with phenotypic traits in mapping diversity[221]. GWAS-identified QTNs are important to further our understanding of the genetic complexity of wheat FHB resistance. The regions discovered in the present study have mined and a few candidate genes have been compared to previously characterized disease resistance genes to provide an element of validation. Second, these QTNs were further used for genomic selection (GS) which provided a second element of validation considering that the breeding values obtained with the QTN sets consistently outperformed the genome-wide random SNP set regardless of the trait or panel [220].

Historically, phenotypic selection has been the most widely used method by wheat breeders to evaluate germplasm and select high-performance individuals [222]. However, phenotyping is expensive, sensitive to measurement errors, often focused on easily measurable traits and applied methods. During the past decades, marker-assisted selection for FHB resistance has been implemented by breeders for major genes only, such as *Fhb1* to *Fhb7* [124, 128, 172-176, 223]. GS is an alternative or a complementary approach to phenotypic selection that is based on genome-wide DNA markers, most often SNPs, to predict the breeding values (BVs) of individuals [206]. Assessment of BVs is important for selecting desirable characters and determining the magnitude and type of gene action(s) on trait expression in breeding programs. GBEVs can be used as a proxy to select for the target trait(s). The use of GP and high-throughput phenotyping has the potential to alleviate the difficulties that come with phenotyping FHB

resistance. GP can improve selection efficiency, speed up breeding cycle, and boost selection intensity [207].

This study evaluated a diverse collection of 279 accessions, consisting of *Triticum* and *Aegilops* species divided into a spring and a winter diversity panel. The purpose of using *Aegilops* species was to investigate them as potential new sources of FHB resistance genes such as those already obtained: *Fhb3*, *Fhb6* and *Fhb7* [128, 129, 174]. This research area is the focus of some Canadian wheat pre-breeding programs.

Disease management strategies include cultural, chemical and genetic control practices, with genetic resistance being the most effective. For QTL mapping, we phenotyped both spring and winter panels at two locations over 3-4 years. The location-year datasets were defined as separate environments for both spring and winter panels. This was paramount to obtaining reliable phenotypic data because there is a large genotype by environment effect for INC, SEV, VRI and DON content [224].

In this thesis, the QTNs identified may be linked to novel candidate genes for FHB resistance. The outcome of this study is expected to guide gene annotation and pre-breeding decisions for the transfer of useful FHB genes for wheat improvement. The identified QTNs explained 0.001 to 42.09% of the phenotypic variation, highlighting the quantitative nature of FHB response and the likely importance of the minor effect QTNs.

These QTNs are predicted to be associated with wheat FHB response in various ways. They could harbor genes that may be involved in disease, defense and stress responses. FHB response in wheat is a complex process. GS could be utilized to improve FHB resistance as it considers the whole genetic make-up of the test plants [225]. Various genetic markers could be developed to track the introgression of favorable QTNs is crosses and backcrosses. New technological advances have facilitated the production of large-scale genome-wide markers to screen for genotypes predicted to have superior FHB resistance.

GS is best suited in plant breeding programs for polygenic trait selection. GS employs genome-wide markers that account for small effects QTLs and are more accurate for use in selection, in addition to the potential for increased genetic gain. GS uses phenotypic and genotypic data from a phenotyped training set to build a model and that model is used to predict

the value of non-phenotyped test individuals. GS models are generally constructed from random genomic marker data spanning the entire genome to predict the values of individuals called genomic estimating breeding values or GEBVs. Here, DON content had the highest *r* among all FHB traits in the winter panel, highlighting the importance of an objectively measured trait in breeding for disease resistant genotypes. An asymptomatic line may be highly infected by *Fusarium* fungi and have a high DON content. Screening for DON content is expensive and time consuming because it requires harvest, threshing, grinding, sample preparation and ELISA test. The main focus should be made on primary phenotyping to improve FHB resistance for low DON content. Varieties with low DON content will be readily adopted because it would alleviate the food safety issue associated with the presence of these mycotoxins in food.

The germplasm in this study comprised elite cultivars, breeding lines, landraces, synthetic hexaploid wheats (SHWs), progenitor species and wild relatives of the *Aegilops* genus. The germplasm mentioned above was targeted for its potential as new sources of resistance that could be introgressed into the primary genepool of wheat [208, 209]. Although wild relatives lack adaptation traits, they remain an excellent source of genetic variation for many traits, as demonstrated for several diseases [210]. The identification of QTLs and QTNs has been made easier by recent developments in technologies and other strategies [226]. These new technological advances have facilitated the production of large-scale genome-wide markers and have consequently resulted in the proliferation of genome-wide association studies [211]. One study showed QTNs identified through single- and multi-locus GWAS models advance GS, thereby maximize the selection of suitable genomic variants for modern plant molecular breeding [213]. In GS, markers are used as a genotypic data to calculate the predictive ability of the traits, allowing selection to be done on the same basis [214]. I believe that genome-based methods will become the strategies of choice in predicting traits. Although obtaining marker data requires cost and time investments, obtaining high-density marker information has become increasingly cost-effective in recent years [215].

GP has been used in crop breeding to improve efficiency and accelerate genetic progress, thereby minimizing the cost of production. A potential application of our GP study is to improve the efficiency of breeding by performing early selection of promising lines from a large number of segregating lines. These results are encouraging from a practical breeding point of view

because selection based on GBEVs could save time and resources associated with phenotyping and still identify the most resistant lines. Controlling FHB on the farm involves a multi-prong approach that considers crop rotation, tilling methods, bio-control monitoring, fungicide treatment, and FHB forecasting algorithms [118]. FHB-resistant cultivars are critical for an effective integrated *Fusarium* management because they contribute to long-term disease control and mycotoxin prevention directly at the start of the production chain: at the farmer's field.

5.0 Limitations and Suggested Future Studies

One of the main limitations of this study is the validation step which is based on the same set of germplasm (five-fold cross-validation). A better validation set would be an independent set of germplasm that would be phenotyped. We hoped to have that with the three bi-parental populations that were tested in Morden in 2021 but, unfortunately, the data was not usable because Morden experienced its worst drought in recorded history which reduced the disease load significantly and did not permit to distinguish the lines phenotypically.

FHB phenotyping is expensive, demanding and requires experienced trained personnel. This phenotyping is also time sensitive. The most susceptible stage is at anthesis. FHB visual symptoms must be measured in multiple environments for precise phenotyping and, even so, there is residual errors caused by variation in anthesis time. The timing of inoculation may impact the disease progression and scientists have a limited control over that. Some of these limitations can be overcome by implementing recent advances in imaging technology for precise disease phenotyping in the field plots. DON content measurement is also costly and labor-intensive. DON analysis kits have measurement ranges of 0.5 to 5 ppm which limits the precision of detection to this range. Other limitations of the study include unequal sample representation and missing phenotypic data. To minimise their impact, the best linear unbiased predictor (BLUP) values, as a “mean” for different environments, was used.

Nine different statistical models were utilised to conduct the GWAS, and this resulted in the discovery of a significant number of QTNs, some of which were unique while others had been reported by others. To narrow down the list of viable candidate genes, functional annotation was performed for RGAs located near major QTNs. While useful, these RGAs remain putative

and functional analysis is still required to confirm their role in FHB resistance. The biological role of discovered candidate genes, including their interaction networks, molecular pathways, and contributions to disease resistance, must be investigated further. However, the QTNs in high linkage disequilibrium with the functional variants can be used as proxies or genetic markers [227].

The reduction in cost of high-throughput sequencing makes the genotyping more accessible and has the potential to expedite the breeding accuracy, reliability and decision making process. The results of our study provides useful information for the improvement of wheat breeding for various agronomic and disease traits. Cross breeding prediction and the phenotyping a large number of plants are major challenges for breeders, and the development of genetic simulations with hundreds to thousands of crosses will help in reducing the error and cost. Future work must concentrate on empirical studies to validate predictive ability to determine the efficacy of GS. With the availability of marker information, breeding values of individuals from genomic selection combined with computer simulations offers a computational approach to simulate segregation populations and predict the genetic performance of different types of crosses by evaluating the potential breeding value of crosses through the use of GS methods.

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6.0 Conclusions

Wheat is one of the major crop of the world; therefore, special attention should be given to its yield and quality attributes. Biotic factors such as diseases are limiting wheat productivity. FHB, caused by the fungus *Fusarium graminearum*, represents a major wheat disease, contributing to significant yield losses. In this study, genetic variations and patterns of haplotype distribution in wheat was concluded. The genomic areas discovered by the marker-trait association analyses are novel findings that have the potential to lead to more research and/or applications upon further validation. Some of these genes, particularly those linked to significant impact and large-effect QTNs can be considered as candidate genes for their linked traits. As QTNs, they can be used in breeding, such as marker-assisted and genomic selection.

The foundation of genomic selection has been laid at the beginning of this century. Since then, it has developed into a very active field of research. Although it has originally been developed in dairy cattle breeding, it rapidly attracted the attention of the plant breeding community and has, by now (2022), developed into an integral component of the breeding techniques of international companies. GS offers a significant opportunity to enhance crop production through shortening the breeding cycles, and through either the selection of superior genotypes or the early removal of inferior genotypes from populations.

The recent development of large-scale and cost-efficient genotyping platforms was the prerequisite for the rise of genomic selection. Its functional principle is based on information shared between individuals. Genetic similarities between individuals are assessed by the use of genomic fingerprints. In practice, implementation of genomic prediction requires the training (calibration) sets consisting of individuals that have been both phenotyped and genotyped, followed by model calibration to determine the breeding value of the population on testing sets. The model is then used to derive predictions of the genomic estimated breeding values for non-phenotyped individuals. These predictions can save time by accelerating the breeding program and cost by reducing resources usually allocated to phenotyping. The positive impact can be seen in the rates of genetic gain through improved accuracy of GEBVs and/or through shorter breeding cycles in polyploid species.

A large body of literature has reported the accuracy of genomic selection for non-phenotyped individuals. However, training population individuals are themselves often times

selection candidates in plant breeding, and there is no conceptual obstacle to apply genomic selection to them, making use of information obtained via marker-based similarities. It is therefore also highly important to assess the predictive ability and to explore possibilities for its improvement. The success of this approach depends on the marker sets, the population structure and the GWAS methods.

In summary, the studies described in this thesis applied GWAS to identify QTNs linked to FHB traits and identified superior datasets and models to use in genomic selection for the improvement of FHB resistance in both spring and winter wheat breeding programs. The GWAS analysis successfully identified several QTNs significantly associated with FHB-related traits. Furthermore, the QTNs found were used for downstream analysis of GS. The GS uses all markers including those with minor effects allowing breeders to obtain GEBVs in order to perform parent selection for breeding purpose. This is especially important for quantitative traits like FHB disease resistance, conferred by a large number of genes, each with a minor effect. Finally, breeders can use plant's genetic makeup along with its visible and measureable traits, known as phenotypic data, to train a model to predict the GEBV without having to plant seeds and wait for them to grow and measure their traits physically. In this way, they can save time and cost by reducing the number of selection cycles.

7.0 References

1. Ali M, Zhang Y, Rasheed A, Wang J, Zhang L: **Genomic prediction for grain yield and yield-related traits in chinese winter wheat**. *International Journal of Molecular Sciences* 2020, **21**(4):1342.
2. Venske E, dos Santos RS, Busanello C, Gustafson P, Costa de Oliveira A: **Bread wheat: a role model for plant domestication and breeding**. *Hereditas* 2019, **156**(1):16.
3. Salamini F, Özkan H, Brandolini A, Schäfer-Pregl R, Martin W: **Genetics and geography of wild cereal domestication in the near east**. *Nature Reviews Genetics* 2002, **3**(6):429-441.
4. Cox TS: **Deepening the wheat gene pool**. *Journal of Crop Production* 1997, **1**(1):1-25.
5. Marcussen T, Sandve SR, Heier L, Spannagl M, Pfeifer M, Jakobsen KS, Wulff BB, Steuernagel B, Mayer KF, Olsen OA: **Ancient hybridizations among the ancestral genomes of bread wheat**. *Science* 2014, **345**(6194):1250092.
6. McFadden E, Sears ER: **The origin of triticum spelta and its free-threshing hexaploid relatives**. *Journal of Heredity* 1946, **37**:81 107.
7. Feldman M, Levy AA: **Genome evolution due to allopolyploidization in wheat**. *Genetics* 2012, **192**(3):763-774.
8. Fernandez R, Takahashi F, Liu Z, Steward R, Stein D, Stanley ER: **The Drosophila shark tyrosine kinase is required for embryonic dorsal closure**. *Gene Development* 2000, **14(5)**:604--614.
9. Shewry PR: **Wheat**. *Journal of Experimental Botany* 2009, **60**(6):1537-1553.
10. Gustafson P, Raskina O, Ma X-F, Nevo E: **Wheat evolution , domestication , and improvement**. In: *In Wheat Science and Trade Eds Carver, B F Wiley-Blackwell Ames Iowa USA Chapter 1: 2009*. 5-23.
11. Feldman M: **Origin of cultivated wheat. In The world wheat book**. *A history of wheat breeding Eds by A P Bonjean and W J Angus Lavoisier Publishing, France Chapter 1* 1997:4-56.
12. Nevo E: **Evolution of wild emmer wheat and crop improvement**. *Journal of Systematics and Evolution* 2014, **52**(6):673-696.
13. Curtis BC: **Wheat in the world. In Bread Wheat Improvement and Production.** *Food and Agriculture Organization of the United Nations, Rome* 2002(ISBN 92-5-104809-6).
14. Gooding MJ, and Davies, W. P.: **Wheat production and utilization.** . *Advance Typesetting Oxford, the University of Cambridge* 1997:335.
15. Glover NM, Redestig H, Dessimoz C: **Homoeologs: What are they and how do we infer them?** *Trends in Plant Science* 2016, **21**(7):609-621.
16. Appels R, Eversole K, Feuillet C, Keller B, Rogers J, Stein N, Pozniak CJ, Stein N, Choulet F, Distelfeld A *et al*: **Shifting the limits in wheat research and breeding using a fully annotated reference genome**. *Science* 2018, **361**(6403).
17. Fatima F: **Genome-wide association study for disease traits in wheat and its wild relatives** *Doctoral dissertation, Univerity of Ottawa* 2020.
18. Osei M, Asante M, A A, Adebayo M, H A-D: **Plant breeding: a tool for achieving food sufficiency**. In., vol. 9: Plants; 2014: 253-274.
19. Dubcovsky J, Dvorak J: **Genome plasticity a key factor in the success of polyploid wheat under domestication**. *Science* 2007, **316**:1862-1866.
20. Akhunov ED, Akhunova AR, Anderson OD, Anderson JA, Blake N, Clegg MT, Coleman-Derr D, Conley EJ, Crossman CC, Deal KR *et al*: **Nucleotide diversity maps**

reveal variation in diversity among wheat genomes and chromosomes. *BMC Genomics* 2010, **11**:702.
21. Eltaher S, Sallam A, Belamkar V, Emara HA, Nower AA, Salem KFM, Poland J, Baenziger PS: **Genetic diversity and population structure of F3:6 nebraska winter wheat genotypes using genotyping-by-sequencing**. *Frontiers in Genetics* 2018, **9**:76.
22. Smýkal P, Nelson NM, Berger DJ, Von Wettberg JBE: **The impact of genetic changes during crop domestication**. *Agronomy* 2018, **8**(7).
23. Gorafi YSA, Kim JS, Elbashir AAE, Tsujimoto H: **A population of wheat multiple synthetic derivatives: an effective platform to explore, harness and utilize genetic diversity of Aegilops tauschii for wheat improvement**. *Theoritical and Applied Genetics* 2018, **131**(8):1615-1626.
24. Heffner E, Sorrells M, Jannink J-L: **Genomic selection for crop improvement**. *Crop Science* 2009, **49**.
25. Ogbonnaya FC, Abdalla OS, Mujeeb-Kazi A, Kazi AG, Xu SS, Gosman N, Lagudah ES, Bonnett D, Sorrells ME, Tsujimoto H: **Synthetic hexaploids: harnessing species of the primary gene pool for wheat improvement**. *Plant Breeding Reviews* 2013, **37**:35-122.
26. Arora S, Singh N, Kaur S, Bains NS, Uauy C, Poland J, Chhuneja P: **Genome-wide association study of grain architecture in wild wheat *Aegilops tauschii***. *Frontiers in Plant Science* 2017, **8**(886).
27. Bhatta M, Morgounov A, Belamkar V, Poland J, Baenziger PS: **Unlocking the novel genetic diversity and population structure of synthetic hexaploid wheat**. *BMC Genomics* 2018, **19**(1):591.
28. Khan Z, Qazi J, Rasheed A, Mujeeb-Kazi A: **Diversity in D-genome synthetic hexaploid wheat association panel for seedling emergence traits under salinity stress**. *Plant Genetic Resources* 2016, **15**(6):488-495.
29. Cossani CM, Reynolds MP: **Heat stress adaptation in elite lines derived from synthetic hexaploid wheat**. *Crop Science* 2015, **55**(6):2719-2735.
30. Misra G, Badoni S, Anacleto R, Graner A, Alexandrov N, Sreenivasulu N: **Whole genome sequencing-based association study to unravel genetic architecture of cooked grain width and length traits in rice**. *Scientific Reports* 2017, **7**(1):12478.
31. Hästbacka J, de la Chapelle A, Kaitila I, Sistonen P, Weaver A, Lander E: **Linkage disequilibrium mapping in isolated founder populations: diastrophic dysplasia in Finland**. *Nature Genetics* 1992, **2**(3):204-211.
32. Risch N, Merikangas K: **The future of genetic studies of complex human diseases**. *Science* 1996, **273**(5281):1516-1517.
33. Altshuler D, Daly MJ, Lander ES: **Genetic mapping in human disease**. *Science (New York, NY)* 2008, **322**(5903):881-888.
34. Thornsberry JM, Goodman MM, Doebley J, Kresovich S, Nielsen D, Buckler ES: **Dwarf8 polymorphisms associate with variation in flowering time**. *Nature Genetics* 2001, **28**(3):286-289.
35. Nordborg M, Weigel D: **Next-generation genetics in plants**. *Nature* 2008, **456**(7223):720-723.
36. Tsai H-Y, Janss LL, Andersen JR, Orabi J, Jensen JD, Jahoor A, Jensen J: **Genomic prediction and GWAS of yield, quality and disease-related traits in spring barley and winter wheat**. *Scientific Reports* 2020, **10**(1):3347.

37. Yu J, Buckler ES: **Genetic association mapping and genome organization of maize**. *Current Opinion in Biotechnology* 2006, **17**(2):155-160.
38. Juliana P, Singh RP, Singh PK, Poland JA, Bergstrom GC, Huerta-Espino J, Bhavani S, Crossa J, Sorrells ME: **Genome-wide association mapping for resistance to leaf rust, stripe rust and tan spot in wheat reveals potential candidate genes**. *Theoretical and Applied Genetics* 2018, **131**(7):1405-1422.
39. Spindel JE, Begum H, Akdemir D, Collard B, Redoña E, Jannink JL, McCouch S: **Genome-wide prediction models that incorporate de novo GWAS are a powerful new tool for tropical rice improvement**. *Heredity* 2016, **116**(4):395-408.
40. Li C, Fu Y, Sun R, Wang Y, Wang Q: **Single-locus and multi-locus genome-wide association studies in the genetic dissection of fiber quality traits in upland cotton (*Gossypium hirsutum* L.)**. *Frontiers in Plant Science* 2018, **9**(1083).
41. Bradbury PJ, Zhang Z, Kroon DE, Casstevens TM, Ramdoss Y, Buckler ES: **TASSEL: software for association mapping of complex traits in diverse samples**. *Bioinformatics* 2007, **23**(19):2633-2635.
42. Yin L, Zhang H, Tang Z, Xu J, Yin D, Zhang Z, Yuan X, Zhu M, Zhao S, Li X *et al*: **Rmvp: A memory-efficient, visualization-enhanced, and parallel-accelerated tool for genome-wide association study**. *Genomics, Proteomics & Bioinformatics* 2021.
43. Li C, Fu Y, Sun R, Wang Y, Wang Q: **Single-locus and multi-locus genome-wide association studies in the genetic dissection of fiber quality traits in upland cotton (*Gossypium hirsutum* L.)**. *Frontiers in Plant Science* 2018, **9**.
44. Kusmec A, Schnable PS: **FarmCPUpp: Efficient large-scale genomewide association studies**. *Plant Direct* 2018, **2**(4):e00053.
45. Wang S-B, Feng J-Y, Ren W-L, Huang B, Zhou L, Wen Y-J, Zhang J, Dunwell JM, Xu S, Zhang Y-M: **Improving power and accuracy of genome-wide association studies via a multi-locus mixed linear model methodology**. *Scientific Reports* 2016, **6**(1):19444.
46. Tamba CL, Zhang Y-M: **A fast mrMLM algorithm for multi-locus genome-wide association studies**. *bioRxiv [Preprint]* 2018:341784.
47. Wen Y-J, Zhang H, Ni Y-L, Huang B, Zhang J, Feng J-Y, Wang S-B, Dunwell JM, Zhang Y-M, Wu R: **Methodological implementation of mixed linear models in multi-locus genome-wide association studies**. *Briefings in Bioinformatics* 2017, **19**(4):700-712.
48. Ren W-L, Wen Y-J, Dunwell JM, Zhang Y-M: **pKWmEB: integration of Kruskal–Wallis test with empirical Bayes under polygenic background control for multi-locus genome-wide association study**. *Heredity* 2018, **120**(3):208-218.
49. Tamba CL, Ni Y-L, Zhang Y-M: **Iterative sure independence screening EM-Bayesian LASSO algorithm for multi-locus genome-wide association studies**. *PLOS Computational Biology* 2017, **13**(1):e1005357.
50. He L, Xiao J, Rashid KY, Yao Z, Li P, Jia G, Wang X, Cloutier S, You FM: **Genome-wide association studies for pasmo resistance in flax (*Linum Usitatissimum* L.)**. *Frontiers in Plant Science* 2019, **9**(1982).
51. Zhao K, Aranzana MJ, Kim S, Lister C, Shindo C, Tang C, Toomajian C, Zheng H, Dean C, Marjoram P *et al*: **An Arabidopsis example of association mapping in structured samples**. *PLoS genetics* 2007, **3**(1):e4-e4.

52. Clark SA, Hickey JM, Daetwyler HD, van der Werf JHJ: **The importance of information on relatives for the prediction of genomic breeding values and the implications for the makeup of reference data sets in livestock breeding schemes**. *Genetics Selection Evolution* 2012, **44**(1):4.
53. Guo G, Zhao F, Wang Y, Zhang Y, Du L, Su G: **Comparison of single-trait and multiple-trait genomic prediction models**. *BMC Genetics* 2014, **15**(1):30.
54. Lorenz AJ, Smith KP, Jannink J-L: **Potential and optimization of genomic selection for *Fusarium* head blight resistance in six-row barley**. *Crop Science* 2012, **52**(4):1609-1621.
55. Liu X, Huang. M, . BF, Buckler. ES, Zhang. Z: **Iterative Usage of Fixed and Random Effect Models for Powerful and Efficient Genome-Wide Association Studies**. *PLOS Genetics* 2016, **12**(2):1-24.
56. Meuwissen THE, Hayes BJ, Goddard ME: **Prediction of total genetic value using genome-wide dense marker maps**. *Genetics* 2001, **157**(4):1819.
57. Crossa J, Pérez-Rodríguez P, Cuevas J, Montesinos-López O, Jarquín D, de Los Campos G, Burgueño J, González-Camacho JM, Pérez-Elizalde S, Beyene Y *et al*: **Genomic selection in plant breeding: methods, models, and perspectives**. *Trends in Plant Science* 2017, **22**(11):961-975.
58. Jannink JL, Lorenz AJ, Iwata H: **Genomic selection in plant breeding: from theory to practice**. *Briefings in Functional Genomics* 2010, **9**(2):166-177.
59. Lorenz AJ, Chao S, Asoro FG, Heffner EL, Hayashi T, Iwata H, Smith KP, Sorrells ME, Jannink J-L: **Chapter two - genomic selection in plant breeding: Knowledge and prospects**. In: *Advances in Agronomy*. Edited by Sparks DL, vol. 110: Academic Press; 2011: 77-123.
60. de los Campos G, Hickey JM, Pong-Wong R, Daetwyler HD, Calus MPL: **Whole-genome regression and prediction methods applied to plant and animal breeding**. *Genetics* 2013, **193**(2):327-345.
61. Bulmer MG: **The effect of selection on genetic variability**. *The American Naturalist* 1971, **105**(943):201-211.
62. Meuwissen TH, Hayes BJ, Goddard ME: **Prediction of total genetic value using genome-wide dense marker maps**. *Genetics* 2001, **157**(4):1819-1829.
63. Zingaretti ML, Monfort A, Pérez-Enciso M: **Psbvb: A versatile simulation tool to evaluate genomic selection in polyploid species**. *G3 Genes|Genomes|Genetics* 2019, **9**(2):327-334.
64. Berro I, Lado B, Nalin RS, Quincke M, Gutiérrez L: **Training population optimization for genomic selection**. *The plant genome* 2019, **12**(3):1-14.
65. Zhong S, Dekkers JC, Fernando RL, Jannink JL: **Factors affecting accuracy from genomic selection in populations derived from multiple inbred lines: a Barley case study**. *Genetics* 2009, **182**(1):355-364.
66. Schrauf MF, de los Campos G, Munilla S: **Comparing genomic prediction models by means of cross validation**. 2021, **12**.
67. Howard R, Carriquiry AL, Beavis WD: **Parametric and nonparametric statistical methods for genomic selection of traits with additive and epistatic genetic architectures**. *G3 Genes|Genomes|Genetics* 2014, **4**(6):1027-1046.

68. Albrecht T, Wimmer V, Auinger H-J, Erbe M, Knaak C, Ouzunova M, Simianer H, Schön C-C: **Genome-based prediction of testcross values in maize**. *Theoretical and Applied Genetics* 2011, **123**(2):339.
69. Guo Z, Tucker DM, Lu J, Kishore V, Gay G: **Evaluation of genome-wide selection efficiency in maize nested association mapping populations**. *Theoretical and Applied Genetics* 2012, **124**(2):261-275.
70. Beyene Y, Semagn K, Mugo S, Tarekegne A, Babu R, Meisel B, Sehabiague P, Makumbi D, Magorokosho C, Oikeh S *et al*: **Genetic gains in grain yield through genomic selection in eight bi-parental maize populations under drought stress**. *Crop Science* 2015, **55**(1):154-163.
71. Slater AT, Cogan NOI, Forster JW, Hayes BJ, Daetwyler HD: **Improving genetic gain with genomic selection in autotetraploid potato**. *The Plant Genome* 2016, **9**(3).
72. Bassi FM, Bentley AR, Charmet G, Ortiz R, Crossa J: **Breeding schemes for the implementation of genomic selection in wheat (*Triticum* spp.)**. *Plant Science* 2016, **242**:23-36.
73. Sverrisdóttir E, Byrne S, Sundmark EHR, Johnsen HØ, Kirk HG, Asp T, Janss L, Nielsen KL: **Genomic prediction of starch content and chipping quality in tetraploid potato using genotyping-by-sequencing**. *Theoretical and Applied Genetics* 2017, **130**(10):2091-2108.
74. Gezan SA, Osorio LF, Verma S, Whitaker VM: **An experimental validation of genomic selection in octoploid strawberry**. *Horticulture Research* 2017, **4**(1):16070.
75. Enciso-Rodriguez F, Douches D, Lopez-Cruz M, Coombs J, de Los Campos G: **Genomic selection for late blight and common scab resistance in tetraploid potato (*solanum tuberosum*)**. *G3 Genes|Genomes|Genetics* 2018, **8**(7):2471-2481.
76. Jannink JL, Lorenz AJ, Iwata H: **Genomic selection in plant breeding: from theory to practice**. *Briefing in Functional Genomics* 2010, **9**(2):166-177.
77. Wang X, Xu Y, Hu Z, Xu C: **Genomic selection methods for crop improvement: Current status and prospects**. *The Crop Journal* 2018, **6**(4):330-340.
78. He L, Xiao J, Rashid YK, Jia G, Li P, Yao Z, Wang X, Cloutier S, You MF: **Evaluation of genomic prediction for pasmo resistance in flax**. *International Journal of Molecular Sciences* 2019, **20**(2).
79. Larkin DL, Lozada DN, Mason RE: **Genomic selection—considerations for successful implementation in wheat breeding programs**. *Agronomy* 2019, **9**(9):479.
80. Alqudah AM, Sallam A, Stephen Baenziger P, Börner A: **GWAS: Fast-forwarding gene identification and characterization in temperate Cereals: lessons from Barley - A review**. *Journal of Advanced Research* 2019, **22**:119-135.
81. Lan S, Zheng C, Hauck K, McCausland M, Duguid SD, Booker HM, Cloutier S, You FM: **Genomic prediction accuracy of seven breeding selection traits improved by qtl identification in flax**. *International Journal of Molecular Sciences* 2020, **21**(5):1577.
82. Rutkoski J, Benson J, Jia Y, Brown-Guedira G, Jannink J-L, Sorrells M: **Evaluation of genomic prediction methods for *Fusarium* head blight resistance in wheat**. *The Plant Genome* 2012, **5**(2):51-61.
83. Deshmukh R, Sonah H, Patil G, Chen W, Prince S, Mutava R, Vuong T, Valliyodan B, Nguyen HT: **Integrating omic approaches for abiotic stress tolerance in soybean**. *Frontiers in Plant Science* 2014, **5**(244).

84. Sarinelli JM, Murphy JP, Tyagi P, Holland JB, Johnson JW, Mergoum M, Mason RE, Babar A, Harrison S, Sutton R *et al*: **Training population selection and use of fixed effects to optimize genomic predictions in a historical USA winter wheat panel**. *Theoretical and Applied Genetics* 2019, **132**(4):1247-1261.
85. Bernardo R: **Genomewide selection when major genes are known**. *Crop Science* 2014, **54**(1):68-75.
86. Rutkoski JE, Poland JA, Singh RP, Huerta-Espino J, Bhavani S, Barbier H, Rouse MN, Jannink J-L, Sorrells ME: **Genomic selection for quantitative adult plant stem rust resistance in wheat**. *The plant genome* 2014, **7**(3):plantgenome2014.2002.0006.
87. Owens BF, Lipka AE, Magallanes-Lundback M, Tiede T, Diepenbrock CH, Kandianis CB, Kim E, Cepela J, Mateos-Hernandez M, Buell CR *et al*: **A foundation for provitamin a biofortification of maize: Genome-wide association and genomic prediction models of carotenoid levels**. *Genetics* 2014, **198**(4):1699-1716.
88. Mason RE, Addison CK, Babar A, Acuna A, Lozada D, Subramanian N, Arguello MN, Miller RG, Brown-Guedira G, Guedira M *et al*: **Diagnostic markers for vernalization and photoperiod loci improve genomic selection for grain yield and spectral reflectance in wheat**. *Crop Science* 2018, **58**(1):242-252.
89. Buerstmayr M, Buerstmayr H: **Comparative mapping of quantitative trait loci for *Fusarium* head blight resistance and anther retention in the winter wheat population Capo × Arina**. *Theoritical and Applied Genetics* 2015, **128**(8):1519-1530.
90. Hoffstetter A, Cabrera A, Sneller C: **Identifying quantitative trait loci for economic traits in an elite soft red winter wheat population**. *Crop Science* 2016, **56**(2):547-558.
91. Mirdita V, He S, Zhao Y, Korzun V, Bothe R, Ebmeyer E, Reif JC, Jiang Y: **Potential and limits of whole genome prediction of resistance to *Fusarium* head blight and Septoria tritici blotch in a vast Central European elite winter wheat population**. *Theoretical and Applied Genetics* 2015, **128**(12):2471-2481.
92. Steiner B, Buerstmayr M, Michel S, Schweiger W, Lemmens M, Buerstmayr H: **Breeding strategies and advances in line selection for *Fusarium* head blight resistance in wheat**. *Tropical Plant Pathology* 2017, **42**(3):165-174.
93. Xu Y, Li P, Zou C, Lu Y, Xie C, Zhang X, Prasanna BM, Olsen MS: **Enhancing genetic gain in the era of molecular breeding**. *Journal of Experimental Botany* 2017, **68**(11):2641-2666.
94. Falconer DSaM, T.F.C.: **Introduction to Quantitative Genetics. 4th Edition, Addison Wesley Longman, Harlow**. *In: Elsevier Current Trends* 1996.
95. Xu Y, Crouch JH: **Marker-assisted selection in plant breeding: From publications to practice**. *Crop Science* 2008, **48**(2):391-407.
96. Collard BCY, Mackill DJ: **Marker-assisted selection: an approach for precision plant breeding in the twenty-first century**. *Philosophical Transactions of the Royal Society B: Biological Sciences* 2008, **363**(1491):557-572.
97. Ogutu JO, Piepho H-P, Schulz-Streeck T: **A comparison of random forests, boosting and support vector machines for genomic selection**. *BMC Proceedings* 2011, **5**(3):S11.
98. Buerstmayr H, Ban T, Anderson JA: **QTL mapping and marker-assisted selection for *Fusarium* head blight resistance in wheat: a review**. *Plant Breeding* 2009, **128**(1):1-26.

99. Pumphrey MO, Bernardo R, Anderson JA: **Validating the fhb1 qtl for Fusarium head blight resistance in near-isogenic wheat lines developed from breeding populations**. *Crop Science* 2007, **47**(1):200-206.
100. Sneller CH, Paul P, Guttieri M: **Characterization of resistance to *Fusarium* head blight in an eastern u.S. Soft red winter wheat population**. *Crop Science* 2010, **50**(1):123-133.
101. Mesterhazy A: **Updating the breeding philosophy of wheat to *Fusarium* head blight (fhb): Resistance components, qtl identification, and phenotyping—a review**. *Plants* 2020, **9**(12):1702.
102. Mesterházy A: **Types and components of resistance to *Fusarium* head blight of wheat**. *Plant Breeding* 1995, **114**(5):377-386.
103. Wang L, Li Q, Liu Z, Surendra A, Pan Y, Li Y, Zaharia LI, Ouellet T, Fobert PR: **Integrated transcriptome and hormone profiling highlight the role of multiple phytohormone pathways in wheat resistance against *Fusarium* head blight**. *PLOS ONE* 2018, **13**(11):e0207036.
104. Zhu Z, Hao Y, Mergoum M, Bai G, Humphreys G, Cloutier S, Xia X, He Z: **Breeding wheat for resistance to *Fusarium* head blight in the Global North: China, USA, and Canada**. *The Crop Journal* 2019, **7**(6):730-738.
105. Herter CP, Ebmeyer E, Kollers S, Korzun V, Miedaner T: **An experimental approach for estimating the genomic selection advantage for *Fusarium* head blight and Septoria tritici blotch in winter wheat**. *Theoritical and Applied Genetics* 2019, **132**(8):2425-2437.
106. Bai G, Shaner G: **Management and resistance in wheat and barley to *Fusarium* head blight**. *Annual Review of Phytopathology* 2004, **42**(1):135-161.
107. Cai J, Wang S, Li T, Zhang G, Bai G: **Multiple minor qtls are responsible for *Fusarium* head blight resistance in chinese wheat landrace haiyanzhong**. *PLoS One* 2016, **11**(9):e0163292.
108. Buerstmayr H, Lemmens M, Fedak G, Ruckenbauer P, Gill CBSJT, Genetics A: **Back-cross reciprocal monosomic analysis of *Fusarium* head blight resistance in wheat (Triticum aestivum L.)**. *Theoretical and Applied Genetics* 1999, **98**:76-85.
109. Miedaner T: **Breeding wheat and rye for resistance to *Fusarium* diseases**. *Plant Breeding* 1997, **116**(3):201-220.
110. Bai GH: **Inheritance of resistance to *Fusarium graminearum* in wheat**. *Theoretical and Applied Genetics* 2000, **v. 100**(no. 1):pp. 1-0-2000 v.2100 no.2001.
111. Bai GH, Chen, L.F. and Shaner, G.E.: **Breeding for resistance to *Fusarium* head blight of wheat in China. In *Fusarium* Head Blight of Wheat and Barley, Leonard, K.J.,**

Bushnell, W.R., Eds.,. *The American Phytopathological Society: St Paul, Minnesota, USA, Chapter 1* 2003:296-317.
112. Buerstmayr H, Lemmens M, Hartl L, Doldi L, Steiner B, Stierschneider M, Ruckenbauer PJT, Genetics A: **Molecular mapping of QTLs for *Fusarium* head blight resistance in spring wheat. I. Resistance to fungal spread (Type II resistance)**. *Theoritical and Applied Genetics* 2002, **104**:84-91.
113. Mesterházy Á, Bartók T, Lamper C: **Influence of wheat cultivar, species of *Fusarium*, and isolate aggressiveness on the efficacy of fungicides for control of *Fusarium* head blight**. *Plant Disease* 2003, **87**(9):1107-1115.

114. Bai G, Shaner G: **Management and resistance in wheat and barley to *Fusarium* head blight**. *Annual Review of Phytopathology* 2004, **42**:135-161.
115. Yu YJ: **Monosomic Analysis for Scab Resistance and Yield Components in the Wheat Cultivar Soo-mo 3.** *Cereal Research Communications* 1982, **10**:185-189.
116. Mesterházy á: **Breeding Wheat for *Fusarium* Head Blight Resistance in Europe**. *In: Leonard, KJ and Bushnell, WR, Eds, Fusarium Head Blight of Wheat and Barley, APS Press, St Paul* 2003:211-240.
117. Ginkel Mv, Schaar, W. van der, Zhuping, Y., & Rajaram, S.: **Inheritance of resistance to scab in two wheat cultivars from Brazil and China.** *Plant Disease* 1996, **80**(863-867).
118. Buerstmayr M, Steiner B, Buerstmayr H: **Breeding for *Fusarium* head blight resistance in wheat—progress and challenges**. *Plant Breeding* 2020, **139**(3):429-454.
119. Sari E, Berraies S, Knox RE, Singh AK, Ruan Y, Cuthbert RD, Pozniak CJ, Henriquez MA, Kumar S, Burt AJ *et al*: **High density genetic mapping of *Fusarium* head blight resistance QTL in tetraploid wheat**. *PLOS ONE* 2018, **13**(10):e0204362.
120. Herter CP, Ebmeyer E, Kollers S, Korzun V, Würschum T, Miedaner T: **Accuracy of within- and among-family genomic prediction for *Fusarium* head blight and Septoria tritici blotch in winter wheat**. *Theoretical and Applied Genetics* 2019, **132**(4):1121-1135.
121. Saini DK, Chopra Y, Singh J, Sandhu KS, Kumar A, Bazzer S, Srivastava P: **Comprehensive evaluation of mapping complex traits in wheat using genome-wide association studies**. *Molecular Breeding* 2021, **42**(1):1.
122. Venske E, Dos Santos RS, Farias DDR, Rother V, da Maia LC, Pegoraro C, Costa de Oliveira A: **Meta-analysis of the qtlome of *Fusarium* head blight resistance in bread wheat: Refining the current puzzle**. *Frontiers in Plant Science* 2019, **10**:727.
123. Cuthbert PA, Somers DJ, Thomas J, Cloutier S, Brulé-Babel A: **Fine mapping Fhb1, a major gene controlling *Fusarium* head blight resistance in bread wheat (Triticum aestivum L.)**. *Theoritical and Applied Genetics* 2006, **112**(8):1465-1472.
124. Cuthbert PA, Somers DJ, Brulé-Babel A: **Mapping of Fhb2 on chromosome 6BS: a gene controlling *Fusarium* head blight field resistance in bread wheat (Triticum aestivum L.)**. *Theoritical and Applied Genetics* 2007, **114**(3):429-437.
125. Qi LL, Pumphrey MO, Friebe B, Chen PD, Gill BS: **Molecular cytogenetic characterization of alien introgressions with gene Fhb3 for resistance to *Fusarium* head blight disease of wheat**. *Theoritical and Applied Genetics* 2008, **117**(7):1155-1166.
126. Xue S, Li G, Jia H, Xu F, Lin F, Tang M, Wang Y, An X, Xu H, Zhang L *et al*: **Fine mapping Fhb4, a major QTL conditioning resistance to *Fusarium* infection in bread wheat (Triticum aestivum L.)**. *Theoritical and Applied Genetics* 2010, **121**(1):147-156.
127. Xue S, Xu F, Tang M, Zhou Y, Li G, An X, Lin F, Xu H, Jia H, Zhang L *et al*: **Precise mapping Fhb5, a major QTL conditioning resistance to *Fusarium* infection in bread wheat (Triticum aestivum L.)**. *Theoritical and Applied Genetics* 2011, **123**(6):1055-1063.
128. Cainong JC, Bockus WW, Feng Y, Chen P, Qi L, Sehgal SK, Danilova TV, Koo DH, Friebe B, Gill BS: **Chromosome engineering, mapping, and transferring of resistance to *Fusarium* head blight disease from Elymus tsukushiensis into wheat**. *Theoritical and Applied Genetics* 2015, **128**(6):1019-1027.

129. Guo J, Zhang X, Hou Y, Cai J, Shen X, Zhou T, Xu H, Ohm HW, Wang H, Li A *et al*: **High-density mapping of the major fhb resistance gene fhb7 derived from thinopyrum ponticum and its pyramiding with fhb1 by marker-assisted selection**. *Theoritical and Applied Genetics* 2015, **128**(11):2301-2316.
130. Miedaner T, Würschum T, Maurer HP, Korzun V, Ebmeyer E, Reif JC: **Association mapping for *Fusarium* head blight resistance in European soft winter wheat**. *Molecular Breeding* 2011, **28**(4):647-655.
131. Rodríguez-Rodríguez DR, Ramírez-Solís R, Garza-Elizondo MA, Garza-Rodríguez MDL, Barrera-Saldaña HA: **Genome editing: A perspective on the application of crispr/cas9 to study human diseases (review)**. *International Journal of Molecular Medicine* 2019, **43**(4):1559-1574.
132. Kaur B, Sandhu KS, Kamal R, Kaur K, Singh J, Röder MS, Muqaddasi QH: **Omics for the improvement of abiotic, biotic, and agronomic traits in major cereal crops: Applications, challenges, and prospects**. *International Journal of Molecular Sciences* 2021, **10**(10):1989.
133. Poland JA, Rife TW: **Genotyping-by-sequencing for plant breeding and genetics**. *The plant genome* 2012, **5**(3).
134. Wang S, Wong D, Forrest K, Allen A, Chao S, Huang BE, Maccaferri M, Salvi S, Milner SG, Cattivelli L *et al*: **Characterization of polyploid wheat genomic diversity using a high-density 90 000 single nucleotide polymorphism array**. *Plant Biotechnology Journal* 2014, **12**(6):787-796.
135. Liu X, Huang M, Fan B, Buckler ES, Zhang Z: **Iterative usage of fixed and random effect models for powerful and efficient genome-wide association studies**. *PLOS Genetics* 2016, **12**(2):e1005767.
136. Yu J, Pressoir G, Briggs WH, Vroh Bi I, Yamasaki M, Doebley JF, McMullen MD, Gaut BS, Nielsen DM, Holland JB *et al*: **A unified mixed-model method for association mapping that accounts for multiple levels of relatedness**. *Nature Genetics* 2006, **38**(2):203-208.
137. Khaki S, Wang L: **Crop yield prediction using deep neural networks**. *Frontiers in Plant Science* 2019, **10**(621).
138. Kimber G: **Genome symbols and plasmon types in the wheat group**. *Proceedings of 7th International Wheat Genetics Symposium Cambridge* 1988, **2**:1209-1210.
139. Stack RW, Mcmullen MV: **A Visual Scale to Estimate Severity of *Fusarium* Head Blight in Wheat**. *Biology* 1998.
140. Abramson D, Gan Z, Clear RM, Gilbert J, Marquardt RR: **Relationships among deoxynivalenol, ergosterol and *Fusarium* exoantigens in Canadian hard and soft wheat**. *International Journal of Food Microbiology* 1998, **45**(3):217-224.
141. Gan Z, R. Marquardt R, Abramson D, Clear RM: **The characterization of chicken antibodies raised against F*usarium* spp. by enzyme-linked immunosorbent assay and immunoblotting**. *International Journal of Food Microbiology* 1997, **38**(2):191-200.
142. Hill NS, Schwarz P, Dahleen LS, Neate SM, Horsley R, Glenn AE, O'Donnell K: **ELISA analysis for *Fusarium* in barley: Development of methodology and field assessment**. *Crop Science* 2006, **v. 46**(no. 6):pp. 2636-2630-2006 v.2646 no.2636.
143. Iyer MS, Cousin MA: **Immunological detection of *Fusarium* species in cornmeal**. *Journal of Food Protection* 2003, **66**(3):451-456.

144. Fatima F, McCallum BD, Pozniak CJ, Hiebert CW, McCartney CA, Fedak G, You FM, Cloutier S: **Identification of new leaf rust resistance loci in wheat and wild relatives by array-based snp genotyping and association genetics**. *Frontiers in Plant Science* 2020, **11**(1728).
145. Bates D, Mächler M, Bolker B, Walker S: **lme4:linear mixed-effects models using Eigen and S4**. *Journal of Statistical Software* 2014(1):1,1-23.
146. Hossain M, Azad M, Alam M, and Eaton T: **Estimation of variability, heritability and genetic advance for phenological, physiological and yield contributing attributes in wheat genotypes under heat stress condition**. *American Journal of Plant Sciences* 2021(12):586-602.
147. Lipka AE, Kandianis CB, Hudson ME, Yu J, Drnevich J, Bradbury PJ, Gore MA: **From association to prediction: statistical methods for the dissection and selection of complex traits in plants**. *Current Opinion in Plant Biology* 2015, **24**:110-118.
148. Pritchard JK, Stephens M, Donnelly P: **Inference of population structure using multilocus genotype data**. *Genetics* 2000, **155**(2):945-959.
149. Frichot E, François O: **LEA: An R package for landscape and ecological association studies**. *Methods in Ecology and Evolution* 2015, **6**(8):925-929.
150. Luu K, Bazin E, Blum MGB: **pcadapt: an R package to perform genome scans for selection based on principal component analysis**. *Molecular Ecology Resources* 2017, **17**(1):67-77.
151. Li P, Quan X, Jia G, Xiao J, Cloutier S, You FM: **RGAugury: a pipeline for genome-wide prediction of resistance gene analogs (RGAs) in plants**. *BMC Genomics* 2016, **17**(1):852.
152. You FM, Cloutier S: **Mapping Quantitative Trait Loci onto Chromosome-Scale Pseudomolecules in Flax**. *Methods and Protocols* 2020, **3**(2):28.
153. Collard BCY, Jahufer MZZ, Brouwer JB, Pang ECK: **An introduction to markers, quantitative trait loci (QTL) mapping and marker-assisted selection for crop improvement: The basic concepts**. *Euphytica* 2005, **142**(1):169-196.
154. Su Z, Bernardo A, Tian B, Chen H, Wang S, Ma H, Cai S, Liu D, Zhang D, Li T *et al*: **A deletion mutation in TaHRC confers Fhb1 resistance to *Fusarium* head blight in wheat**. *Nature Genetics* 2019, **51**(7):1099-1105.
155. Rawat N, Pumphrey MO, Liu S, Zhang X, Tiwari VK, Ando K, Trick HN, Bockus WW, Akhunov E, Anderson JA *et al*: **Wheat Fhb1 encodes a chimeric lectin with agglutinin domains and a pore-forming toxin-like domain conferring resistance to *Fusarium* head blight**. *Nature Genetics* 2016, **48**(12):1576-1580.
156. Guo P-G, Bai G-H, Li R-H, Shaner G, Baum M: **Resistance gene analogs associated with *Fusarium* head blight resistance in wheat**. *Euphytica* 2006, **151**(2):251-261.
157. Wisser RJ, Sun Q, Hulbert SH, Kresovich S, Nelson RJJG: **Identification and characterization of regions of the rice genome associated with broad-spectrum, quantitative disease resistance**. *Genetics* 2005, **169**:2277 - 2293.
158. Gu L, Si W, Zhao L, Yang S, Zhang X: **Dynamic evolution of NBS–LRR genes in bread wheat and its progenitors**. *Molecular Genetics and Genomics* 2015, **290**(2):727-738.
159. Steuernagel B, Witek K, Krattinger SG, Ramirez-Gonzalez RH, Schoonbeek H-j, Yu G, Baggs E, Witek A, Yadav I, Krasileva KV *et al*: **- The NLR-Annotator tool enables annotation of the intracellular immune receptor repertoire**: Plant Physiology 2020.

160. Saijo Y, Tintor N, Lu X, Rauf P, Pajerowska-Mukhtar K, Häweker H, Dong X, Robatzek S, Schulze-Lefert P: **Receptor quality control in the endoplasmic reticulum for plant innate immunity**. *The Embo Journal* 2009, **28**(21):3439-3449.
161. Lin F, Xue SL, Zhang ZZ, Zhang CQ, Kong ZX, Yao GQ, Tian DG, Zhu HL, Li CJ, Cao Y *et al*: **Mapping QTL associated with resistance to *Fusarium* head blight in the Nanda2419 × Wangshuibai population. II: Type I resistance**. *Theoretical and Applied Genetics* 2006, **112**(3):528-535.
162. Tang G, Zhang C, Ju Z, Zheng S, Wen Z, Xu S, Chen Y, Ma Z: **The mitochondrial membrane protein FgLetm1 regulates mitochondrial integrity, production of endogenous reactive oxygen species and mycotoxin biosynthesis in *Fusarium* graminearum**. *Molecular Plant Pathology* 2018, **19**(7):1595-1611.
163. Apel K, Hirt H: **Reactive oxygen species: metabolism, oxidative stress, and signal transduction**. *Annual Review of Plant Biology* 2004, **55**:373-399.
164. Jiang C, Zhang S, Zhang Q, Tao Y, Wang C, Xu JR: **FgSKN7 and FgATF1 have overlapping functions in ascosporogenesis, pathogenesis and stress responses in *Fusarium* graminearum**. *Environmental Microbiology* 2015, **17**(4):1245-1260.
165. Montibus M, Ducos C, Bonnin-Verdal MN, Bormann J, Ponts N, Richard-Forget F, Barreau C: **The bZIP transcription factor Fgap1 mediates oxidative stress response and trichothecene biosynthesis but not virulence in *Fusarium* graminearum**. *PLoS One* 2013, **8**(12):e83377.
166. Bönnighausen J, Gebhard D, Kröger C, Hadeler B, Tumforde T, Lieberei R, Bergemann J, Schäfer W, Bormann J: **Disruption of the GABA shunt affects mitochondrial respiration and virulence in the cereal pathogen *Fusarium* graminearum**. *Molecular Microbiology* 2015, **98**(6):1115-1132.
167. Liu Y, Salsman E, Fiedler JD, Hegstad JB, Green A, Mergoum M, Zhong S, Li X: **Genetic mapping and prediction analysis of fhb resistance in a hard red spring wheat breeding population**. *Frontiers in Plant Science* 2019, **10**.
168. Verges VL, Brown-Guedira GL, Sanford DAV: **Genome-wide association studies combined with genomic selection as a tool to increase *Fusarium* head blight resistance in wheat**. *Crop Breeding, Genetics and Genomics* 2021, **3**(4):e210007.
169. Beukert U, Thorwarth P, Zhao Y, Longin CFH, Serfling A, Ordon F, Reif JC: **Comparing the potential of marker-assisted selection and genomic prediction for improving rust resistance in hybrid wheat**. *Frontiers in Plant Science* 2020, **11**(1650).
170. Bakhsh A, Mengistu N, Baenziger PS, Dweikat I, Wegulo SN, Rose DJ, Bai G, Eskridge KM: **Effect of *Fusarium* head blight resistance gene fhb1 on agronomic and end-use quality traits of hard red winter wheat**. *Crop Science* 2013, **53**(3):793-801.
171. Bai G, Su Z, Cai J: **Wheat resistance to *Fusarium* head blight**. *Canadian Journal of Plant Pathology* 2018, **40**(3):336-346.
172. Cuthbert PA, Somers DJ, Thomas J, Cloutier S, Brulé-Babel A: **Fine mapping Fhb1, a major gene controlling *Fusarium* head blight resistance in bread wheat (Triticum aestivum L.)**. *Theoretical and Applied Genetics* 2006, **112**(8):1465-1472.
173. Liu S, Zhang X, Pumphrey MO, Stack RW, Gill BS, Anderson JA: **Complex microcolinearity among wheat, rice, and barley revealed by fine mapping of the genomic region harboring a major QTL for resistance to *Fusarium* head blight in wheat**. *Functional & Integrative Genomics* 2006, **6**(2):83-89.

174. Qi LL, Pumphrey MO, Friebe B, Chen PD, Gill BS: **Molecular cytogenetic characterization of alien introgressions with gene Fhb3 for resistance to *Fusarium* head blight disease of wheat**. *Theoretical and Applied Genetics* 2008, **117**(7):1155-1166.
175. Xue S, Li G, Jia H, Xu F, Lin F, Tang M, Wang Y, An X, Xu H, Zhang L *et al*: **Fine mapping Fhb4, a major QTL conditioning resistance to *Fusarium* infection in bread wheat (Triticum aestivum L.)**. *Theoretical and Applied Genetics* 2010, **121**(1):147-156.
176. Xue S, Xu F, Tang M, Zhou Y, Li G, An X, Lin F, Xu H, Jia H, Zhang L *et al*: **Precise mapping Fhb5, a major QTL conditioning resistance to *Fusarium* infection in bread wheat (*Triticum aestivum* L.)**. *Theoretical and Applied Genetics* 2011, **123**(6):1055-1063.
177. Clark AJ, Sarti-Dvorjak D, Brown-Guedira G, Dong Y, Baik B-K, Van Sanford DA: **Identifying rare fhb-resistant segregants in intransigent backcross and f2 winter wheat populations**. *Frontiers in Microbiology* 2016, **7**(277).
178. Verges VL: **Heritability estimates and response to selection for *Fusarium* head blight resistance in soft red winter wheat**. *Crop science* 2006, **v. 46**(no. 4):pp. 1587-1580-2006 v.1546 no.1584.
179. Hao Y, Rasheed A, Zhu Z, Wulff BBH, He Z: **Harnessing wheat fhb1 for *Fusarium* resistance**. *Trends in Plant Science* 2020, **25**(1):1-3.
180. Ghimire B, Sapkota S, Bahri B, Martinez-Espinoza A, Buck J, Mergoum MJFiPS: ***Fusarium* head blight and rust diseases in soft red winter wheat in the southeast united states: State of the art, challenges and future perspective for breeding**. *Frontiers in Plant Science* 2020, **11**.
181. Galiano-Carneiro AL, Boeven PHG, Maurer HP, Würschum T, Miedaner T: **Genome-wide association study for an efficient selection of *Fusarium* head blight resistance in winter triticale**. *Euphytica* 2018, **215**(1):4.
182. Rutkoski J, Benson J, Jia Y, Brown-Guedira G, Jannink J-L, Sorrells M: **Evaluation of genomic prediction methods for *Fusarium* head blight resistance in wheat**. *The plant genome* 2012, **5**(2).
183. Meuwissen THE, Hayes BJ, Goddard ME: **Prediction of Total Genetic Value Using Genome-Wide Dense Marker Maps**. *Genetics* 2001, **157**(4):1819-1829.
184. Robertsen CD, Hjortshøj RL, Janss LL: **Genomic Selection in Cereal Breeding**. *Agronomy* 2019, **9**(2):95.
185. Sandhu KS, Mihalyov PD, Lewien MJ, Pumphrey MO, Carter AH: **Combining genomic and phenomic information for predicting grain protein content and grain yield in spring wheat**. *Frontiers in Plant Science* 2021, **12**(170).
186. Fritsche-Neto R, Galli G, Borges KLR, Costa-Neto G, Alves FC, Sabadin F, Lyra DH, Morais PPP, Braatz de Andrade LR, Granato I *et al*: **Optimizing genomic-enabled prediction in small-scale maize hybrid breeding programs: A roadmap review**. *Frontiers in Plant Science* 2021, **12**:658267-658267.
187. Arruda MP, Lipka AE, Brown PJ, Krill AM, Thurber C, Brown-Guedira, G. D, Y.,, Foresman BJ, & Kolb FL: **Comparing genomic selection and marker-assisted selection for *Fusarium* head blight resistance in wheat (*Triticum aestivum* L.)**. *Molecular Breeding* 2016, **36(7)**:1-11.

188. Dong H, Wang R, Yuan Y, Anderson J, Pumphrey M, Zhang Z, Chen J: **Evaluation of the potential for genomic selection to improve spring wheat resistance to *Fusarium* head blight in the pacific northwest**. *Frontiers in Plant Science* 2018, **9**(911).
189. Arruda MP, Brown PJ, Lipka AE, Krill AM, Thurber C, & Kolb FL: **Genomic selection for predicting *Fusarium* head blight resistance in a wheat breeding program**. *The plant genome* 2015, **8(3)**:1-12.
190. Bernardo R: **Prediction of maize single-cross performance using RFLPs and information from related hybrids**. *Crop Science* 1994, **34**(1):cropsci1994.0011183X003400010003x.
191. Bernardo R: **Bandwagons I, too, have known**. *Theoretical and Applied Genetics* 2016, **129**(12):2323-2332.
192. Endelman JB: **Ridge regression and other kernels for genomic selection with r package rrblup**. *The plant genome* 2011, **4**(3).
193. Sandhu KS, Lozada DN, Zhang Z, Pumphrey MO, Carter AH: **Deep learning for predicting complex traits in spring wheat breeding program**. *Frontiers in Plant Science* 2021, **11**:613325.
194. Montesinos-López OA, Montesinos-López A, Crossa J, Toledo FH, Pérez-Hernández O, Eskridge KM, Rutkoski J: **A genomic bayesian multi-trait and multi-environment model**. *G3: Genes, Genomes, Genetics* 2016, **6**(9):2725-2744.
195. Pérez P, de los Campos G: **Genome-wide regression and prediction with the BGLR statistical package**. *Genetics* 2014, **198**(2):483-495.
196. Tibshirani R: **Regression shrinkage and selection via the lasso**. *Journal of the Royal Statistical Society: Series B (Methodological)* 1996, **58**(1):267-288.
197. Bellot P, de Los Campos G, Pérez-Enciso M: **Can deep learning improve genomic prediction of complex human traits?** *Genetics* 2018, **210**(3):809-819.
198. LeCun Y, Bengio Y, Hinton G: **Deep learning**. *Nature* 2015, **521**(7553):436-444.
199. Wang H, Cimen E, Singh N, Buckler E: **Deep learning for plant genomics and crop improvement**. *Current Opinion in Plant Biology* 2020, **54**:34-41.
200. Sandhu KS, Aoun M, Morris CF, Carter AH: **Genomic selection for end-use quality and processing traits in soft white winter wheat breeding program with machine and deep learning models**. *Biology* 2021, **10**(7):689.
201. VanRaden PM: **Efficient methods to compute genomic predictions**. *Journal of Dairy Science* 2008, **91**(11):4414-4423.
202. Pérez-Rodríguez P, Gianola D, González-Camacho JM, Crossa J, Manès Y, Dreisigacker S: **Comparison between linear and non-parametric regression models for genome-enabled prediction in wheat**. *G3 Genes|Genomes|Genetics* 2012, **2**(12):1595-1605.
203. Pérez P, de Los Campos G, Crossa J, Gianola D: **Genomic-enabled prediction based on molecular markers and pedigree using the bayesian linear regression package in r**. *The plant genome* 2010, **3**(2):106-116.
204. Wu Q, Ye Y, Liu Y, Ng MK: **SNP selection and classification of genome-wide SNP data using stratified sampling random forests**. *IEEE Transactions on Nanobioscience* 2012, **11**(3):216-227.
205. Covarrubias-Pazaran G: **Genome-assisted prediction of quantitative traits using the r package sommer**. *PLOS ONE* 2016, **11**(6):e0156744.
206. Desta ZA, Ortiz R: **Genomic selection: genome-wide prediction in plant improvement**. *Trends in Plant Science* 2014, **19**(9):592-601.

207. Heffner EL, Lorenz AJ, Jannink J-L, Sorrells ME: **Plant breeding with genomic selection: Gain per unit time and cost**. *Crop Science* 2010, **50**(5):1681-1690.
208. Gao L, Turner MK, Chao S, Kolmer J, Anderson JA: **Genome wide association study of seedling and adult plant leaf rust resistance in elite spring wheat breeding lines**. *PLOS ONE* 2016, **11**(2):e0148671.
209. Riaz A, Athiyannan N, Periyannan SK, Afanasenko O, Mitrofanova OP, Platz GJ, Aitken EAB, Snowdon RJ, Lagudah ES, Hickey LT *et al*: **Unlocking new alleles for leaf rust resistance in the Vavilov wheat collection**. *Theoretical and Applied Genetics* 2018, **131**(1):127-144.
210. McCallum B, Hiebert C, Huerta-Espino J, Cloutier S: **"Wheat leaf rust," in Disease Resistance in Wheat**. *ed I Sharma (Wallingford: CAB International)* 2012:33–62.
211. Zhang J, Zhang P, Dodds P, Lagudah E: **How target-sequence enrichment and sequencing (tenseq) pipelines have catalyzed resistance gene cloning in the wheat-rust pathosystem**. *Frontiers in Plant Science* 2020, **11**(678).
212. Larkin DL, Mason RE, Moon DE, Holder AL, Ward BP, Brown-Guedira G: **Predicting *Fusarium* head blight resistance for advanced trials in a soft red winter wheat breeding program with genomic selection**. *Frontiers in Plant Science* 2021, **12**:715314-715314.
213. Kumar D, Kumar A, Chhokar V, Gangwar OP, Bhardwaj SC, Sivasamy M, Prasad SVS, Prakasha TL, Khan H, Singh R *et al*: **Genome-wide association studies in diverse spring wheat panel for stripe, stem, and leaf rust resistance**. *Frontiers in Plant Science* 2020, **11**(748).
214. Jonas E, Fikse F, Rönnegård L, Mouresan EF: **Genomic Selection**. In: *Population genomics: Concepts, approaches and applications.* Edited by Rajora OP. Cham: Springer International Publishing; 2019: 427-480.
215. Xu Y, Liu X, Fu J, Wang H, Wang J, Huang C, Prasanna BM, Olsen MS, Wang G, Zhang A: **Enhancing genetic gain through genomic selection: From livestock to plants**. *Plant Communications* 2020, **1**(1):100005.
216. Ali M, Zhang Y, Rasheed A, Wang J, Zhang L: **Genomic prediction for grain yield and yield-related traits in chinese winter wheat**. *International Journal of Molecular Sciences* 2020, **21**(4):1342.
217. Heffner EL, Jannink J-L, Sorrells ME: **Genomic selection accuracy using multifamily prediction models in a wheat breeding program**. *The plant genome* 2011, **4**(1).
218. Ma W, Qiu Z, Song J, Li J, Cheng Q, Zhai J, Ma C: **A deep convolutional neural network approach for predicting phenotypes from genotypes**. *Planta* 2018, **248**(5):1307-1318.
219. Dong H, Wang R, Yuan Y, Anderson J, Pumphrey M, Zhang Z, Chen J: **Evaluation of the potential for genomic selection to improve spring wheat resistance to *Fusarium* head blight in the pacific northwest**. *Frontiers in Plant Science* 2018, **9**.
220. Krishnappa G, Savadi S, Tyagi BS, Singh SK, Mamrutha HM, Kumar S, Mishra CN, Khan H, Gangadhara K, Uday G *et al*: **Integrated genomic selection for rapid improvement of crops**. *Genomics* 2021, **113**(3):1070-1086.
221. Korte A, Farlow A: **The advantages and limitations of trait analysis with GWAS: a review**. *Plant Methods* 2013, **9**:29.
222. Moose SP, Mumm RH: **Molecular plant breeding as the foundation for 21st century crop improvement**. *Plant Physiology* 2008, **147**(3):969-977.

223. Van Sanford D, Anderson J, Campbell K, Costa J, Cregan P, Griffey C, Hayes P, Ward R: **Discovery and deployment of molecular markers linked to *Fusarium* head blight resistance: An integrated system for wheat and barley**. *Crop Science* 2001, **41**(3):638-644.
224. Sip V, Chrpova J, Leisova L, Sykorova S, Kucera L, Ovesna J: **Implications for *Fusarium* head blight control from study of factors determining pathogen and don content in grain of wheat cultivars**. In: *Wheat Production in Stressed Environments: 2007// 2007; Dordrecht*. Springer Netherlands: 281-287.
225. Wenjing H, Luping F, Derong G, Yong Z, Chunmei Z: **Pyramiding three qtl improved *Fusarium* head blight resistance in wheat (*Triticum Aestivum l.*)**. *Molecular Breeding* 2022.
226. Jia H, Zhou J, Xue S, Li G, Yan H, Ran C, Zhang Y, Shi J, Jia L, Wang X *et al*: **A journey to understand wheat *Fusarium* head blight resistance in the Chinese wheat landrace Wangshuibai**. *The Crop Journal* 2018, **6**(1):48-59.
227. Myles S, Peiffer J, Brown PJ, Ersoz ES, Zhang Z, Costich DE, Buckler ES: **Association Mapping: Critical Considerations Shift from Genotyping to Experimental Design**. *The Plant Cell* 2009, **21**(8):2194-2202.

8.0 Appendix

Appendix 1 List of accessions included in this study

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
14	CN12232	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Czech	PGRC
33	AS11527	ABD	*T. aestivum ssp. spelta*	Cultivated	Hexaploid	Spring	Czech	PGRC
34	AS11585	ABD	*T. aestivum ssp. spelta*	Cultivated	Hexaploid	Spring	Canada	PGRC
35	CN12223	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Czech	PGRC
36	CN32158	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	United States of America	PGRC
37	CN2644	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Portugal	PGRC
38	CN51254	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC
39	CN1748	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
40	CN10545	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
41	CN10547	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
42	CN11002	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
43	CN11003	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
44	CN11573	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
45	CN11579	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
46	CN12222	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Czech	PGRC
47	CN12224	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
48	CN12225	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Czech	PGRC
50	CN12227	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Czech	PGRC
51	CN12230	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Czech	PGRC
53	CN51246	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Canada	PGRC
54	CN51253	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC
55	CN51255	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC
56	CN51256	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC
57	CN51257	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC
58	CN51258	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC
59	CN51259	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC
60	CN51263	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
61	CN51265	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Russia	PGRC
62	CN51269	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	United States of America	PGRC
67	CN38247	A	*T. urartu*	Progenitor	Diploid	Winter	Iraq	PGRC
72	CN11649	Am	*T. monococcum ssp. monococcum*	Progenitor	Diploid	Winter	Hungary	PGRC
73	CN37617	Am	*T. monococcum ssp. monococcum*	Progenitor	Diploid	Winter	Bulgaria	PGRC
74	CN12440	Am	*T. monococcum ssp. monococcum*	Progenitor	Diploid	Winter	France	PGRC
76	CN11756	Am	*T. monococcum ssp. monococcum*	Progenitor	Diploid	Winter		PGRC
78	CN37611	Am	*T. monococcum ssp. monococcum*	Progenitor	Diploid	Winter	United Kingdom	PGRC
87	CWI19490_RL5444	Am	*T. monococcum*	Progenitor	Diploid	Winter	Turkey	AAFC
88	CWI16956	Am	*T. monococcum*	Progenitor	Diploid	Winter	Russia	AAFC
89	BW31215	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
90	BW31221	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
91	BW31225	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
94	BW31236	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
95	BW31260	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
97	BW31261	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
99	CN1837	AB	*T. turgidum ssp. turgidum*	Cultivated	Tetraploid	Spring	Portugal	PGRC
100	CN11761	AB	*T. turgidum ssp. turgidum*	Cultivated	Tetraploid	Spring		PGRC
101	CN33858	AB	*T. turgidum ssp. turgidum*	Cultivated	Tetraploid	Spring	United States of America	PGRC
102	CN40845	AB	*T. turgidum ssp. turgidum*	Cultivated	Tetraploid	Spring	Ethiopia	PGRC
103	CN40848	AB	*T. turgidum ssp. turgidum*	Cultivated	Tetraploid	Spring	Ethiopia	PGRC
104	DW7195	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	Spain	AAFC
105	CN32492	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Israel	PGRC
106	CN32493	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Spain	PGRC
107	CN32494	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Hungary	PGRC
108	CN32495	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Switzerland	PGRC
110	CWI18234	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Armenia	AAFC
111	CWI15345	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring		AAFC
112	CWI16984	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Spain	AAFC
113	CWI17128	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Spain	AAFC
116	CWI18902	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Turkey	AAFC
117	5310	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Mexico	AAFC

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
118	5315	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Mexico	AAFC
119	CN7773	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Israel	PGRC
120	CN7786	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Tunisia	PGRC
121	CN10163	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	South Africa	PGRC
124	CN2652	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Romania	PGRC
125	CN32482	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Armenia	PGRC
126	CN32483	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Saudi Arabia	PGRC
127	CN32486	AB	*T. turgidum ssp. dicoccoides*	Progenitor	Tetraploid	Spring	Ethiopia	PGRC
128	CN32488	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Serbia	PGRC
131	CWI19155	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Ethiopia	AAFC
132	DW7193	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Tunisia	AAFC
133	DW7191	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Tunisia	AAFC
134	CN32496	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Georgia	PGRC
135	CN32498	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Hungary	PGRC
136	CN32500	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Iraq	PGRC
137	CN32502	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Armenia	PGRC
138	CN40686	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Georgia	PGRC
139	DW7196	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Jordan	AAFC
140	CWI5222	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring		AAFC
141	CWI44150	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring		AAFC
142	CWI44453	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Russia	AAFC
143	CWI44562	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Russia	AAFC
144	CWI44563	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Iran	AAFC
145	CWI44464	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring	Russia	AAFC
146	Blackbird	AB	*T. turgidum ssp. carthlicum*	Cultivated	Tetraploid	Spring		
147	CN1839	AB	*T. turgidum ssp. turanicum*	Cultivated	Tetraploid	Spring		PGRC
148	CN10543	AB	*T. turgidum ssp. turanicum*	Cultivated	Tetraploid	Spring	Canada	PGRC
149	CN11587	AB	*T. turgidum ssp. turanicum*	Cultivated	Tetraploid	Spring		PGRC
151	CN45831	AB	*T. turgidum ssp. polonicum*	Cultivated	Tetraploid	Spring		PGRC
152	CN51900	AB	*T. turgidum ssp. polonicum*	Cultivated	Tetraploid	Spring		PGRC
154	CN51933	AB	*T. turgidum ssp. polonicum*	Cultivated	Tetraploid	Spring		PGRC
160	CN1849	ABD	*T. aestivum ssp. spelta*	Cultivated	Hexaploid	Spring		PGRC

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
161	CN2758	ABD	*T. aestivum ssp. spelta*	Cultivated	Hexaploid	Spring		PGRC
162	CN12229	ABD	*T. aestivum ssp. spelta*	Cultivated	Hexaploid	Spring	Czech	PGRC
163	CN12261	ABD	*T. aestivum ssp. spelta*	Cultivated	Hexaploid	Spring	Czech	PGRC
164	CN37599	ABD	*T. aestivum ssp. spelta*	Cultivated	Hexaploid	Spring	Sweden	PGRC
165	CN2674	ABD	*T. aestivum ssp. compactum*	Cultivated	Hexaploid	Spring		PGRC
166	CN12213	ABD	*T. aestivum ssp. compactum*	Cultivated	Hexaploid	Spring		PGRC
168	CWI42787	ABD	*T. aestivum ssp. compactum*	Cultivated	Hexaploid	Spring	Denmark	AAFC
169	CN11647	ABD	*T. aestivum ssp. sphaerococcum*	Cultivated	Hexaploid	Spring	Hungary	PGRC
171	CN33803	ABD	*T. aestivum ssp. sphaerococcum*	Cultivated	Hexaploid	Spring	Georgia	PGRC
172	CN33892	ABD	*T. aestivum ssp. sphaerococcum*	Cultivated	Hexaploid	Spring	Pakistan	PGRC
173	CN33893	ABD	*T. aestivum ssp. sphaerococcum*	Cultivated	Hexaploid	Spring	India	PGRC
174	G3893	ABD	*T. aestivum ssp. sphaerococcum*	Cultivated	Hexaploid	Spring		PGDC
175	CWI42988	ABD	*T. aestivum ssp. sphaerococcum*	Cultivated	Hexaploid	Spring		PGRC
176	CN99032	ABD	*T. aestivum ssp. macha*	Cultivated	Hexaploid	Spring		PGRC
359	01C0203285	ABD	*T. aestivum ssp. lutescense*	Cultivated	Hexaploid	Spring	Czech	RICP,CZECH
365	01C0105549	ABD	*T. aestivum ssp. lutescense*	Cultivated	Hexaploid	Spring	Yugoslavia	RICP,CZECH
455	01C0203832	ABD	*T. aestivum ssp. lutescense*	Cultivated	Hexaploid	Spring	Australia	RICP,CZECH
456	01C0204206	ABD	*T. aestivum ssp. graecum*	Cultivated	Hexaploid	Spring	Australia	RICP,CZECH
483	RL5265	D	*Ae. tauschii var. anathera*	Progenitor	Diploid	Winter		AAFC
490	RL5499	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
491	RL5524	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Azerbaijan	AAFC
493	RL5534	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Azerbaijan	AAFC
494	RL5536	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Azerbaijan	AAFC
495	RL5537	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Azerbaijan	AAFC
498	RL5554	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Uzbekistan	AAFC
521	RL5686	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter	Azerbaijan	AAFC
527	RL5695	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter	Azerbaijan	AAFC
528	RL5699	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter	Iran	AAFC
529	RL5736	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
534	RL5741	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
535	RL5744	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
536	RL5745	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
538	RL5747	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
539	RL5750	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
545	RL5761	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter	Iran	AAFC
549	RL5768	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter	Iran	AAFC
550	RL5769	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter	Iran	AAFC
553	RL5772	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
556	RL5775	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
559	RL5778	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
562	RL5781	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
564	RL5783	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
567	RL5786	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
572	RL5791	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter		AAFC
574	RL5793	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter		AAFC
575	RL5794	D	*Ae. tauschii var. strangulata*	Progenitor	Diploid	Winter		AAFC
578	RL5798	D	*Ae. tauschii var. mayeri*	Progenitor	Diploid	Winter		AAFC
579	C34	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	AS2296/AS2388	AAFC
580	C35	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	AS2313/AS2388	AAFC
581	C36	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	AS2382/AS2388	AAFC
582	C37	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	AS2255/AS2395	AAFC
583	C38	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	AS2255/AS2393	AAFC
584	C39	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	AS285/AS2386	AAFC
585	C40	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	AS285/AS2404	AAFC
586	C41	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	AS286/AS66	AAFC
587	C42	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	AS286/AS2386	AAFC
588	C43	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	AS286/AS2407	AAFC
589	C44	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	Langdon/AS2386	AAFC
590	C45	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	Langdon/AS2399	AAFC
591	C46	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	Langdon/AS2404	AAFC
592	C48	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	AS2240/AS77	AAFC
593	C49	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI94666/AS2405	AAFC
594	C50	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI94627/AS2386	AAFC
595	C51	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	PI94650/AS2404	AAFC

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
596	C52	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI94655/AS2404	AAFC
597	C53	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI94655/AS2407	AAFC
598	C54	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI94666/AS2407	AAFC
599	C55	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI94675/AS2405	AAFC
600	C56	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI113961/AS2404	AAFC
601	C57	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI113961/AS2386	AAFC
602	C61	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI352335/AS2386	AAFC
603	C62	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	PI355465/AS2405	AAFC
604	C63	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	PI355476/AS2404	AAFC
605	C64	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	AS2399/PI355527	AAFC
606	C65	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI377655/AS2399	AAFC
607	C66	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI377655/AS2386	AAFC
608	C67	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Winter	PI355465/AS60	AAFC
609	C68	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	PI211681AS2386	AAFC
610	M321	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring		
611	AS2240	AB	*T. turgidum*	Cultivated	Tetraploid	Spring		AAFC
612	PI94614	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Ukraine	NSGC
613	PI94627	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Asia Minor	NSGC
614	PI94650	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Czech	NSGC
615	PI94655	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Bulgaria	NSGC
616	PI94666	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Russia	NSGC
617	PI94675	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	United States of America	NSGC
618	PI113961	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Georgia	NSGC
619	PI113963	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Georgia	NSGC
620	PI352335	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	United States of America	NSGC
621	PI355465	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Belgium	NSGC
622	PI355476	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Belgium	NSGC
623	PI377655	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Serbia	NSGC
624	PI415152	AB	*T. turgidum ssp. dicoccum*	Progenitor	Tetraploid	Spring	Israel	NSGC
625	PI211691	AB	*T. turgidum ssp. turanicum*	Cultivated	Tetraploid	Spring	Turkey	NSGC
626	Langdon	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	United States of America	USDA-ARS
627	AS2255	AB	*T. turgidum*	Cultivated	Tetraploid	Spring	China	AAFC

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
628	AS285	AB	*T. turgidum*	Cultivated	Tetraploid	Spring		AAFC
629	AS286	AB	*T. turgidum*	Cultivated	Tetraploid	Spring		AAFC
630	AC Avonlea	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	AAFC: SPARC
631	AC Morse	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	AAFC: CRC
632	AC Napoleon	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	CDC
634	AC Pathfinder	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	AAFC: SPARC
635	Commander	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	AAFC: SPARC
636	DT 773 (Brigade)	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	AAFC: SPARC
637	DT 776 (Eurostar)	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	AAFC: SPARC
638	Kyle	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	AAFC: SPARC
639	Strongfield	AB	*T. turgidum ssp. durum*	Cultivated	Tetraploid	Spring	Canada	AAFC: SPARC
671	Thatcher	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	United States of America	PGRC
693	Chinese Spring	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	China	USDA-ARS
694	Superb	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: CRC
695	Sumai3	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	China	NSGC
696	BW278	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring		AAFC
697	White Glenlea	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	PSD
698	AC Karma	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: SPARC
699	Opata	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Mexico	NSGC
700	Synthetic W7984	ABD	*T. aestivum (synt)*	SHW	Hexaploid	Spring	Mexico	AAFC
701	RL4452	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC
702	AC Domain	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	CDC
703	Roblin	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: CRC
704	AC Reed	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: LRC

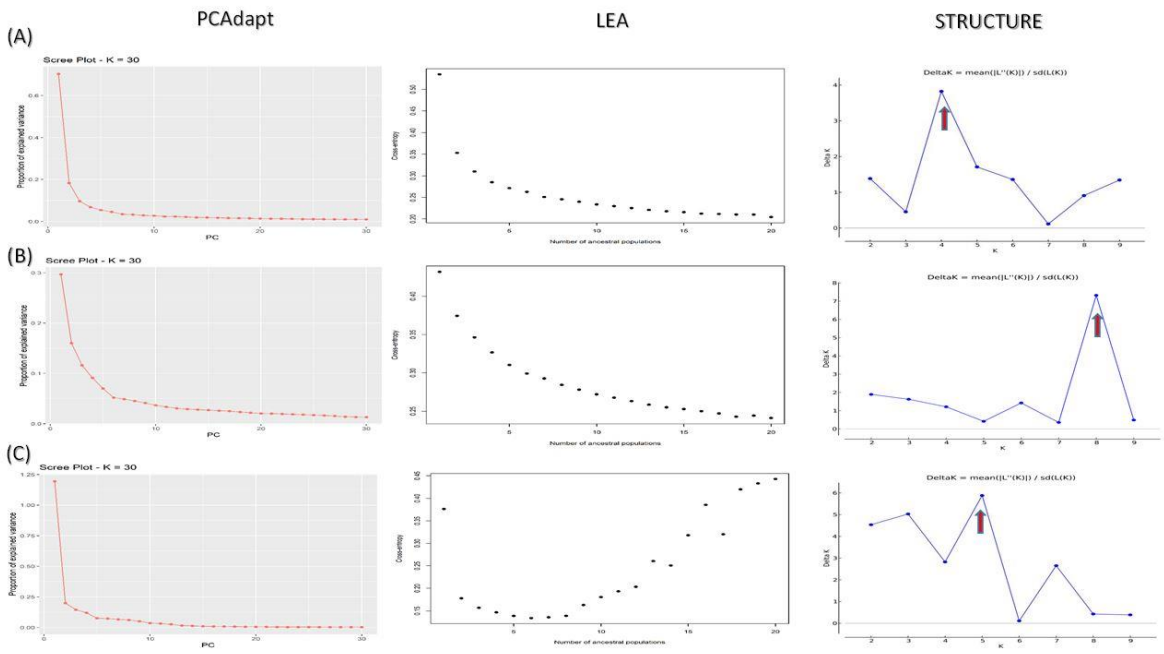
Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
705	Grandin	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: CRC
706	Foremost	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: SPARC & LRC
707	RL6058	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring		AAFC
708	RL6091	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring		AAFC
709	RL6159	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring		AAFC
710	RL6069	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring		AAFC
711	RL6070	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring		AAFC
712	RL6106	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring		AAFC
713	RL6077	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring		AAFC
714	RL6050	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Iran	AAFC
715	Frontana	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Brazil	NSGC
716	Terenzio	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Portugal	USDA-ARS
717	PI58548	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	China	NSGC
718	Fielder-1	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	United States of America	AAFC: LRC
719	Lalbahadur (Pavon 1B)	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Mexico	AAFC
720	AC Minto	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: CRC
721	Pasqua	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: CRC
722	Selkirk	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC
723	CN44011_Toropi	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Brazil	PGRC
724	01C0204206_Sunbird	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Australia	RICP,CZECH
725	AC Barrie	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: SPARC
726	AC Vista	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: SPARC
727	Aus30426_Otane	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	New Zealand	AWCC
728	Bluesky	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: Beaverlodge
729	BW31100	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Mexico	AAFC
730	BW779_Tezanos	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Argentina	AAFC

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
	Printos Precoz							
731	Carberry BW874	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Mexico	AAFC: SPARC
732	CN10719_Kenya Farmer	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Kenya	PGRC
733	CN11057_Maria Escobar	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Peru	PGRC
734	CN11189_Neepawa	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	PGRC
735	CN32076_Nobeoka Bozu	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Japan	PGRC
736	CN32077_Nyu Bay	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Japan	PGRC
737	CN9591_Bage	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Uruguay	PGRC
738	Fieldstar BW365	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: CRC
739	CN10112_Gabo	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Australia	PGRC
740	HARTOG	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Australia	AWCC
741	CN38927_Katepwa	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	PGRC
742	LEADER (BW535)	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: SPARC
743	Lillian = BW776	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: SPARC
744	ND694=Parshall	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	United States of America	NDSU
745	Pavon 76	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Mexico	AAFC
746	Peace(PT416)	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: CRC
747	Regent	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC: CRC
748	Salamouni	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Lebanon	USDA-ARS
749	Sonora 64	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Mexico	AAFC
750	Stanley	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	CDC
751	Weebill-1	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Mexico	AAFC
752	Wilgoyne	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Australia	AAFC
754	AC Cora	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	CDC
755	JANZ	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Australia	AWCC
756	HC374	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Spring	Canada	AAFC

Entry[1]	Accession	Genome	Category	Growth habit	Ploidy	Panel	Origin/Pedigree	Seed source
757	C23 (AS60)	D	*Ae. tauschii*	Progenitor	Diploid	Winter	Iran	AAFC
758	C24 (AS66)	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
759	C26 (AS2386)	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
760	C29 (AS2395)	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
761	C30 (AS2399)	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
762	C31 (AS2404)	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
763	C32 (AS2405)	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
764	C33 (AS2407)	D	*Ae. tauschii*	Progenitor	Diploid	Winter		AAFC
801	EKC135_RL5716	DC	*Ae. cylindrica*	Wild	Tetraploid	Winter		AAFC
805	EKC154_RL5853	MU	*Ae. geniculata (=ovata)*	Wild	Tetraploid	Winter		AAFC
806	EKC158_RL5857	MU	*Ae. geniculata (=ovata)*	Wild	Tetraploid	Winter		AAFC
807	EKC159_RL5858	MU	*Ae. geniculata (=ovata)*	Wild	Tetraploid	Winter		AAFC
808	EKC160_RL5859	MU	*Ae. geniculata (=ovata)*	Wild	Tetraploid	Winter		AAFC
810	EKC163_RL5017	SU	*Ae. peregrina (= variabilis)*	Wild	Tetraploid	Winter		AAFC
811	EKC164_RL5849	SU	*Ae. peregrina (= variabilis)*	Wild	Tetraploid	Winter		AAFC
815	EKC186_RL5847	UM	*Ae. biuncialis = (Ae. lorentii)*	Wild	Tetraploid	Winter		AAFC
871	Emerson-1	ABD	*T. aestivum ssp. aestivum*	Cultivated	Hexaploid	Winter	Canada	AAFC: LRC

The species names and genome symbols are according to Kimber and Tsunewaki [138].

[1] Entry numbers that were a part of the 279 accession used in this study



Appendix 2 Scree plots for determining the number of subpopulations k for the following diversity panels: combined (A), spring (B) and winter (C). PCAdapt (left graphs) plots the number of principal components (PCs) against the proportion of variance explained. LEA (center graphs) plots cross-entropy validation errors against the number of ancestral populations (k). STRUCTURE calculates a number of components based on the second-order rate of change of the likelihood (ΔK). The ΔK shows a clear peak at the true value of k.

Appendix 3 Quantitative trait nucleotides (QTNs) that were identified for four traits on three panels; Combined, Spring and Winter using nine GWAS models

QTN	Chromosome	Position	Trait	Models	R^2 (%)
Combined Panel					
11655_BS00090277_51	6B	478949875	DON	FASTmrEMMA	7.06
11935_BS00095515_51	3B	788060470	VRI	FASTmrMLM	0.08
12462_BS00110052_51	1D	342788950	DON	pLARmEB	2.15
12504_BS00110278_51	1B	601013650	SEV	ISIS EM-BLASSO	9.09
12915_CAP11_c4929_135	7A	271525529	VRI	MLM	3.73
13180_CAP12_c118_95	6A	557441706	SEV	pLARmEB	1.34
13266_CAP12_c1981_78	1B	462097527	SEV	ISIS EM-BLASSO	11.30
14041_CAP7_c3664_305	4B	5666429	INC	FarmCPU	5.21
14141_CAP7_c4879_249	1A	534934626	DON	FarmCPU	2.16
15120_D_contig00171_426	2D	519171206	SEV	pKWmEB	5.60
15288_D_contig03979_394	7D	515988498	INC	ISIS EM-BLASSO	0.14
16242_D_contig25796_184	4D	149409210	INC	GLM	0.03
16351_D_contig28176_605	5D	285598548	INC	GLM	0.03
16410_D_contig29364_197	1D	50541491	INC	GLM	0.06
16611_D_contig34321_189	4D	34683294	INC	GLM	0.03
16708_D_contig36996_183	4D	261455568	INC	GLM	0.03
16786_D_contig39568_111	7D	198286171	INC	GLM	0.07
16918_D_contig58377_170	5D	372287609	VRI	ISIS EM-BLASSO	0.13
17047_D_contig67724_316	4D	62962288	INC	GLM	0.03
17105_D_contig71738_238	6D	103996137	INC	GLM	0.05
17937_D_GB5Y7FA01A570E_178	2D	13089524	INC	GLM	0.05
1796_BobWhite_c24069_257	2A	767750947	DON	pKWmEB	5.06
17971_D_GB5Y7FA01BHBOA_91	2D	380223009	INC	FASTmrMLM	0.35
18187_D_GBB4FNX01BUWXG_371	7D	312153542	INC	GLM	1.49
18201_D_GBB4FNX01CNX9D_254	6D	42442831	INC	GLM	2.43
18219_D_GBB4FNX01DLV3V_102	6D	476758547	INC	GLM	1.49
18510_D_GBQ4KXB01C9DNU_246	1D	114815650	INC	GLM	0.90
20257_Ex_c24766_653	6B	577220314	SEV	GLM	2.22
20710_Ex_c4348_113	2A	157642581	VRI	MLM	3.73
21963_Excalibur_c11795_902	5A	320927775	VRI	MLM	3.93
22159_Excalibur_c12835_2524	5A	284243685	VRI	pKWmEB	1.81
22293_Excalibur_c13603_1389	1A	594835828	VRI	MLM	3.73
23178_Excalibur_c18644_566	2D	614462146	VRI	FASTmrMLM	3.86
2399_BobWhite_c30760_262	3D	287930031	VRI	MLM	9.09
24589_Excalibur_c27531_122	7B	36457447	VRI	MLM	3.73
24840_Excalibur_c29425_457	3A	715273236	VRI	MLM	9.09
25440_Excalibur_c34269_460	6A	518485246	VRI	MLM	3.73
25829_Excalibur_c37474_179	1A	308680724	DON	GLM	14.70
26899_Excalibur_c47756_323	6B	725266977	VRI	MLM	3.73
27335_Excalibur_c52772_1592	3D	238259506	DON	GLM	4.55
27682_Excalibur_c56854_181	7B	34503448	VRI	MLM	0.23
27951_Excalibur_c60262_359	2D	568340040	INC	GLM	6.21

QTN	Chromosome	Position	Trait	Models	R^2 (%)
28718_Excalibur_c74403_580	7A	21158431	VRI	ISIS EM-BLASSO	0.93
29365_Excalibur_c9183_1397	7D	13416701	VRI	ISIS EM-BLASSO	2.32
29617_Excalibur_c98275_372	2D	617994904	SEV	GLM	13.38
30022_Excalibur_rep_c104322_115	5A	659174173	VRI	MLM	3.73
30482_Excalibur_rep_c110450_286	1D	234966752	DON	GLM	0.01
30867_Excalibur_rep_c67473_320	5B	509742704	VRI	GLM	0.33
31276_Excalibur_rep_c77404_218	1A	35385737	DON	ISIS EM-BLASSO	0.70
31535_Excalibur_s111897_126	6B	123536882	VRI	pKWmEB	8.38
32140_GENE-0910_153	2A	683486986	DON	mrMLM	12.70
32618_GENE-1733_358	3B	598510365	VRI	MLM	19.89
32785_GENE-2058_43	3D	507743875	VRI	MLM	19.89
34012_GENE-4564_387	7A	699200157	INC	mrMLM	7.08
34665_IAAV3115	4A	100654839	INC	pKWmEB	0.06
35131_IAAV6011	1B	682639244	INC	FASTmrEMMA	0.30
35578_IAAV902	3A	574651540	INC	pKWmEB	1.03
35687_IACX11310	3B	601609681	DON	GLM	5.07
35946_IACX419	6B	531514116	VRI	GLM	1.88
36458_Jagger_c1781_99	3A	208155305	VRI	MLM	9.09
36611_Jagger_c4901_95	3B	53703225	SEV	pLARmEB	4.46
36640_Jagger_c550_91	5D	409443434	DON	GLM	3.12
36738_Jagger_c736_109	4A	201413600	VRI	MLM	3.73
36771_Jagger_c8121_167	5B	698065732	DON	GLM	0.08
37431_JD_c4128_277	3B	73391914	INC	mrMLM	1.36
38451_Ku_c14376_1164	3A	512336082	VRI	MLM	3.73
39346_Ku_c3907_2063	2A	209712279	VRI	MLM	3.73
39560_Ku_c51309_212	2B	723230788	DON	GLM	16.79
40414_Kukri_c107483_476	3D	89939402	INC	GLM	22.33
40694_Kukri_c119_295	3B	781032674	VRI	FASTmrMLM	0.10
42295_Kukri_c20269_422	3A	167512111	VRI	MLM	3.73
42528_Kukri_c21628_1215	7B	613149662	DON	pKWmEB	0.39
42531_Kukri_c2164_1856	3B	847968058	DON	mrMLM	0.01
42938_Kukri_c24194_2659	4A	748723146	DON	ISIS EM-BLASSO	16.76
43566_Kukri_c2842_248	6D	331186031	SEV	FASTmrEMMA	7.25
43875_Kukri_c30641_403	7A	292086334	VRI	FarmCPU	3.48
44197_Kukri_c33403_781	3A	608539373	VRI	MLM	3.73
44878_Kukri_c39041_574	7A	504075930	VRI	MLM	3.73
45001_Kukri_c40134_451	4A	38694592	VRI	MLM	3.73
45188_Kukri_c42075_156	3D	64885909	DON	ISIS EM-BLASSO	2.29
45329_Kukri_c43306_282	7D	4394602	INC	FASTmrEMMA	25.20
46152_Kukri_c51296_438	7B	570077904	SEV	mrMLM	4.07
46165_Kukri_c51474_334	1B	463285004	DON	GLM	0.08
46288_Kukri_c5282_622	2A	19787961	DON	FASTmrEMMA	20.17
46344_Kukri_c53506_1141	6B	485988986	VRI	pLARmEB	17.55
46794_Kukri_c59403_339	2D	77411184	VRI	mrMLM	0.29
47439_Kukri_c67939_649	1B	690717518	INC	ISIS EM-BLASSO	2.00

QTN	Chromosome	Position	Trait	Models	R^2 (%)
47539_Kukri_c7244_303	2A	202098196	VRI	MLM	3.73
47776_Kukri_c7874_1096	5A	535804823	VRI	pLARmEB	14.02
48175_Kukri_c8929_2161	2A	531255241	VRI	MLM	3.73
49061_Kukri_rep_c105016_212	4A	509326342	DON	GLM	12.04
49397_Kukri_rep_c109210_417	3D	235052801	DON	GLM	12.67
50253_Kukri_rep_c73149_106	3D	444204837	SEV	FASTmrEMMA	0.00
50449_Kukri_rep_c86638_306	3B	257382735	VRI	MLM	3.73
50643_Kukri_s112441_279	3A	205619000	VRI	MLM	3.73
52304_Ra_c58279_702	2A	45928203	VRI	FASTmrMLM	0.58
52729_Ra_c8311_1521	5A	353947508	VRI	MLM	3.73
53059_RAC875_c104483_216	5A	605186837	VRI	ISIS EM-BLASSO	0.32
53194_RAC875_c10866_1219	7D	342117441	VRI	MLM	4.15
53989_RAC875_c14732_461	5D	555935295	DON	GLM	7.67
54091_RAC875_c15402_286	3A	222954082	VRI	MLM	3.73
55230_RAC875_c21750_784	2D	630191012	VRI	pKWmEB	3.76
55515_RAC875_c23473_165	2B	59105039	VRI	MLM	3.73
55566_RAC875_c23799_2056	5A	105916657	VRI	MLM	3.73
55860_RAC875_c25864_770	7A	659499214	VRI	MLM	3.73
56084_RAC875_c27611_467	2B	9233744	SEV	ISIS EM-BLASSO	7.73
56355_RAC875_c29540_413	1A	505749527	VRI	pKWmEB	1.05
57442_RAC875_c38693_319	7B	752222562	INC	pKWmEB	0.40
57507_RAC875_c39339_400	3B	817272964	DON	mrMLM	5.66
57676_RAC875_c41113_144	1A	344959765	DON	GLM	0.18
57892_RAC875_c430_1072	3B	404475194	VRI	GLM	5.22
58728_RAC875_c51216_804	4B	645758432	VRI	MLM	3.73
58832_RAC875_c52458_454	2A	696313164	VRI	pLARmEB	5.48
59332_RAC875_c57998_165	2D	191033496	VRI	MLM	3.73
59530_RAC875_c60522_1342	5A	567522493	DON	ISIS EM-BLASSO	0.64
59983_RAC875_c65882_668	2B	771847837	DON	GLM	8.57
60785_RAC875_c8494_93	4A	500153212	INC	pKWmEB	25.18
610_BobWhite_c14222_571	2B	104978464	DON	mrMLM	13.17
61219_RAC875_c9789_1341	3B	270790420	DON	GLM	12.67
61335_RAC875_rep_c102406_205	4A	508192004	DON	GLM	12.04
61500_RAC875_rep_c105780_64	2B	240717510	VRI	MLM	12.04
61888_RAC875_rep_c109516_822	7A	88401545	VRI	MLM	3.73
63043_RAC875_rep_c74891_340	4A	509324648	DON	GLM	3.73
63061_RAC875_rep_c75748_172	3D	277424368	DON	GLM	12.04
63392_RAC875_s105188_92	1B	462883976	DON	GLM	12.67
63497_RAC875_s119017_245	2D	547202763	DON	GLM	14.57
63667_RFL_Contig1511_612	7A	134396890	VRI	MLM	3.73
64322_RFL_Contig337_1645	2B	645411473	DON	GLM	0.26
64957_RFL_Contig5277_888	2A	775589238	VRI	ISIS EM-BLASSO	5.76
65349_TA001138-1003	5B	589168687	VRI	pLARmEB	1.83
65469_TA001874-1495	2B	37419792	INC	FarmCPU	1.40
65683_TA003419-0641	6D	487522708	VRI	FASTmrMLM	1.08

QTN	Chromosome	Position	Trait	Models	R^2 (%)
66386_Tdurum_contig102328_129	7B	569916476	SEV	GLM	3.33
67051_Tdurum_contig11382_669	3A	549601573	VRI	MLM	3.73
68232_Tdurum_contig14482_423	2B	786378366	DON	FASTmrEMMA	2.79
68352_Tdurum_contig15126_286	4A	730542903	DON	pKWmEB	0.06
68412_Tdurum_contig15438_231	2A	19671792	DON	GLM	24.50
68456_Tdurum_contig15718_314	3B	659430855	VRI	MLM	3.73
68558_Tdurum_contig16612_337	6A	500011565	VRI	MLM	3.73
6859_BS00022014_51	2B	645301133	DON	GLM	0.20
68934_Tdurum_contig21518_326	4A	499103420	VRI	MLM	3.73
6908_BS00022107_51	5B	35393045	DON	pKWmEB	0.00
69162_Tdurum_contig25674_294	5A	563619393	VRI	MLM	3.73
69960_Tdurum_contig30108_218	3A	435143001	VRI	MLM	3.73
69963_Tdurum_contig30121_240	1A	91282013	SEV	FarmCPU	0.23
70065_Tdurum_contig30621_328	7A	71357685	INC	FASTmrMLM	21.27
70129_Tdurum_contig30916_316	3B	816074580	VRI	MLM	3.73
7079_BS00022432_51	1A	505746422	VRI	pLARmEB	5.57
72711_Tdurum_contig60165_722	5B	698075859	DON	GLM	7.25
74007_Tdurum_contig97656_120	3B	24979593	DON	ISIS EM-BLASSO	22.45
74043_Tdurum_contig98478_494	4B	118610356	SEV	ISIS EM-BLASSO	1.75
75364_wsnp_BE404601B_Ta_2_1	2B	172041850	SEV	pKWmEB	2.14
75644_wsnp_BF292596A_Ta_1_3	3A	647674948	SEV	pLARmEB	5.12
76700_wsnp_Ex_c15188_23387523	1A	307813760	DON	GLM	0.02
77039_wsnp_Ex_c19525_28494827	6B	721736556	DON	FASTmrEMMA	14.39
77908_wsnp_Ex_c3681_6715277	1B	462883977	DON	GLM	0.00
78002_wsnp_Ex_c40019_47165575	5B	607884903	VRI	FASTmrMLM	0.36
78397_wsnp_Ex_c54092_57099525	5B	554616628	DON	ISIS EM-BLASSO	1.97
78528_wsnp_Ex_c5936_10411877	6B	564587592	VRI	pKWmEB	2.42
78871_wsnp_Ex_c852_1657319	1B	461128280	VRI	ISIS EM-BLASSO	16.46
79457_wsnp_Ex_rep_c68712_67571580	1B	462183400	VRI	GLM	16.56
79666_wsnp_JD_c14769_14413046	4A	105196277	DON	GLM	14.23
80064_wsnp_Ku_c12701_20446223	7D	82497413	INC	GLM	7.05
80235_wsnp_Ku_c21275_31007309	5A	567525415	DON	pKWmEB	3.88
80328_wsnp_Ku_c2797_5284087	1B	687093328	SEV	FarmCPU	1.97
80996_wsnp_Ra_c2027_3945713	1B	462883485	DON	GLM	11.24
80997_wsnp_Ra_c2027_3945764	1B	462883434	INC	GLM	15.82
81057_wsnp_Ra_c2730_5190076	6B	131945815	SEV	pLARmEB	1.88
81112_wsnp_Ra_c34203_42948357	1A	305745550	DON	FarmCPU	0.02
81493_wsnp_RFL_Contig3344_3442711	3A	37135958	VRI	ISIS EM-BLASSO	9.12
8213_BS00040246_51	3B	108863021	DON	FarmCPU	12.48
8296_BS00041947_51	1B	440499591	INC	mrMLM	18.00
8329_BS00043071_51	1B	206557175	DON	mrMLM	12.69
9653_BS00065865_51	2A	721702719	DON	mrMLM	5.45
Spring Panel					
10435_BS00070051_51	1B	27126042	INC	ISIS EM-BLASSO	0.37
11021_BS00078597_51	6B	181966848	INC	GLM	3.09

QTN	Chromosome	Position	Trait	Models	R^2 (%)
11655_BS00090277_51	6B	478949875	DON	ISIS EM-BLASSO	4.62
11935_BS00095515_51	3B	788060470	INC	pKWmEB	0.74
12462_BS00110052_51	1D	342788950	INC	GLM	17.44
12504_BS00110278_51	1B	601013650	SEV	FarmCPU	12.29
12651_CAP11_c1022_117	3A	714842763	DON	GLM	3.86
12652_CAP11_c1022_66	3A	714842712	DON	GLM	4.35
13266_CAP12_c1981_78	1B	462097527	INC	GLM	18.43
14141_CAP7_c4879_249	1A	534934626	DON	FarmCPU	10.39
15079_CAP8_rep_c9413_186	3B	234539883	SEV	GLM	5.21
1796_BobWhite_c24069_257	2A	767750947	DON	GLM	9.92
19609_Ex_c104539_35	2B	490569231	INC	ISIS EM-BLASSO	11.37
20257_Ex_c24766_653	6B	577220314	SEV	GLM	8.00
22159_Excalibur_c12835_2524	5A	284243685	INC	mrMLM	0.43
24303_Excalibur_c25368_372	1B	71130493	SEV	GLM	6.65
24662_Excalibur_c28045_741	1A	626327417	DON	ISIS EM-BLASSO	0.44
25627_Excalibur_c35713_106	2D	60807566	DON	mrMLM	3.56
25829_Excalibur_c37474_179	1A	308680724	DON	GLM	1.60
27335_Excalibur_c52772_1592	3D	238259506	DON	GLM	5.04
27534_Excalibur_c55096_613	1D	735448298	DON	mrMLM	8.89
28718_Excalibur_c74403_580	7A	21158431	SEV	FASTmrMLM	1.34
30482_Excalibur_rep_c110450_286	1D	234966752	DON	GLM	2.71
31276_Excalibur_rep_c77404_218	1A	35385737	DON	FarmCPU	21.84
31535_Excalibur_s111897_126	6B	123536882	INC	pLARmEB	4.44
32140_GENE-0910_153	2A	683486986	DON	GLM	1.83
33266_GENE-3158_262	5A	644395007	INC	GLM	2.96
33468_GENE-3569_500	1A	17317956	VRI	ISIS EM-BLASSO	2.81
34422_IAAV1673	6B	131948570	SEV	GLM	3.17
34512_IAAV2161	6B	416649770	VRI	GLM	16.07
35131_IAAV6011	1B	682639244	INC	GLM	10.01
35457_IAAV8279	6B	522166024	VRI	GLM	17.16
35555_IAAV8886	2D	125754699	SEV	pKWmEB	2.22
35578_IAAV902	3A	574651540	INC	pKWmEB	2.69
35946_IACX419	6B	531514116	VRI	GLM	3.45
36146_IACX6349	2D	482420019	INC	pKWmEB	6.19
37431_JD_c4128_277	3B	73391914	INC	mrMLM	9.07
38978_Ku_c2606_779	6B	181960036	INC	GLM	2.37
41483_Kukri_c15912_2330	3A	680860234	SEV	pKWmEB	3.77
43087_Kukri_c25145_332	7D	632780685	VRI	GLM	1.31
43566_Kukri_c2842_248	6D	331186031	INC	GLM	18.34
44603_Kukri_c36747_195	5D	340132967	SEV	GLM	8.47
45087_Kukri_c4097_898	1B	569236929	INC	pKWmEB	0.01
46152_Kukri_c51296_438	7B	570077904	SEV	GLM	7.20
46165_Kukri_c51474_334	1B	463285004	INC	GLM	12.55
46288_Kukri_c5282_622	2A	19787961	DON	GLM	16.38
46344_Kukri_c53506_1141	6B	485988986	INC	FarmCPU	20.64

QTN	Chromosome	Position	Trait	Models	R^2 (%)
47612_Kukri_c74912_285	2D	431336433	INC	FASTmrEMMA	2.98
49061_Kukri_rep_c105016_212	4A	509326342	DON	GLM	3.14
49397_Kukri_rep_c109210_417	3D	235052801	DON	GLM	5.04
50813_Ra_c106376_879	1B	142977868	INC	pLARmEB	5.90
51731_Ra_c29107_289	2D	18602466	INC	pKWmEB	0.70
51875_Ra_c33766_656	3B	834159260	INC	GLM	4.11
52304_Ra_c58279_702	2A	45928203	INC	pLARmEB	1.02
52751_Ra_c8677_465	6B	182399198	INC	GLM	1.91
53989_RAC875_c14732_461	5D	555935295	INC	GLM	3.44
54254_RAC875_c16255_570	6A	17374497	DON	GLM	1.43
54537_RAC875_c17747_1380	3B	235783172	SEV	GLM	5.21
56662_RAC875_c3187_873	1B	642203581	INC	pKWmEB	5.34
57892_RAC875_c430_1072	3B	404475194	INC	GLM	3.19
58832_RAC875_c52458_454	2A	696313164	INC	mrMLM	3.73
59635_RAC875_c61892_309	3D	265592552	DON	GLM	5.54
60140_RAC875_c6798_467	1A	538108963	DON	ISIS EM-BLASSO	5.14
61219_RAC875_c9789_1341	3B	270790420	DON	GLM	5.04
61335_RAC875_rep_c102406_205	4A	508192004	DON	GLM	3.14
63043_RAC875_rep_c74891_340	4A	509324648	DON	GLM	3.14
63061_RAC875_rep_c75748_172	3D	277424368	DON	GLM	5.04
63392_RAC875_s105188_92	1B	462883976	INC	GLM	11.31
63897_RFL_Contig2290_184	1B	567447186	INC	ISIS EM-BLASSO	0.04
64322_RFL_Contig337_1645	2B	645411473	DON	GLM	7.82
65469_TA001874-1495	2B	37419792	INC	GLM	0.74
65683_TA003419-0641	6D	487522708	SEV	FarmCPU	0.44
66386_Tdurum_contig102328_129	7B	569916476	SEV	GLM	6.33
68412_Tdurum_contig15438_231	2A	19671792	DON	GLM	20.65
6859_BS00022014_51	2B	645301133	DON	GLM	6.58
68811_Tdurum_contig19512_292	7B	331690915	DON	GLM	3.13
70635_Tdurum_contig41096_416	1B	421151869	INC	GLM	10.82
71745_Tdurum_contig47006_2711	1A	598092152	SEV	pKWmEB	3.72
71780_Tdurum_contig47269_904	2D	601674145	VRI	ISIS EM-BLASSO	0.98
71916_Tdurum_contig48695_527	3A	461797507	VRI	pLARmEB	6.78
72169_Tdurum_contig51313_408	3A	176407029	INC	mrMLM	0.17
72711_Tdurum_contig60165_722	5B	698075859	SEV	GLM	3.01
74007_Tdurum_contig97656_120	3B	24979593	DON	GLM	20.19
74456_tplb0032a08_1721	3D	5664733	INC	FarmCPU	0.46
76127_wsnp_CD454706B_Ta_1_1	2D	610294752	INC	mrMLM	0.08
76700_wsnp_Ex_c15188_23387523	1A	307813760	DON	GLM	1.60
77039_wsnp_Ex_c19525_28494827	6B	721736556	SEV	pKWmEB	3.60
77908_wsnp_Ex_c3681_6715277	1B	462883977	INC	GLM	11.31
78871_wsnp_Ex_c852_1657319	1B	461128280	INC	GLM	16.81
79037_wsnp_Ex_rep_c101757_87064771	5A	438395139	VRI	GLM	2.67
79457_wsnp_Ex_rep_c68712_67571580	1B	462183400	INC	GLM	17.96
80074_wsnp_Ku_c13069_20938717	1D	77498631	SEV	pKWmEB	1.67

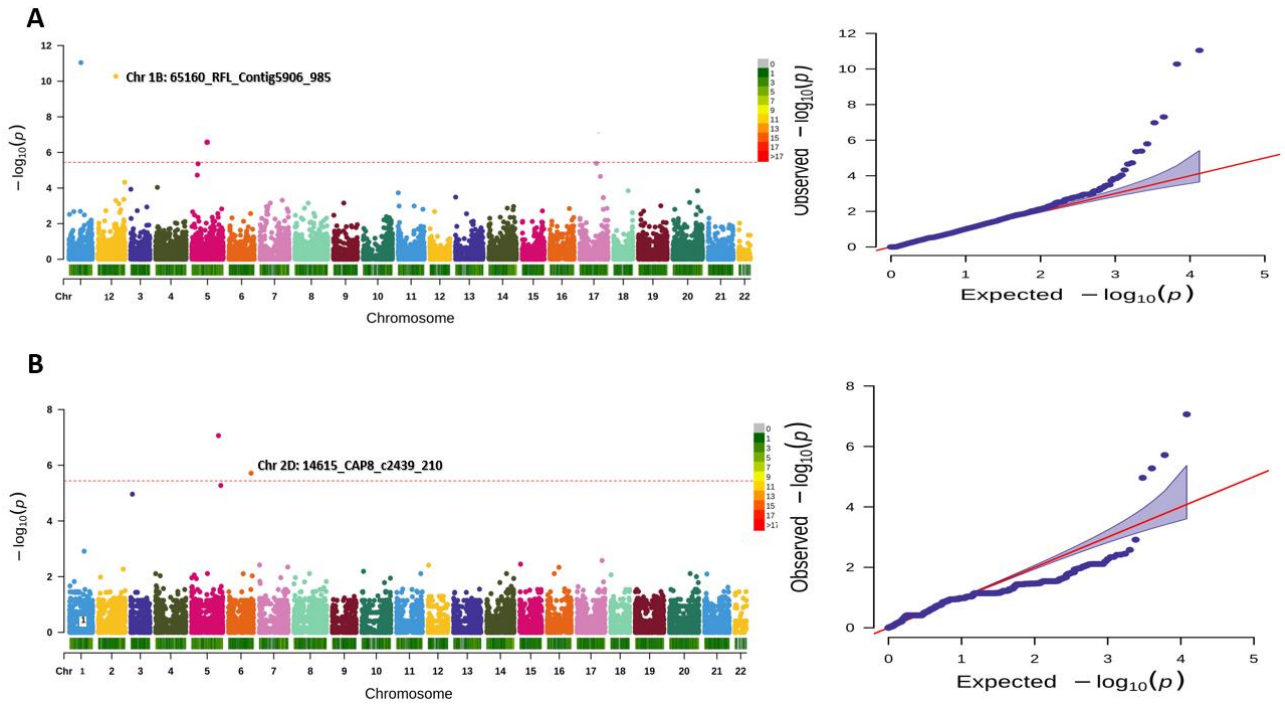
QTN	Chromosome	Position	Trait	Models	R^2 (%)
80285_wsnp_Ku_c25372_35336531	7D	14163144	VRI	FarmCPU	1.26
80328_wsnp_Ku_c2797_5284087	1B	687093328	SEV	FarmCPU	2.57
80485_wsnp_Ku_c4045_7380115	1A	553440047	VRI	ISIS EM-BLASSO	6.03
80742_wsnp_Ku_c9971_16598986	1B	463291533	INC	GLM	14.95
80996_wsnp_Ra_c2027_3945713	1B	462883485	INC	GLM	14.14
80997_wsnp_Ra_c2027_3945764	1B	462883434	INC	FarmCPU	19.82
81057_wsnp_Ra_c2730_5190076	6B	131945815	SEV	GLM	3.17
81112_wsnp_Ra_c34203_42948357	1A	305745550	DON	GLM	1.60
81493_wsnp_RFL_Contig3344_3442711	3A	37135958	SEV	FASTmrMLM	8.75
8296_BS00041947_51	1B	440499591	INC	pLARmEB	15.79
8340_BS00043474_51	5A	445375706	VRI	GLM	16.00
8649_BS00054690_51	5A	70950202	VRI	GLM	1.01
931_BobWhite_c1656_186	2A	8355223	DON	pKWmEB	1.29
9653_BS00065865_51	2A	721702719	DON	mrMLM	3.48
9955_BS00067096_51	5A	36408984	SEV	GLM	18.81
Winter Panel					
10344_BS00068556_51	3D	164810641	SEV	GLM	0.9478
10753_BS00074662_51	1B	57365453	SEV	GLM	0.6269
10901_BS00076782_51	5B	15178680	VRI	GLM	0.2696
12308_BS00107222_51	5B	46332832	SEV	GLM	0.529
12915_CAP11_c4929_135	7A	271525529	VRI	GLM	0.2696
14615_CAP8_c2439_210	2D	590124023	VRI	FarmCPU	38.46
15600_D_contig11034_559	1D	122256021	INC	ISIS EM-BLASSO	5.15
1704_BobWhite_c23392_496	3A	13799472	SEV	GLM	1.3986
20645_Ex_c4003_1351	3B	148256131	SEV	GLM	7.5799
20659_Ex_c40727_927	6A	235845903	VRI	GLM	0.2696
20710_Ex_c4348_113	2A	157642581	VRI	GLM	0.2696
21421_Ex_c9042_1461	1A	132114818	SEV	GLM	7.3144
21963_Excalibur_c11795_902	5A	320927775	VRI	GLM	0.2696
22019_Excalibur_c12004_215	2B	140474375	DON	GLM	1.29
22118_Excalibur_c12576_541	2A	553657013	SEV	GLM	1.5533
22120_Excalibur_c12586_152	6D	72290247	VRI	GLM	0.2696
22293_Excalibur_c13603_1389	1A	594835828	VRI	GLM	0.2696
2233_BobWhite_c28819_517	2A	578845725	SEV	GLM	1.507
23273_Excalibur_c19240_1885	3B	195671718	VRI	GLM	7.9972
23932_Excalibur_c23214_583	1A	408212350	SEV	GLM	1.5533
2399_BobWhite_c30760_262	3D	287930031	VRI	GLM	0.2696
24589_Excalibur_c27531_122	7B	36457447	VRI	GLM	0.2696
24840_Excalibur_c29425_457	3A	715273236	VRI	GLM	0.2696
25048_Excalibur_c30992_257	6A	88466460	VRI	GLM	0.2696
25289_Excalibur_c33031_1755	5D	72075420	DON	GLM	0.17
25440_Excalibur_c34269_460	6A	518485246	VRI	GLM	0.2696
25795_Excalibur_c37239_916	2B	147189293	DON	GLM	0.1
25813_Excalibur_c37358_741	6A	371618518	VRI	GLM	0.2696
25891_Excalibur_c37897_473	7A	71510110	SEV	GLM	0.9478

QTN	Chromosome	Position	Trait	Models	R^2 (%)
26084_Excalibur_c39833_163	5A	479299750	VRI	GLM	0.2696
26470_Excalibur_c43604_205	3D	603680165	VRI	GLM	0.2696
26899_Excalibur_c47756_323	6B	725266977	VRI	GLM	0.2696
26988_Excalibur_c488_4121	2D	580104364	SEV	GLM	0.583
27303_Excalibur_c52196_235	6A	598278249	SEV	GLM	7.5799
27452_Excalibur_c54128_125	6A	26004818	VRI	GLM	0.2696
27682_Excalibur_c56854_181	7B	34503448	VRI	GLM	0.2696
27929_Excalibur_c60064_496	2A	550765011	VRI	GLM	0.2696
28347_Excalibur_c65152_572	1B	639809358	SEV	GLM	1.5533
28746_Excalibur_c75305_436	4B	482953291	SEV	GLM	1.5533
2905_BobWhite_c37402_506	2A	755687907	VRI	GLM	0.2696
29091_Excalibur_c8399_516	6A	41263040	VRI	GLM	0.2696
29551_Excalibur_c96447_465	5B	687731542	SEV	GLM	1.5533
30022_Excalibur_rep_c104322_115	5A	659174173	VRI	GLM	0.2696
30108_Excalibur_rep_c105284_110	2A	54169820	SEV	GLM	2.8117
30732_Excalibur_rep_c66349_1292	7A	576735399	DON	GLM	1.1
3168_BobWhite_c41378_234	7A	704309661	SEV	GLM	8.3011
32373_GENE-1304_388	2A	774564580	VRI	GLM	0.2696
32618_GENE-1733_358	3B	598510365	VRI	GLM	0.2696
32785_GENE-2058_43	3D	507743875	VRI	GLM	0.2696
33213_GENE-3078_101	4D	346189159	VRI	GLM	0.4435
33396_GENE-3447_333	5D	393839692	SEV	GLM	1.5533
33946_GENE-4414_102	3B	783595303	SEV	GLM	7.8453
35571_IAAV8980	2B	443867659	DON	FarmCPU	0.85
3586_BobWhite_c47340_313	5D	68859544	DON	GLM	0.02
36458_Jagger_c1781_99	3A	208155305	VRI	GLM	0.2696
36738_Jagger_c736_109	4A	201413600	VRI	GLM	0.2696
37327_JD_c30831_466	5D	251967443	SEV	GLM	1.5533
38451_Ku_c14376_1164	3A	512336082	VRI	GLM	0.2696
39158_Ku_c3169_1250	6A	618329085	DON	FarmCPU	6.43
39346_Ku_c3907_2063	2A	209712279	VRI	GLM	0.2696
40125_Ku_c892_676	1B	555353159	SEV	GLM	1.5533
40468_Kukri_c10915_383	2B	699316217	VRI	FarmCPU	18.47
40510_Kukri_c110735_106	6D	110485159	SEV	GLM	1.5533
4087_BobWhite_c6094_447	5B	514737766	VRI	GLM	0.2696
42062_Kukri_c19017_1039	1D	487714262	SEV	GLM	7.5799
42295_Kukri_c20269_422	3A	167512111	VRI	GLM	0.2696
42481_Kukri_c21384_1333	5D	568635787	DON	GLM	0.13
4251_BobWhite_c6885_1480	2D	394080697	SEV	GLM	1.5533
42810_Kukri_c23451_737	5D	27327078	VRI	GLM	0.2696
42918_Kukri_c2408_857	1A	1340611	SEV	GLM	0.9478
4342_BobWhite_c744_614	2B	587915389	SEV	GLM	7.3144
43654_Kukri_c29138_749	7A	513276292	VRI	GLM	0.2696
43875_Kukri_c30641_403	7A	292086334	SEV	GLM	31.1962
43993_Kukri_c31596_412	3A	145360059	INC	pKWmEB	22.6

QTN	Chromosome	Position	Trait	Models	R^2 (%)
44197_Kukri_c33403_781	3A	608539373	VRI	GLM	0.2696
44858_Kukri_c38926_370	1D	589499802	SEV	mrMLM	6.64
44878_Kukri_c39041_574	7A	504075930	VRI	GLM	0.2696
45001_Kukri_c40134_451	4A	38694592	VRI	GLM	0.2696
45195_Kukri_c4213_1202	2B	115063180	DON	FASTmrEMMA	1.61
45886_Kukri_c48570_149	3A	158837969	SEV	GLM	1.5533
4595_BobWhite_c930_1135	2B	20383875	DON	GLM	0.19
47009_Kukri_c627_695	4B	310943991	SEV	GLM	1.5533
47215_Kukri_c65249_58	2B	108844773	SEV	GLM	29.0121
47539_Kukri_c7244_303	2A	202098196	VRI	GLM	0.2696
47644_Kukri_c7569_1371	7A	275419561	SEV	GLM	1.5533
48175_Kukri_c8929_2161	2A	531255241	VRI	GLM	0.2696
48685_Kukri_rep_c101778_438	1D	681605997	INC	pKWmEB	0
48969_Kukri_rep_c104267_68	2A	778842896	VRI	GLM	0.2696
49386_Kukri_rep_c109145_695	1A	573958234	VRI	GLM	0.2696
50449_Kukri_rep_c86638_306	3B	257382735	VRI	GLM	0.2696
50643_Kukri_s112441_279	3A	205619000	VRI	GLM	0.2696
51134_Ra_c1485_2580	6B	421338113	VRI	GLM	0.2696
51268_Ra_c17341_2119	7A	87166428	VRI	GLM	0.2696
51667_Ra_c27593_460	1A	48796937	SEV	GLM	9.7854
52072_Ra_c42858_503	3A	653428186	VRI	GLM	2.5216
52729_Ra_c8311_1521	5A	353947508	VRI	GLM	0.2696
52957_RAC875_c10225_667	5D	416250795	VRI	GLM	0.2696
53062_RAC875_c104514_534	2B	66092784	DON	ISIS EM-BLASSO	37.7
54091_RAC875_c15402_286	3A	222954082	VRI	GLM	0.2696
55515_RAC875_c23473_165	2B	59105039	VRI	GLM	0.2696
55566_RAC875_c23799_2056	5A	105916657	VRI	GLM	0.2696
55860_RAC875_c25864_770	7A	659499214	VRI	GLM	0.2696
56691_RAC875_c32059_1790	7A	111365587	VRI	GLM	0.2696
57324_RAC875_c3770_970	7A	695383398	SEV	GLM	1.5533
57515_RAC875_c39432_1007	4A	323310265	VRI	GLM	0.2696
58549_RAC875_c49349_1074	7B	44003133	SEV	GLM	0.529
58728_RAC875_c51216_804	4B	645758432	VRI	GLM	0.2696
59116_RAC875_c55445_92	6B	721147245	SEV	GLM	29.0121
59332_RAC875_c57998_165	2D	191033496	VRI	GLM	0.2696
60102_RAC875_c6747_104	7B	635263652	SEV	GLM	1.5533
60305_RAC875_c7166_279	6A	149470653	SEV	GLM	1.5533
60475_RAC875_c77534_451	5B	671028119	SEV	GLM	7.5799
60851_RAC875_c8647_200	2D	94133205	DON	GLM	2.26
61500_RAC875_rep_c105780_64	2B	240717510	VRI	GLM	0.2696
61578_RAC875_rep_c106337_522	5D	393494214	SEV	GLM	29.0121
61616_RAC875_rep_c106596_127	2B	29764155	DON	GLM	7.15
61878_RAC875_rep_c109306_735	1B	148842486	VRI	GLM	0.2696
61888_RAC875_rep_c109516_822	7A	88401545	VRI	GLM	0.2696
62843_RAC875_rep_c72158_264	2B	779063796	SEV	GLM	1.5533

QTN	Chromosome	Position	Trait	Models	R^2 (%)
63667_RFL_Contig1511_612	7A	134396890	VRI	GLM	0.2696
64160_RFL_Contig2980_972	3B	166791730	SEV	GLM	7.5799
64971_RFL_Contig5302_405	7B	738310288	SEV	GLM	1.5533
65160_RFL_Contig5906_985	1B	16958960	DON	ISIS EM-BLASSO	42.09
66562_Tdurum_contig10506_261	4B	314358511	SEV	GLM	1.5533
67051_Tdurum_contig11382_669	3A	549601573	VRI	GLM	0.2696
68357_Tdurum_contig15150_2456	3B	683245415	VRI	GLM	0.2696
68431_Tdurum_contig15610_321	1B	404565122	DON	pLARmEB	2.58
68456_Tdurum_contig15718_314	3B	659430855	VRI	GLM	0.2696
68548_Tdurum_contig16482_124	7B	597731414	DON	GLM	2.35
68550_Tdurum_contig1653_190	2B	107712458	SEV	GLM	2.3541
68558_Tdurum_contig16612_337	6A	500011565	VRI	GLM	0.2696
68771_Tdurum_contig19102_84	1A	281617671	SEV	GLM	2.1759
68934_Tdurum_contig21518_326	4A	499103420	VRI	GLM	0.2696
69162_Tdurum_contig25674_294	5A	563619393	VRI	GLM	0.2696
69384_Tdurum_contig27942_125	2A	81899936	VRI	GLM	0.2696
69960_Tdurum_contig30108_218	3A	435143001	VRI	GLM	0.2696
69963_Tdurum_contig30121_240	1A	91282013	VRI	GLM	0.2696
70064_Tdurum_contig30610_268	2B	413833782	SEV	GLM	1.5533
70065_Tdurum_contig30621_328	7A	71357685	VRI	GLM	13.3545
	7A	71357685	SEV	GLM	10.0862
	7A	71357685	INC	FASTmrEMMA	17.12
70129_Tdurum_contig30916_316	3B	816074580	VRI	GLM	0.2696
7136_BS00022524_51	1D	10383887	VRI	pLARmEB	13.95
71731_Tdurum_contig46877_76	7B	49447278	SEV	GLM	0.529
71919_Tdurum_contig48766_257	5A	445357311	SEV	GLM	9.5957
72090_Tdurum_contig50576_1223	7B	41600671	DON	GLM	0.81
72499_Tdurum_contig56281_261	1A	613121920	INC	ISIS EM-BLASSO	19.97
72518_Tdurum_contig56538_140	1A	549464722	SEV	GLM	9.3438
72586_Tdurum_contig57566_1653	1A	535410856	DON	ISIS EM-BLASSO	5.86
72868_Tdurum_contig62141_496	6A	36798202	SEV	GLM	0.3744
73944_Tdurum_contig95708_332	4B	275668961	VRI	GLM	0.2696
78230_wsnp_Ex_c4769_8510104	3B	496418477	SEV	GLM	2.1304
78270_wsnp_Ex_c4921_8764106	5A	475040948	SEV	GLM	0.531
	5A	475040948	SEV	pLARmEB	3.91
79013_wsnp_Ex_rep_c101269_86664147	3A	712607958	VRI	pLARmEB	14.18
80334_wsnp_Ku_c28245_38183393	5A	456607896	SEV	GLM	1.0554
80468_wsnp_Ku_c3956_7237642	3A	513760600	SEV	GLM	0.9478
8117_BS00037784_51	6B	672525118	SEV	GLM	7.5799
8353_BS00043866_51	7A	543527851	SEV	GLM	7.5799
9877_BS00066756_51	2A	36477315	SEV	GLM	1.5886
	2A	36477315	SEV	FASTmrEMMA	1.55

INC, incidence; SEV, SEV; VRI, visual rating index; DON, deoxynivalenol content



Appendix 4 Manhattan and quantile-quantile (QQ) plots showing QTNs that are significantly associated obtained by the model ISIS-EM_BLASSO and FarmCPU using the stringent Bonferroni criterion for both datasets; **(A)** DON content; **(B)** VRI. The horizontal dotted lines indicate the threshold $P = 0.05/n$, where 'n' is the number of markers. The *y*-axis represents the *p*-value of the marker–trait association on a −log10 scale. The horizontal line represents the threshold for declaring a marker as significant. Colors in Manhattan plot indicate the 21 chromosomes of wheat in order and the chromosome 22 is defined as unknown SNPs.

Appendix 5 Numbers of quantitative trait nucleotides (QTNs) that were pleiotropic on two or three of the four traits.

QTN	No. of Trait	No of Models
Combined Panel		
8329_BS00043071_51	2	SEV, DON
81112_WSNP_RA_C34203_42948357	3	SEV, VRI, DON
15288_D_CONTIG03979_394	2	INC, VRI
47776_KUKRI_C7874_1096	2	INC, VRI
52304_RA_C58279_702	2	INC, VRI
46344_KUKRI_C53506_1141	3	INC, SEV, VRI
53059_RAC875_C104483_216	3	INC, SEV, VRI
65469_TA001874-1495	3	INC, SEV, VRI
12504_BS00110278_51	2	SEV, VRI
16918_D_CONTIG58377_170	2	SEV, VRI
13266_CAP12_C1981_78	2	INC, VRI
65349_TA001138-1003	2	SEV, VRI
65683_TA003419-0641	2	SEV, VRI
12504_BS00110278_51		
Spring Panel		
35578_IAAV902	2	INC, SEV
13266_CAP12_C1981_78	2	INC, VRI
80997_WSNP_RA_C2027_3945764	2	INC, VRI
46344_KUKRI_C53506_1141	3	INC, , SEV, VRI
56662_RAC875_C3187_873	2	SEV, VRI
22159_EXCALIBUR_C12835_2524	3	INC, SEV, VRI
12504_BS00110278_51	3	SEV, VRI, DON
81112_WSNP_RA_C34203_42948357	3	INC, SEV, VRI
81493_WSNP_RFL_CONTIG3344_3442711	2	VRI, SEV
Winter Panel		
70065_Tdurum_contig30621_328	**3**	INC, VRI, SEV

INC, incidence; SEV, SEV; VRI, visual rating index; DON, deoxynivalenol content

Appendix 6 Numbers of QTNs identified by each statistical model for incidence (INC), SEV (SEV), visual rating index (VRI) and deoxynivalenol content (DON) in the combined, spring and winter panels

Model	**INC**			**SEV**			**VRI**			**DON**			**Total**		
Single-locus	Combined	Spring	Winter	Combined	Spring	Winter	Combined	Spring	Winter	Combined	Spring	Winter	Combined	Spring	Winter
GLM	17	20	0	3	11	54	4	7	73	27	23	11	51	61	138
MLM	0	0	0	0	0	0	43	0	0	0	0	0	43	0	0
Multi-locus															
FASTmrEMMA	2	1	1	2	0	1	0	0	0	4	0	1	8	1	3
FASTmrMLM	2	0	0	0	2	0	6	0	0	0	0	0	8	2	0
ISIS EM-BLASSO	2	3	2	4	0	0	7	3	0	6	3	3	19	9	5
mrMLM	3	5	0	1	0	1	1	0	0	6	3	0	11	8	1
pKWmEB	4	6	2	2	5	0	5	0	0	5	1	0	16	12	2
pLARmEB	0	4	0	4	0	1	5	1	2	1	0	1	10	5	4
FarmCPU	2	3	0	2	3	0	1	1	2	3	2	2	8	9	4
Total	32	42	5	18	21	57	72	12	77	52	32	18	174	107	157

INC, incidence; SEV, SEV; VRI, visual rating index; DON, deoxynivalenol content

Appendix 7 List of quantitative trait nucleotides (QTNs) and co-located candidate genes associated with FHB resistance traits.

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Spring	INC	10435_BS00070051_51	1B	27126042	*TraesCS1B01G045400*	RLK	43097	0.37
Spring	INC	10435_BS00070051_51	1B	27126042	*TraesCS1B01G045600*	RLK	521518	0.37
Spring	INC	10435_BS00070051_51	1B	27126042	*TraesCS1B01G045800*	CNL	531383	0.37
Spring	INC	10435_BS00070051_51	1B	27126042	*TraesCS1B01G046000*	CNL	655533	0.37
Winter	VRI	10901_BS00076782_51	5B	15178680	*TraesCS5B01G015200*	RLK	439657	0.2
Spring	INC	12462_BS00110052_51	1D	342788950	*TraesCS1D01G247800*	TM-CC	94401	17.44
Spring	DON	12651_CAP11_c1022_117	3A	714842763	*TraesCS3A01G485600*	RLK	424981	3.86
Spring	DON	12651_CAP11_c1022_117	3A	714842763	*TraesCS3A01G486400*	RLK	479805	3.86
Spring	DON	12652_CAP11_c1022_66	3A	714842712	*TraesCS3A01G485600*	RLK	425032	4.35
Spring	DON	12652_CAP11_c1022_66	3A	714842712	*TraesCS3A01G486400*	RLK	479856	4.35
Spring	INC	13266_CAP12_c1981_78	1B	462097527	*TraesCS1B01G259000*	TM-CC	78817	18.43
Spring	DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G346300*	RLK	330529	10.39
Spring	DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345700*	RLK	477893	10.39
Spring	DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345500*	RLK	494538	10.39
Spring	DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345400*	RLK	505992	10.39
Spring	DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345100*	RLK	611658	10.39
Spring	DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G345000*	RLK	633184	10.39
Spring	DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G344600*	RLK	771934	10.39
Spring	DON	14141_CAP7_c4879_249	1A	534934626	*TraesCS1A01G344400*	RLK	840298	10.39
Winter	VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G486800*	RLK	169583	38.46
Winter	VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G486400*	RLK	283319	38.46
Winter	VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G486300*	RLK	335284	38.46
Winter	VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G486200*	RLK	343954	38.46
Winter	VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G485700*	RLK	765187	38.46
Winter	VRI	14615_CAP8_c2439_210	2D	590124023	*TraesCS2D01G485400*	RLK	967045	38.46
Combined	VRI	16918_D_contig58377_170	5D	372287609	*TraesCS5D01G263300*	RLK	365140	0.13
Combined	VRI	16918_D_contig58377_170	5D	372287609	*TraesCS5D01G265800*	RLK	724681	0.13
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G116100*	RLK	233351	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G116000*	RLK	251068	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G115800*	RLP	266815	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G115700*	RLK	331044	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G116500*	RLP	480553	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G116600*	RLP	486867	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G116700*	RLP	538152	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G115300*	RLK	542647	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G116900*	RLP	575637	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G117200*	RLP	641341	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G117400*	RLP	676417	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G115000*	RLK	708701	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G117600*	RLP	721218	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G114600*	RLK	794182	0.05
Combined	INC	17105_D_contig71738_238	6D	103996137	*TraesCS6D01G114400*	TM-CC	803343	0.05
Combined	INC	17937_D_GB5Y7FA01A570E_178	2D	13089524	*TraesCS2D01G033400*	RLK	36019	0.05
Combined	INC	17937_D_GB5Y7FA01A570E_178	2D	13089524	*TraesCS2D01G032600*	RLK	92355	0.05
Combined	INC	17937_D_GB5Y7FA01A570E_178	2D	13089524	*TraesCS2D01G032200*	TM-CC	139857	0.05
Combined	INC	17937_D_GB5Y7FA01A570E_178	2D	13089524	*TraesCS2D01G031600*	RLK	233379	0.05
Combined	INC	17937_D_GB5Y7FA01A570E_178	2D	13089524	*TraesCS2D01G034200*	RLK	336958	0.05

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Combined	INC	18201_D_GBB4FNX01CNX9D_254	6D	42442831	*TraesCS6D01G066400*	CN	0	2.43
Combined	INC	18219_D_GBB4FNX01DLV3V_102	6D	476758547	*TraesCS6D01G365100*	NL	717617	1.49
Combined	INC	18510_D_GBQ4KXB01C9DNU_246	1D	114815650	*TraesCS1D01G116300*	RLK	362252	0.9
Combined	INC	18510_D_GBQ4KXB01C9DNU_246	1D	114815650	*TraesCS1D01G115400*	RLK	631334	0.9
Winter	DON	22019_Excalibur_c12004_215	2B	140474375	*TraesCS2B01G158800*	RLK	93167	1.29
Winter	DON	22019_Excalibur_c12004_215	2B	140474375	*TraesCS2B01G158700*	RLK	100768	1.29
Winter	VRI	22128_Excalibur_c12644_58	5A	3332382	*TraesCS5A01G003500*	TM-CC	3422	0.34
Winter	VRI	22128_Excalibur_c12644_58	5A	3332382	*TraesCS5A01G005300*	RLK	942784	0.34
Winter	VRI	22293_Excalibur_c13603_1389	1A	594835828	*TraesCS1A01G440300*	RLP	852985	0.2
Winter	VRI	2399_BobWhite_c30760_262	3D	287930031	*TraesCS3D01G213300*	TM-CC	755922	0.2
Winter	VRI	24589_Excalibur_c27531_122	7B	36457447	*TraesCS7B01G036100*	RLK	873477	0.2
Winter	VRI	24840_Excalibur_c29425_457	3A	715273236	*TraesCS3A01G485600*	RLK	3151	0.2
Winter	VRI	24840_Excalibur_c29425_457	3A	715273236	*TraesCS3A01G486400*	RLK	49332	0.2
Winter	DON	25289_Excalibur_c33031_1755	5D	72075420	*TraesCS5D01G073900*	RLK	978791	0.17
Winter	VRI	25440_Excalibur_c34269_460	6A	518485246	*TraesCS6A01G285100*	TM-CC	161221	0.2
Spring	DON	25627_Excalibur_c35713_106	2D	60807566	*TraesCS2D01G106800*	RLK	700721	3.56
Winter	VRI	25891_Excalibur_c37897_473	7A	71510110	*TraesCS7A01G111200*	CN	104615	0.95
Winter	VRI	25891_Excalibur_c37897_473	7A	71510110	*TraesCS7A01G111100*	CN	145179	0.95
Winter	VRI	25891_Excalibur_c37897_473	7A	71510110	*TraesCS7A01G111000*	CN	157908	0.95
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G463900*	NL	29237	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G463700*	NL	41123	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G464300*	CN	72776	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G463600*	CNL	73643	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G464400*	NL	82794	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G464500*	CN	92019	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G463500*	NL	110022	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G462600*	RLK	248602	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G465000*	CN	314582	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G465100*	CN	327829	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G465800*	CN	467445	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G462000*	NL	655423	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G461900*	CN	722154	0.2
Winter	VRI	26899_Excalibur_c47756_323	6B	725266977	*TraesCS6B01G461700*	CN	827393	0.2
Winter	VRI	27452_Excalibur_c54128_125	6A	26004818	*TraesCSU01G184100*	RLP	767808	0.2
Winter	VRI	27452_Excalibur_c54128_125	6A	26004818	*TraesCS6A01G044300*	RLP	925878	0.2
Winter	VRI	27452_Excalibur_c54128_125	6A	26004818	*TraesCS6A01G044000*	RLP	974936	0.2
Winter	VRI	27452_Excalibur_c54128_125	6A	26004818	*TraesCS6A01G043900*	RLK	996394	0.2
Winter	VRI	27929_Excalibur_c60064_496	2A	550765011	*TraesCS2A01G319800*	TM-CC	472275	0.2
Combined	INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G457600*	RLK	216686	6.21
Combined	INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G457400*	RLK	229288	6.21
Combined	INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G457300*	RLK	252722	6.21
Combined	INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G456800*	RLK	825810	6.21
Combined	INC	27951_Excalibur_c60262_359	2D	568340040	*TraesCS2D01G456700*	RLK	863007	6.21
Winter	VRI	28347_Excalibur_c65152_572	1B	639809358	*TraesCS1B01G403300*	CNL	544010	1.55
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G044200*	NL	247408	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G044300*	NL	272075	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G044400*	NL	279409	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G044500*	RLK	292703	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G044600*	RLK	299655	1.34

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G044700*	CN	343913	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G044800*	CN	371680	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G044900*	NL	384951	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G045100*	NL	437530	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G045200*	NL	446193	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G045300*	CN	462936	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G045600*	NL	493330	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G045700*	NL	507971	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G045800*	NL	514787	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G046100*	CN	744186	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G046300*	CN	758289	1.34
Spring	SEV	28718_Excalibur_c74403_580	7A	21158431	*TraesCS7A01G041700*	CN	840332	1.34
Winter	VRI	2905_BobWhite_c37402_506	2A	755687907	*TraesCS2A01G537900*	RLK	500298	0.2
Winter	VRI	2905_BobWhite_c37402_506	2A	755687907	*TraesCS2A01G537200*	RLK	692997	0.2
Winter	VRI	29089_Excalibur_c8399_344	6A	41262867	*TraesCS6A01G072100*	RLK	977246	0.34
Winter	VRI	29089_Excalibur_c8399_344	6A	41262867	*TraesCS6A01G072300*	RLK	992580	0.34
Winter	VRI	29091_Excalibur_c8399_516	6A	41263040	*TraesCS6A01G072100*	RLK	977073	0.2
Winter	VRI	29091_Excalibur_c8399_516	6A	41263040	*TraesCS6A01G072300*	RLK	992407	0.2
Winter	VRI	29551_Excalibur_c96447_465	5B	687731542	*TraesCS5B01G526200*	RLK	843649	1.55
Winter	VRI	29551_Excalibur_c96447_465	5B	687731542	*TraesCS5B01G528200*	RLK	854755	1.55
Winter	VRI	29551_Excalibur_c96447_465	5B	687731542	*TraesCS5B01G528300*	RLK	859157	1.55
Winter	VRI	29551_Excalibur_c96447_465	5B	687731542	*TraesCS5B01G526000*	RLK	887073	1.55
Winter	VRI	32373_GENE-1304_388	2A	774564580	*TraesCS2A01G580900*	RLK	34432	0.2
Winter	VRI	32373_GENE-1304_388	2A	774564580	*TraesCS2A01G580000*	RLK	789931	0.2
Winter	VRI	32373_GENE-1304_388	2A	774564580	*TraesCS2A01G579900*	CN	800756	0.2
Winter	VRI	32383_GENE-1322_33	2D	79161450	*TraesCS2D01G131600*	RLK	8905	0.57
Winter	VRI	32785_GENE-2058_43	3D	507743875	*TraesCS3D01G391600*	RLK	394691	0.2
Winter	VRI	32851_GENE-2239_535	5A	592439004	*TraesCS5A01G396200*	CNL	519764	0.57
Spring	INC	33266_GENE-3158_262	5A	644395007	*TraesCS5A01G463200*	RLK	697218	2.96
Spring	INC	33266_GENE-3158_262	5A	644395007	*TraesCS5A01G463300*	RLK	704061	2.96
Combined	INC	34012_GENE-4564_387	7A	699200157	*TraesCS7A01G507700*	RLK	94366	7.08
Combined	INC	34012_GENE-4564_387	7A	699200157	*TraesCS7A01G507500*	RLK	158108	7.08
Combined	INC	34012_GENE-4564_387	7A	699200157	*TraesCS7A01G507400*	RLK	183080	7.08
Combined	INC	34012_GENE-4564_387	7A	699200157	*TraesCS7A01G507100*	RLK	314963	7.08
Combined	INC	34665_IAAV3115	4A	100654839	*TraesCS4A01G092500*	RLK	753960	0.06
Spring	INC	35131_IAAV6011	1B	682639244	*TraesCS1B01G460100*	CN	200412	10.01
Combined	SEV	36611_Jagger_c4901_95	3B	53703225	*TraesCS3B01G072100*	RLP	28535	4.46
Combined	SEV	36611_Jagger_c4901_95	3B	53703225	*TraesCS3B01G072900*	RLK	377619	4.46
Combined	SEV	36611_Jagger_c4901_95	3B	53703225	*TraesCS3B01G073100*	RLK	483119	4.46
Winter	VRI	3722_BobWhite_c5092_422	6A	14459261	*TraesCS6A01G027400*	RLK	742287	0.09
Winter	VRI	3722_BobWhite_c5092_422	6A	14459261	*TraesCS6A01G027600*	CNL	920424	0.09
Winter	VRI	3722_BobWhite_c5092_422	6A	14459261	*TraesCS6A01G024200*	RLP	981427	0.09
Spring	INC	37431_JD_c4128_277	3B	73391914	*TraesCS3B01G093800*	RLK	551594	9.07
Winter	DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G168400*	NL	2436	6.43
Winter	DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G203600*	NL	3864	6.43
Winter	DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G213400*	CN	18927	6.43
Winter	DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G194700*	CN	85659	6.43
Winter	DON	39158_Ku_c3169_1250	6A	618329085	*TraesCSU01G219700*	CN	95793	6.43
Winter	DON	39158_Ku_c3169_1250	6A	618329085	*TraesCS6A01G414300*	NL	414235	6.43

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Winter	DON	39158_Ku_c3169_1250	6A	618329085	*TraesCS6A01G414400*	CNL	424027	6.43
Winter	DON	39158_Ku_c3169_1250	6A	618329085	*TraesCS6A01G415000*	CN	595973	6.43
Winter	VRI	39160_Ku_c31764_136	4D	48541991	*TraesCS4D01G073300*	RLK	103064	25.64
Winter	VRI	39160_Ku_c31764_136	4D	48541991	*TraesCS4D01G074100*	RLK	590673	25.64
Winter	VRI	39346_Ku_c3907_2063	2A	209712279	*TraesCS2A01G217600*	RLK	312498	0.2
Winter	VRI	40125_Ku_c892_676	1B	555353159	*TraesCS1B01G323100*	RLK	417299	1.55
Winter	VRI	40510_Kukri_c110735_106	6D	110485159	*TraesCS6D01G124200*	RLK	0	1.55
Winter	VRI	40510_Kukri_c110735_106	6D	110485159	*TraesCS6D01G124600*	RLP	61250	1.55
Winter	VRI	40510_Kukri_c110735_106	6D	110485159	*TraesCS6D01G124700*	RLP	152627	1.55
Winter	VRI	40510_Kukri_c110735_106	6D	110485159	*TraesCS6D01G124800*	RLP	157695	1.55
Winter	VRI	40510_Kukri_c110735_106	6D	110485159	*TraesCS6D01G124900*	RLP	164846	1.55
Combined	VRI	40694_Kukri_c119_295	3B	781032674	*TraesCS3B01G522800*	CN	0	0.1
Combined	VRI	40694_Kukri_c119_295	3B	781032674	*TraesCS3B01G522700*	CN	7603	0.1
Combined	VRI	40694_Kukri_c119_295	3B	781032674	*TraesCS3B01G522600*	CN	41956	0.1
Combined	VRI	40694_Kukri_c119_295	3B	781032674	*TraesCS3B01G522500*	CN	75565	0.1
Combined	VRI	40694_Kukri_c119_295	3B	781032674	*TraesCS3B01G523300*	CN	171455	0.1
Combined	VRI	40694_Kukri_c119_295	3B	781032674	*TraesCS3B01G523400*	CN	184050	0.1
Combined	VRI	40694_Kukri_c119_295	3B	781032674	*TraesCS3B01G522200*	NL	345437	0.1
Winter	VRI	4087_BobWhite_c6094_447	5B	514737766	*TraesCS5B01G326400*	CN	993693	0.2
Spring	SEV	41483_Kukri_c15912_2330	3A	680860234	*TraesCS3A01G437800*	CN	664737	3.77
Winter	VRI	42062_Kukri_c19017_1039	1D	487714262	*TraesCS1D01G440300*	RLP	916886	7.58
Winter	DON	42481_Kukri_c21384_1333	5D	568635787	*TraesCS5D01G560500*	CN	29890	0.13
Winter	DON	42481_Kukri_c21384_1333	5D	568635787	*TraesCS5D01G561200*	CN	217276	0.13
Winter	DON	42481_Kukri_c21384_1333	5D	568635787	*TraesCS5D01G561300*	CN	244424	0.13
Combined	DON	42528_Kukri_c21628_1215	7B	613149662	*TraesCS7B01G352400*	RLP	180033	0.39
Winter	VRI	42810_Kukri_c23451_737	5D	27327078	*TraesCS5D01G029100*	RLP	419730	0.2
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G487300*	NL	0	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G487000*	RLK	46055	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488000*	RLK	93458	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488200*	NL	174722	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G486500*	CNL	198340	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488400*	NL	254501	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488700*	CN	317324	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488800*	CNL	343532	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G488900*	RLK	372156	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G489800*	CN	626723	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G490200*	NL	683358	16.76
Combined	DON	42938_Kukri_c24194_2659	4A	748723146	*TraesCS4A01G490500*	NL	775036	16.76
Spring	VRI	43087_Kukri_c25145_332	7D	632780685	*TraesCS7D01G538200*	CNL	596677	1.31
Spring	VRI	43087_Kukri_c25145_332	7D	632780685	*TraesCSU01G105400*	RLK	827109	1.31
Spring	INC	43566_Kukri_c2842_248	6D	331186031	*TraesCS6D01G219000*	RLK	639208	18.34
Spring	SEV	44603_Kukri_c36747_195	5D	340132967	*TraesCS5D01G230000*	RLK	35666	8.47
Spring	SEV	44603_Kukri_c36747_195	5D	340132967	*TraesCS5D01G230300*	TM-CC	154597	8.47
Winter	VRI	44608_Kukri_c3676_483	7D	633888185	*TraesCS7D01G538200*	CNL	507089	0.59
Winter	VRI	44608_Kukri_c3676_483	7D	633888185	*TraesCS7D01G540500*	CNL	668252	0.59
Winter	VRI	44608_Kukri_c3676_483	7D	633888185	*TraesCS7D01G540600*	CN	687061	0.59
Winter	VRI	44878_Kukri_c39041_574	7A	504075930	*TraesCS7A01G340700*	RLK	224007	0.2
Combined	INC	45329_Kukri_c43306_282	7D	4394602	*TraesCS7D01G007600*	NL	289893	25.2
Combined	INC	45329_Kukri_c43306_282	7D	4394602	*TraesCS7D01G007000*	NL	426217	25.2

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Combined	INC	45329_Kukri_c43306_282	7D	4394602	*TraesCS7D01G006800*	NL	467351	25.2
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G045800*	NL	43225	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G045700*	NL	49763	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G045600*	NL	65357	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G045300*	CN	93316	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G045200*	NL	111591	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G045100*	NL	120257	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G044900*	NL	169546	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G044800*	CN	181775	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G046100*	CN	183648	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G046300*	CN	197751	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G044700*	CN	214978	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G044600*	RLK	258358	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G044500*	RLK	264417	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G044400*	NL	276658	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G044300*	NL	285508	0.09
Winter	VRI	45392_Kukri_c43901_220	7A	21718969	*TraesCS7A01G044200*	NL	310553	0.09
Winter	DON	4595_BobWhite_c930_1135	2B	20383875	*TraesCS2B01G039700*	NL	177644	0.19
Winter	DON	4595_BobWhite_c930_1135	2B	20383875	*TraesCS2B01G039500*	CN	204531	0.19
Winter	DON	4595_BobWhite_c930_1135	2B	20383875	*TraesCS2B01G039200*	CN	243724	0.19
Winter	DON	4595_BobWhite_c930_1135	2B	20383875	*TraesCS2B01G038900*	NL	377186	0.19
Winter	DON	4595_BobWhite_c930_1135	2B	20383875	*TraesCS2B01G038400*	RLK	484197	0.19
Winter	DON	4595_BobWhite_c930_1135	2B	20383875	*TraesCS2B01G038100*	CN	612271	0.19
Spring	DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G042500*	NL	349140	16.38
Spring	DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G042600*	NL	360251	16.38
Spring	DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G042700*	NL	432113	16.38
Spring	DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G043000*	CNL	467813	16.38
Spring	DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G044100*	CNL	854485	16.38
Spring	DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G044200*	NL	861844	16.38
Spring	DON	46288_Kukri_c5282_622	2A	19787961	*TraesCS2A01G044500*	NL	919670	16.38
Winter	VRI	46761_Kukri_c5873_645	2B	399582727	*TraesCS2B01G281900*	RLK	612780	0.97
Winter	VRI	46761_Kukri_c5873_645	2B	399582727	*TraesCS2B01G284500*	CN	792058	0.97
Combined	VRI	46794_Kukri_c59403_339	2D	77411184	*TraesCS2D01G129100*	RLK	711	0.29
Combined	VRI	46794_Kukri_c59403_339	2D	77411184	*TraesCS2D01G129000*	RLK	8490	0.29
Combined	VRI	46794_Kukri_c59403_339	2D	77411184	*TraesCS2D01G130500*	RLK	744169	0.29
Combined	VRI	46794_Kukri_c59403_339	2D	77411184	*TraesCS2D01G130600*	RLK	747435	0.29
Winter	VRI	47210_Kukri_c6519_97	6D	487999676	*TraesCS6D01G392500*	RLK	454861	0.59
Winter	VRI	47210_Kukri_c6519_97	6D	487999676	*TraesCS6D01G392600*	RLK	457344	0.59
Winter	VRI	47644_Kukri_c7569_1371	7A	275419561	*TraesCS7A01G268000*	NL	738334	1.55
Winter	VRI	47776_Kukri_c7874_1096	5A	535804823	*TraesCS5A01G323800*	RLK	377540	25.64
Winter	VRI	47776_Kukri_c7874_1096	5A	535804823	*TraesCS5A01G323900*	RLK	385996	25.64
Winter	VRI	49386_Kukri_rep_c109145_695	1A	573958234	*TraesCS1A01G409400*	CN	726913	0.2
Winter	VRI	51268_Ra_c17341_2119	7A	87166428	*TraesCS7A01G130100*	RLK	195737	0.2
Spring	INC	51731_Ra_c29107_289	2D	18602466	*TraesCS2D01G048600*	CN	351394	0.7
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G588900*	CNL	0	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G588800*	NL	44594	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G589100*	NL	81717	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G589300*	NL	135377	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G589500*	NL	224315	4.11

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G589700*	CN	370858	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G588200*	RLK	379207	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G588100*	NL	479158	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G587900*	CN	495325	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G587600*	NL	687078	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G587400*	CNL	699938	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCSU01G245300*	CN	894257	4.11
Spring	INC	51875_Ra_c33766_656	3B	834159260	*TraesCS3B01G590700*	CN	941769	4.11
Spring	INC	52304_Ra_c58279_702	2A	45928203	*TraesCS2A01G088800*	RLK	906853	1.02
Spring	INC	53989_RAC875_c14732_461	5D	555935295	*TraesCS5D01G540500*	CNL	203216	3.44
Spring	INC	53989_RAC875_c14732_461	5D	555935295	*TraesCS5D01G542500*	CN	828638	3.44
Spring	INC	53989_RAC875_c14732_461	5D	555935295	*TraesCS5D01G542600*	RLK	887547	3.44
Spring	DON	54254_RAC875_c16255_570	6A	17374497	*TraesCS6A01G030100*	CN	78209	1.43
Spring	DON	54254_RAC875_c16255_570	6A	17374497	*TraesCS6A01G029700*	TM-CC	190542	1.43
Spring	DON	54254_RAC875_c16255_570	6A	17374497	*TraesCS6A01G029200*	RLP	286494	1.43
Spring	DON	54254_RAC875_c16255_570	6A	17374497	*TraesCS6A01G029100*	RLK	292815	1.43
Combined	VRI	55230_RAC875_c21750_784	2D	630191012	*TraesCS2D01G551800*	RLP	15066	3.76
Combined	VRI	55230_RAC875_c21750_784	2D	630191012	*TraesCS2D01G551700*	RLP	76358	3.76
Combined	VRI	55230_RAC875_c21750_784	2D	630191012	*TraesCS2D01G551600*	RLP	81193	3.76
Combined	VRI	55230_RAC875_c21750_784	2D	630191012	*TraesCS2D01G551200*	RLK	436039	3.76
Combined	VRI	55230_RAC875_c21750_784	2D	630191012	*TraesCS2D01G550900*	RLK	649652	3.76
Winter	VRI	55857_RAC875_c25839_225	6D	313735680	*TraesCS6D01G206100*	RLK	136696	25.64
Winter	VRI	55857_RAC875_c25839_225	6D	313735680	*TraesCS6D01G205900*	RLK	290151	25.64
Combined	SEV	56084_RAC875_c27611_467	2B	9233744	*TraesCS2B01G001600*	RLK	0	7.73
Winter	VRI	57324_RAC875_c3770_970	7A	695383398	*TraesCS7A01G499600*	NL	729314	1.55
Combined	INC	57442_RAC875_c38693_319	7B	752222562	*TraesCS7B01G482300*	CNL	15509	0.4
Combined	INC	57442_RAC875_c38693_319	7B	752222562	*TraesCS7B01G482800*	NL	937985	0.4
Combined	DON	57507_RAC875_c39339_400	3B	817272964	*TraesCS3B01G567500*	RLK	649227	5.66
Spring	INC	58832_RAC875_c52458_454	2A	696313164	*TraesCS2A01G442900*	RLK	92745	3.73
Winter	VRI	59116_RAC875_c55445_92	6B	721147245	*TraesCS6B01G454800*	RLK	471266	29.01
Winter	VRI	59116_RAC875_c55445_92	6B	721147245	*TraesCS6B01G454900*	RLK	473981	29.01
Winter	VRI	59332_RAC875_c57998_165	2D	191033496	*TraesCS2D01G221400*	RLK	25704	0.2
Winter	VRI	59332_RAC875_c57998_165	2D	191033496	*TraesCS2D01G221900*	RLK	485817	0.2
Winter	VRI	59332_RAC875_c57998_165	2D	191033496	*TraesCS2D01G222000*	RLK	547913	0.2
Winter	VRI	59332_RAC875_c57998_165	2D	191033496	*TraesCS2D01G222100*	RLK	560982	0.2
Winter	VRI	59332_RAC875_c57998_165	2D	191033496	*TraesCS2D01G222300*	RLK	576879	0.2
Winter	VRI	59332_RAC875_c57998_165	2D	191033496	*TraesCS2D01G222500*	RLK	719633	0.2
Winter	VRI	59332_RAC875_c57998_165	2D	191033496	*TraesCS2D01G222700*	RLK	861575	0.2
Combined	DON	59530_RAC875_c60522_1342	5A	567522493	*TraesCS5A01G365400*	RLK	48049	0.64
Combined	DON	59530_RAC875_c60522_1342	5A	567522493	*TraesCS5A01G365300*	RLK	56431	0.64
Combined	DON	59530_RAC875_c60522_1342	5A	567522493	*TraesCS5A01G365600*	RLK	63146	0.64
Spring	DON	60140_RAC875_c6798_467	1A	538108963	*TraesCS1A01G354100*	CN	642146	5.14
Spring	DON	60140_RAC875_c6798_467	1A	538108963	*TraesCS1A01G354200*	CN	646687	5.14
Winter	VRI	60475_RAC875_c77534_451	5B	671028119	*TraesCS5B01G502500*	RLK	623556	7.58
Winter	VRI	60475_RAC875_c77534_451	5B	671028119	*TraesCS5B01G502400*	RLK	645212	7.58
Combined	INC	60785_RAC875_c8494_93	4A	500153212	*TraesCS4A01G207800*	RLK	7414	25.18
Combined	DON	610_BobWhite_c14222_571	2B	104978464	*TraesCS2B01G129700*	NL	50121	13.17
Winter	VRI	61578_RAC875_rep_c106337_522	5D	393494214	*TraesCS5D01G292500*	RLK	667086	29.01
Winter	DON	61616_RAC875_rep_c106596_127	2B	29764155	*TraesCS2B01G054500*	RLK	177271	7.15

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Winter	DON	61616_RAC875_rep_c106596_127	2B	29764155	*TraesCS2B01G053500*	RLK	456094	7.15
Winter	DON	61616_RAC875_rep_c106596_127	2B	29764155	*TraesCS2B01G056500*	NL	786443	7.15
Winter	DON	61616_RAC875_rep_c106596_127	2B	29764155	*TraesCS2B01G057100*	TM-CC	975923	7.15
Winter	VRI	61888_RAC875_rep_c109516_822	7A	88401545	*TraesCS7A01G132200*	CNL	414106	0.2
Winter	VRI	62843_RAC875_rep_c72158_264	2B	779063796	*TraesCS2B01G581400*	RLP	857726	1.55
Winter	VRI	62843_RAC875_rep_c72158_264	2B	779063796	*TraesCS2B01G581300*	RLP	866153	1.55
Spring	INC	63392_RAC875_s105188_92	1B	462883976	*TraesCS1B01G259000*	TM-CC	699769	11.31
Spring	INC	63897_RFL_Contig2290_184	1B	567447186	*TraesCS1B01G333300*	RLK	793007	0.04
Combined	VRI	64957_RFL_Contig5277_888	2A	775589238	*TraesCS2A01G580900*	RLK	987841	5.76
Winter	DON	65160_RFL_Contig5906_985	1A	16958960	*TraesCS1A01G031800*	RLK	828996	42.09
Winter	VRI	65349_TA001138-1003	5B	589168687	*TraesCS5B01G410900*	RLK	599426	0.96
Spring	INC	65469_TA001874-1495	2B	37419792	*TraesCS2B01G067000*	RLK	920318	0.74
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G039700*	RLP	4316	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G039600*	TM-CC	12946	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G039500*	RLK	25573	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G039400*	RLK	30094	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G039300*	RLK	66992	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G039200*	RLK	119021	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G039100*	RLK	126157	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G038700*	RLP	215414	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G038800*	RLP	222305	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G038500*	RLP	232737	1.51
Winter	VRI	65471_TA001885-0568	3A	21599951	*TraesCS3A01G037200*	RLK	812240	1.51
Spring	SEV	65683_TA003419-0641	6D	487522708	*TraesCS6D01G392500*	RLK	931829	0.44
Spring	SEV	65683_TA003419-0641	6D	487522708	*TraesCS6D01G392600*	RLK	934312	0.44
Combined	DON	68352_Tdurum_contig15126_286	4A	730542903	*TraesCS4A01G456900*	RLK	724740	0.06
Combined	DON	68352_Tdurum_contig15126_286	4A	730542903	*TraesCS4A01G458300*	RLK	741158	0.06
Spring	DON	68412_Tdurum_contig15438_231	2A	19671792	*TraesCS2A01G042500*	NL	465309	20.65
Spring	DON	68412_Tdurum_contig15438_231	2A	19671792	*TraesCS2A01G042600*	NL	476420	20.65
Spring	DON	68412_Tdurum_contig15438_231	2A	19671792	*TraesCS2A01G042700*	NL	548282	20.65
Spring	DON	68412_Tdurum_contig15438_231	2A	19671792	*TraesCS2A01G043000*	CNL	583982	20.65
Spring	DON	68412_Tdurum_contig15438_231	2A	19671792	*TraesCS2A01G044100*	CNL	970654	20.65
Spring	DON	68412_Tdurum_contig15438_231	2A	19671792	*TraesCS2A01G044200*	NL	978013	20.65
Combined	DON	6908_BS00022107_51	5B	35393045	*TraesCS5B01G031600*	CN	0	0
Combined	DON	6908_BS00022107_51	5B	35393045	*TraesCS5B01G032400*	RLK	978861	0
Winter	VRI	69162_Tdurum_contig25674_294	5A	563619393	*TraesCS5A01G359800*	TM-CC	152110	0.2
Winter	VRI	69384_Tdurum_contig27942_125	2A	81899936	*TraesCS2A01G128900*	RLK	42099	0.2
Winter	VRI	69384_Tdurum_contig27942_125	2A	81899936	*TraesCS2A01G129700*	RLK	340186	0.2
Winter	VRI	69845_Tdurum_contig29607_294	6D	483997120	*TraesCS6D01G384800*	RLP	315208	25.64
Winter	INC	70065_Tdurum_contig30621_328	7A	71357685	*TraesCS7A01G111100*	CN	5258	17.12
Winter	INC	70065_Tdurum_contig30621_328	7A	71357685	*TraesCS7A01G111000*	CN	5483	17.12
Winter	INC	70065_Tdurum_contig30621_328	7A	71357685	*TraesCS7A01G111200*	CN	37773	17.12
Winter	VRI	70129_Tdurum_contig30916_316	3B	816074580	*TraesCS3B01G565900*	RLP	209386	0.2
Winter	VRI	70129_Tdurum_contig30916_316	3B	816074580	*TraesCS3B01G567500*	RLK	546637	0.2
Winter	VRI	7136_BS00022524_51	1D	10383887	*TraesCS1D01G025200*	CN	432705	13.95
Winter	VRI	7136_BS00022524_51	1D	10383887	*TraesCS1D01G026000*	CNL	697944	13.95
Winter	VRI	7136_BS00022524_51	1D	10383887	*TraesCS1D01G021200*	NL	887992	13.95
Winter	VRI	7136_BS00022524_51	1D	10383887	*TraesCS1D01G021000*	CNL	940173	13.95
Winter	VRI	71614_Tdurum_contig45504_166	6A	104735561	*TraesCS6A01G130200*	RLP	3900	25.64

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Winter	VRI	71614_Tdurum_contig45504_166	6A	104735561	*TraesCS6A01G130000*	RLP	14323	25.64
Winter	VRI	71614_Tdurum_contig45504_166	6A	104735561	*TraesCS6A01G129900*	RLP	294166	25.64
Spring	VRI	71780_Tdurum_contig47269_904	2D	601674145	*TraesCS2D01G504300*	RLK	23749	0.98
Spring	VRI	71780_Tdurum_contig47269_904	2D	601674145	*TraesCS2D01G504400*	RLK	79658	0.98
Spring	VRI	71780_Tdurum_contig47269_904	2D	601674145	*TraesCS2D01G504500*	NL	91855	0.98
Spring	VRI	71780_Tdurum_contig47269_904	2D	601674145	*TraesCS2D01G503300*	RLK	471807	0.98
Spring	VRI	71780_Tdurum_contig47269_904	2D	601674145	*TraesCS2D01G505200*	RLK	562058	0.98
Spring	INC	72169_Tdurum_contig51313_408	3A	176407029	*TraesCS3A01G161800*	RLK	955991	0.17
Winter	DON	72586_Tdurum_contig57566_1653	1A	535410856	*TraesCS1A01G346300*	RLK	806759	5.86
Winter	DON	72586_Tdurum_contig57566_1653	1A	535410856	*TraesCS1A01G345700*	RLK	954123	5.86
Winter	DON	72586_Tdurum_contig57566_1653	1A	535410856	*TraesCS1A01G345500*	RLK	970768	5.86
Winter	DON	72586_Tdurum_contig57566_1653	1A	535410856	*TraesCS1A01G345400*	RLK	982222	5.86
Spring	DON	74007_Tdurum_contig97656_120	3B	24979593	*TraesCS3B01G040100*	CN	143317	20.19
Spring	DON	74007_Tdurum_contig97656_120	3B	24979593	*TraesCS3B01G039200*	CNL	475733	20.19
Combined	SEV	74043_Tdurum_contig98478_494	4B	118610356	*TraesCS4B01G106000*	RLK	25045	1.75
Spring	INC	74456_tplb0032a08_1721	3D	5664733	*TraesCS3D01G014500*	RLK	678873	0.46
Spring	INC	74456_tplb0032a08_1721	3D	5664733	*TraesCS3D01G014200*	RLK	696941	0.46
Spring	INC	74456_tplb0032a08_1721	3D	5664733	*TraesCS3D01G013600*	RLK	735435	0.46
Combined	SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G401300*	RLP	30111	5.12
Combined	SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G401200*	RLP	34777	5.12
Combined	SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G401100*	RLP	84233	5.12
Combined	SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G401000*	RLP	86386	5.12
Combined	SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G400900*	RLP	113324	5.12
Combined	SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G400800*	RLP	115806	5.12
Combined	SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G400700*	RLP	237154	5.12
Combined	SEV	75644_wsnp_BF292596A_Ta_1_3	3A	647674948	*TraesCS3A01G400500*	TM-CC	316978	5.12
Spring	INC	76127_wsnp_CD454706B_Ta_1_1	2D	610294752	*TraesCS2D01G516700*	RLP	613014	0.08
Spring	INC	76127_wsnp_CD454706B_Ta_1_1	2D	610294752	*TraesCS2D01G516900*	RLP	622873	0.08
Spring	INC	76127_wsnp_CD454706B_Ta_1_1	2D	610294752	*TraesCS2D01G517100*	RLP	833735	0.08
Spring	SEV	77039_wsnp_Ex_c19525_28494827	6B	721736556	*TraesCS6B01G454900*	RLK	113347	3.6
Spring	SEV	77039_wsnp_Ex_c19525_28494827	6B	721736556	*TraesCS6B01G454800*	RLK	115647	3.6
Spring	SEV	77039_wsnp_Ex_c19525_28494827	6B	721736556	*TraesCS6B01G457400*	CN	776538	3.6
Spring	SEV	77039_wsnp_Ex_c19525_28494827	6B	721736556	*TraesCS6B01G457700*	CNL	821568	3.6
Winter	VRI	77134_wsnp_Ex_c20695_29781602	7A	5319681	*TraesCS7A01G012100*	RLP	178345	0.34
Winter	VRI	77134_wsnp_Ex_c20695_29781602	7A	5319681	*TraesCS7A01G011400*	RLP	197677	0.34
Winter	VRI	77134_wsnp_Ex_c20695_29781602	7A	5319681	*TraesCS7A01G012700*	RLP	433381	0.34
Winter	VRI	77134_wsnp_Ex_c20695_29781602	7A	5319681	*TraesCS7A01G013000*	RLP	567839	0.34
Winter	VRI	77854_wsnp_Ex_c3530_6459532	6A	14464729	*TraesCS6A01G027400*	RLK	736819	0.35
Winter	VRI	77854_wsnp_Ex_c3530_6459532	6A	14464729	*TraesCS6A01G027600*	CNL	914956	0.35
Winter	VRI	77854_wsnp_Ex_c3530_6459532	6A	14464729	*TraesCS6A01G024200*	RLP	986895	0.35
Spring	INC	77908_wsnp_Ex_c3681_6715277	1B	462883977	*TraesCS1B01G259000*	TM-CC	699770	11.31
Combined	DON	78397_wsnp_Ex_c54092_57099525	5B	554616628	*TraesCS5B01G374600*	TM-CC	404430	1.97
Winter	VRI	78537_wsnp_Ex_c5979_10480527	4A	466403334	*TraesCS4A01G187300*	RLK	719741	0.34
Winter	VRI	79013_wsnp_Ex_rep_c101269_86664147	3A	712607958	*TraesCS3A01G479400*	CN	983723	14.18
Spring	VRI	79037_wsnp_Ex_rep_c101757_87064771	5A	438395139	*TraesCS5A01G222200*	RLK	86247	2.67
Spring	VRI	79037_wsnp_Ex_rep_c101757_87064771	5A	438395139	*TraesCS5A01G222300*	RLK	138757	2.67
Spring	VRI	79037_wsnp_Ex_rep_c101757_87064771	5A	438395139	*TraesCS5A01G222500*	TM-CC	202838	2.67
Spring	INC	79457_wsnp_Ex_rep_c68712_67571580	1B	462183400	*TraesCS1B01G259000*	TM-CC	0	17.96
Winter	VRI	80119_wsnp_Ku_c14920_23377027	6A	618589681	*TraesCS6A01G414300*	NL	153639	0.49

Panel	Trait	QTN	Chr	Position	Candidate gene	Functional annotation	Distance to gene (bp)	R^2 (%)
Winter	VRI	80119_wsnp_Ku_c14920_23377027	6A	618589681	*TraesCS6A01G414400*	CNL	163431	0.49
Winter	VRI	80119_wsnp_Ku_c14920_23377027	6A	618589681	*TraesCSU01G219700*	CN	163900	0.49
Winter	VRI	80119_wsnp_Ku_c14920_23377027	6A	618589681	*TraesCSU01G213400*	CN	240751	0.49
Winter	VRI	80119_wsnp_Ku_c14920_23377027	6A	618589681	*TraesCSU01G168400*	NL	263032	0.49
Winter	VRI	80119_wsnp_Ku_c14920_23377027	6A	618589681	*TraesCSU01G203600*	NL	264460	0.49
Winter	VRI	80119_wsnp_Ku_c14920_23377027	6A	618589681	*TraesCS6A01G415000*	CN	335377	0.49
Winter	VRI	80119_wsnp_Ku_c14920_23377027	6A	618589681	*TraesCSU01G194700*	CN	346255	0.49
Combined	DON	80235_wsnp_Ku_c21275_31007309	5A	567525415	*TraesCS5A01G365400*	RLK	50971	3.88
Combined	DON	80235_wsnp_Ku_c21275_31007309	5A	567525415	*TraesCS5A01G365300*	RLK	59353	3.88
Combined	DON	80235_wsnp_Ku_c21275_31007309	5A	567525415	*TraesCS5A01G365600*	RLK	60224	3.88
Spring	VRI	80285_wsnp_Ku_c25372_35336531	7D	14163144	*TraesCS7D01G029200*	RLK	969745	1.26
Spring	SEV	80328_wsnp_Ku_c2797_5284087	1B	687093328	*TraesCS1B01G469500*	CNL	0	2.57
Spring	SEV	80328_wsnp_Ku_c2797_5284087	1B	687093328	*TraesCS1B01G469400*	RLK	48553	2.57
Spring	SEV	80328_wsnp_Ku_c2797_5284087	1B	687093328	*TraesCS1B01G468200*	TM-CC	927100	2.57
Spring	VRI	80485_wsnp_Ku_c4045_7380115	1A	553440047	*TraesCS1A01G379100*	CN	106750	6.03
Spring	INC	80996_wsnp_Ra_c2027_3945713	1B	462883485	*TraesCS1B01G259000*	TM-CC	699278	14.14
Spring	INC	80997_wsnp_Ra_c2027_3945764	1B	462883434	*TraesCS1B01G259000*	TM-CC	699227	19.82
Winter	VRI	8117_BS00037784_51	6B	672525118	*TraesCS6B01G388800*	CN	190865	7.58
Spring	SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G060000*	RLK	15888	8.75
Spring	SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G060100*	RLK	58312	8.75
Spring	SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G060200*	RLK	69206	8.75
Spring	SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G059400*	RLP	157389	8.75
Spring	SEV	81493_wsnp_RFL_Contig3344_3442711	3A	37135958	*TraesCS3A01G061400*	RLK	427107	8.75
Winter	VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G365500*	RLK	94542	7.58
Winter	VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G365400*	RLK	137622	7.58
Winter	VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G366000*	RLK	303408	7.58
Winter	VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G366200*	RLK	308246	7.58
Winter	VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G366300*	RLK	383507	7.58
Winter	VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G366600*	RLK	656634	7.58
Winter	VRI	8353_BS00043866_51	7A	543527851	*TraesCS7A01G364500*	RLK	768805	7.58
Winter	VRI	8809_BS00060541_51	6B	165407501	*TraesCS6B01G157800*	RLP	616475	8
Spring	DON	9653_BS00065865_51	2A	721702719	*TraesCS2A01G479800*	TM-CC	62768	3.48
Spring	SEV	9955_BS00067096_51	5A	36408984	*TraesCS5A01G037500*	RLP	374845	18.81

Appendix 8 Predictive ability ($r \pm s$) of four FHB related traits with each marker sets along with their panels. GBLUP was used to estimate *r*-values

Panels	Marker sets	MLM	GLM	FarmCPU	FASTmrMLM	FASTmrEMMA	ISIS EM-BLASSO	mrMLM	pkWmEB	pLARmEB	No. of markers	$r \pm s$
		p-value					LOD score					
Combined	Set1	0.05	0.05	3	3	3	3	3	3	3	174	**0.79±0.05a**
	Set5	0.03	0.03	1	1	1	1	1	1	1	4251	0.71±0.08b
	Set4	0.02	0.02	2	2	2	2	2	2	2	3498	0.71±0.08bc
	Set6	0.06	0.06	1	1	1	1	1	1	1	5495	0.70±0.07bc
	Set3	0.01	0.01	2	2	2	2	2	2	2	1459	0.70±0.08bc
	Set7	0.08	0.08	1	1	1	1	1	1	1	7158	0.68±0.09bcd
	Set8	0.1	0.1	1	1	1	1	1	1	1	8287	0.69±0.08bcd
	Set9	0.15	0.15	1	1	1	1	1	1	1	11310	0.68±0.07cd
	Set2	0.005	0.01	2	2	2	2	2	2	2	484	0.66±0.11de
	Set10 (All SNPs)	0.05	0.05	3	3	3	3	3	3	3	13760	0.63±0.09e
Spring	Set1	0.05	0.05	3	3	3	3	3	3	3	107	**0.82±0.05a**
	Set6	0.06	0.06	1	1	1	1	1	1	1	4686	0.67±0.09b
	Set7	0.08	0.08	1	1	1	1	1	1	1	6279	0.67±0.08b
	Set8	0.1	0.1	1	1	1	1	1	1	1	7469	0.67±0.09b
	Set9	0.15	0.15	1	1	1	1	1	1	1	11484	0.67±0.09b
	Set4	0.02	0.02	2	2	2	2	2	2	2	920	0.66±0.09bc
	Set5	0.03	0.03	1	1	1	1	1	1	1	1993	0.65±0.09bc
	Set10 (All SNPs)	0.05	0.05	3	3	3	3	3	3	3	13760	0.62±0.07bcd
	Set2	0.005	0.01	2	2	2	2	2	2	2	198	0.61±0.13cd
	Set3	0.01	0.01	2	2	2	2	2	2	2	454	0.59±0.19d
Winter	Set1	0.05	0.05	3	3	3	3	3	3	3	157	**0.62±0.25a**
	Set3	0.01	0.01	2	2	2	2	2	2	2	807	0.58±0.21b
	Set4	0.02	0.02	2	2	2	2	2	2	2	1058	0.58±0.23b
	Set5	0.03	0.03	1	1	1	1	1	1	1	1672	0.58±0.20b

Panels	Marker sets	MLM	GLM	FarmCPU	FASTmrMLM	FASTmrEMMA	ISIS EM-BLASSO	mrMLM	pkWmEB	pLARmEB	No. of markers	*r* ± *s*
	Set7	0.08	0.08	1	1	1	1	1	1	1	3420	0.59±0.170b
	Set8	0.1	0.1	1	1	1	1	1	1	1	4027	0.58±0.20b
	Set9	0.15	0.15	1	1	1	1	1	1	1	9209	0.58±0.18b
	Set2	0.005	0.01	2	2	2	2	2	2	2	592	0.53±0.19bc
	Set6	0.06	0.06	1	1	1	1	1	1	1	2222	0.52±0.23bc
	Set10 (All SNPs)	0.05	0.05	3	3	3	3	3	3	3	10421	0.50±0.18bc

Appendix 9 Predictive ability ($r \pm s$) of four traits using quantitative trait loci (QTN) of three panels identified by ten statistical GS models

Trait	Model	$r \pm s$ (Combined)	Model	$r \pm s$ (Spring)	Model	$r \pm s$ (Winter)
INC	RR_BLUP	0.82±0.04a	BayesC	0.83±0.04a	BayesA	0.51±0.24a
	GBLUP	0.81±0.04a	GBLUP	0.82±0.04a	RFR	0.51±0.22a
	BRR	0.81±0.04a	BRR	0.82±0.04a	BayesB	0.49±0.26b
	BL	0.81±0.04a	BL	0.82±0.04a	BayesC	0.47±0.26c
	BayesA	0.81±0.04a	BayesA	0.82±0.04a	RR_BLUP	0.46±0.24cd
	BayesB	0.81±0.05a	BayesB	0.82±0.05a	BRR	0.46±0.27cd
	BayesC	0.81±0.05a	RR_BLUP	0.82±0.04ab	BL	0.42±0.25d
	SVR	0.80±0.05a	RKHS	0.81±0.05b	GBLUP	0.41±0.27d
	RKHS	0.78±0.05	SVR	0.77±0.06c	RKHS	0.40±0.26d
	RFR	0.75±0.06b	RFR	0.74±0.08d	SVR	0.35±0.31e
SEV	GBLUP	0.76±0.05a	BayesC	0.83±0.05a	RFR	0.63±0.19a
	RR_BLUP	0.76±0.05a	GBLUP	0.82±0.05ab	RKHS	0.54±0.2b
	BRR	0.76±0.05a	RR_BLUP	0.82±0.05ab	GBLUP	0.52±0.18bc
	BL	0.76±0.05a	BRR	0.82±0.05ab	RR_BLUP	0.52±0.17bc
	BayesA	0.76±0.05a	BL	0.82±0.05ab	BRR	0.51±0.18bc
	BayesB	0.76±0.05a	BayesA	0.82±0.05ab	BL	0.51±0.18bc
	BayesC	0.76±0.05a	BayesB	0.82±0.05ab	BayesC	0.50±0.18c
	RKHS	0.76±0.05a	RKHS	0.81±0.05b	BayesA	0.49±0.2cd
	SVR	0.73±0.06b	SVR	0.77±0.05c	BayesB	0.48±0.21cd
	RFR	0.67±0.07c	RFR	0.71±0.07d	BayesA	0.47±0.27d
VRI	GBLUP	0.80±0.05a	GBLUP	0.80±0.05a	RFR	0.62±0.21a
	RR_BLUP	0.80±0.05a	RR_BLUP	0.80±0.05a	RKHS	0.58±0.19ab
	BRR	0.80±0.05a	BRR	0.80±0.05a	RR_BLUP	0.55±0.23b
	BL	0.80±0.05a	BL	0.80±0.05a	BayesA	0.52±0.25bc
	BayesA	0.80±0.05a	BayesA	0.80±0.05a	GBLUP	0.51±0.21c
	BayesC	0.80±0.05a	BayesC	0.80±0.05a	BRR	0.51±0.22c
	RKHS	0.80±0.05a	RKHS	0.80±0.05a	BayesC	0.50±0.23c
	BayesB	0.79±0.05ab	BayesB	0.79±0.05ab	BL	0.49±0.25cd
	SVR	0.78±0.06b	SVR	0.78±0.06b	BayesB	0.48±0.28cd
	RFR	0.70±0.07c	RFR	0.70±0.07c	SVR	0.42±0.29d

Trait	Model	$r \pm s$ (Combined)	Model	$r \pm s$ (Spring)	Model	$r \pm s$ (Winter)
DON	GBLUP	0.80±0.05a	GBLUP	0.77±0.06a	RKHS	0.64±0.25a
	RR_BLUP	0.80±0.05a	RR_BLUP	0.78±0.06a	RFR	0.59±0.24b
	BRR	0.80±0.05a	BRR	0.77±0.06a	GBLUP	0.58±0.29ab
	BL	0.79±0.05a	BL	0.78±0.06a	BRR	0.58±0.28ab
	BayesA	0.79±0.05a	BayesA	0.78±0.06a	RR_BLUP	0.57±0.28ab
	BayesB	0.79±0.05a	BayesB	0.78±0.06a	BayesC	0.57±0.29ab
	BayesC	0.79±0.05a	BayesC	0.78±0.06a	BL	0.56±0.28ab
	RKHS	0.79±0.05ab	RKHS	0.75±0.06b	BayesA	0.55±0.28ab
	SVR	0.77±0.05b	RFR	0.74±0.06bc	BayesB	0.53±0.29ab
	RFR	0.76±0.05c	SVR	0.73±0.06c	SVR	0.49±0.22b

INC, incidence; SEV, SEV; VRI, visual rating index; DON, deoxynivalenol content

Appendix 10 Analysis of variance (ANOVA) for statistical significance of various sources constructed by different traits, statistical models of GWAS and panels

Source	Degree of freedom	Sum of Square	Mean Square	F-value	Pr(>F)
Traits	3	7.6	2.54	168.782	< 2e-16 ***
Models	9	11.5	1.28	85.045	< 2e-16 ***
Panels	2	213.6	106.78	7083.207	< 2e-16 ***
Traits:Models	27	1.6	0.06	3.922	2.87e-11 ***
Models:Panels	18	10.3	0.57	37.899	< 2e-16 ***
Traits:Panels	6	19.2	3.2	212.346	< 2e-16 ***
Residuals	29934	451.3	0.02		

Appendix 11 Predictive ability ($r \pm s$) of four traits using all single nucleotide polymorphisms (All-SNPs) of three panels identified by ten statistical GS models

Trait	No. of markers	Model	*r±s* (Combined)	Model	*r±s* (Spring)	Model	*r±s* (Winter)
INC	All-SNPs	GBLUP	0.63±0.09a	GBLUP	0.64±0.09a	RR_BLUP	0.54±0.15a
		RR_BLUP	0.63±0.09a	RR_BLUP	0.64±0.09a	BayesA	0.54±0.16ab
		BRR	0.63±0.09a	BRR	0.64±0.09a	BL	0.53±0.14ab
		BL	0.63±0.09a	BL	0.64±0.09a	GBLUP	0.53±0.15ab
		BayesA	0.63±0.09a	BayesA	0.64±0.09a	BRR	0.53±0.15ab
		BayesB	0.63±0.09a	BayesB	0.64±0.09a	BayesB	0.53±0.15ab
		BayesC	0.63±0.09a	BayesC	0.64±0.09a	BayesC	0.53±0.16ab
		RFR	0.63±0.09a	SVR	0.64±0.09a	SVR	0.52±0.16abc
		RKHS	0.63±0.09a	RFR	0.63±0.09ab	RKHS	0.51±0.16bc
		SVR	0.61±0.10b	RKHS	0.61±0.09b	RFR	0.49±0.16c
SEV	All-SNPs	GBLUP	0.56±0.08a	GBLUP	0.61±0.1a	GBLUP	0.51±0.20a
		RR_BLUP	0.55±0.08a	BRR	0.61±0.1a	BRR	0.51±0.20a
		BRR	0.56±0.08a	BL	0.61±0.1a	BayesA	0.51±0.20a
		BL	0.56±0.08a	BayesA	0.61±0.1a	RR_BLUP	0.51±0.21a
		BayesA	0.56±0.08a	BayesB	0.61±0.1a	BayesB	0.51±0.21a
		BayesB	0.56±0.08a	BayesC	0.61±0.1a	BayesC	0.50±0.21a
		BayesC	0.56±0.08a	SVR	0.61±0.1ab	SVR	0.49±0.25ab
		RFR	0.55±0.08a	RFR	0.59±0.09ab	BL	0.48±0.20bc
		SVR	0.54±0.09a	RR_BLUP	0.60±0.1ab	RKHS	0.48±0.23bc
		RKHS	0.56±0.08a	RKHS	0.58±0.1b	RFR	0.46±0.26c
VRI	All-SNPs	GBLUP	0.61±0.08a	BayesB	0.62±0.1a	BRR	0.59±0.14a
		RR_BLUP	0.60±0.08a	BayesC	0.62±0.09a	BayesA	0.59±0.14a
		BRR	0.61±0.08a	GBLUP	0.62±0.09ab	BayesB	0.59±0.14a
		BL	0.60±0.08a	BRR	0.62±0.1ab	BL	0.58±0.12a
		BayesA	0.61±0.08a	BL	0.62±0.09ab	GBLUP	0.58±0.14a
		BayesB	0.61±0.08a	BayesA	0.62±0.1ab	RR_BLUP	0.58±0.14a
		BayesC	0.61±0.08a	SVR	0.61±0.09ab	BayesC	0.58±0.14a
		RFR	0.60±0.08a	RR_BLUP	0.61±0.1abc	RFR	0.58±0.17a
		SVR	0.58±0.09a	RFR	0.59±0.1bc	RKHS	0.56±0.14b
		RKHS	0.61±0.08a	RKHS	0.58±0.11c	SVR	0.56±0.16b

Trait	No. of markers	Model	*r±s* (Combined)	Model	*r±s* (Spring)	Model	*r±s* (Winter)
DON	All-SNPs	RFR	0.67±0.07a	RFR	0.63±0.09a	RKHS	0.63±0.3
		GBLUP	0.65±0.08ab	RR_BLUP	0.62±0.09a	GBLUP	0.62±0.3
		BRR	0.65±0.07ab	GBLUP	0.61±0.09ab	BRR	0.62±0.29
		BL	0.65±0.07ab	BRR	0.61±0.09ab	BayesC	0.62±0.29
		BayesC	0.65±0.07ab	BL	0.61±0.09ab	BayesA	0.6±0.28
		RKHS	0.65±0.08ab	BayesA	0.61±0.09ab	BL	0.59±0.28
		RR_BLUP	0.65±0.07b	BayesB	0.61±0.09ab	BayesB	0.59±0.28
		BayesA	0.64±0.08b	BayesC	0.61±0.09ab	SVR	0.59±0.26
		BayesB	0.64±0.08b	RKHS	0.61±0.09ab	RR_BLUP	0.58±0.29
		SVR	0.63±0.08b	SVR	0.59±0.09b	RFR	0.53±0.27

INC, incidence; SEV, SEV; VRI, visual rating index; DON, deoxynivalenol content