

Association Mapping and Genomic Selection for Stem Rust Resistance of Western Canadian Winter Wheat

By

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TABLE OF CONTENTS

ACKNOWLEDGEMENTS	i
TABLE OF CONTENTS	iii
LIST OF TABLES	vi
LIST OF FIGURES	vii
ABSTRACT	x
FOREWORD	xii
Chapter 1: GENERAL INTRODUCTION	1
Chapter 2: LITERATURE REVIEW	4
2.1 Wheat	4
2.1.1 Evolution of Wheat	4
2.1.2 Wheat Production in Canada	4
2.1.3 Winter Wheat	5
2.2 Stem Rust of Wheat	6
2.2.1 Life cycle of <i>Puccinia graminis</i> f. sp. <i>tritici</i>	7
2.2.2 Epidemiology	8
2.2.3 Resistance	9
2.2.4 Differential Sets, Nomenclature and Rating Scales	11
2.2.5 Epidemics	11
2.2.6 Pathogen Control	14
2.3 Marker Assisted Selection	16
2.4 Quantitative Trait Loci (QTL) Analysis	18
2.5 Genome-Wide Association Studies	19
2.6 Genomic Selection	21
Chapter 3: GENOME-WIDE ASSOCIATION STUDY FOR STEM RUST RESISTANCE IN WESTERN CANADIAN WINTER WHEAT (<i>Triticum aestivum</i> L.)	24
3.1 Abstract	24
3.2 Introduction	26
3.3 Materials and Methods	28
3.3.1 Germplasm	28

3.3.2	Phenotypic Evaluation - Inoculated Field Trials.....	31
3.3.3	Seedling Assays	33
3.3.4	Statistical Analysis	35
3.3.5	Data Transformation	36
3.3.6	DNA Extraction and Genotyping.....	36
3.3.7	Population Structure and Linkage Disequilibrium	38
3.3.8	Genome-Wide Association Study	38
3.3.9	QTL Stacking.....	39
3.4	Results.....	41
3.4.1	Phenotypic Assessment – Field Trials.....	41
3.4.2	Phenotypic Assessment – Seedling Assays.....	45
3.4.3	Genotyping, SNP Filtering, and Validation.....	47
3.4.4	Population Structure and Linkage Disequilibrium	47
3.4.5	Genome-Wide Association Study Results	50
3.4.6	Comparison of Identified QTL and Reported <i>Sr</i> Genes and QTLs	63
3.5	QTL Stacking.....	65
3.5.1	Effects of Individual QTL.....	65
3.5.2	Effect of Identified QTL Combinations	67
3.6	Discussion	70
3.7	Conclusion	81
Chapter 4: EVALUATION OF GENOMIC SELECTION FOR STEM RUST		
RESISTANCE IN WESTERN CANADIAN WINTER WHEAT (<i>Triticum aestivum</i> L.)		83
4.1	Abstract.....	83
4.2	Introduction.....	85
4.3	Materials and Methods.....	87
4.3.1	Plant materials, Phenotypic Data, and Genotypic Data	87
4.3.2	Genomic Selection Analyses.....	88
4.3.3	Cross-validation	91
4.4	Results.....	91
4.5	Phenotypic Distribution and Heritability of Field Traits	91
4.6	Prediction Accuracies for Adult Plant Resistance.....	93

4.7	Assessment of Predictive Performance and Model Fit	95
4.8	Phenotypic Distribution of Seedling Evaluations	97
4.9	Prediction Accuracies for Seedling Resistance	98
4.10	Discussion	102
4.11	Conclusion	104
Chapter 5: GENERAL DISCUSSION		106
LITERATURE CITED		110
APPENDICES		123
A.1	List of Abbreviations	123
A.2	Supplemental Figures	125
A3.	Raw Phenotypic Field Data	128
A4.	Seedling Assay Phenotypic Data	197
A5.	List of Genotypes used for Genomic Selection	208

LIST OF TABLES

Table 3.1 Summary statistics of best linear unbiased predictions (BLUPs) for stem rust severity, infection type, and coefficient of infection calculated from the association mapping population field trials. BLUPS were estimated from the raw data, across each site-year independently and the pooled dataset across environments.....	42
Table 3.2 Summary statistics of rank-based inverse normal transformed best linear unbiased predictions (BLUPs) for stem rust severity, infection type, and coefficient of infection calculated from the association mapping population field trials.....	43
Table 3.3 Comparative statistics of the seven <i>Pgt</i> races evaluated independently for infection type to 421 genotypes of the association mapping population	45
Table 3.4 Peak quantitative trait loci (QTL) for the field stem rust resistance traits identified in the association mapping population using Mixed Linear Model (MLM) GWAS analysis. Peak QTL identified by the most significant marker identified for each trait in each site-year.....	55
Table 3.5 Peak quantitative trait loci (QTL) identified in the association mapping population at the seedling stage through controlled environment inoculations using Mixed Linear Model (MLM) GWAS analysis. Peak QTL identified by the most significant marker identified for each race evaluated.	61
Table 3.6 Comparison of identified QTL that coincide for stem rust resistance of wheat in previously identified QTL.....	64
Table 4.1 Heritability estimates for the three field traits used to evaluate adult plant resistance in the association mapping population. The heritability was calculated from the pooled dataset. ...	92
Table 4.2 Prediction accuracies reported as r (Pearson's correlation) for the eight GS models evaluated using the pooled phenotypic data from the field trials.	94
Table 4.3 Prediction accuracies reported as r (Pearson's correlation) for the eight GS models evaluated using the results from the seedling evaluation where seven <i>Pgt</i> races were examined.	100

LIST OF FIGURES

Figure 3.3.1 Pedigree relationships among western Canadian winter wheat lines illustrating the ancestry of germplasm used in breeding programs. Permission granted for use from Dr. Robert Graf.	30
Figure 3.3.2 Infection types (IT) of wheat seedlings inoculated with <i>Puccinia graminis</i> f. sp. <i>tritici</i> , illustrating the Stakman 0–4 rating scale Based upon the Stakman scale, the ratings are as follows, “0” no presence of uredinia; “;” is indicative of a hypersensitive fleck; “1” indicates the presence of small uredinia with necrosis; “2” = small to medium uredinia, chlorosis is present; “3” indicates medium uredinia; and “4” indicates the presence of large uredinia. Individuals categorized as a “1” or “2” were considered resistant, while individuals identified to be a “3” or “4” were considered susceptible	35
Figure 3.4.1 Frequency distributions of variation in phenotypic evaluations for stem rust severity, infection type, and coefficient of infection in the association mapping population across all site-years and the pooled dataset. Frequency distributions were plotted for the original data and the data transformed using RNOmni. CRM23, Carman 2023 untransformed data (a); CRM23, transformed data (b); WPG24, Winnipeg 2024 untransformed data (c); WPG24, transformed data (d); CRM24, Carman 2024 untransformed data (e); CRM24 transformed data (f); trait data pooled over all site-years, untransformed (g); pooled transformed data (h).	44
Figure 3.4.2 Histograms comparing the phenotypic distributions of the association mapping population to each of the seven <i>Pgt</i> races. Ratings were converted from the Stakman scale to a 1-9 linear scale. The means of two lines with varying resistance, Flourish (intermediate) and CDC Ptarmigan (susceptible) are indicated.	46
Figure 3.4.3 Stacked bar plot of the seven <i>Pgt</i> races, demonstrating the frequency (%) of the population that falls into the three categories of susceptible, intermediate, and resistant.	46
Figure 3.4.4 Cross-entropy values obtained from sNMF analysis used to infer population structure within the mapping association panel. Cross-entropy was calculated for values of K 1 to 10, where K represents the number of inferred genetic populations. The minimum cross-entropy value indicates the most likely number of ancestral populations explaining the genetic structure of the population.	48
Figure 3.4.5 Population structure inferred from sNMF analysis at K =2. Each vertical bar represents an individual within the population. The colours indicate the proportion of the ancestry assigned to one of the two genetic subpopulations identified.	49
Figure 3.4.6 Linkage disequilibrium (LD) of the association mapping population plotted as pairwise r^2 against the physical distance in base pairs. The red curve represents the non-linear regression fit using the Hill and Weir model. The dashed horizontal orange line indicates the half-decay of $r^2 = 0.23$ and the blue dashed line indicating the background decay of $r^2 = 0.06$. The corresponding vertical lines indicate the physical distances at which the thresholds are reached. Respectively these at 2.75 and 18.5 Mbp. The half-decay distance was doubled to provide the approximate size of the linkage block, which is 5.5 Mbp.	50
Figure 3.4.7 Manhattan and quantile-quantile (QQ) plots produced from the genome-wide association analysis of the pooled dataset from all site-years. On the Manhattan plots the y-axis indicates the $-\log_{10}(p)$ and the x-axis indicates the chromosomal position of the markers. The dashed line indicates the false discovery rate threshold (FDR) of $-\log_{10}(p) = 3.76$. The QQ plots illustrate the observed $-\log_{10}(p)$ against the expected $-\log_{10}(p)$ for the pooled dataset. A total of 29,766 markers were used. Severity is represented in panels a-b, infection type in c-d, and coefficient of infection in e-f.	54
Figure 3.4.8 Manhattan plots produced from the genome-wide association analysis of the seven <i>Pgt</i> races evaluated for infection type on the association mapping population at the seedling stage. The y-axis	

indicates the $-\log_{10}(p)$ and the x-axis indicates the chromosomal position of the markers. The dashed line indicates the false discovery rate threshold (FDR) of $\log = 4.36$. A total of 29,766 markers were used in the analysis. The races are denoted as MCCFC (a), QTHSF (b), RHTSF (c), RKQSF (d), RTHSF (e), TMRTF (f), and TPMKC (g).59

Figure 3.4.9 Quantile-quantile (QQ) plots produced from the genome-wide association analysis of the seven *Pgt* races evaluated for infection type on the association mapping population at the seedling stage. The QQ plots illustrate the observed $-\log_{10}(p)$ against the expected $-\log_{10}(p)$. A total of 29,766 markers were used. The races are denoted as MCCFC (a), QTHSF (b), RHTSF (c), RKQSF (d), RTHSF (e), TMRTF (f), and TPMKC (g).60

Figure 3.5.1 Box plots illustrating the effect of individual QTL on stem rust severity (%), infection type (IT), and coefficient of infection (COI) in the association mapping population based on the pooled field BLUPs. For each QTL evaluated, genotypes were grouped based on the absence or presence of the identified alleles contributing to resistance.66

Figure 3.5.2 Boxplots showing the effect of the selected QTL combinations on stem rust severity (%), infection type (IT), and coefficient of infection (COI) based on the pooled field BLUPs. Genotypes were grouped according to the presence of combinations of *QSr.umb-1B.1*, *QSr.umb-2A.1*, *QSr.umb-1D*, and *QSr.umb-3D* with sample sizes (n) indicated for each group.69

Figure 4.5.1 Distribution of the phenotypic values for the three field traits, stem rust severity, infection type, and coefficient of infection evaluated in the association mapping population (n = 326). Values represent BLUP estimates calculated from the pooled environmental dataset.....93

Figure 4.6.1 Relationship between computational runtime and prediction accuracy (r) for genomic selection models evaluated for field traits, including severity, infection type (IT), and coefficient of infection (COI). Each point represents the prediction accuracy of a single model for a specific trait. Runtime (hours) is presented on a $\log_{10}$ scale to account for the wide range in computational time among models. Models are distinguished by colour and shape. Abbreviations: GBLUP: Genomic Best Unbiased Linear Predictions; RR: Ridge Regression; LASSO: Least Absolute Shrinkage and Selection Operator; BA/BB/BC: Bayesian methods; RKHS: Reproducing Kernel Hilbert Spaces (RKHS); and RF: Random Forest.95

Figure 4.7.1 Observed versus predicted phenotypes for the BayesB (BB) model under ten-fold cross-validation for severity, infection type (IT), and coefficient of infection (COI) in the genomic selection subset (n = 326). Each point represents an individual genotype, with predicted values obtained from validation folds. Solid lines represent the line of best fit. Pearson's correlation coefficients (r) and coefficients of determination (R^2) are shown for each trait. These plots reflect the predictive ability of the model.96

Figure 4.7.2 Observed phenotypes versus genomic estimated breeding values (GEBVs) predicted by the BayesB (BB) model using the full dataset for severity, infection type (IT), and coefficient of infection (COI) (n = 326). Each point represents an individual genotype. Solid lines represent the line of best fit, and dashed lines indicate the line of equality ($y = x$). Pearson's correlation coefficients (r) and coefficients of determination (R^2) are shown for each trait. These plots represent in-sample model fit and, therefore, show inflated correlations relative to cross-validation results.97

Figure 4.8.1 Phenotypic distributions of seedling infection type responses across the seven *Pgt* races evaluated in the genomic selection subset (n = 326). Histograms represent the frequency of discrete infection type scores (0–9) for each race.98

Figure 4.9.1 Relationship between computational runtime and prediction accuracy (r) for genomic selection models evaluated using seedling resistance data across seven *Pgt* races. Each point represents the prediction accuracy of a single model for an individual race. Runtime (hours) is presented on a $\log_{10}$ scale to account

for the wide range in computational time among models. Models are distinguished by colour and shape. Abbreviations: GBLUP: Genomic Best Unbiased Linear Predictions; RR: Ridge Regression; LASSO: Least Absolute Shrinkage and Selection Operator; BA/BB/BC: Bayesian methods; RKHS: Reproducing Kernel Hilbert Spaces (RKHS); and RF: Random Forest. 101

ABSTRACT

Stem rust, caused by *Puccinia graminis* f. sp. *tritici* (*Pgt*), remains a significant threat to wheat (*Triticum aestivum* L.) production worldwide. Although the disease has been largely controlled in western Canada through the deployment of resistant cultivars and the eradication of the alternate host *Berberis vulgaris*, the emergence of new virulent races globally highlights the need for continued improvement of genetic resistance. The objectives of this study were to (i) identify genomic regions associated with stem rust resistance using genome-wide association studies (GWAS), and (ii) evaluate the potential of genomic selection (GS) for predicting stem rust resistance in winter wheat.

An association mapping panel of 440 winter wheat lines was evaluated for stem rust resistance in both artificially inoculated field trials and greenhouse seedling assays. Field trials were conducted across multiple environments using a mixture of seven *Pgt* races, and infection type, severity, and coefficient of infection were recorded. Seedling assays were conducted using the individual *Pgt* races. Genotyping was performed using high-density SNP arrays, and GWAS analysis identified multiple quantitative trait loci (QTL) associated with resistance across several chromosomes, including regions corresponding to known *Sr* genes. The main QTL identified reside on chromosomes 1D, 1B, 2A, 2D, and 3D. The position of these QTL suggests the presence of *Sr6*, *Sr24*, *Sr31*, *Sr33*, and *Sr38* in the population.

Genomic selection models, including parametric, semi-parametric, and machine learning approaches, were evaluated using cross-validation. Prediction accuracies for adult plant resistance traits ranged from $r = 0.74$ – 0.81 , while seedling resistance showed more variable accuracies across the races ($r = 0.53$ – 0.77). These results reflect differences in the genetic basis of resistance of the

population, with field resistance exhibiting a more polygenic basis compared to race-specific seedling resistance.

Overall, this study demonstrates that stem rust resistance in western Canadian winter wheat is controlled by multiple loci and that genomic selection may be an effective tool for predicting resistance. The integration of GWAS and GS provides a comprehensive framework for improving disease resistance and supports the implementation of GS in winter wheat breeding programs.

FOREWORD

This thesis has been written in the paper style outlined by the Department of Plant Science and Faculty of Graduate and Postdoctoral Studies at the University of Manitoba. The thesis includes a general introduction, a literature review, and two manuscripts. Each manuscript includes an abstract, introduction, material and methods, results, discussion, and a conclusion. Following the manuscripts are a general discussion, the bibliography, and supplementary information provided in the appendix.

Chapter 1: GENERAL INTRODUCTION

Wheat (*Triticum aestivum* L.) is a staple crop around the world and approximately 20% of the world's food production relies on the wheat that is harvested each year (Enghiad et al. 2017; Figueroa et al. 2018). Canada is a leading producer and exporter of wheat. Canada produced 40.0 million tonnes of wheat in 2025, which was up from the 35.0 million tonnes produced in 2024 (Statistics Canada 2024, 2025). The majority of wheat produced in Canada is spring type, with a small proportion being winter wheat. Of the total wheat produced in Canada in 2025, only 3.6 million tonnes (9%) was from winter wheat production (Statistics Canada 2025).

Winter wheat differs from spring wheat in that it requires a vernalization period. Vernalization is prolonged exposure to cold temperatures and is a genetic requirement for winter wheat plants to transition from vegetative to reproductive growth (Fowler and Gusta 1979; Thomas et al. 1988; Mahfoozi et al. 2001; McCallum and DePauw 2008). Canada Western Red Winter (CWRW) is a hard red winter wheat class for western Canada defined by the Canadian Grain Commission. CWRW is the main class of winter wheat grown in western Canada, and the only milling class of winter wheat in western Canada. Hard winter wheat is used in the production of bread and noodles, while soft winter is used for pastries, cereals, cakes, and crackers (Government of Canada 2019; Statistics Canada 2023).

The fungal pathogen *Puccinia graminis* Pers.:Pers. f. sp. *tritici* Eriks. & E. Henn. (*Pgt*) is responsible for causing stem rust on wheat. Hosts of *Pgt* include cereal crops such as wheat, durum (*Triticum turgidum* L.), barley (*Hordeum vulgare* L.) and triticale ($\times$ *Triticosecale*). The alternate host required for sexual reproduction is *Berberis vulgaris* (common barberry) (Peterson 1958; Roelfs et al. 1992). Historically, stem rust has been one of the most devastating diseases affecting wheat production (Roelfs 1989).

Outbreaks of stem rust in Canada primarily occur in the southern region of Manitoba and into eastern Saskatchewan. Inoculum is typically airborne and comes from the southern United States and northern Mexico, where it overwinters on winter crops and travels northward into Canada as the season progresses (Peturson 1958; Fetch et al. 2011a; Brar et al. 2019; Aboukhaddour et al. 2020). Stem rust can be controlled using host resistance (incorporation of genetic resistance into cultivars), cultural practices, and the application of fungicides. Each of these methods pose varying costs to producers (Fetch et al. 2011a). To date, more than 60 genes associated with resistance to stem rust have been identified. These can be broken down into two categories: (1) seedling (all-stage) resistance genes, and (2) adult plant resistance genes (APR). Seedling genes are typically race-specific and confer resistance from the seedling stage into adulthood for the plants. APR genes only confer resistance at the adult plant stage and are divided into categories of race-specific and non-race-specific (Person et al. 1962; Leonard and Szabo 2005; Ellis et al. 2014; Periyannan et al. 2017; Karelov et al. 2022). Most of the *Sr* genes that have been identified fall under the umbrella of seedling resistance genes. These genes have been identified to originate both within bread wheat and in related species of wheat. The introgression of genes from related species has played a role in diversifying resistance (Karelov et al. 2022).

Genomic selection (GS) utilizes genome-wide markers and phenotypic data from a population to predict the genomic estimated breeding values (GEBV) of different genotypes derived from the same breeding gene pool (Meuwissen et al. 2001; Heffner et al. 2009; Crossa et al. 2011). There are a number of different models that can be utilized to conduct GS; each model possesses its own distinct advantages and limitations. The implementation of GS can potentially allow for the selection of superior individuals in a faster and more efficient manner compared to

the use of traditional phenotyping in breeding programs. This is particularly advantageous in winter wheat, where traditional breeding approaches are limited to a single generation per year.

The overall objective of this study was to utilize genome-wide association studies (GWAS) to identify quantitative trait loci (QTL) that are associated with resistance to stem rust within the association mapping population, that is primarily made up of Canadian Western Red Winter (CWRW) wheat. The identified QTL can then be used to identify candidate genes that may reside in the population and find important loci that influence resistance. Following the use of GWAS the goal was to evaluate the use of genomic selection (GS) models to determine their suitability to be implemented into the winter wheat breeding program at the University of Manitoba, aiding in selection of resistant lines within the breeding program.

Chapter 2: LITERATURE REVIEW

2.1 Wheat

2.1.1 Evolution of Wheat

Domesticated wheat, known as bread wheat (*Triticum aestivum* L.), arose through hybridization events taking place between *Aegilops* and *Triticum* species (Dubcovsky & Dvorak 2007). Bread wheat is an allohexaploid species ($2n = 6x = 42$), consisting of three subgenomes. The subgenomes (A, B, D) of wheat originated in diploid species independent of one another and came together through a series of hybridization events. The first event is believed to have occurred between *Triticum urartu* ($2n = 2x = 14$, AA) and an unknown but closely related relative of *Aegilops speltoides* ($2n = 2x = 14$, BB), to give rise to the tetraploid emmer wheat known as *Triticum turgidum* ($2n = 4x = 28$, AABB) (Marcussen et al. 2014; Petersen et al. 2006; Salamini et al. 2002). The tetraploid *T. turgidum* (AABB) eventually hybridized with the diploid species *Aegilops tauschii* Coss. ($2n = 2x = 14$, DD), leading to the development of what is known globally as bread wheat (*T. aestivum*, $2n = 6x = 42$, AABBDD) (Marcussen et al. 2014).

2.1.2 Wheat Production in Canada

Wheat is one of the most important crops globally, making it one of the leading food sources. Close to 20% of the world's food production is reliant on the wheat crops that are harvested each year (Enghiad et al. 2017; Figueroa et al. 2018). More than 75% of the global production of wheat is imported and consumed in developing nations. It is expected that the world's population will exceed more than nine billion people by 2050, indicating that cereal production needs to substantially increase to meet demands (Enghiad et al. 2017; Figueroa et al. 2018). Many factors regularly challenge wheat production, including the reduction in the availability of farmland, changing climate, and abiotic and biotic stresses that vary depending on the global region. As

breeders have worked to establish high performing varieties, some of the genetic diversity present in wheat has been lost. This has led to pathogens emerging that put wheat crops at risk (Singh et al. 2016; Figueroa et al. 2018; Aboukhaddour et al. 2020).

Three main types of wheat are produced in western Canada: spring wheat (*T. aestivum*), winter wheat (*T. aestivum*) and durum wheat (*T. turgidum*), with spring wheat being the most widely grown (McCallum and DePauw 2008). In 2024, Canada produced 35.0 million tonnes of wheat with spring wheat accounting for more than half of this at 26.1 million tonnes produced. Manitoba produced 5.5 million tonnes of wheat during the 2024 growing season (Statistics Canada 2024). During the 2025 growing season, Canada produced 40.0 million tonnes of wheat, which was an increase from both 2023 and 2024. Manitoba was responsible for 5.9 million tonnes of the total wheat produced in 2025 (Statistics Canada 2025).

2.1.3 Winter Wheat

Winter wheat (*T. aestivum*) differs from the more commonly grown spring wheat in that it has a genetic requirement for prolonged cold exposure, known as vernalization. Vernalization is required for winter wheat to go from its vegetative state, to be reproductive. Varieties grown in western Canada will have cold tolerance, that allow them to survive the harsh winters. Winter wheat has been grown in Canada since the early 1600s (Fowler and Gusta 1979; Thomas et al. 1988; Mahfoozi et al. 2001; McCallum and DePauw 2008). In Canada, there are two main types of winter wheat grown, hard and soft. Hard winter wheat is used in the production of bread and noodles, while soft winter is used for pastries, cereals, cakes, and crackers (Statistics Canada 2022). These can be further broken down into further classes such as Canada Western Red Winter (CWRW), Canada Eastern Hard Red Winter (CEHRW), Canada Eastern Soft Red Winter (CESRW), and Canada Eastern White Winter (CEWW) (Government of Canada 2019). Of the four classes of

winter wheat grown in Canada, only CWRW is grown in western Canada. In Canada the record high production for winter wheat occurred in 2008 where 4.8 million tonnes were produced. In 2022, winter wheat was ranked as the tenth largest field crop to be produced in Canada (Statistics Canada 2023). Winter wheat production in western Canada has been limited due to varying factors, this includes harsh winters and limited snow cover resulting in winterkill of the crop and the economic benefits of planting other crops such as spring wheat (McCallum and DePauw 2008).

2.2 Stem Rust of Wheat

The fungal pathogen *Puccinia graminis* Pers.: Pers. f. sp. Eriks. & E. Henn. *tritici* (*Pgt*) is the causal agent of stem rust on wheat. Hosts include common wheat, barley (*Hordeum vulgare* L.), and triticale ($\times$ *Triticosecale*). *Pgt* is a heteroecious fungus that requires two separate and unrelated hosts to complete its full life cycle, going through both sexual and asexual reproduction (Leonard and Szabo 2005). The alternate host required for sexual reproduction is *Berberis vulgaris* L. (common barberry), *Berberis canadensis*, and some species of *Mohania* (Peterson 1958; Roelfs et al. 1992).

Historically, stem rust has been one of the most devastating diseases to wheat production (Roelfs 1989). Within a matter of weeks, a healthy crop could be decimated if infected with *Pgt*, resulting in substantial yield losses due to the presence of shrivelled grains and broken stems (Roelfs et al. 1992). Stem rust can cause harm to the plants in a variety of different ways. Following infection by spores, the pathogen produces pustules (uredinia) that can cover many areas of tissue on a healthy wheat plant, this includes the leaves, leaf sheaths, peduncle, glumes, awns and the stems. If a large portion of the plant tissue is covered with pustules, this reduces the area available for photosynthesis to occur. (Roelfs and Bushnell 1984; Roelfs et al. 1992; Kolmer et al. 2009). Each pustules ruptures the epidermis, leading to significant water loss from the plant. In addition,

the pathogen utilizes nutrients and diverts photosynthate from surrounding host tissue that would normally contribute to grain filling, which induces stress in the plant. This results in reduced tillering, smaller head size, and the production of shrivelled grains. Severe infection on the stems can lead to lodging. Lodging results in yield loss, with machinery being unable to collect heads at ground level (Roelfs and Bushnell 1984; Roelfs et al. 1992; Leonard and Szabo 2005).

2.2.1 Life cycle of *Puccinia graminis* f. sp. *tritici*

The life cycle of *Pgt* is macrocyclic with five distinct spore types. The sexual cycle of *Pgt* requires two unrelated hosts as the pathogen is heteroecious, common barberry is required for sexual reproduction and wheat for asexual reproduction (Roelfs et al. 1992; Leonard and Szabo 2005; Kolmer et al. 2009). For the sexual life cycle, *Pgt* will overwinter as teliospores, within which karyogamy occurs to produce a diploid (2N) nucleus. The teliospores typically require cooler temperatures to germinate. Once germinated, meiosis occurs in the basidium formed from the teliospores. This results in the formation of basidiospores (Roelfs and Bushnell 1984; Roelfs et al. 1992; Kolmer et al. 2009). The basidiospores are haploid (N) and will travel via wind to the alternate host (Roelfs and Bushnell 1984; Leonard and Szabo 2005; Kolmer et al. 2009). After the basidiospores infect and invade the host tissue, they produce pycnia on the upper side of the leaves which are responsible for generating pycniospores. The pycnia will be one of two mating types (+ or -). The pycniospores are transferred to neighbouring host plants through moisture in the form of rain or dew, or through insects (Kolmer et al. 2009). The pycniospores will undergo plasmogamy (fertilization) with receptive hyphae of the appropriate mating type counterpart. Through plasmogamy, a dikaryotic (N + N) cell containing two genetically distinct haploid nuclei is formed (Roelfs et al. 1992; Kolmer et al. 2009). The fertilized structure, known as aecium, will develop on the lower side of the leaf that the pathogen has colonized. It is here where the aeciospores are

produced. Aeciospores are dikaryotic and are the result of genetic recombination that occurred earlier on in the life cycle (i.e., basidiospores), allowing for variation amongst the aeciospores (Roelfs et al. 1992). Aeciospores are produced in large amounts; they are released into the air where they can travel long distances to find a suitable host (wheat). Once the aeciospores come into contact with the appropriate host, they will germinate and invade the host through their stomata (Kolmer et al. 2009). As a result of the aeciospores germinating, they form a pustule that is known as a uredinium, which houses urediniospores ($N + N$). The uredinium and urediniospores are responsible for the asexual life cycle of *Pgt*. Urediniospores can continually infect the same host, allowing for epidemics to take place (Roelfs and Bushnell 1984; Roelfs et al. 1992; Leonard and Szabo 2005; Kolmer et al. 2009). Towards the end of the growing season, the uredinia can develop into telia and produce teliospores. The teliospores, which are resistant to drying and the cold, will overwinter on host plant residues, this cycle then continues in the next growing season (Kolmer et al. 2009). In regions where barberry is absent, the sexual life cycle can be bypassed as the pathogen can overwinter in the uredinial stage on fall-sown crops or volunteer plants, assuming that the weather conditions are not too harsh (Leonard and Szabo 2005).

2.2.2 Epidemiology

In Canada, stem rust primarily occurs in the region of southern Manitoba and eastern Saskatchewan, this region is commonly referred to as the “Rust zone” (Aboukhaddour et al. 2020). Below this region, is a stretch of land through the Great Plains of the United States, into Southern Texas and Northern Mexico known as the “Puccinia Pathway” (Eversmeyer and Kramer 2000; Aboukhaddour et al. 2020). Historically, urediniospores of *Pgt* will over winter in northern Mexico and southern Texas. As the season progresses, the urediniospores will move north with the winds, infecting winter wheats in the south and the spring wheats in the north eventually infecting

susceptible crops in southern Manitoba and eastern Saskatchewan (Peturson 1958; Eversmeyer and Kramer 2000; Kolmer 2005; Aboukhaddour et al. 2020).

2.2.3 Resistance

The gene-for-gene concept for host-pathogen interactions was first proven by Flor, demonstrating this using *Melampsora lini* (Ehrenb.) Lév. (flax rust) and *Linum usitatissimum* L. (flax) (Flor 1956, 1971). In gene-for-gene systems, genes in the host that convey resistance (R) will have corresponding genes in the pathogen for avirulence (Av) (Flor 1956, 1971; Person et al. 1962; Kolmer and Dyck 1994; Leonard and Szabo 2005). When a host is homozygous or heterozygous for a dominant resistance gene (RR or Rr) and the pathogen is homozygous or heterozygous for avirulence (AvAv or Avav), an incompatible reaction occurs (host resistance) (Kolmer and Dyck 1994). If the pathogen has the corresponding virulence genes (avav) regardless of the presence or lack of presence of resistance genes in the host, a compatible reaction will occur (host susceptibility) (Kolmer and Dyck 1994; Van Der Biezen and Jones 1998). Resistance genes and avirulence genes are generally dominant, but exceptions occur.

Genes that convey resistance to *Pgt* can be divided into two categories: non-race-specific and race-specific resistance (Ellis et al. 2014). Non-race-specific resistance is where the host has resistance to all races of the pathogen. This form of resistance is typically quantitative. Varying phenotypes for resistance can be observed (Periyannan et al. 2017). Non-race-specific resistance is typically associated with plants that are developmentally mature, often being referred to as adult plant resistance (APR) (Periyannan et al. 2017). However, APR genes can be race-specific or non-race-specific. Many APR genes have been effective at providing long-term protection against *Pgt* due to being durable and having not broken down; one of the most widely known APR genes for this is *Sr2* (Ellis et al. 2014; Periyannan et al. 2017; Karelov et al. 2022).

Race-specific resistance genes (R genes) convey resistance to certain races of *Pgt*, whereas they have no effect against others. The level of the resistance provided from these genes to *Pgt* can vary depending on the combination of the resistance and avirulence genes present (Leonard and Szabo 2005; Ellis et al. 2014; Periyannan et al. 2017). R genes can function and provide protection from the seedling stage into adult growth stages; these genes are typically race-specific (Ellis et al. 2014; Periyannan et al. 2017). When breeding for resistance to *Pgt* was occurring in the early 20th century, many of the genes utilized were R genes. As new varieties with R genes were released, they were often overcome within a few years as new and more virulent races of *Pgt* arose (Ellis et al. 2014).

Several resistance genes for *Pgt* have been identified some of which are thought to have originated in wheat, whereas others have been introgressed from other species. Genes that originated in wheat include *Sr6*, *Sr9*, *Sr15*, *Sr16*, *Sr19*, *Sr20*, *Sr23*, *Sr29*, *Sr30*, *Sr41*, *Sr42*, *Sr48*, *Sr49*, *Sr54*, *Sr57*, *SrCad*, and *SrTmp* (Karelov et al. 2022). The introgression of genes from other species into wheat has allowed for diversity and an increase in the number of resistance genes to *Pgt*. Introgressed resistance genes include, but are not limited to, *Sr2*, *Sr24*, *Sr31*, *Sr38*, *Sr45*, and *Sr52* (Karelov et al. 2022). The majority of the *Sr* genes can be categorized as seedling resistance genes, while a smaller portion of genes can be categorized as APR genes that are non-race-specific (Karelov et al. 2022)

Many of the *Sr* genes that have been identified were found to be present in spring cultivars, the presence of these genes is less understood in winter wheat. A number of *Sr* genes have been identified to be present in the winter wheat germplasm found in North America and Europe; these genes include *Sr2*, *Sr6*, *Sr24*, *Sr38*, and *Sr57* (Mourad et al. 2018; Laroche et al. 2019; Miedaner et al. 2024).

2.2.4 Differential Sets, Nomenclature and Rating Scales

Studies on the variation of virulence in *Pgt* populations found in Canada have been an annual occurrence since 1919, allowing for the detection of new races that could pose a threat to wheat production in Canada (Fetch et al. 2011b; Menzies et al. 2024). A key for the identification of designated physiological races of *Pgt* and nomenclature was first published in 1922, here a set of twelve differential hosts were utilized (Roelfs 1988; Stakman and Levine 1992). Physiological races are broken down into host reaction classes, on a scale of 0 to 4. The resistant infection types are denoted as “0”, “;”, “1”, and “2”. Susceptible infection types are rated as “3” and “4”. Each of these infection types can have the designation of plus or minus (+/-) which are indicative that the uredinia size is either greater than or less than normal. A mesothetic reaction, abbreviated as “X”, can also occur in which some uredinia on a leaf are consistent with a resistant response, while other uredinia on the same leaf are consistent with a susceptible resistance when inoculated with a single *Pgt* isolate (Roelfs and Bushnell 1984; Roelfs 1988; Stakman and Levine 1992). A reaction of low (L) or high (H) is assigned to each differential line. The lines are organized into a hexadecimal system where the combination of a L or H reaction is then assigned to a specific letter, resulting in a five letter code to identify races of *Pgt* (Fetch and Jin 2007; Jin et al. 2008; Fetch et al. 2015). Today, five sets of four wheat single-gene differential lines for *Pgt* resistance have been utilized - *Sr5*, *Sr21*, *Sr9e*, *Sr7b*; *Sr11*, *Sr6*, *Sr8a*, *Sr9g*; *Sr36*, *Sr9b*, *Sr30*, *Sr17*; *Sr9a*, *Sr9d*, *Sr10*, *SrTmp*; *Sr24*, *Sr31*, *Sr38*, *SrMcN* (Jin et al. 2008; Fetch et al. 2018).

2.2.5 Epidemics

Damage from rust has been recorded as early as 1891 in western Canada, with smaller epidemics occurring frequently up until 1916. Increased losses were observed in 1919 and 1921, with severe losses reported in 1923 (Brar et al. 2019). This led to the development of the Dominion Rust

Research Laboratory in Winnipeg, Manitoba in 1925–26 (Brar et al. 2019). Varieties resistant to *Pgt* were first introduced to western Canada in 1936, from 1939 to 1951 little to no losses due to stem rust were experienced as a result of growing resistant varieties (Peturson 1958). An increase in race 15B during 1952 led to *Pgt* overcoming the resistant varieties. With a virulent race and favourable weather conditions, this resulted in crop losses throughout Manitoba and Saskatchewan. Epidemics then followed from 1953 – 1955 leading to great losses, with 1954 experiencing the greatest loss to wheat production. During this time it is estimated that more than two billion dollars was lost (Peturson 1958; Aboukhaddour et al. 2020). In 1956, new genetic resistance was deployed into commercial varieties, largely controlling stem rust outbreaks (Brar et al. 2019; Aboukhaddour et al. 2020). Since deploying genetic resistance into cultivars and the eradication the alternate host barberry, stem rust has largely been controlled in North America (Kolmer 2005).

In the late 1990's, greater than normal stem rust infections were observed at the Kalengyere Research Station in Uganda. Much of the material grown in this nursery was known to be resistant to stem rust, largely due to the presence of *Sr31* in the material (Pretorius et al. 2000; Singh et al. 2011). Urediniospores were collected from the nursery and were screened in a greenhouse on a set of wheat differentials, phenotyping for virulence/avirulence (Pretorius et al. 2000; Singh et al. 2011) This race became known as Ug99 because it originated in Uganda in 1999. Based upon the results from the phenotyping of the differential sets, Ug99 was designated as TTKSK (Jin et al. 2007; Singh et al. 2011). Ug99 is virulent to many resistance genes in wheat (some originating from wheat, others introgressed into wheat) including, but not limited to, *Sr10*, *Sr16*, *Sr24*, *Sr31*, *Sr36*, *Sr38*, and *SrMcN* (Singh et al. 2008, 2011). Since first being identified, Ug99 has spread and been confirmed in other countries. These include Uganda, Kenya, Ethiopia, Sudan, Yemen, and

Iran. Variants of Ug99 have also been identified (Singh et al. 2011). The identification of Ug99 and its variants led to the development of the Borlaug Global Rust Initiative with the goal of coordinating and advocating for global efforts to reduce the threat of Ug99 (Singh et al., 2011).

Outside of the emergence of Ug99, stem rust has been relatively well controlled in most parts of the world. In recent years more outbreaks have been occurring globally. During the 2013–2014 growing season in Ethiopia, 27 of their wheat growing districts were found to be severely affected by stem rust (Olivera et al. 2015). Digalu, a spring wheat variety, was the most widely grown cultivar during this timeframe. It was known to be resistant to stripe rust (*Puccinia striiformis*) and to races of stem rust in the Ug99 group. In addition to resistance, Digalu had good yield potential. More than 100,000 hectares of wheat were impacted by the stem rust epidemic with 20–40% of those fields having been Digalu. Yield losses of up to 92% were reported for some growing districts that had planted Digalu (Olivera et al. 2015). The race responsible for this outbreak was identified as TKTTF (Olivera et al. 2015, 2017).

In Germany during the 2013 growing season, an outbreak of stem rust occurred for the first time in decades. The symptoms were first reported on winter wheat. Two weeks later symptoms were observed on spring wheat (Olivera et al. 2017; Lewis et al. 2018). Six races were identified as being responsible for the outbreak, none belonging to the Ug99 race group (Olivera et al. 2017). During this time, the first occurrence of stem rust in the United Kingdom (UK) was observed for the first time in more than 60 years. It was reported that stem rust had been identified on an individual plant in the UK. The race responsible for infecting this individual plant was closely related to the one responsible for the epidemic in Ethiopia (Lewis et al. 2018).

A few years after the outbreak in Germany and the first confirmed case of stem rust in the UK in over 60 years, a large outbreak occurred in Sicily, Italy that affected both bread and durum

wheat (Patpour et al. 2022). The outbreak led to the loss of thousands of hectares of wheat in Sicily and to the identification of the stem rust race TTTTF (Bhattacharya 2017). More recently the regions of western Siberia and northern Kazakhstan have been majorly impacted by severe epidemics of stem rust occurring between the years of 2015 to 2019, causing devastating losses within these regions (Olivera et al. 2022a).

2.2.6 Pathogen Control

Over time, a variety of methods have been utilized to control stem rust infections on wheat. These include the eradication of common barberry, early seeding dates, the application of fungicides, breeding for resistance and implementing crop rotations (Forsyth and Peturson 1958; Roelfs and Bushnell 1984; Roelfs 1989; Loughman et al. 2005; McCallum et al. 2007). Dating back to the 1660s, the eradication of common barberry, the alternate host for *Pgt*, was a major concern with laws against growing barberry arising in France (Roelfs and Bushnell 1984). In North America through the late 1700s there were laws implemented against growing barberry in certain states, these laws were not highly enforced once a decline in wheat production occurred (Roelfs 1982). It was not until 1865 when Heinrich Anton de Bary demonstrated the connection between wheat and *Pgt* (Roelfs and Bushnell 1984). In 1916 a severe epidemic plagued the United States, resulting in laws being passed and enforced over the next three years in major wheat growing states (Roelfs 1982). Elimination of barberry was successful in controlling stem rust by allowing for a delay in the occurrence of the disease; reducing the opportunities for sexual reproduction to occur for the pathogen, resulting in a decreased number of pathogen races; reducing the inoculum levels present in the environment and stabilizing the pathogenic races allowing for easier development of resistant cultivars (Roelfs 1982; Roelfs and Bushnell 1984).

The most widely used method to control stem rust to date has been to breed for resistance in cultivars that are being grown; breeding for resistance became more of a commonly used tool in the early 1900s (Roelfs and Bushnell 1984; McCallum et al. 2007). In Canada breeding for resistance to stem rust saw its first attempts at the University of Saskatchewan in 1915, followed years later in 1925 with the development of the Dominion Rust Research Laboratory in Winnipeg (Martens and Dyck 1989). Thatcher was one of the first cultivars released that was resistant to stem rust and was developed in Minnesota. Following its release, Thatcher was one of the primary cultivars grown in Canada up until the mid-1950s when severe epidemics hit (Peterson 1958; Martens and Dyck 1989; Roelfs et al. 1992; McCallum et al. 2007). The release of the cultivar Selkirk in 1954, in conjunction with the barberry eradication programs in North America, brought about durable resistance to farmers. Through the continued release of resistant cultivars and stacking of effective resistance genes in these cultivars, durable resistance has lasted more than 50 years in Canada and been the primary control for stem rust (Roelfs et al. 1992; McCallum et al. 2007; Fetch et al. 2011a)

Barberry eradication and the implementation of resistant cultivars have been the two main drivers toward continued stem rust control in North America. There are additional practices that can be utilized by producers, including application of fungicides to wheat crops (McCallum et al. 2007; Fetch et al. 2011a). Many producers spray their crops to protect against fusarium head blight (FHB) which has caused severe epidemics in certain regions of Canada, such as Manitoba. The application of pesticides used to control FHB also provide protection against stem rust (McCallum et al. 2007; Fetch et al. 2011a). However, the use of fungicides is an added expense for producers and overuse may result in the development of fungicide resistant races of the pathogen (Roelfs and Bushnell 1984; Roelfs et al. 1992; McCallum et al. 2007; Fetch et al. 2011a). Early seeding of

crops in Canada may aid in mitigating infection from spores that have travelled from the United States on the ‘Puccinia Pathway’ as the inoculum will be present at lower levels earlier in the growing season (Roelfs and Bushnell 1984; McCallum et al. 2007; Fetch et al. 2011a). Additional means of control include the removal of volunteer wheat plants from fields, and diversifying the types of crops being grown between fields and on farms (Roelfs and Bushnell 1984).

2.3 Marker Assisted Selection

Marker-assisted selection (MAS) is a form of biotechnology that utilizes DNA sequence variations linked to desirable traits to assist in plant breeding, allowing for increased proficiency and efficiency (Collard and Mackill 2007; Pandurangan et al. 2022). The overarching goal of plant breeding is to develop and select plants with superior combinations of desirable traits. Part of this selection process largely relies on traditional phenotyping methods in greenhouse and/or replicated field trials (Collard and Mackill 2007; Song et al. 2023). Phenotypic selection is often a costly undertaking, timewise and financially. Breeding lines are often not able to be tested until later generations, when the lines are homozygous. As a result, several generations are needed before accurate selection can occur, contributing to the lengthy breeding cycle. Consequently, it can take ten or more years to develop a new variety (Collard and Mackill 2007; Pandurangan et al. 2022).

As molecular marker technology has progressed through the years, DNA markers have become widely used in plant breeding. DNA markers are utilized to detect the presence or absence of genes and their allelic variants, increasing the efficiency of plant breeding. This technology is known as MAS (Collard and Mackill 2007). There are many forms of molecular markers that can be utilized for MAS. These include hybridization-based markers such as restriction fragment length polymorphisms (RFLPs), polymerase chain reaction (PCR) based markers of which there are many forms of markers such as simple sequence repeats (SSRs), sequence-tagged sites (STS)

and amplified fragment length polymorphisms (AFLPs) among others. Finally there are DNA sequence based markers like single nucleotide polymorphisms (SNP) (Gupta et al. 1999; Collard et al. 2005; Song et al. 2023). Among the markers mentioned above, SNPs are one of the most common forms of DNA polymorphisms found in plants and animals. SNPs are widely used in plant breeding programs and are considered to be third-generation molecular markers (Collard et al. 2005; Khan et al. 2014; Song et al. 2023). Advances in DNA technology has allowed for the development of SNP-based technology for genotyping, offering high throughput results and enhancing molecular based applications (Langridge et al. 2001; Song et al. 2023). SNP arrays have been widely used due to their cost effectiveness and for being high throughput (Ganal et al. 2012; Song et al. 2023; Burridge et al. 2024).

Depending on the goals of the breeding program, MAS can serve several purposes, these include the screening of breeding material to identify traits of interest, pyramiding of genes, marker-assisted introgression (MAI), and marker-assisted back-cross (MAB) (Collard and Mackill 2007; Hospital 2009). Five major considerations to take into account, when utilizing MAS, are assay reliability, quality and quantity of DNA required, methodology being used, the level of polymorphism at the marker locus, and assay cost (Collard and Mackill 2007; Hasan et al. 2021). Markers should be sufficiently polymorphic to discriminate between genotypes within the breeding material. For markers to be considered reliable they should be within 5 centi-Morgans (cM) of the target loci. Depending on the marker technique being utilized, the amount of DNA required can vary and the quality of the DNA can impact outcome. When determining methodology, time constraints and the simplicity of the technique involved are key factors taken into consideration. The marker should be able to be selective between the genotypes utilized within the breeding material. Finally, the DNA markers being utilized should be affordable due to the

large number of lines a breeding program will require to genotype (Collard and Mackill 2007; Nadeem et al. 2018; Hasan et al. 2021).

MAS can improve the efficiency of plant breeding by allowing for selection of individual plants with target traits prior to phenotyping, pyramiding of R genes, selection of recessive genes in earlier generations without requiring extensive phenotyping, and cost effective selection of genes in breeding programs compared to traditional phenotyping (Liu et al. 2007; Collard and Mackill 2007; Hospital 2009; Song et al. 2023).

The limitations of MAS include requiring polymorphisms within the germplasm (parents) used to develop the breeding lines (Liu et al. 2007). Traditional phenotypic screening will evaluate lines for resistance against multiple diseases, while to utilize MAS, multiple markers for resistance will need to be identified through genetic mapping studies in order to successfully ensure resistance against multiple diseases (Liu et al. 2007; Heffner et al. 2009).

2.4 Quantitative Trait Loci (QTL) Analysis

QTL analysis is a method of identifying associations often between polygenic traits that are quantitative and alleles that are segregating within a population. This can be broken down into identifying a specific traits association with DNA markers and then mapping the markers to their region within the genome (Geldermann 1975; Kearsey and Farquhar 1998; Collard et al. 2005; Cavanagh et al. 2008). The closer that a marker is to a QTL, the more likely it is that they will be inherited together and the lower the chance of recombination occurring between the QTL and the marker (Kearsey and Farquhar 1998; Collard et al. 2005). If the marker is not tightly linked to the QTL or they are unlinked, then independent segregation will occur (Collard et al. 2005). The populations used for QTL analysis typically consist of bi-parental crosses where each parent will have a contrasting phenotype for the trait of interest. These populations may be developed from

doubled haploids, recombinant inbred lines (RIL), F₂ and back-cross populations, or near-isogenic lines (Tanksley and Nelson 1996; Mohan et al. 1997; Collard et al. 2005; Dhingani et al. 2015). Each type of population can have varying advantages and disadvantages, such as the time required to compose the population. Development of a RIL population will take much longer compared to that of a F₂ population (Kearsey and Farquhar 1998; Collard et al. 2005; Cavanagh et al. 2008; Dhingani et al. 2015). The most commonly used methods for the detection of QTL for mapping are: single marker regression (simplest method), simple interval mapping (SIM), and composite interval mapping (CIM) (Collard et al. 2005). Single marker analysis detects QTL associated with individual markers, SIM utilizes linkage maps and evaluates the intervals between linked markers along the chromosomes, and CIM is an extension of SIM that includes additional markers (Collard et al. 2005). One of the disadvantages of QTL mapping is limited mapping resolution due to the amount of recombination occurring in the development of the population (Korte and Farlow 2013). Association mapping is a means to increase this resolution, though the trade off can be its ability to detect QTL may not be as effective as QTL mapping (Yu et al. 2006; Cavanagh et al. 2008; Korte and Farlow 2013; Tibbs Cortes et al. 2021).

2.5 Genome-Wide Association Studies

Genome-wide association study (GWAS) is a form of association mapping that is utilized to identify marker-trait associations (MTA) in populations that have a large amount of genetic diversity. This practice may be utilized to confirm the association of important loci that have been identified through other means and to detect associations that may not have been previously identified (Tibbs Cortes et al. 2021). The basic concept of GWAS is a trait of interest is evaluated in the population being examined, the population is genotyped, and through statistical methods the markers that are significantly associated with the phenotype of interest are identified (Korte and

Farlow 2013; Tibbs Cortes et al. 2021; Soto-Cerda et al. 2022). GWAS has been implemented in both human genetics and for agricultural purposes when examining trait associations within plants and animals, including crops such as wheat, rice, and maize (Korte and Farlow 2013; Hasan et al. 2021). GWAS relies on linkage disequilibrium (LD), which is the non-random association of alleles throughout the genome (Gaut and Long 2003; Sorrells and Yu 2009; Tibbs Cortes et al. 2021). SNPs or other marks identified may reside near the loci of interest, these can be in high LD with one another, demonstrating their association with the phenotype of interest (Pritchard et al. 2000b; Tibbs Cortes et al. 2021). One of the requirements for GWAS to be successful is for the marker density to be higher than the LD, this is influenced by the population being utilized and the amount of the recombination events that have taken place within the population (Brachi et al. 2011).

In addition to LD being a major component for association mapping methodologies, population structure is an equally important component that cannot be overlooked. A weak population structure is ideal, as it indicates limited genetic subdivision within the population, which can help in the correction of false positives and provide the greatest statistical power. If numerous sub-populations are present (strong population structure), then some individuals will be more closely related than others and this increases the risk of false-positives occurring (Pritchard et al. 2000b; Sorrells and Yu 2009; Brachi et al. 2011; Soto-Cerda et al. 2022). Population structure is corrected for within a mapping population to reduce the likelihood of statistical errors occurring, particularly for use of populations developed in breeding programs where the relatedness among individuals can be highly variable (Sorrells and Yu 2009). To correct for relatedness within the population, there are varying models of GWAS that can be utilized that take into account the population structure and the kinship present. One of the most commonly used models for this is

the mixed linear model (MLM), MLM controls for false positives by having population structure as a fixed effect and using kinship as a random effect (Yu et al. 2006; Korte and Farlow 2013; Tibbs Cortes et al. 2021). Limitations of GWAS include the occurrence of false positives and its inability to detect rare alleles (minor allele frequencies $< 5\text{--}10\%$) affecting the trait (Wang et al. 2005; Korte and Farlow 2013; Tibbs Cortes et al. 2021).

2.6 Genomic Selection

Genomic selection (GS) is a method that utilizes genome-wide markers and phenotypic trait data to calculate genomic estimated breeding values (GEBVs) of progeny, allowing for selection of genetically superior individuals in a breeding population (Meuwissen et al. 2001; Heffner et al. 2009; Crossa et al. 2011). In GS, a training population is tested for traits of interest and genotyped with DNA markers spanning the genome to identify relationships (i.e., association) between the DNA markers and phenotypic data (Heffner et al. 2009; Lorenz et al. 2011). As such, the training population sets the parameters of the GS model to calculate the GEBVs for individuals where only their genotypic information is known. These individuals are referred to as the selection candidates or the testing population (Heffner et al. 2009; Lorenz et al. 2011; Crossa et al. 2011). For GS to be effective, the training population should consist of individuals that are representative of the testing population. Based upon the calculated GEBVs of the testing population, individuals deemed superior for the desired trait of interest can be selected for use within breeding programs (Heffner et al. 2009; Crossa et al. 2017). GS can allow for a reduction in the costs associated with phenotyping for breeding programs, which to date has been primarily used by breeders to select for improved resistance/traits in their breeding programs. There is also the added benefit of potentially being able to reduce the number of years between making initial crosses and selecting new parents among the progeny of the initial crosses (Heffner et al. 2009; Crossa et al. 2017).

There are many different models that can be utilized when conducting GS. Two of the main approaches include the use of parametric and non-parametric models. Parametric models make specific assumptions about the data that is being utilized, such as the distribution of the data in terms of normality (Gianola et al. 2006; De Los Campos et al. 2013; Howard et al. 2014; Crossa et al. 2017). While non-parametric models do not make assumptions regarding the data distribution or require a fixed number of parameters, these models are more flexible compared to parametric models (Gianola et al. 2006; Howard et al. 2014).

Ridge regression best linear unbiased prediction (RR-BLUP) is a parametric method that can estimate the effects of all the markers being utilized simultaneously. The markers are assumed to be random effects and treated as if they have a common variance. In addition the effects of all of the markers are shrunken towards zero, reducing the impact of their estimated effects and helping to prevent overfitting of the model (Whittaker et al. 2000; Meuwissen et al. 2001; Heffner et al. 2009; Lorenz et al. 2011).

Bayesian methods are another form of parametric methods that can be utilized for GS, and they can be broken down into different subsets of methods. Bayesian models differ from RR-BLUP in that each marker has its own variance estimated rather than a common variance being applied. The variance for each marker is assumed to follow a specified prior distribution (Meuwissen et al. 2001; Heffner et al. 2009; Lorenz et al. 2011). Two types of prior distribution are utilized with the Bayesian methods, BayesA and BayesB (Meuwissen et al. 2001).

BayesA determines that each marker effect is derived from a normal distribution and have their own variances, allowing each marker to be shrunken towards zero independently of one another. The variance parameters are derived from an inverted chi square distribution (Meuwissen et al. 2001; Xu 2003; Heffner et al. 2009; Lorenz et al. 2011). BayesB is derived from BayesA;

however, it differs in that it assumes most markers have no effect on the trait, while only a subset of markers contribute to genetic variation (Meuwissen et al. 2001; Heffner et al. 2009; Lorenz et al. 2011).

Nonparametric models do not require a predefined relationship between DNA markers and the response variable, and are capable of modelling complex trait architectures (Lorenz et al. 2011; Howard et al. 2014). Semi-parametric models are available as an alternative to parametric and non-parametric models. These include the method known as reproducing kernel Hilbert spaces (RKHS) regression. This is a method that utilizes a mixed model approach by utilizing aspects of nonparametric models with kernel (mathematical) functions (Gianola et al. 2006; Habier et al. 2007; Lorenz et al. 2011; Howard et al. 2014)

Cross validation (CV) is the method of splitting the working dataset into a training set and a validation set (also referred to as a test set). Typically, the data will be split into five different sets, four of these are combined to form the training set and the remaining set acts as the validation set. This division is random (Lorenz et al. 2011; Crossa et al. 2017). Each of the subsets of data will act as the validation set at least once through this process, calculating the GEBVs and developing the model that will be utilized. CV is commonly used to determine the accuracy of different GS methods to calculate GEBVs (Lorenz et al. 2011; Crossa et al. 2017).

Chapter 3: GENOME-WIDE ASSOCIATION STUDY FOR STEM RUST RESISTANCE IN WESTERN CANADIAN WINTER WHEAT (*Triticum aestivum* L.)

3.1 Abstract

The fungal pathogen *Puccinia graminis* Pers.:Pers. f. sp. *tritici* Eriks. & E. Henn. (*Pgt*) is the causal agent of stem rust on wheat (*Triticum aestivum* L.). *Pgt* has the potential to cause significant losses to wheat production in Canada, making it an economically important disease. No stem rust epidemics have occurred since the 1950s in western Canada due to the cultivation of resistant varieties and the eradication of the alternate host *Berberis vulgaris* L. Due to its destructive potential, stem rust is a priority-one disease for the Prairie Recommending Committee for Wheat, Rye, and Triticale, and breeding lines without stem rust resistance are unlikely to be registered for production in western Canada. The genetic basis for stem rust resistance in most Canadian wheat cultivars is not fully understood. The objective of this study is to identify the stem rust resistance genes (*Sr*) currently deployed in the winter wheat germplasm present in western Canada. The population utilized in the study consists of approximately 300 hard red winter wheats from western Canada, and 100 hard red winter wheats from the United States, eastern Canada, and Europe. The population was phenotyped for stem rust resistance in inoculated field trials and seedling assays. Field stem rust nurseries were grown at two locations, Winnipeg and Carman, Manitoba in 2022–2023 and 2023–2024. Field trials were evaluated as randomized complete block designs with two replicates per trial. Plots were 1m long and inoculated with a mixture of *Pgt* races. Nurseries were mist irrigated to promote disease development. Data were collected on stem rust field severity and infection response. Infection type data was recorded from indoor seedling inoculations with individual races. The SGS TraitGenetics 25K Infinium and the Wheat Barley 40K Infinium single nucleotide polymorphism (SNP) arrays were used to genotype the population. Genome-wide

association study (GWAS) analyses identified quantitative trait loci (QTL) for stem rust resistance on chromosomes 1D, 1B, 2A, 2D, and 3D. The position of these QTL suggests the presence of *Sr6*, *Sr24*, *Sr31*, *Sr33*, and *Sr38* in the population.

3.2 Introduction

In 2025, wheat (*Triticum aestivum* L.) was the most widely grown field crop in Canada with a total of 40.0 million tonnes produced, representing an increase of 11.2% from the 2024 growing season (Statistics Canada 2025). Spring wheat is the predominant type of wheat grown in Canada, followed closely by durum (*Triticum turgidum* L.), whereas winter wheat accounts for a much smaller portion of the total production. During the 2024–2025 growing season, winter wheat production was 3.6 million tonnes in Canada, which was an increase of 17% compared to production in 2023–2024 (Statistics Canada 2025). In western Canada, Saskatchewan and Alberta are the primary wheat producers, with a combined production of 30.5 million tonnes. Comparatively, Manitoba produces a small amount of wheat with a total of 5.9 million tonnes produced in 2025 (Government of Canada 2025). During the 2024–2025 growing season a total of 41,000 acres of winter wheat were seeded in Manitoba. Out of the acres seeded in Manitoba, the winter wheat cultivars AAC Wildfire, AAC Vortex, and Emerson represent approximately 75% of the total acres seeded (Manitoba Crop Alliance 2025).

With wheat being a major crop in Canada, the threat of pathogens must be evaluated and considered to prevent major losses for producers. Of the pathogens that can create yield loss in wheat, there are five priority-one diseases in western Canada that all breeders must incorporate some form of resistance when developing their lines. These diseases are stem rust, leaf rust, stripe rust, fusarium head blight (FHB) and common bunt (Laroche et al. 2019; Aboukhaddour et al. 2020; Manitoba Crop Alliance 2024). For a winter wheat line to be supported for registration, the breeding line must be rated “intermediate” to all three rust diseases, which highlights the priority for maintaining effective resistance in new wheat varieties (Laroche et al. 2019).

Puccinia graminis Pers.: Pers. f. sp. Eriks. & E. Henn. *tritici* (*Pgt*) is a fungal pathogen and the causal agent of stem rust on wheat. Hosts of *Pgt* include common wheat, barley (*Hordeum vulgare* L.), and triticale ($\times$ *Triticosecale*). The alternate host required for sexual reproduction is *Berberis vulgaris* (common barberry), *B. canadensis*, and some *Mohania* species (Peturson 1958; Roelfs et al. 1992). *Pgt* is a heteroecious fungus that requires two separate and unrelated hosts to complete its full life cycle, going through both sexual and asexual reproduction (Leonard and Szabo 2005). Stem rust has historically been one of the most devastating diseases to wheat production (Roelfs 1989). No major losses due to stem rust have occurred in Canada since the 1950s, largely due to the incorporation of genetic resistance into the cultivars that are being widely grown in Canada (Peturson 1958; McCallum and DePauw 2008; Laroche et al. 2019). Globally, in recent years epidemics have become a more common occurrence with outbreaks taking place in Germany (2013), Italy (2016), and more recently in western Siberia and northern Kazakhstan (Bhattacharya 2017; Lewis et al. 2018; Patpour et al. 2022; Olivera et al. 2022a). These recent events emphasize the importance of maintaining and continuing to look for sources of resistance to stem rust.

Resistance to stem rust can be categorized into seedling (all-stage) resistance and adult plant resistance (APR). Seedling resistance genes are effective through all developmental stages and are race-specific, while APR genes are only effective at the adult plant stage and tend to be non-race-specific (Leonard and Szabo 2005; Ellis et al. 2014; Periyannan et al. 2017). APR genes are less likely to be defeated in contrast to race-specific seedling genes, due to their generally non-specific nature to *Pgt* races (Laroche et al. 2019). With races of *Pgt* continually evolving, it is more important than ever to ensure that multiple resistance genes are present in lines being developed so that broad spectrum and durable resistance can be maintained.

Genome-wide association studies (GWAS) are a form of association mapping that are utilized to identify marker-trait associations (MTA) by detecting the statistical association between molecular markers and specific traits in the population being evaluated. This allows for a better understanding of the traits being evaluated (Tibbs Cortes et al. 2021). The exact make up of the resistance genes present in the winter wheat germplasm of western Canada is not fully known. It is important to identify these resistance genes to allow winter wheat breeders to have a more comprehensive understanding of the resistance genes present in their breeding programs. Phenotypic data collected at the seedling and adult plant stage, combined with high density SNP marker data were used to identify these resistance loci. Further analysis will allow for the identification of resistance genes present in the association mapping population and loci associated with resistance that may provide novel sources of resistance

The objective of this study is to conduct GWAS on a winter wheat association mapping population that is primarily comprised of the breeding germplasm from western Canada to identify quantitative trait loci (QTL) that are associated with stem rust resistance loci.

3.3 Materials and Methods

3.3.1 Germplasm

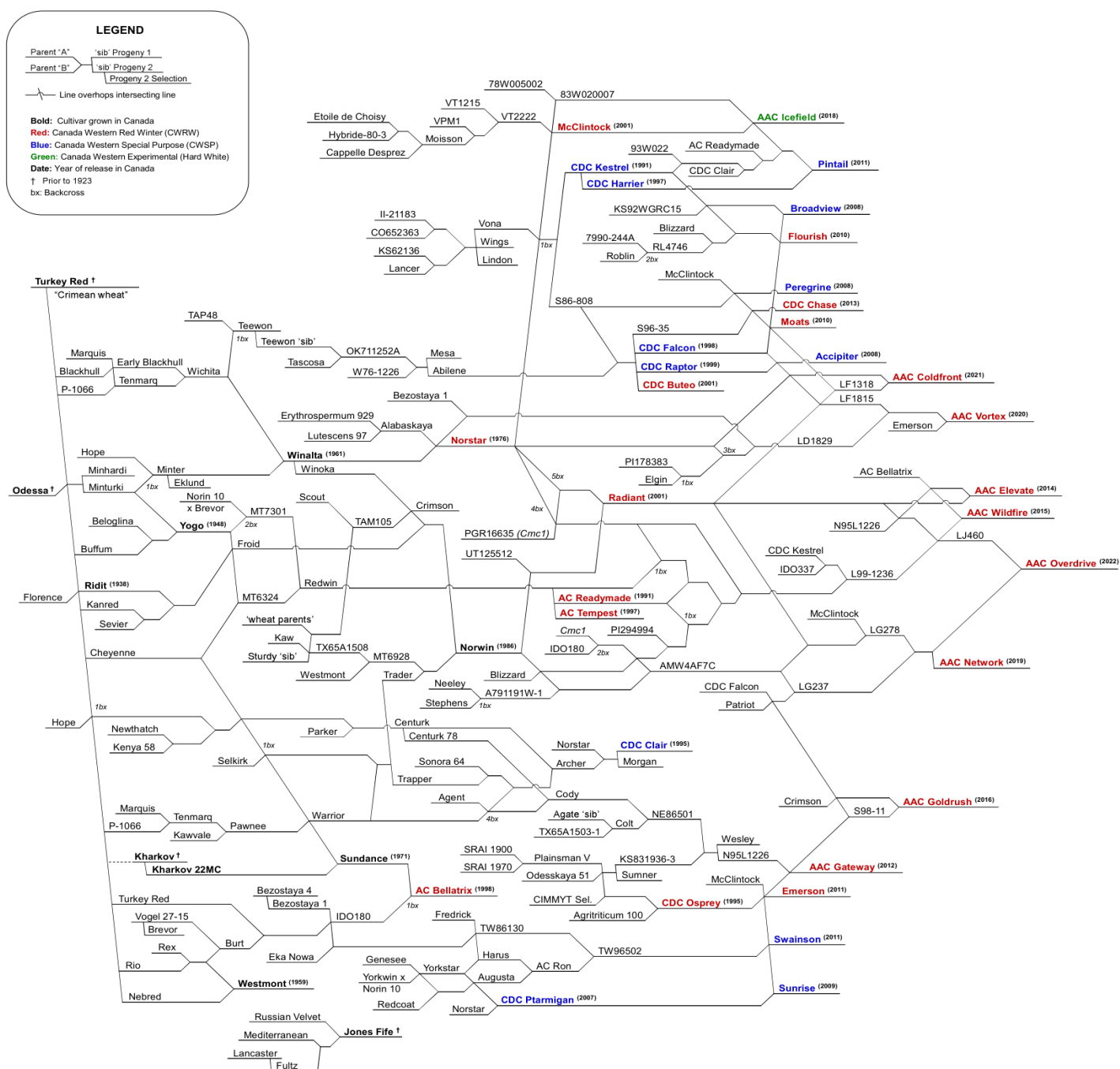
The association mapping population was comprised of 440 lines, primarily consisting of hard red winter wheat varieties. Approximately 300 individuals in the population originated from western Canada. The remaining lines were from the United States, Europe, and eastern Canada. The lines from western Canada consisted of breeding germplasm that were closely related to one another, the lines from other regions were often unrelated; however, some of the material acquired from the United States had previously been used in breeding schemes in western Canada to introduce novel sources of resistance (McCallum and DePauw 2008). An in-depth look at the relatedness amongst

many of the recently released Canada Western Red Winter (CWRW) wheat cultivars is provided in Figure 3.3.1. This ancestry chart provides information to show the pedigree relationships among cultivars currently and formerly utilized in Canada and aids in providing context for the genetic structure of the association mapping population.

Seed utilized for field trials originated from one plant that was grown in a controlled environment. A single seed was sown into the cell of a 5-cell root trainer sleeve containing Sunshine Mix #5 (SunGro Horticulture, Canada, Seba Beach, Alberta). Root trainers were maintained in a controlled environment (20°C day, 18°C night; 16-hour light, 8-hour dark) for 21 days. Leaves were trimmed down to approximately 1-inch in length. Root trainers were moved into vernalization (4°C; 16-hour light, 8-hour dark) for nine weeks. Following the vernalization period, root trainers were moved into a controlled environment growth room (15°C day, 12°C night; 16-hour day, 8-hour day) for one week. Seedlings were then transplanted into 1-gallon pots with a 2:2:1 mix (soil; sand; peat), plants were then maintained at temperatures ranging from 15 – 22°C in a controlled environment growth room.

Glassine bags were placed on individual spikes on each plant prior to anthesis, ensuring no outcrossing occurred and allowing for seed being utilized to be traced back to the original plant. Seed increases of genotypes took place in the field prior to disease testing in the field to ensure adequate amounts of seed were available. The individual lines were seeded in 1 m rows, consisting of 70 seeds per row with row spacing of 30 cm. Rows of winter wheat alternated with fall rye, reducing chances of seed mixing when seeding and likelihood of outcrossing. At the time of harvesting, compressed air was used to clean the combine between threshing samples.

The Ancestry of Western Canadian Winter Wheat Cultivars



Ancestry created by R.J. Graf, Ph.D.
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Figure 3.3.1 Pedigree relationships among western Canadian winter wheat lines illustrating the ancestry of germplasm used in breeding programs. Permission granted for use from Dr. Robert Graf.

3.3.2 Phenotypic Evaluation - Inoculated Field Trials

Fields trials took place over the 2022–2023 and 2023–2024 growing seasons in Carman, MB at the Ian N. Morrison Research Farm and in Winnipeg, MB on the University of Manitoba Fort Garry campus. Seeding took place from late September to early October. Field trials were seeded as an alpha lattice with two replicates per trial. During the 2022–2023 growing season, 256 lines were evaluated, while 440 lines were evaluated during the 2023–2024 growing season. Rows were sown to be 1m in length, with approximately 70 seeds per row and row spacing of 30 cm. CDC Ptarmigan (susceptible) was utilized for spreader rows and borders of the nurseries. Five checks were utilized within each field trial, these include AAC Elevate (moderately resistant), AAC Vortex (resistant), AAC Coldfront (resistant), CDC Ptarmigan (susceptible), and Moats (resistant).

Inoculations took place from late May to early June, ideally when the overnight temperatures were above 10°C. A mist irrigation system was set up within the nurseries, to provide conditions suitable for urediniospore germination and infection of host tissue following inoculations. A mixture of seven *Pgt* races MCCFC (1541), QTHSF (1347), RHTSF (1562), RKQSF (1312), RTHSF (1561), TMRTF (1311), and TPMKC (1373) were used to inoculate the nurseries. Inoculum for the field trials was provided by the Morden Research and Development Centre (MRDC, Agriculture and Agri-Food Canada). The inoculum consisted of equal portions of the seven *Pgt* races being examined in the field, the vials were stored at -80°C until required. The inoculum was placed into a heat bath at 40°C for 10 minutes to heat shock the spores. Once removed from the water bath, the vials were opened and the inoculum was then passed through an 80-mesh sieve. From the inoculum retained 0.5g of the urediniospores were weighed out. The weighed-out inoculum was kept in a glass container until inoculations took place that day. At the time of inoculation, 1.5L of Soltrol oil (Soltrol 170 Isoparaffin, Chevron Philips, The Woodlands,

Texas, USA) was added to the spores. The bottle of inoculum was then shaken and attached to a Microfit Herbiflex sprayer (MiCRON Group, Bromyard, Herefordshire, United Kingdom). Each individual row was inoculated with a fine mist dispensed by the Herbiflex. The mist system was then run overnight and as needed following the inoculations.

Ratings took place mid-to-late July. Plants were phenotyped using the modified Cobb scale. This scale looks at severity, denoted as a percentage from 0–100% of the area of the stem affected and infection type (IT). IT was denoted as follows; “0” represents no visible infection; “R” indicates the presence of necrosis, but no pustules are visible; “MR” indicates the presence of small pustules with the presence of chlorosis and/or necrosis; “MS” indicating the presence of medium to large pustules surrounded by chlorosis; and “S” indicated the presence of large pustule with no visible chlorosis and necrosis. A combination of these ratings is acceptable too (Peterson et al. 1948).

Coefficient of infection (COI) was then calculated by multiplying the severity by the IT rating for each line. The infection type ratings were converted to a numerical scale for this multiplication as follows: R = 0.1, R-MR= 0.25, MR = 0.4, MR-MS =0.55, MS= 0.7, MS-S =0.85, S=1.

Before proceeding with the statistical analysis, the data was curated to remove lines with low spring stand. Spring stand was visually estimated on each row as the percentage of the row that survived the winter. Some wheat lines from countries with moderate climates were not winter hardy and did not survive the harsh Manitoba winters well. For the field trial that took place in the summer of 2023, there were 256 lines sown in the fall of 2022. With two replicates this would mean there were a total of 512 rows. Rows that had less than 10% spring stand were removed from the analysis, because there would not be enough plants for accurate stem rust ratings. As a result,

17 rows were removed from the analysis of the Carman 2022–2023 field trial. For the 2023–2024 field season, there was an increase in the number of lines seeded for the population, due to increased seed stores of genotypes being available for analysis. In both locations, a total of 440 lines were sown, with two replicates per trial. A total of 880 rows were sown in each nursery. A total of 63 and 126 rows were lost due to a combination of winterkill and wildlife damage in Carman and Winnipeg during this field season, respectively.

3.3.3 Seedling Assays

Seedling assays took place at the Morden Research and Development Centre in Morden, MB. Seed from field seed increases was used to conduct the seedling tests. A total of 421 lines were tested. Seedlings were grown in a controlled environment where five seeds per line were sowed into individual cone-tainers (RL98, Stuewe & Sons, Inc., Tangent, Oregon, USA) containing Sunshine Mix #5 (SunGro Horticulture, Canada, Seba Beach, Alberta), seedlings were grown at 20°C for 16 hours of light, 8 hours dark (Menzies et al. 2024). Once the first leaf fully emerged, the seedlings were inoculated with individual *Pgt* races (MCCFC, QTHSF, RHTSF, RKQSF, RTHSF, TMRTE, and TPMKC). Approximately 3 mg of the urediniospores for each race was suspended in 0.7 mL of Soltrol oil (Soltrol 170 Isoparaffin: Chevron Philips, The Woodlands, Texas USA) and sprayed onto the seedling once the first leaf emerged. Following inoculation, seedlings were placed into a dew chamber overnight. The following morning, seedlings were exposed to light for a minimum of 2 hours inside the dew chamber, before being moved to a controlled environment at 20°C with 16 hours light and 8 hours dark. Infection type (IT) ratings were then determined 14 days after inoculation on the underside of the first leaves using the 0–4 Stakman scale (Stakman et al. 1962; Menzies et al. 2024). Based upon the Stakman scale, the ratings are as follows, “;” is indicative of a hypersensitive fleck; “0” no presence of uredinia; “1” indicates the presence of small uredinia

with necrosis; “2” = small to medium uredinia, chlorosis is present; “3” indicates medium uredinia; and “4” indicates the presence of large uredinia. The addition of “+” or “-” to the ratings indicates that the uredinia may have been either larger or smaller than what would be expected for the applicable IT. In this study, individuals categorized as a “1” or “2” were considered resistant, while individuals identified to be a “3” or “4” were considered susceptible (Figure 3.3.2). A combination of the IT ratings was acceptable (Stakman et al. 1962; Roelfs 1988) Inoculations took place over a two month period from January – February 2024.

Seedling ratings were utilized for data analysis when based upon three or more plants. This resulted in 417 genotypes being evaluated for *Pgt* race MCCFC, 406 for QTHSF, 386 for RHTSF, 396 for RKQSF, 392 for RTHSF, 396 for TMRTF, and 403 for TPMKC. For the purpose of GWAS analysis, the traditional IT scores recorded following the Stakman scale were converted into a 1–9 scale (Fatima et al. 2020). The scale is as follows where “0/0;/” = 1, “01-/ 0;1-/1 /;1-” = 2, “1-/1;/1/1+/1-/1;/1-1/11-/11/1+1/11+” = 3, “;12-/;12/1-2/12/21/21-” = 4, “2-/2/2+/22+/22-/2+2/2-2” = 5, “23/2+3/2-3/23+/23-/23+/3-2” = 6, “3-/3-3/33-/3/3+/3+3/33+” = 7, “3-4/34” = 8, and “4/43” = 9. Scores 1 to 4 are considered resistant, scores 5 to 6 are intermediate, and 7 to 9 are deemed susceptible.

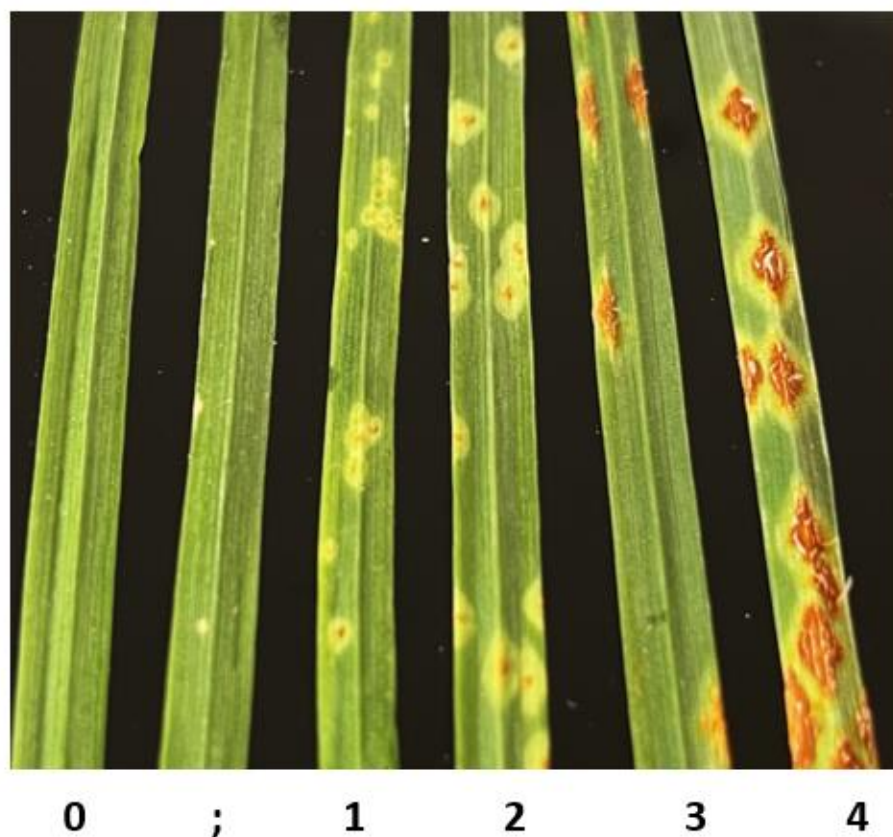


Figure 3.3.2 Infection types (IT) of wheat seedlings inoculated with *Puccinia graminis* f. sp. *tritici*, illustrating the Stakman 0–4 rating scale Based upon the Stakman scale, the ratings are as follows, “0” no presence of uredinia; “;” is indicative of a hypersensitive fleck; “1” indicates the presence of small uredinia with necrosis; “2” = small to medium uredinia, chlorosis is present; “3” indicates medium uredinia: and “4” indicates the presence of large uredinia. Individuals categorized as a “1” or “2” were considered resistant, while individuals identified to be a “3” or “4” were considered susceptible

3.3.4 Statistical Analysis

Statistical analysis was performed for each field trial. The program Multi-Environment Trait Analysis in R (META-R) was used to calculate adjusted means of stem rust severity, IT, and COI. These were calculated as best linear unbiased predictors (BLUPs) (Alvarado et al. 2020). BLUPs were used as the adjusted means for phenotypic data collected for GWAS. The field trials were randomized as an alpha lattice but were analyzed as randomized complete block design (RCBD) because a number of plots were removed from the analysis due to low spring stand, poor winter survival, or animal damage. META-R was also used to analyze phenotypic and genetic correlations

among environments and the correlations of traits by environment or across all environments. The broad-sense heritability (H^2) for each field trait (severity, IT, COI) was estimated for each environment and across the pooled environments. Broad-sense heritability for an individual trait is calculated as follows:

$$H^2 = \frac{\sigma_g^2}{\sigma_g^2 + \sigma_\epsilon^2/nRep}$$

Here σ_g^2 and σ_ϵ^2 represent the genotype and error variance components, while $nRep$ is the number of replicates. For the combined analyses the calculation of broad-sense heritability changes to the following:

$$H^2 = \frac{\sigma_g^2}{\sigma_g^2 + \sigma_{ge}^2/nLoc + \sigma_\epsilon^2/(nLoc \times nRep)}$$

Taking into consideration the genotype $\times$ environment ($G \times E$) interaction variance component denoted as σ_{ge}^2 and $nLoc$ reflecting the number of environments.

3.3.5 Data Transformation

Following the calculation of the BLUPs, the phenotypic data for the field trait was transformed using rank-based inverse normal transformation implemented in the R package RNOmni (McCaw et al. 2020). This form of transformation is applied to improve the distribution of traits that are not normally distributed, such as disease ratings. This ensures that Type I errors (false positive) are appropriately controlled for in genome-wide association studies (McCaw et al. 2020).

3.3.6 DNA Extraction and Genotyping

Tissue was collected from each of the plants grown in the controlled environment, at the seedling stage. Tissue was collected in 2.5cm sections, it was then placed in liquid nitrogen, followed by

lyophilization. Tissue was stored in a -80°C freezer until the DNA was extracted. At the time of extraction, two 2.5 cm long sections of leaf tissue were placed into 1.2 ml collection tubes with a 3 mm stainless steel grinding ball, the plates were then processed using a Qiagen Tissue Lyser II™ (Qiagen, Mississauga, ON), running for 3 minutes and 30 oscillations per minute. Once the tissue had been ground into a fine powder, the Patricia Warner Wheat Barley DNA extraction protocol was followed (Pallotta et al. 2003). Extracted DNA was quantified using Hoescht 33258 Stain (Invitrogen by Thermo Fisher Scientific, Waltham, Massachusetts, USA), for the quantification of genomic DNA. The Hoescht stain was diluted to a concentration of 1 mg/ml. A quantification buffer of 10 mM Tris-HCl, 1 mM EDTA, 1 M NaCl was utilized. Aliquots of 4 µl for each DNA sample were placed into 200 µl of the dilute stain. Samples were then quantified using a Tecan Infinite F500 (Tecan Group, Männedorf, Switzerland). Following quantification, DNA samples were diluted with 0.1X TE to a concentration of 55 ng/ul for genotyping purposes.

Two oligonucleotide arrays were utilized to carry out genotyping of the population, the first being the Illumina Infinium Wheat Barley 40K v1.0 BeadChip (Keeble-Gagnère et al. 2021) and the second being 25K Wheat Infinium Array from SGS TraitGenetics (Gatersleben, Germany). The 40K array was tested at MRDC and the 25K array was tested by SGS TraitGenetics. The wheat SNPs from the 40K wheat barley Infinium array were analyzed in GenomeStudio 2.0.5 (Illumina, San Diego, California, USA) using the Polyploid Genotyping module with the PolyGenTrain algorithm set to an inbred population. The wheat SNPs from the 25K SGS TraitGenetics were analyzed by SGS TraitGenetics. Markers present on both arrays were identified, and only the duplicate data from the 40K wheat barley Infinium array were retained. A total of 43,362 markers were scored and utilized for downstream analyses.

3.3.7 Population Structure and Linkage Disequilibrium

Linkage Disequilibrium (LD) was calculated using TASSELv5.2.96 (Bradbury et al. 2007). LD was estimated with the standardized disequilibrium coefficient (D'), squared allele frequency correlation (r^2), and the calculated p -values (Bradbury et al. 2007). Following calculation of the LD, RStudio was utilized to remove any missing values denoted as 'NaN', conduct model fitting, and calculate the LD decay threshold.

Population structure was assessed using sparse non-negative matrix factorization (sNMF) implemented in the R package LEA v3.14.0 (Frichot et al. 2014). sNMF will assume that the genetic data for the population originates from an admixture of the ancestral populations (K). K represents the number of ancestral populations and is unknown. The algorithm is able to provide ancestry coefficient estimates from multi-locus genotype data. The program employs a cross-entropy criterion to determine the number of ancestral populations present; this evaluates the model fit. Assessing how well the model can predict masked genotype data, the lower the cross-entropy values, the more indicative of how well the model can perform (Pritchard et al. 2000a; Frichot et al. 2014).

3.3.8 Genome-Wide Association Study

GWAS was conducted using the single-locus test Mixed Linear Model (MLM) implemented in the R package GAPIT v3.4.0 (Wang and Zhang 2021). SNP loci were retained based on a minimum minor allele frequency (MAF) of 0.05. The MLM model used five principal components to control for population structure and a kinship matrix was derived to account for relatedness between individuals, which reduce the likelihood of type I errors (Zhu et al. 2008; Wang and Zhang 2021). Principal component analysis (PCA) was performed using the R package LEA, and the first five principal components were included in the MLM to account for population structure (Frichot et al.

2014). GWAS was performed independently for each site year and for the pooled data of all field trial locations. This analysis was conducted for each of the field traits being examined in this study: severity, IT, and COI. GWAS was also independently conducted for reaction to each *Pgt* race from the seedling assays.

Following GWAS, significant SNPs were identified for each of the tests. The R package *qvalue* v2.34.0 (Storey and Tibshirani 2003) was used to calculate the false discovery rate (FDR). This provides the threshold above which SNP are statistically significant. GAPIT uses the Bonferroni correction as its internal cut off for significant SNP. This threshold is often considered too stringent, not identifying QTL where they exist (type II errors). The FDR threshold significance values ($-\log_{10}(p)$) for the field traits was calculated to be 3.76, for the seedling assays it was respectively calculated to be 4.36.

Once significant SNPs were identified, LD analysis was conducted using PLINK (Purcell et al. 2007) to assess the accuracy of the a priori location assigned to each marker. An r^2 value of 0.8–1.0 between markers was considered evidence of strong LD. Marker locations was then assigned based upon consensus between the marker consideration and all other markers. SNPs from the 90K wheat Infinium SNP chip were previously mapped (Wang et al. 2014), which provided an additional check for the location of their assigned chromosome. Following the various analyses Manhattan and quantile-quantile (QQ) plots were visualized with the R package CMplot (Yin et al. 2021).

3.3.9 QTL Stacking

Following the identification of significant SNP markers, the effects of QTL stacking were explored. QTL stacking analysis was performed in R studio using selected loci that were

associated with stem rust resistance. For each QTL, a favourable allele was determined based on the allele associated with the lowest mean disease response across significant markers of the identified QTL. Lines were then scored with the absence or presence of the favourable allele. Lines with missing marker data were excluded from the analysis. A QTL stack count was calculated for each line by summing the number of favourable QTL present, and disease responses were compared among stack combinations to evaluate the effects of QTL combinations on stem rust resistance.

3.4 Results

The GWAS panel was comprised of 440 winter wheat accessions that were evaluated for their response to *Pgt* inoculations, at the adult and seedling stage. The panel was evaluated at the seedling stage in a controlled environment; 421 accessions were evaluated for their response to seven *Pgt* races independent of another. Infection type was evaluated at the seedling stage. Field trials were conducted to evaluate 440 winter wheat accessions (including checks) for their response to a mixture of the seven *Pgt* races at the adult plant stage. At the adult stage, infection type, severity (%), and coefficient of infection (COI) were evaluated. Field trials were conducted from 2022–2023 and 2023–2024 in Winnipeg and Carman, Manitoba. Site-years are referred to as Carman 2023 (CRM23), Carman 2024 (CRM24), and Winnipeg 2024 (WPG24). The calculated BLUPs were utilized to conduct the analysis. Each site-year was analyzed independently, and the data were also pooled to evaluate responses across all site-years.

3.4.1 Phenotypic Assessment – Field Trials

Summary statistics of the GWAS population were calculated for each site-year and for the pooled dataset across all environments utilizing the estimated BLUPs. Severity ratings observed across each site-year, and the pooled dataset ranged from 1.62% to 70.39%, infection type ranged from 0.04 to 0.97, and coefficient of infection from 1.09 to 70.04 (Table 3.1). The mean severity ratings observed across site-years, and the pooled dataset ranged from 19.78 to 23.62%, mean infection type ranged from 0.46 to 0.53, and mean coefficient of infection ranged from 15.39 to 18.80. The phenotypic observations indicate that a range of phenotypes were present in the population. Severity and coefficient of infection tended to be skewed towards the left on the frequency distributions, while infection type often exhibited a bimodal distribution (Figure 3.4.1).

The frequency distribution of the three stem rust resistance traits evaluated in the field trials initially displayed non-normal distributions. The data was transformed using the R package RNOmni (McCaw et al. 2020) to improve normality of the data prior to analysis. The phenotypic data were transformed using a rank-based inverse normal transformation, where values were ranked in ascending order from smallest to largest. These ranks were then converted to percentile scores within the dataset and subsequently mapped onto a standard normal distribution, resulting in the transformed values ranging from -3 to +3. Comparison of the frequency distribution of the original untransformed dataset to that of the transformed data can be observed in Figure 3.4.1. Summary statistics based upon the estimated BLUPs following transformation are presented in Table 3.2.

Table 3.1 Summary statistics of best linear unbiased predictions (BLUPs) for stem rust severity, infection type, and coefficient of infection calculated from the association mapping population field trials. BLUPS were estimated from the raw data, across each site-year independently and the pooled dataset across environments.

Trait—environment combinations ^a		Mean	SD ^b	Min	Max
Severity (%)	CRM23	23.62	15.92	4.66	70.39
	CRM24	19.78	17.37	2.39	68.39
	WPG24	23.42	17.15	3.54	69.74
	Pooled	23.26	17.47	1.62	67.00
Infection Type	CRM23	0.52	0.20	0.10	0.91
	CRM24	0.46	0.34	0.04	0.95
	WPG24	0.53	0.32	0.06	0.95
	Pooled	0.52	0.30	0.04	0.97
Coefficient of Infection	CRM23	15.39	14.34	2.46	60.97
	CRM24	15.72	17.6	1.15	68.21
	WPG24	18.80	18.43	1.65	70.04
	Pooled	17.51	17.73	1.09	65.58

^aCRM23, Carman 2023; CRM24, Carman 2024; WPG24, Winnipeg 2024

^bStandard deviation

Table 3.2 Summary statistics of rank-based inverse normal transformed best linear unbiased predictions (BLUPs) for stem rust severity, infection type, and coefficient of infection calculated from the association mapping population field trials.

Trait—environment combinations ^a		Mean	SD ^b	Min	Max
Severity (%)					
	CRM23	0.00	0.99	-2.49	2.81
	CRM24	0.01	0.96	-1.51	2.66
	WPG24	0.00	0.98	-2.01	2.96
	Pooled	0.00	1.00	-2.78	2.97
Infection Type					
	CRM23	0.00	0.98	-2.49	2.61
	CRM24	0.00	0.93	-1.51	1.59
	WPG24	-0.01	0.96	-2.01	1.64
	Pooled	0.00	0.99	-2.78	2.43
Coefficient of Infection					
	CRM23	0.00	0.99	-2.49	2.81
	CRM24	0.01	0.97	-2.97	2.66
	WPG24	0.00	0.99	-2.96	2.96
	Pooled	0.00	1.00	-2.78	2.97

^aCRM23, Carman 2023; CRM24, Carman 2024; WPG24, Winnipeg 2024

^bStandard deviation

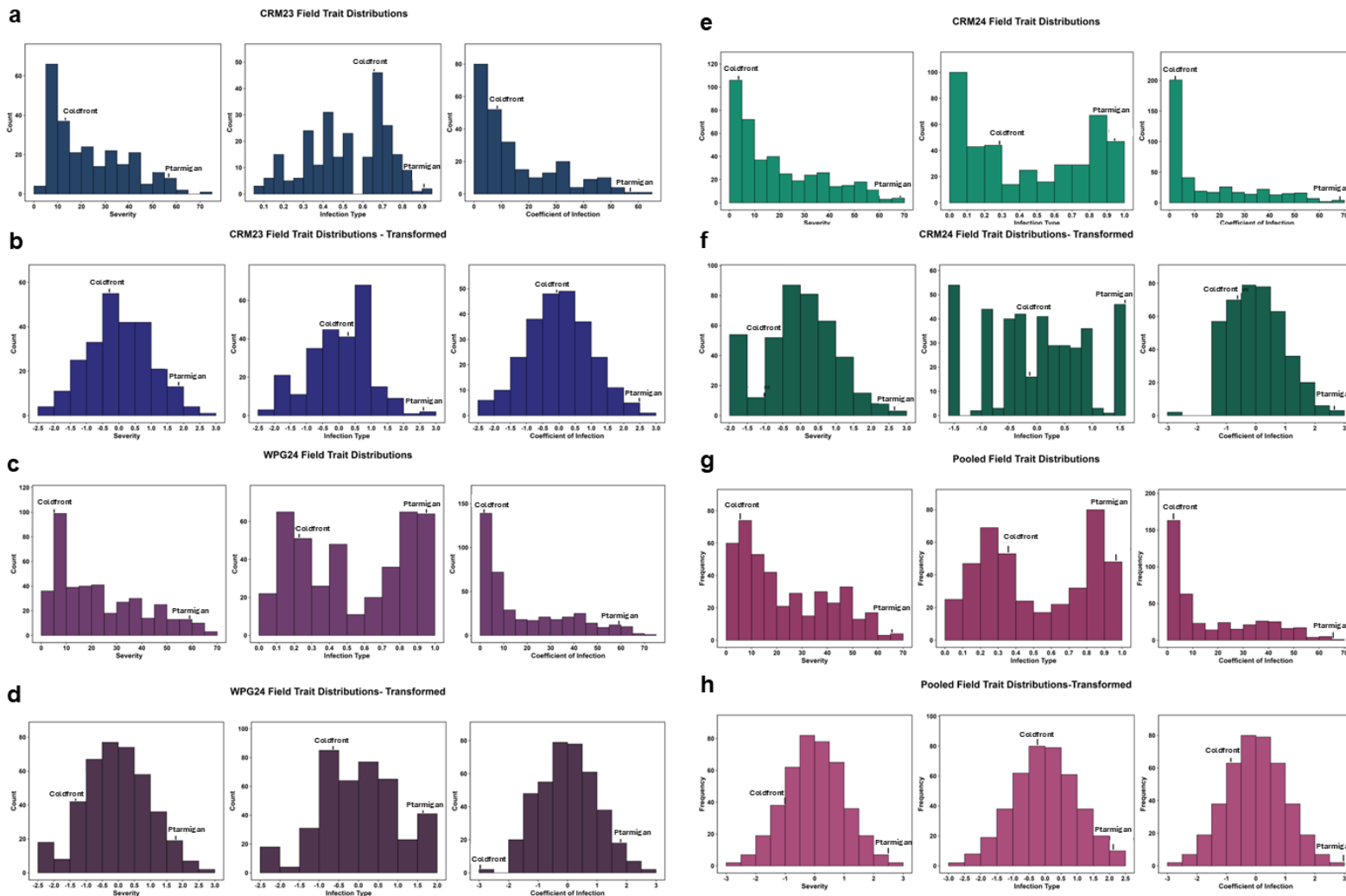


Figure 3.4.1 Frequency distributions of stem rust severity, infection type, and coefficient of infection in the association mapping population across all site-years and the pooled dataset. Frequency distributions were plotted for the original data and the data transformed using R package RNOmni. CRM23, Carman 2023 untransformed data (a); CRM23, transformed data (b); WPG24, Winnipeg 2024 untransformed data (c); WPG24, transformed data (d); CRM24, Carman 2024 untransformed data (e); CRM24 transformed data (f); trait data pooled over all site-years, untransformed (g); pooled transformed data (h). The means of two lines with varying resistance, Coldfront (resistant) and CDC Ptarmigan (susceptible) are indicated.

3.4.2 Phenotypic Assessment – Seedling Assays

A broad spectrum of phenotypic responses was observed in the association mapping panel, with infection type scores ranging from 1 (resistant) to 9 (highly susceptible). The mean infection type scores observed across all lines were 4.97 for MCCFC, 5.59 for QTHSF, 5.64 for RHTSF, 5.00 for RKQSF, 6.21 for RTHSF, 6.48 for TMRTF, and 6.56 for TPMKC (Table 3.3). The phenotypic distributions varied among the races. The population was skewed to more resistant IT responses when inoculated with RKQSF. MCCFC and QTHSF split the population between resistant and susceptible responses. RHTSF and RTHSF exhibited a similar split between the resistant and susceptible IT responses, but with an increase in wheat lines exhibiting an intermediate reaction. In contrast, the population was skewed towards susceptibility when inoculated with TMRTF and TPMKC (Figure 3.4.2). Of the wheat lines tested with each race, 51.3%, 47.5%, 40.4%, 54.5%, 34.9%, 31.6%, 29.0% were resistant to *Pgt* races MCCFC, QTHSF, RHTSF, RKQSF, RTHSF, TMRTF, and TPMKC, respectively (Figure 3.4.3). Overall, a broad range of phenotypes was observed across the population and between the seven *Pgt* races being evaluated.

Table 3.3 Comparative statistics of the seven *Pgt* races evaluated independently for infection type to 421 genotypes of the association mapping population

Race	N ^a	Mean	SD ^b	Median	Min	Max
MCCFC	417	4.97	3.09	3	1	9
QTHSF	406	5.59	2.86	5	1	9
RHTSF	386	5.64	2.72	5	1	9
RKQSF	396	5.00	3.17	3	1	9
RTHSF	392	6.21	2.73	7	1	9
TMRTF	396	6.48	2.87	8	1	9
TPMKC	403	6.56	2.9	9	1	9

^aNumber of genotypes evaluated

^bStandard deviation

Seedling Infection Type Distributions by Race

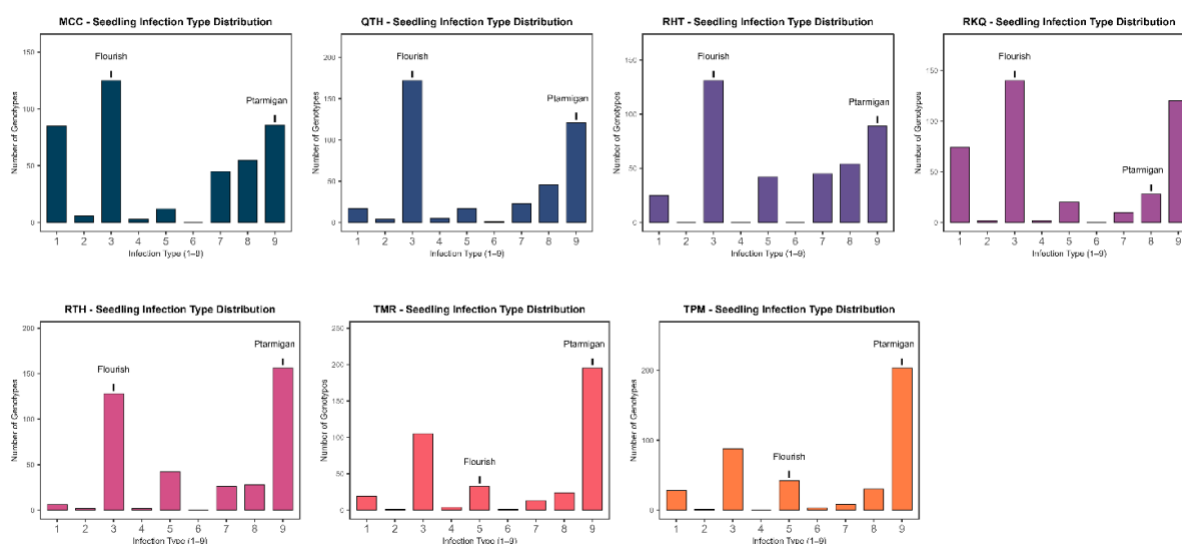


Figure 3.4.2 Histograms comparing the phenotypic distributions of the association mapping population to each of the seven *Pgt* races. IT ratings were converted from the Stakman scale to a 1-9 linear scale. The means of two lines with varying resistance, Flourish (intermediate) and CDC Ptarmigan (susceptible) are indicated. *Pgt* races are denoted as follows MCC (MCCFC), QTH (QTHSF), RHT (RHTSF), RKQ (RKQSF), RTH (RTHSF), TMR (TMRTF), AND TPM (TPMKC).

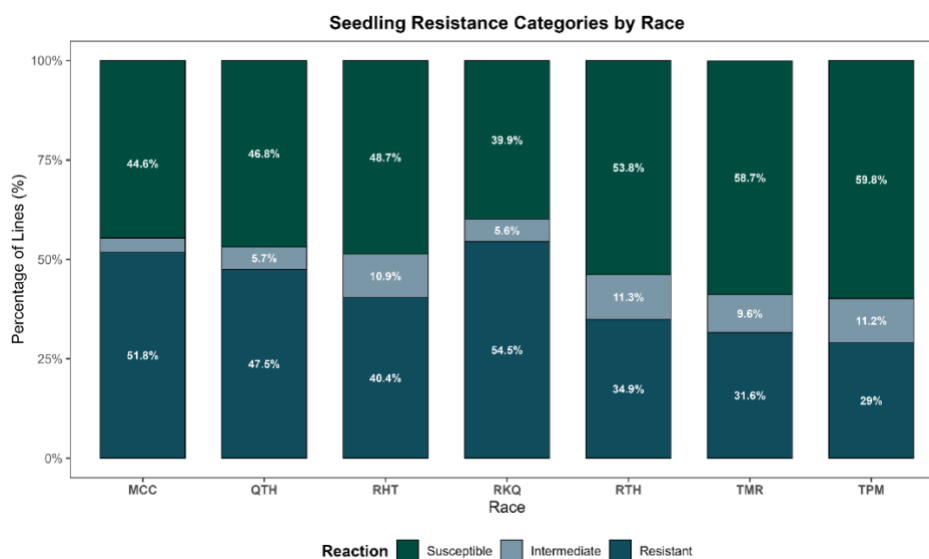


Figure 3.4.3 Stacked bar plots for each of the seven *Pgt* races, demonstrating the frequency (%) of the population that falls into the three categories of susceptible, intermediate, and resistant. *Pgt* races are denoted as follows MCC (MCCFC), QTH (QTHSF), RHT (RHTSF), RKQ (RKQSF), RTH (RTHSF), TMR (TMRTF), AND TPM (TPMKC).

3.4.3 Genotyping, SNP Filtering, and Validation

For the purpose of this study the SNP marker data retained from the Illumina Infinium Wheat Barley 40K v1.0 BeadChip (Keeble-Gagnère et al. 2021) and the 25K Wheat Infinium array from SGS TraitGenetics were assembled into a single HapMap file. This file contained a total of 43,362 SNPs. The SNPs marker were filtered prior to analysis in GAPIT (Wang and Zhang 2021) using a minor allele frequency (MAF) threshold of 0.05. Markers falling below this threshold were removed from the dataset. After filtering, 29,766 markers were retained and utilized for this study.

Linkage disequilibrium (LD) was determined for the markers utilized in the study. Based on the LD results, markers were reassigned to different chromosomes when they were in strong LD with markers previously placed on other chromosomes. Following the LD analysis, the markers that were reassigned to different chromosomes were placed against the Chinese Spring reference genome v1.0 (IWGSC et al. 2018) using Basic Local Alignment Search Tool (BLAST) to confirm their alignments. Marker positions were then updated accordingly. The markers were also compared to markers that had been mapped using the 90K Infinium Array as a secondary measure of position confirmation (Wang et al. 2014).

3.4.4 Population Structure and Linkage Disequilibrium

Population structure of the association mapping population was assessed using the sparse non-negative matrix factorization (sNMF) function implemented in the R package Landscape and Ecological Association (LEA) v3.14.0 (Frichot et al. 2014). Cross-entropy was evaluated across $K = 1-10$ to determine the most likely number of ancestral subpopulations. As the cross-entropy values decreased and K increased it was determined that $K = 2$ (Figure 3.4.4). Indicating the population is best described by two subpopulations. As K increased beyond 2, the cross-entropy values showed minimal improvement.

An ancestry coefficient plot was derived from $K=2$ (Figure 3.4.5) this is inferred as two genetic clusters; however, individuals were not clearly separated explicitly into the two distinct groups. A continuous gradient of ancestry proportions was observed, indicating that some individuals within the population share genetic background between the two subpopulations.

Of the two subpopulations identified 59.3% fell into cluster 1 and 40.7% fell into cluster 2. This distribution likely reflects the mixed breeding origins of the population, where large proportions of the population are derived from western Canada and the United States, resulting in two partially overlapping genetic groups rather than clearly distinct populations.

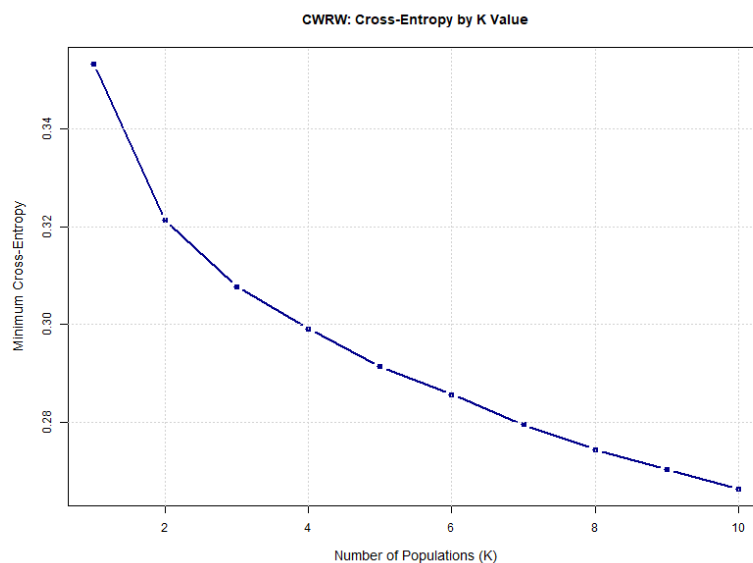


Figure 3.4.4 Cross-entropy values obtained from sNMF analysis used to infer population structure within the mapping association panel. Cross-entropy was calculated for values of K 1 to 10, where K represents the number of inferred genetic populations. The minimum cross-entropy value indicates the most likely number of ancestral populations explaining the genetic structure of the population.

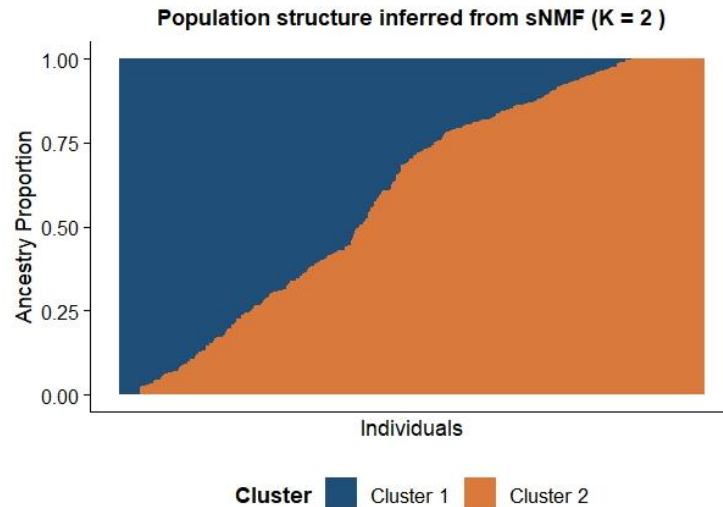


Figure 3.4.5 Population structure inferred from sNMF analysis at $K = 2$. Each vertical bar represents an individual within the population. The colours indicate the proportion of the ancestry assigned to one of the two genetic subpopulations identified.

Linkage disequilibrium (LD) was assessed by plotting the pairwise r^2 values against physical distance. This was fitted using non-linear regression by the Hill and Weir model (Hill and Weir 1986). LD decayed rapidly at short distances. An r^2 value of approximately 0.23 at ~2.75 Mbp was identified as the half-decay point. Background LD was observed with an r^2 of 0.06 at 18.5 Mbp. Based upon these results, an LD window of 5.5 Mbp was used to define QTL regions for downstream analysis. This allowed for markers within these 5.5 Mbp regions to be grouped together indicating linkage (Figure 3.4.6).

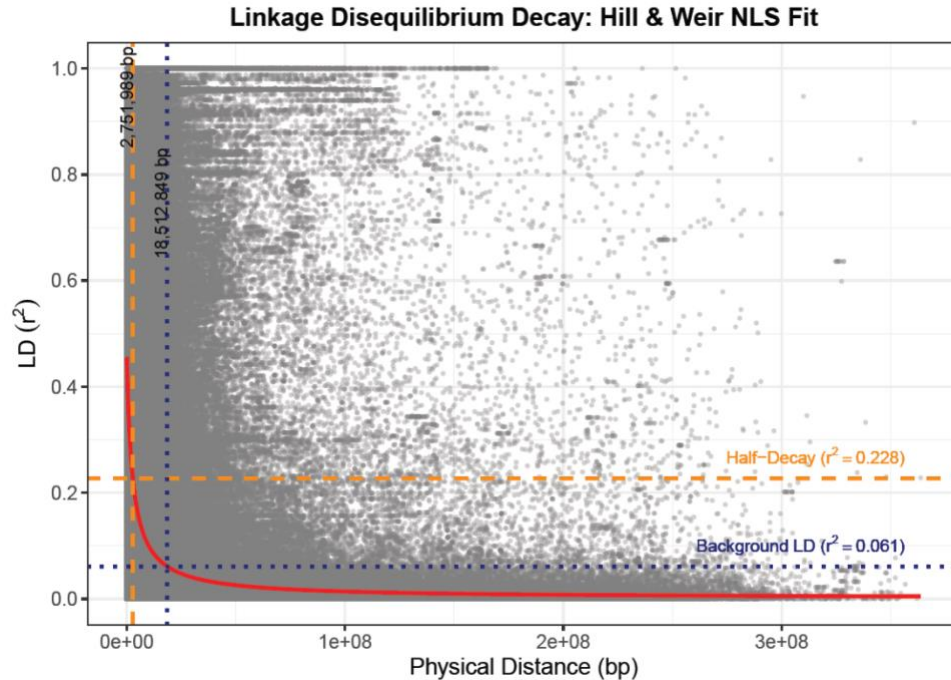


Figure 3.4.6 Linkage disequilibrium (LD) of the association mapping population plotted as pairwise r^2 against the physical distance in base pairs. The red curve represents the non-linear regression fit using the Hill and Weir model. The dashed horizontal orange line indicates the half-decay of $r^2 = 0.23$ and the blue dashed line indicating the background decay of $r^2 = 0.06$. The corresponding vertical lines indicate the physical distances at which the thresholds are reached. Respectively these at 2.75 and 18.5 Mbp. The half-decay distance was doubled to provide the approximate size of the linkage block, which is 5.5 Mbp.

3.4.5 Genome-Wide Association Study Results

GWAS analyses identified 16 QTL of known chromosome locations from the field trials evaluating stem rust severity, infection type, and coefficient of infection (Table 3.4). QTL were numbered according to their ascending physical position within each chromosome; numbering was maintained across both the seedling and field trait analyses to allow for comparison. The results from the GWAS analyses were visualized using Manhattan and QQ plots (Figure 3.4.7; Figure S1-S3). The Manhattan plots revealed several strong association peaks across the environments and the QQ plots demonstrated that population structure was accounted for within the association mapping population.

For each QTL, the top marker was identified. In several cases, the top marker associated with a given QTL varied between the environment or trait being examined. There was overlap between some environment-trait combinations. In total, QTL were detected across 9 chromosomes (1A, 1B, 1D, 2A, 2B, 2D, 3B, 3D, and 5A). Additional marker-trait associations were identified and placed into an ‘unknown’ category, for markers that did not have an assigned physical location.

The calculated $-\log_{10}(p)$ values ranged from 3.77–17.24. Many of the QTL were associated with multiple stem rust traits. The number of QTL were distributed amongst the traits in the following manner: seven QTL were associated with severity, twelve with infection type, and ten with COI. Looking beyond the number of QTLs identified for each trait several were identified to be present in all the site-years and the pooled dataset, these include: *QSr.umb-1B.1*, *QSr.umb-2A.1*, *QSr.umb-2A.4*, and *QSr.umb-2D.1*. Four were associated with all three traits being evaluated in the field: *QSr.umb-1B.1*, *QSr.umb-2A.1*, *QSr.umb-2A.4*, and *QSr.umb-2D.1*. The calculated phenotypic variation for the top markers identified across all QTL ranged from 1.18–28.12% (Table 3.4).

Among the QTL identified, the most significant association was observed for *QSr.umb-2A.1*, represented by marker ‘D_contig01272_220’, which reached a $-\log_{10}(p)$ value of 13.49 for severity in the CRM23 environment. This locus also explained the largest proportion of phenotypic variance among the identified loci, accounting for 28.12% of the variation for severity in CRM23. This same region was detected across multiple environments and traits.

Several significant markers could not be assigned a physical position in the CSv1.0 reference genome and were therefore classified as having unknown chromosomal position and not given a QTL designation. These markers were absent from the reference genome assembly or did not return informative BLAST hits. Despite this, a number of these markers were in linkage disequilibrium with markers of known chromosomal location, allowing them to be tentatively

associated with previously identified QTL regions. For example, ‘AX-158596552’, ‘Excalibur_c18324_390’, ‘Excalibur_c25599_358’, and ‘Kukri_c24446_306’ were associated with *Qs.umb-2A.1*, while ‘AVRIG24133’ and ‘AVRIG24187’ were associated with significant markers on chromosome 3A and *Qs.umb-3D*, ‘AVRIG24577’ with *Qs.umb-3B.1*, and ‘AVRIG24644’ and ‘AVRIG26841’ with *Qs.umb-3D*. Interestingly, the marker ‘AVRIG26841’ produced the strongest statistical association overall ($-\log_{10}(p) = 17.24$) for severity in the pooled dataset. Results from GWAS are indicative that field resistance in the association mapping population was controlled by multiple genomic regions.

The seedling GWAS analyses identified a total of 22 unique QTL across all seven *Pgt* races that were evaluated in the study (Table 3.5). Evaluation of each race identified three QTL associated with MCCFC, six with QTHSF, four with RHTSF, two with RKQSF, eight with RTHSF, and ten with both TMRTF and TPMKC. The results from the GWAS analyses were visualized utilizing Manhattan and QQ plots (Figure 3.4.8 and Figure 3.4.9). Marker significance was determined using the FDR threshold ($-\log_{10}(p) = 4.36$). The Manhattan plots revealed several strong association peaks for each *Pgt* race being evaluated and the QQ plots demonstrated that population structure was accounted for within the association mapping population.

The seedling resistance QTL identified were spread across 14 chromosomes: 1A, 1B, 1D, 2A, 2B, 2D, 3A, 3B, 3D, 5B, 6A, 6B, 7A and 7B. Several of the QTLs identified provided resistance to multiple races. *Qs.umb-3D.1* was detected in six races, *Qs.umb-1B.1* and *Qs.umb-6A* were detected in five races, *Qs.umb-3B* was detected in four races, and *Qs.umb-2D.2* in three races. Three of the QTL only contributed resistance to one race of the seven races evaluated, these include *Qs.umb-7A.1* detected by MCCFC, *Qs.umb-2A.2* by TPMKC, and *Qs.umb-2B.4* by TPMKC.

Amongst all unique QTLs identified the phenotypic variance ranged from 2.36–15.00%, with the largest effect resulting from *QSr.umb-3D.1* (Table 3.5). The calculated $-\log_{10}(p)$ values ranged from 4.31 to 14.23, with the strongest association detected for ‘AVRIG20916’ detected in *QSr.umb-2D.2* in response to TPMKC.

Comparison of QTL identified in the field and seedling GWAS analyses identified several QTL were detected at both growing stages. These include *QSr.umb-1B.1*, *QSr.umb-2A.2*, *QSr.umb-3B.1*, and *QSr.umb-3D.1*. The detection of these QTL in both the seedling assays and the field trials suggest these QTL may contribute to resistance across multiple stages of plant development.

Several QTL were detected only in the seedling analyses, these include *QSr.umb-1B.2*, *QSr.umb-5B*, *QSr.umb-6A*, *QSr.umb-6B*, *QSr.umb-7B.1*, and *QSr.umb-7B.2*. Conversely, *QSr.umb-1A.2*, *QSr.umb-1A.3*, *QSr.umb-1A.4*, *QSr.umb-2A.1*, *QSr.umb-2A.3*, *QSr.umb-2A.4*, *QSr.umb-2B.1*, *QSr.umb-2B.2*, *QSr.umb-2B.3*, *QSr.umb-2D.1*, *QSr.umb-3D.2*, and *QSr.umb-5A* were only detected in the field trials, suggesting they may contribute to resistance that is primarily expressed at the adult plant stage under field conditions.

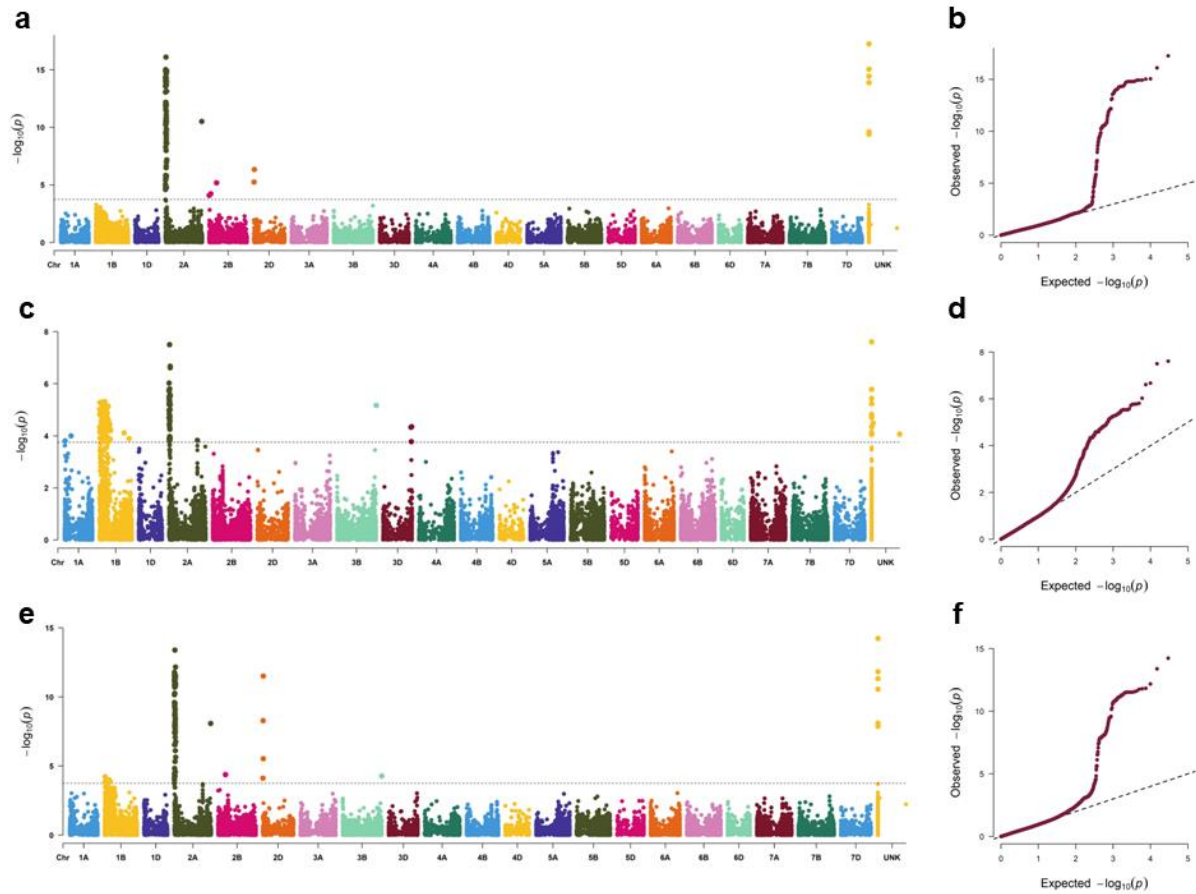


Figure 3.4.7 Manhattan and quantile-quantile (QQ) plots produced from the genome-wide association analysis of the pooled dataset from all site-years. On the Manhattan plots the y-axis indicates the $-\log_{10}(p)$ and the x-axis indicates the chromosomal position of the markers. The dashed line indicates the false discovery rate threshold (FDR) of $-\log_{10}(p) = 3.76$. The QQ plots illustrate the observed $-\log_{10}(p)$ against the expected $-\log_{10}(p)$ for the pooled dataset. A total of 29,766 markers were used. Severity is represented in panels a-b, infection type (IT) in c-d, and coefficient of infection (COI) in e-f.

Table 3.4 Peak quantitative trait loci (QTL) for the field stem rust resistance traits identified in the association mapping population using Mixed Linear Model (MLM) GWAS analysis. Peak QTL identified by the most significant marker identified for each trait in each site-year.

QTL designation	Trait—environment combinations ^a	Marker	Position (bp)	Effect	PVE% ^b	-log ₁₀ (p)
<i>QSr.umb-1A.2</i>	Pooled_IT	AX-94844830	11,822,296	0.17	2.00	3.81
<i>QSr.umb-1A.3</i>	WPG24_IT	wsnp_Ku_rep_c101863_88951284	135,970,175	0.35	2.33	4.21
<i>QSr.umb-1A.4</i>	Pooled_IT	AVRIG14435	302,899,167	-0.3	1.27	3.77
<i>QSr.umb-1B.1</i>	CRM23_IT	RAC875_c63624_753	11,859,589	0.22	7.92	6.35
	CRM23_COI	RAC875_c30138_595	4,321,944	0.21	3.49	3.79
	CRM24_Sev	AVRIG15510	100,234,150	0.39	2.28	3.83
	CRM24_COI			0.47	2.82	4.71
	CRM24_IT	AVRIG15442	69,906,281	0.55	3.92	5.44
	WPG24_IT	AVRIG15579	117,172,879	0.42	3.05	5.27
	Pooled_IT			0.40	2.97	5.31
	Pooled_COI	TA004380-0460	10,204,634	0.38	2.21	3.89
<i>QSr.umb-1D</i>	CRM23_IT	Ra_c69552_1419	8,721,360	0.14	4.66	4.53
<i>QSr.umb-2A.1</i>	CRM23_Sev	D_contig01272_220	21,262,961	0.54	28.12	13.49
	CRM23_COI	AVRIG17409	22,127,659	0.39	17.47	10.17
	CRM24_Sev	AVRIG17384	12,011,806	0.52	14.91	13.10
	CRM24_IT			0.40	8.42	7.89
	CRM24_COI			0.50	12.41	11.42
	WPG24_Sev			0.47	15.88	13.11
	WPG24_IT			0.37	9.59	8.88
	WPG24_COI			0.49	14.61	13.10
	Pooled_Sev			0.53	17.97	16.09
	Pooled_IT			0.32	7.67	7.50
<i>QSr.umb-2A.2</i>	Pooled_IT	Tdurum_contig30451_88	603,687,273	0.24	1.18	3.82
<i>QSr.umb-2A.3</i>	WPG24_COI	RAC875_c96399_610	758,388,484	0.25	1.20	4.06
<i>QSr.umb-2A.4</i>	CRM23_Sev	BS00067382_51	772,221,017	0.44	19.40	9.69
	CRM24_Sev			0.33	6.39	6.55
	CRM24_COI			0.31	5.06	4.14
	WPG24_Sev			0.39	11.54	10.47
	WPG24_IT			0.28	5.50	5.69
	WPG24_COI			0.39	9.93	9.82
	Pooled_Sev			0.39	10.19	10.50
	Pooled_COI			0.34	7.35	8.08

QTL designation	Trait—environment combinations ^a	Marker	Position (bp)	Effect	PVE% ^b	-log10(p)
<i>QSr.umb-2B.1</i>	WPG24_Sev	RAC875_c5577_1682	3,406,018	-0.17	2.28	4.21
	Pooled_Sev			-0.16	1.91	4.08
<i>QSr.umb-2B.2</i>	CRM24_Sev	AX-94739692	35,389,355	-0.19	1.94	5.34
	CRM24_COI			-0.19	1.94	4.14
	Pooled_Sev			-0.16	1.90	4.08
<i>QSr.umb-2B.3</i>	CRM23_Sev	AVRIG19145	155,883,958	-0.25	5.90	5.72
	CRM23_COI			-0.18	3.75	4.07
	WPG24_COI			-0.18	2.09	4.29
	Pooled_Sev			-0.19	2.43	5.17
	Pooled_COI			-0.17	1.97	4.37
<i>QSr.umb-2D.1</i>	CRM23_Sev	AVRIG20775	10,174,086	-0.23	5.17	5.63
	CRM23_COI			-0.17	3.45	4.17
	CRM24_Sev			-0.20	2.61	4.93
	CRM24_COI			-0.19	1.96	4.50
	WPG24_Sev			-0.19	2.97	5.98
	WPG24_IT			-0.15	1.80	3.97
	WPG24_COI			-0.20	2.72	5.90
	Pooled_Sev			-0.19	2.74	6.33
	Pooled_COI			0.44	11.88	11.51
	CRM23_IT			0.43	13.23	5.58
<i>QSr.umb-3B.2</i>	WPG24_IT	AVRIG24284	829,196,468	0.37	3.52	4.56
	Pooled_IT			0.37	3.84	5.16
	Pooled_COI			0.36	2.77	4.28
	CRM23_IT			0.29	7.90	4.94
<i>QSr.umb-3D</i>	Pooled_IT	AVRIG24644	612,890,894	0.23	2.47	4.73
	CRM23_IT			0.13	4.11	4.15
<i>QSr.umb-5A</i>	CRM24_IT	IAAV3514	478,822,209	0.39	2.22	3.86
Unknown	WPG24_IT	AVRIG21099	NA	0.35	2.32	4.01
	CRM23_IT	AVRIG21480	NA	0.19	5.53	4.81
	CRM23_Sev	AVRIG26841	NA	0.5	23.54	12.35
	CRM23_COI			0.37	15.47	9.1
	CRM24_Sev			0.51	14.16	13.73
	CRM24_IT			0.38	7.29	7.54
	CRM24_COI			0.49	11.76	11.89
	WPG24_Sev			0.47	15.33	13.91

QTL designation	Trait—environment combinations ^a	Marker	Position (bp)	Effect	PVE% ^b	-log10(p)
	WPG24_IT			0.36	8.77	8.94
	WPG24_COI			0.48	13.74	13.57
	Pooled_Sev			0.53	17.49	17.24
	Pooled_IT			0.31	7.09	7.61
	Pooled_COI			0.48	13.53	14.23
	CRM23_Sev	AVRIG32301	NA	0.48	22.52	11.37
	CRM23_COI			0.34	13.73	7.86
	CRM24_Sev			0.47	12.36	11.35
	CRM24_IT			0.32	5.55	5.63
	CRM24_COI			0.43	9.20	8.99
	WPG24_Sev			0.43	13.44	11.6
	WPG24_IT			0.29	5.83	5.89
	WPG24_COI			0.43	11.34	10.74
	Pooled_Sev			0.48	14.79	13.88
	Pooled_IT			0.24	4.41	4.75
	CRM23_Sev	AX-158596552	NA	0.41	16.55	9.91
	CRM23_COI			0.30	10.78	7.23
	CRM24_Sev			0.32	6.19	8.45
	CRM24_IT			0.27	4.13	5.81
	CRM24_COI			0.31	5.31	7.52
	WPG24_Sev			0.27	5.48	7.15
	WPG24_IT			0.19	2.76	4.13
	WPG24_COI			0.27	4.68	6.68
	Pooled_Sev			0.31	6.61	9.4
	Pooled_IT			0.19	3.10	4.7
	Pooled_COI			0.28	5.22	7.88
	CRM23_Sev	Excalibur_c18324_390	NA	0.51	25.47	12.29
	CRM23_COI			0.38	16.75	9.08
	CRM24_Sev			0.49	13.77	12.19
	CRM24_IT			0.36	6.80	6.59
	CRM24_COI			0.47	10.77	10.1
	WPG24_Sev			0.43	13.46	11.36
	WPG24_IT			0.30	6.18	6.07
	WPG24_COI			0.43	11.37	10.54
	Pooled_Sev			0.50	15.90	14.44

QTL designation	Trait—environment combinations ^a	Marker	Position (bp)	Effect	PVE% ^b	-log10(p)
	Pooled_IT			0.26	5.27	5.43
	Pooled_COI			0.44	11.52	11.33
	CRM23_Sev	Excalibur_c25599_358	NA	0.53	26.84	12.72
	CRM23_COI			0.39	17.18	9.19
	CRM24_Sev			0.50	13.87	12.33
	CRM24_IT			0.36	6.98	6.79
	CRM24_COI			0.47	10.77	10.3
	WPG24_Sev			0.45	14.34	12.07
	WPG24_IT			0.31	6.77	6.61
	WPG24_COI			0.44	12.13	11.22
	Pooled_Sev			0.51	16.56	15.04
	Pooled_IT			0.27	5.62	5.78
	Pooled_COI			0.45	11.52	11.82
	CRM23_Sev	Kukri_c24446_306	NA	0.40	15.93	9.7
	CRM23_COI			0.30	10.48	7.13
	CRM24_Sev			0.32	6.37	8.86
	CRM24_IT			0.27	4.16	5.93
	CRM24_COI			0.32	5.46	7.84
	WPG24_Sev			0.26	5.39	7.17
	WPG24_IT			0.19	2.79	4.24
	WPG24_COI			0.26	4.66	6.77
	Pooled_Sev			0.31	6.61	9.6
	Pooled_IT			0.19	3.13	4.82
	Pooled_COI			0.28	5.28	8.11
	CRM24_IT	Tdurum_contig100344_184	NA	0.46	2.76	4.57
	WPG24_IT			0.39	2.54	4.27
	Pooled_IT			0.41	3.11	5.23
	CRM24_IT	TGWA25K-TG0025	NA	0.47	2.72	3.99
	WPG24_IT			0.43	2.90	4.16
	Pooled_IT			0.41	2.93	4.34

^aSev, severity; IT, infection type; COI, coefficient of infection; CRM23, Carman 2023; CRM24, Carman 2024; WPG24, Winnipeg 2024

^bPVE, phenotypic variance explained

****Note:** Blank cells following a listed marker indicate that the same marker was identified as the top marker for the subsequent environments shown.

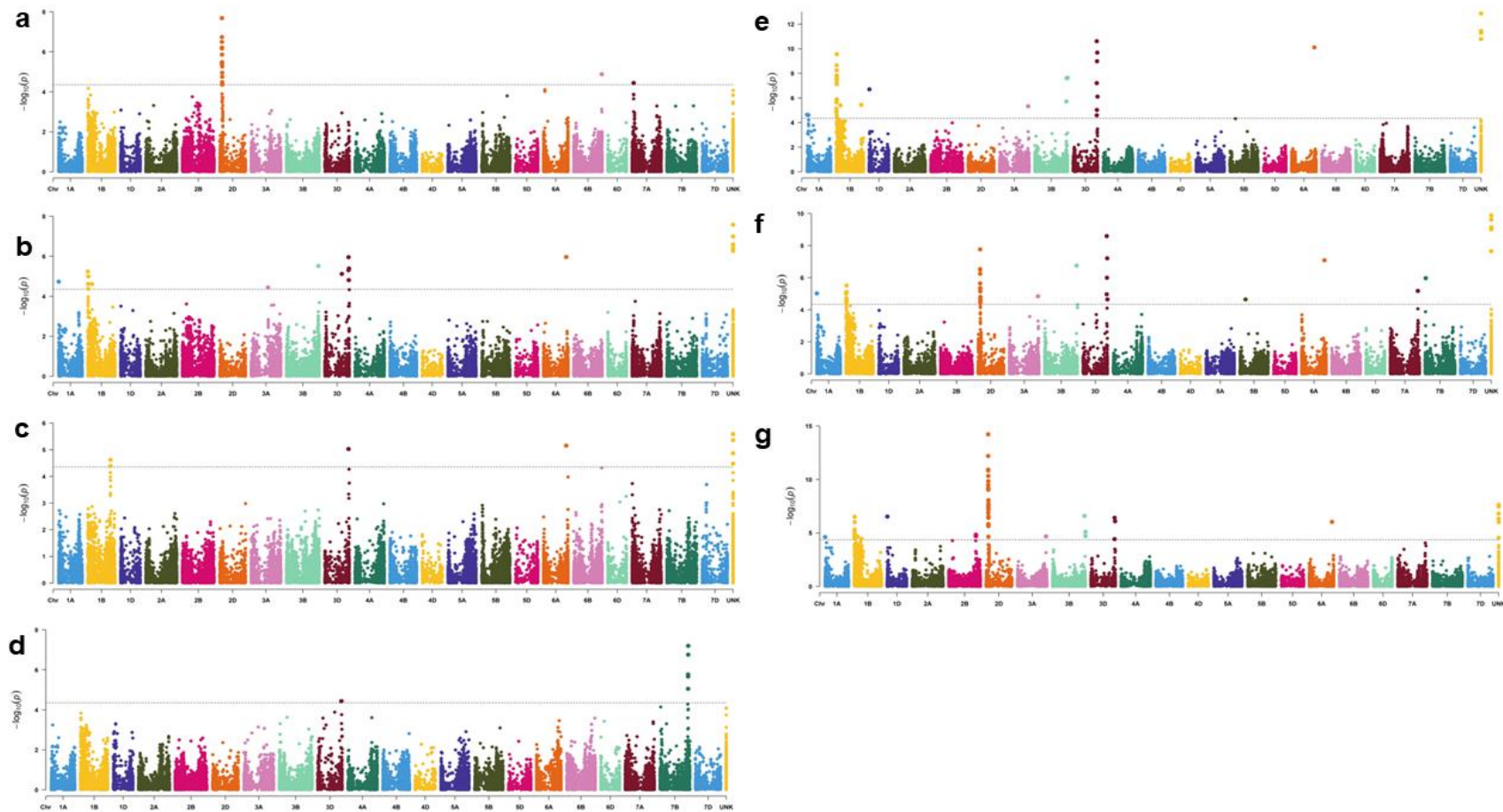


Figure 3.4.8 Manhattan plots produced from the genome-wide association analysis of the seven *Pgt* races evaluated for infection type on the association mapping population at the seedling stage. The y-axis indicates the $-\log_{10}(p)$ and the x-axis indicates the chromosomal position of the markers. The dashed line indicates the false discovery rate threshold (FDR) of $\log = 4.36$. A total of 29,766 markers were used in the analysis. The races are denoted as MCCFC (a), QTHSF (b), RHTSF (c), RKQSF (d), RTHSF (e), TMRTF (f), and TPMKC (g).

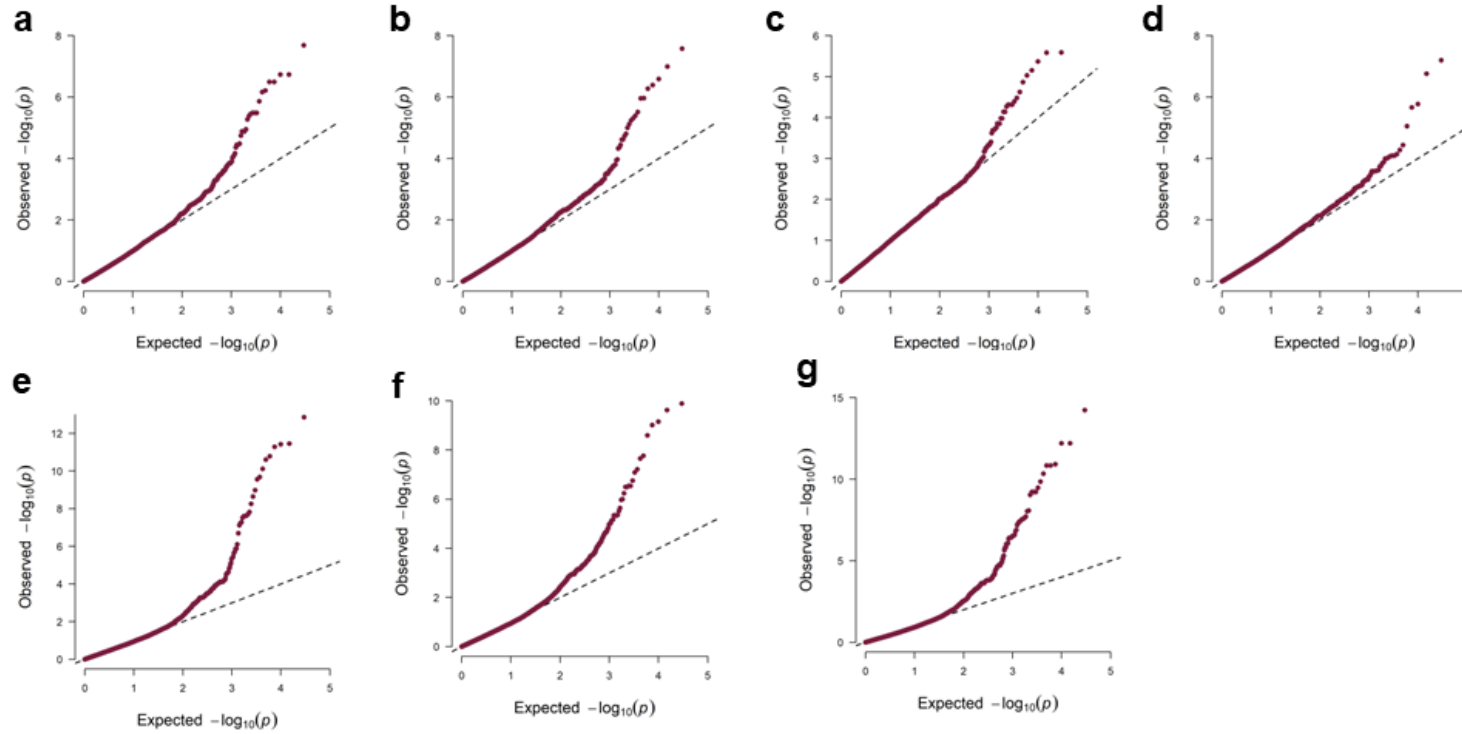


Figure 3.4.9 Quantile-quantile (QQ) plots produced from the genome-wide association analysis of the seven *Pgt* races evaluated for infection type on the association mapping population at the seedling stage. The QQ plots illustrate the observed $-\log_{10}(p)$ against the expected $-\log_{10}(p)$. A total of 29,766 markers were used. The races are denoted as MCCFC (a), QTHSF (b), RHTSF (c), RKQSF (d), RTHSF (e), TMRTF (f), and TPMKC (g).

Table 3.5 Peak quantitative trait loci (QTL) identified in the association mapping population at the seedling stage through controlled environment inoculations using Mixed Linear Model (MLM) GWAS analysis. Peak QTL identified by the most significant marker identified for each race evaluated.

Race	QTL designation	Marker	Position (bp)	Effect	PVE% ^a	-log ₁₀ (p)
MCCFC	<i>QSr.umb-2D.2</i>	AVRIG20916	56,239,548	1.93	5.50	7.69
	<i>QSr.umb-6B</i>	AVRIG32120	709,216,825	0.96	2.86	4.88
	<i>QSr.umb-7A.1</i>	AVRIG32612	39,817,754	-0.79	2.94	4.44
QTHSF	<i>QSr.umb-1A.1</i>	Kukri_c26168_423	1,208,261	1.01	4.38	4.73
	<i>QSr.umb-1B.1</i>	Kukri_c76307_182	14,030,921	1.16	4.54	5.00
	<i>QSr.umb-3A.1</i>	AVRIG21718	424,079,759	1.30	2.85	4.44
	<i>QSr.umb-3B.1</i>	AVRIG24190	804,245,497	2.23	8.73	5.51
	<i>QSr.umb-3D</i>	D_GDS7LZN02IJRXZ_309	594,069,919	1.98	7.64	5.95
	<i>QSr.umb-6A</i>	AVRIG30577	572,169,030	1.26	5.58	5.96
	Unknown	AVRIG21480	NA	1.51	7.24	7.58
		AVRIG24644	NA	1.39	6.38	6.99
		AVRIG24577	NA	1.49	6.73	6.59
		AVRIG24187	NA	1.33	5.92	6.39
		AVRIG24133	NA	1.33	5.69	6.27
RHTSF	<i>QSr.umb-1B.2</i>	wsnp_Ex_c5101_9053178	570,841,321	0.97	2.98	4.62
	<i>QSr.umb-3D</i>	D_GDS7LZN02IJRXZ_309	594,069,919	1.69	5.84	5.03
	<i>QSr.umb-6A</i>	AVRIG30577	572,169,030	1.06	4.24	5.15
	<i>QSr.umb-6B</i>	AVRIG32120	709,216,825	0.82	2.67	4.32
	Unknown	AVRIG24644	NA	1.12	4.43	5.59
		AVRIG21480	NA	1.17	4.60	5.59
		AVRIG24133	NA	1.12	4.30	5.37

Race	QTL designation	Marker	Position (bp)	Effect	PVE% ^a	-log10(p)
RKQSF		AVRIG24577	NA	1.15	4.24	4.87
		AVRIG24187	NA	0.99	3.55	4.48
		<i>Qs.umb-3D</i>	D_GDS7LZN02IJRXZ_309	1.75	5.02	4.44
		<i>Qs.umb-7B.2</i>	tplb0035h03_1251	1.37	5.22	7.20
RTHSF		<i>Qs.umb-1A.2</i>	RFL_Contig5637_1008	1.3	7.74	7.82
		<i>Qs.umb-1B.1</i>	Kukri_c76307_182	1.60	9.60	9.56
		<i>Qs.umb-1D</i>	Ra_c69552_1419	0.95	5.70	6.70
		<i>Qs.umb-3A.2</i>	AVRIG22488	0.87	3.72	5.33
		<i>Qs.umb-3B.2</i>	AVRIG24284	2.15	9.94	7.64
		<i>Qs.umb-3D</i>	D_GDS7LZN02IJRXZ_309	2.64	15.00	10.61
		<i>Qs.umb-5B</i>	AX-158526498	0.72	2.84	4.31
		<i>Qs.umb-6A</i>	AVRIG30577	1.61	10.16	10.11
		Unknown	AVRIG21480	1.95	13.49	12.86
			AVRIG24133	1.79	11.46	11.45
			AVRIG24577	1.93	12.71	11.42
			AVRIG24644	1.76	11.34	11.29
			AVRIG24187	1.68	10.62	10.79
TMRTF		<i>Qs.umb-1A.1</i>	Kukri_c26168_423	0.99	4.20	5.02
		<i>Qs.umb-1B.1</i>	Kukri_c76307_182	1.18	4.73	5.52
		<i>Qs.umb-2D.2</i>	AVRIG20916	1.92	6.21	7.76
		<i>Qs.umb-3A.2</i>	AVRIG22488	0.85	3.21	4.84
		<i>Qs.umb-3B.1</i>	AVRIG24190	2.35	9.93	6.75
		<i>Qs.umb-3D</i>	D_GDS7LZN02IJRXZ_309	2.37	10.71	8.59
		<i>Qs.umb-5B</i>	AX-158526498	0.78	2.97	4.65
		<i>Qs.umb-6A</i>	AVRIG30577	1.34	6.26	7.09
		<i>Qs.umb-7A.2</i>	AX-109338271	0.79	2.57	5.18
		<i>Qs.umb-7B.1</i>	wsnp_CAP7_c44_26549	1.09	3.04	5.97
		Unknown	AVRIG24133	1.67	8.85	9.89
			AVRIG21480	1.68	8.83	9.63
			AVRIG24644	1.57	8.05	9.15
			AVRIG24577	1.70	8.83	9.02

Race	QTL designation	Marker	Position (bp)	Effect	PVE% ^a	-log ₁₀ (p)
TPMKC		AVRIG24187	NA	1.41	6.67	7.66
	<i>QSr.umb-1A.2</i>	RFL_Contig5637_1008	9,580,143	1.07	4.65	5.27
	<i>QSr.umb-1B.1</i>	AX-94692514	11,633,627	0.95	5.02	6.50
	<i>QSr.umb-1D</i>	Ra_c69552_1419	8,721,360	0.95	5.05	6.53
	<i>QSr.umb-2B.5</i>	wsnp_Ex_c40976_47910672	709,836,593	1.38	2.36	4.70
	<i>QSr.umb-2B.4</i>	wsnp_Ra_c13679_21569624	687,205,349	1.42	2.56	4.84
	<i>QSr.umb-2D.2</i>	AVRIG20916	56,239,548	2.61	11.87	14.23
	<i>QSr.umb-3A.2</i>	AVRIG22488	723,444,477	0.85	3.10	4.69
	<i>QSr.umb-3B.1</i>	AVRIG24190	804,245,497	2.33	9.57	6.59
	<i>QSr.umb-3D</i>	D_GDS7LZN02IJRXZ_309	594,069,919	1.97	7.47	6.41
	<i>QSr.umb-6A</i>	AVRIG30577	572,169,030	1.21	5.15	6.03
	Unknown	AVRIG24133	NA	1.44	6.58	7.62
		AVRIG21480	NA	1.46	6.73	7.55
		AVRIG24577	NA	1.56	7.34	7.47
		AVRIG24644	NA	1.34	5.83	6.81
		Tdurum_contig100344_184	NA	1.81	3.81	4.54

^aPVE, phenotypic variance explained

3.4.6 Comparison of Identified QTL and Reported *Sr* Genes and QTLs

Comparison of the identified QTL with previously reported stem rust resistance showed that several regions in this study co-localized with previously published associations. Most prevalent are *QSr.umb-1A.2*, *QSr.umb-1B.1*, *QSr.umb-2A.1*, *QSr.umb-2A.3*, *QSr.umb-2B.1*, and *QSr.umb-3B* were located near loci reported by Miedaner et al. (2024) (Table 3.6). *QSr.umb-2D.1* nearly overlapped with a previously reported locus from Mourad et al. (2018) and *QSr.umb-3B.1* was located near a region associated with seedling resistance in Pradhan et

al. (2023). The close proximity of QTL identified in this study to those of previously published literature, supports the reliability of the associations identified. In contrast, several additional QTL were identified in the population, but they did not correspond closely to what was reported in the literature.

Table 3.6 Comparison of identified QTL that coincide for stem rust resistance of wheat in previously identified QTL.

Dataset	Chromosome	QTL	Position (bp)	Reference QTL position (bp)	Reference
Seedling/Field	1A	<i>QSr.umb-1A.2</i>	9,580,143	7,660,991	(Miedaner et al. 2024)
Seedling/Field	1B	<i>QSr.umb-1B.1</i>	11,859,589	9,555,825	(Miedaner et al. 2024)
Field	2A	<i>QSr.umb-2A.1</i>	12,011,806	12,544,152	(Miedaner et al. 2024)
Seedling	2A	<i>QSr.umb-2A.3</i>	709,836,593	713,630,879	(Miedaner et al. 2020)
Field	2B	<i>QSr.umb-2B.1</i>	3,406,018	7,676,708	(Miedaner et al. 2024)
Seedling	2D	<i>QSr.umb-2D.2</i>	56,239,548	56,190,286	(Mourad et al. 2018)
Seedling	3B	<i>QSr.umb-3B.1</i>	804,245,497	802,928,978	(Pradhan et al. 2023)
Seedling/Field	3D	<i>QSr.umb-3D</i>	594,069,919	613,700,556	(Miedaner et al. 2024)

3.5 QTL Stacking

Based upon the literature review and the GWAS analysis, four key QTL that were identified were evaluated to look at the impacts of the QTL on the field traits if they were absent compared to being present and when they were used in different combinations. The effects of QTL stacking were evaluated in the following: *QSr.umb-1B.1*, *QSr.umb-2A.1*, *QSr.umb-1D*, and *QSr.umb-3D*.

3.5.1 Effects of Individual QTL

Evaluation of the four key QTL identified shows that each had varying results on the field traits evaluated and at the seedling stage, if applicable. Among the QTL evaluated *QSr.umb-2A.1* exhibited the largest effect, with lines carrying this locus showing substantial decreases in severity, infection type, and COI compared to the lines that were lacking this QTL (Figure 3.12). *QSr.umb-3D* showed a strong effect across all three field traits and in six of the seven *Pgt* races evaluated at the seedling stage, *QSr.umb-1B.1* had a more moderate impact in comparison. Finally, *QSr.umb-1D* exhibited a smaller and more variable effect on resistance (Figure 3.5.1). Arguably, *QSr.umb-2A.1* provides the best resistance in terms of the impact it has on severity and COI; while *QSr.umb-3D* provides a slight advantage in decreasing IT. Suggesting that stacking these two QTL in breeding lines could provide more durable and broad-spectrum resistance rather than having individual QTL.

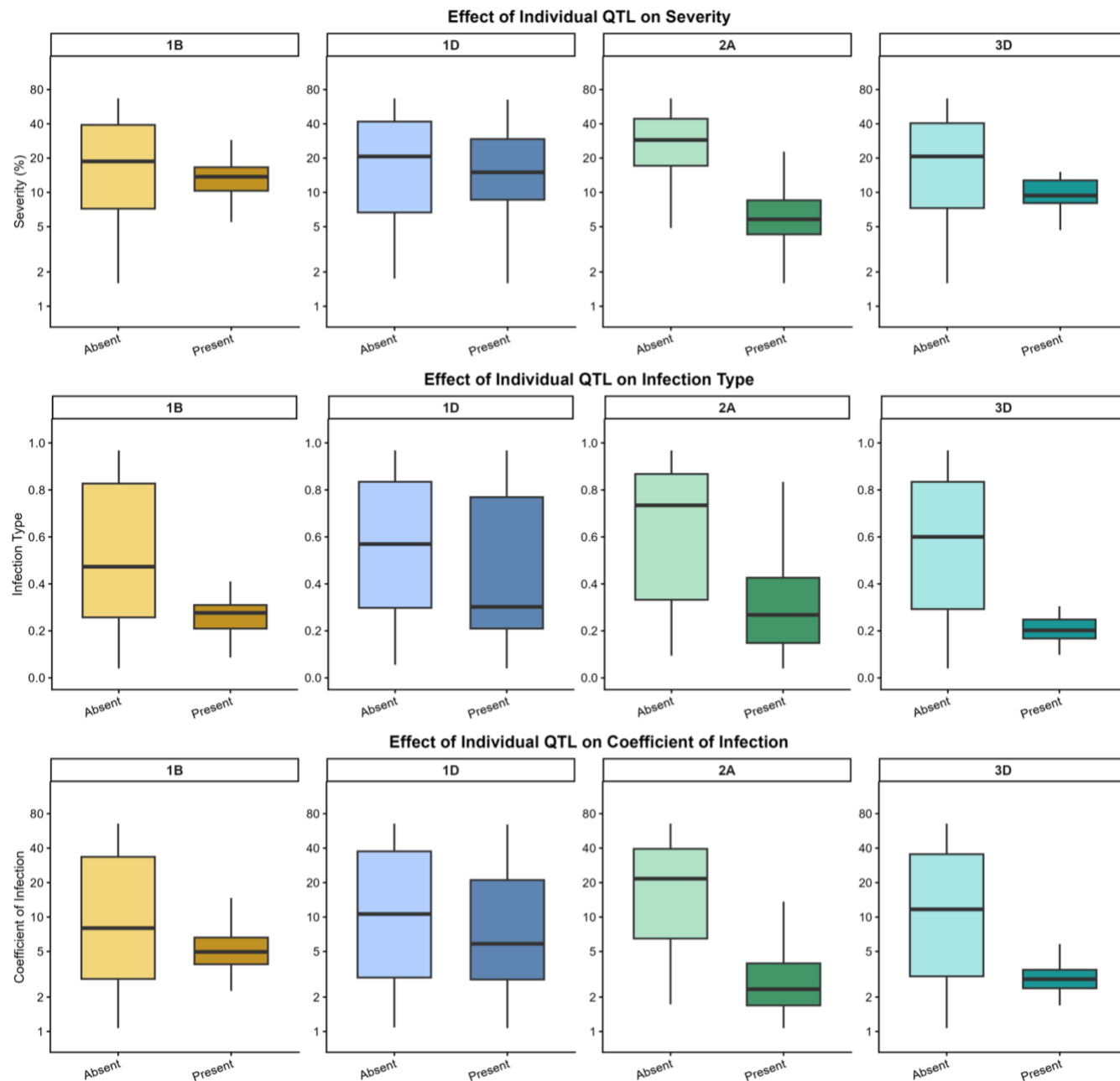


Figure 3.5.1 Box plots illustrating the effect of individual QTL on stem rust severity (%), infection type (IT), and coefficient of infection (COI) in the association mapping population based on the pooled field BLUPs. For each QTL evaluated, genotypes were grouped based on the absence or presence of the identified alleles contributing to resistance.

3.5.2 Effect of Identified QTL Combinations

Evaluation of specific QTL combinations revealed clear differences in resistance levels depending on the loci present. Among the QTL evaluated, *QSr.umb-2A.1* exhibited the largest effect, with lines carrying this QTL showing substantial reduction in severity, infection type, and COI compared to lines lacking this QTL or possessing other combinations. A total of 90 lines were identified that possessed this QTL alone (Figure 3.5.2). When *QSr.umb-2A.1* is used combination with other identified QTL, stronger resistance responses are observed. In particular, the combination of *QSr.umb-1D* and *QSr.umb-2A.1* showed the strongest resistance response across all three traits. *QSr.umb-1D* when alone exhibits comparatively high levels of disease, similar to lines that are lacking the presence of any of the identified QTL. This suggests that *QSr.umb-1D* by itself provides limited stem rust resistance.

Combinations involving three loci, such as *QSr.umb-1B*, *QSr.umb-1D*, and *QSr.umb-2A.1* showed strong resistance; however, the combination of these three QTL were only observed in a small number of lines ($n = 5$). This combination provides the largest decrease in IT and COI. The combination of *QSr.umb-1D/QSr.umb-3D* ($n = 4$) and *QSr.umb-1B/QSr.umb-1D* ($n = 16$) were also identified in the population. Both QTL combinations provide an increase in resistance responses across all three traits, though notable differences are observed in IT and COI. Many other QTL combinations were not observed in the population that could provide strong resistance, such as *QSr.umb-2A.1* and *QSr.umb-3D*.

Overall, the results are indicative that resistance within the association mapping population is enhanced through the combination of multiple QTL, compared to individual QTL alone. The improvement in resistance largely depends on the combination of specific QTL working together.

In particular, *Q_{Sr.umb-2A.1}* appears to play a central role in conferring resistance, with the presence of additional QTL providing further increases to the resistance present.

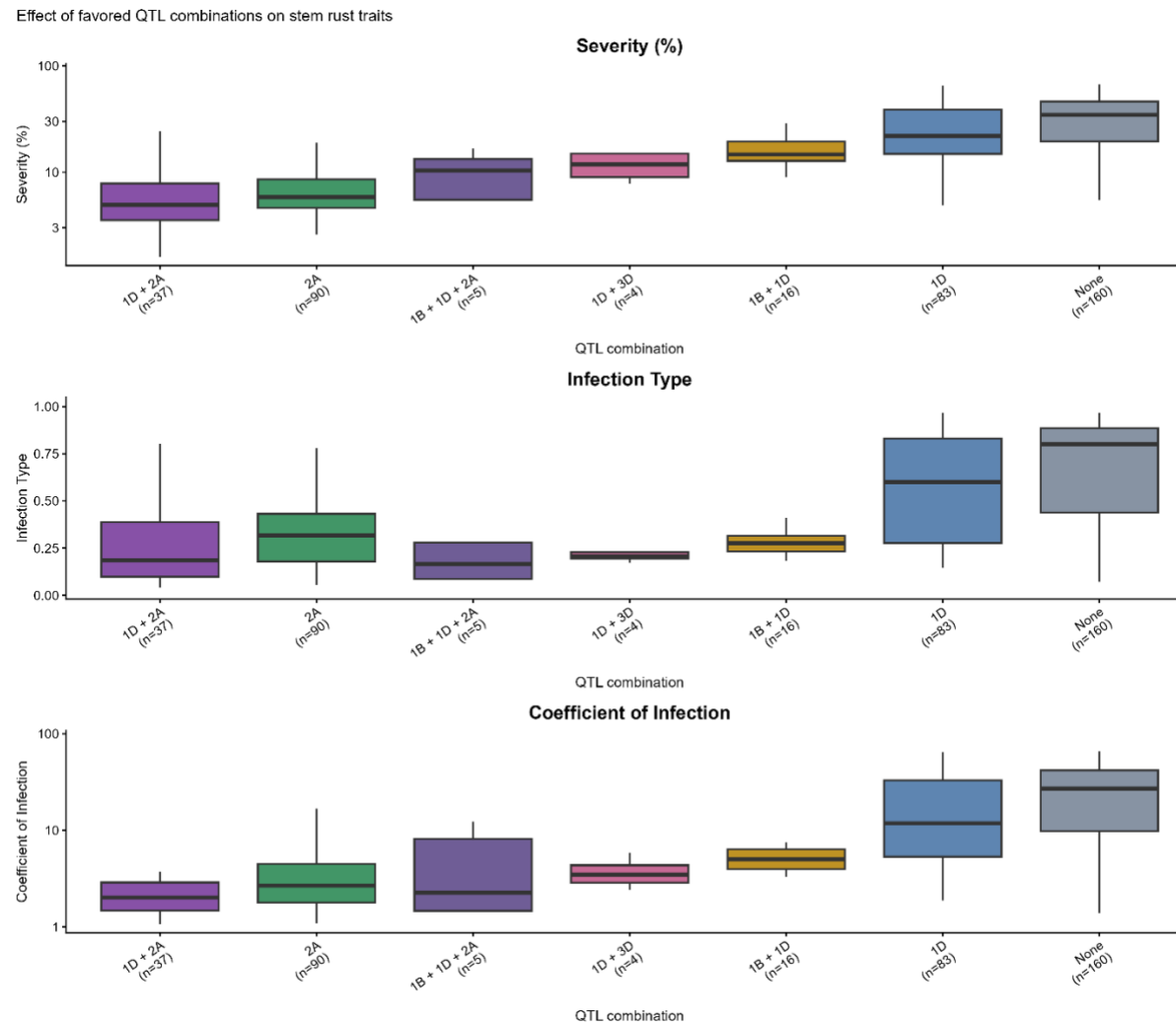


Figure 3.5.2 Boxplots showing the effect of the selected QTL combinations on stem rust severity (%), infection type (IT, and coefficient of infection (COI) based on the pooled field BLUPs. Genotypes were grouped according to the presence of combinations of *QsSr.umb-1B.1*, *QsSr.umb-2A.1*, *QsSr.umb-1D*, and *QsSr.umb-3D* with sample sizes (n) indicated for each group.

3.6 Discussion

The association mapping population exhibited a wide range of phenotypic variation for stem rust resistance across both seedling and adult plant evaluations, providing support for the application of genome-wide association analysis of this population. In the field trials, severity and COI exhibited left-skewed distributions, with ratings for severity ranging from 2.39–70.39 and COI from 1.09–70.04 across all site-years and the pooled dataset prior to transformation. Infection type exhibited a bimodal distribution with observations ranging from 0.04–0.97, indicating the population displayed resistant and susceptible reactions based on the classification of infection types.

At the seedling stage, phenotypic distributions were varied across the seven *Pgt* races evaluated. Some races were highly virulent on the population, while others showed a greater tendency towards avirulence. The differences observed highlight the presence of race-specific resistance at the seedling stage and suggest that distinct resistance alleles or genes are present. Overall, the broad phenotypic variation observed across both seedling and adult developmental stages indicates that the population has a wide spectrum of reactions to the races being examined in this study.

Population structure analysis of the association mapping population identified the presence of two ancestral subpopulations using sNMF analysis. However, individuals were not clearly separated into two discrete groups; the ancestry coefficients showed that individuals often exhibited varying amounts of genetic relatedness between the two identified subpopulations. Given that the population is comprised primarily of breeding lines from western Canada, with the remaining individuals originating from the United States, Europe, and eastern Canada, it is

unsurprising that two subpopulations would be identified. Much of the breeding material utilized in this study originated from the Lethbridge Research and Development Centre (LeRDC), part of Agriculture and Agri-Food Canada. It is known that much of the genetic background for the lines developed at LeRDC originated from the Central Great Plains/Pacific Northwest regions of the United States and from Europe (Laroche et al. 2019) This provides context for the identification of the two ancestral subpopulations.

Linkage disequilibrium analysis further supported the suitability of the population for GWAS. LD decayed relatively rapidly at short physical distances, with a half-decay point observed at approximately 2.75 Mbp, whereas background LD was reached at 18.5 Mbp. This pattern indicates that the population contains sufficient historical recombination to provide useful mapping resolution, while also showing that associated markers may represent broader genomic regions rather than single causal genes. Based on this result, a 5.5 Mbp window was used to define QTL regions; this provides a threshold for identifying linked markers.

GWAS of the population at the adult stage in the field trials identified multiple QTL associated with stem rust resistance, indicating that resistance within this population may be controlled through the effects of multiple genes or loci associated with resistance. A total of 16 QTL were detected across nine chromosomes, with certain QTL explaining larger proportions of the phenotypic variance observed in the population. Of the QTL identified from GWAS, several were detected across multiple environments, including the pooled dataset, and in multiple traits. These were *Q_{Sr.umb-1B.1}*, *Q_{Sr.umb-2A.1}*, *Q_{Sr.umb-2A.4}*, and *Q_{Sr.umb-2D.1}*. The detection of these QTL suggests that they may represent stable sources of resistance within the population.

In the seedling evaluations, a total of 22 unique QTL were identified across the races being evaluated for seedling resistance within the population. The number of QTL identified varied

among individual races, indicating differences in the virulence of the races. Several of the seedling QTL were identified in multiple races; these include *QSr.umb-3D*, *QSr.umb-1B.1*, and *QSr.umb-6A*, indicating that these QTL may confer a broader spectrum of resistance to the races being evaluated. In contrast, other QTL were detected in only a single race, such as *QSr.umb-7A.1*, *QSr.umb-1B.2*, and *QSr.umb-2B.4*.

Two classes of resistance genes are associated with rust resistance in wheat; this includes race-specific (*R* genes) and adult plant resistance (APR) genes. The *R* genes function and provide protection from the seedling stage into the adult growth stages. These genes are often referred to as seedling or all-stage resistance genes and tend to be race-specific. APR genes are often race-non-specific and are only effective at the adult stage (Ellis et al. 2014; Periyannan et al. 2017; Karelov et al. 2022). Among the QTL identified in this study, several coincide with known race-specific resistance genes based upon the significant markers identified in the GWAS analysis, these include *Sr38* (*QSr.umb-2A.1*), *Sr31* (*QSr.umb-1B.1*), *Sr24* (*QSr.umb-3D*), *Sr6* (*QSr.umb-2D.2*), and a candidate for *Sr33* (*QSr.umb-1D*). These are discussed in more detail below.

The most prominent QTL identified from the field trials was that of *QSr.umb-2A.1*, which was detected in each site-year and the pooled dataset. Additionally, it was associated with all three field traits evaluated. This QTL had PVE values ranging from 7.67–28.12% (Table 3.4). Many wild wheat relatives have been used to introduce sources of resistance into bread wheat, one example being *Aegilops ventricosa*, which was utilized in an attempt to introduce resistance for eyespot into wheat (Bariana and McIntosh 1993; Helguera et al. 2003; Gao et al. 2021). A translocation occurred on the short arm of chromosome 2A in wheat, giving rise to the 2AS/2NS translocation which provides resistance to stem, stripe and leaf rust. This translocation carries the resistance gene *Sr38*, which is perfectly linked with *Yr17* and *Lr37* (Bariana and McIntosh 1993;

Helguera et al. 2003). These genes have been deployed into a number of lines globally and are known to be present within the breeding material here in western Canada; this translocation has been relied upon heavily by the hard winter wheat breeding program in Kansas (Bariana and McIntosh 1993; McIntosh et al. 1995; Helguera et al. 2003; Laroche et al. 2019; Gao et al. 2021; Karelov et al. 2022). A number of markers in this study were identified to be significant on the short arm of chromosome 2A, with positions ranging from 259,213 base pairs up to 27.2 Mbp. The estimated size of the translocation is believed to be 32.6 – 33.5 Mbp, and the significant markers detected in this study all fall within these estimated parameters (Gao et al. 2021). Due to the presence of the translocation on 2AS, the 5.5 Mbp LD window used to define boundaries for if markers belong to the same QTL does not apply. Since the translocation is an alien chromosomal segment, recombination is suppressed and markers across the region remain in high LD, causing them to be inherited as a single block. Consequently, many of these significant markers could be used to detect the presence of this translocation in future breeding lines. This translocation has been identified in winter wheat cultivars Emerson and AAC Icefield, which were developed at LeRDC and are present within the population (Laroche et al. 2019). Interestingly, *Sr38* is known to be an all-stage seedling resistance gene, yet no QTL identified at the seedling stage were associated with this gene. Studies evaluating the stem rust resistance genes present in winter wheat cultivars and elite breeding lines present in the United States identified that *Sr38* was the most prevalent gene detected in their winter wheats and it was the most effective at the adult stage in their field tests. Based upon seedling evaluations and previous literature, *Sr38* appears to provide mixed infection type responses at the seedling stage despite being effective against many of the North American *Pgt* races (McIntosh et al. 1995; Jin et al. 2007; Olson et al. 2010; Zhang et al. 2014). In the seedling evaluations conducted, mixed results were observed with the differential

sets, *Sr38* provided a mixed response to the different races evaluated ranging from susceptible to resistant. Interestingly, knowing that *Sr38* has been confirmed to be present in Emerson and AAC Icefield, the seedling evaluations showed that Emerson was susceptible to all of the races evaluated while AAC Icefield had resistant responses to four of the seven races evaluated and was susceptible to the remaining three. These results agree with the conclusion from other studies that *Sr38* is more effective at the adult plant stage (Bariana and McIntosh 1993; McIntosh et al. 1995; Jin et al. 2007; Olson et al. 2010; Zhang et al. 2014; Laroche et al. 2019).

Q_{Sr.umb-1B.1} was the next major QTL identified across all environments, site-years, and traits. This QTL was also identified in the seedling evaluations of races QTHSF, RTHSF, TMRTF, and TPMKC. In the field trials this QTL had PVE values ranging from 2.21–7.92%, (Table 3.4). For the seedling evaluations, the PVEs were 4.54%, 9.60%, 4.73% and 4.65% (Table 3.5). Similar to the QTL identified on 2AS, this QTL was associated with markers across the short arm of chromosome 1B with positions ranging from 6.7 to 236 Mbp. On 1BL, a translocation from the short arm of chromosome 1 from *Secale cereale* (rye) is present in many wheat cultivars globally; this translocation is known as 1BL.1RS and it spans the short arm of chromosome 1B in wheat (Mettin et al. 1973; Zeller 1973; McIntosh et al. 1995; Burnett et al. 1995). The length of chromosome arm 1BS is estimated to be 315 Mbp (Janda et al. 2006). As with the 2AS/2NS translocation, markers within the 1BL.1RS translocation are inherited together with little to no recombination, so the typical 5.5 Mbp LD decay window is not applicable to markers in this region since they are inherited as a single linkage block. On this translocation resides the gene *Sr31* which until early 1999 widely conferred resistance to races of *Pgt* around the world. However, a new race designated TTKSK (also known as Ug99) arose that was virulent on *Sr31* (Pretorius et al. 2000; Fetch et al. 2021). In addition to the presence of *Sr31* on this translocation, the genes *Lr26* and *Yr9*

are present too (McIntosh et al. 1995). Further use of the 1BL.1RS translocation is not recommended for use within breeding programs, because this translocation is associated with negative impacts on grain quality. In particular, dough made from wheats with the translocation are often found to have increased stickiness which is undesirable (Burnett et al. 1995; Zhai et al. 2024). While *Sr31* conveys resistance to all *Pgt* races present in North America, the fact it contributes to decreased quality, breeders may wish to prioritize other resistance genes and select against lines that contain this gene.

The next notable QTL identified was *Q_{Sr.umb-3D}* which was detected in both field and seedling evaluations across multiple races. In the field, this QTL was associated with IT, it was detected in CRM23 and the pooled dataset. In the field evaluations this QTL had a PVE value of 7.90% in CRM23 and 2.47% in the pooled dataset (Table 3.4). This QTL was also identified in the majority of the seedling evaluations; it was detected in six of the seven races evaluated in this study. These races were QTHSF, RHTSF, RKQSF, RTHSF, TMRTF, and TPMKC. This QTL had PVE values of 7.64%, 5.84%, 5.02%, 15.00%, 10.71%, and 7.47%, respectively (Table 3.5). Multiple significant markers were identified spanning from 594 – 614 Mbp on the long arm of chromosome 3D. A translocation of 3Ag from *Agropyron elongatum* into wheat previously occurred, and present on this translocation is the *Sr24* gene which is linked to *Lr24* (McIntosh et al. 1977, 1995; Mago et al. 2005; Karelov et al. 2022). As with the previously discussed translocations, markers with this introgressed region are inherited together with recombination not occurring, meaning the typical 5.5 Mbp LD window does not apply here. Previous studies have identified the following winter wheat cultivars to have the presence of *Sr24/Lr24*, these include Broadview, Flourish, AAC Gateway, AAC Elevate, and AAC Goldrush (Laroche et al. 2019). *Sr24* is an all-stage gene, in the field evaluations it was only detected in CRM23 and the pooled dataset,

suggesting that this gene may be present in the population but at a relatively low frequency. The number of lines in the population increased from 2023 to 2024, which may have altered the frequency of this resistance loci between the 2023 and 2024 datasets. In the previous literature, it has been shown that *Sr24* is effective against all the races present in North America; the testing of the differential lines confirms this (Olson et al. 2010; Zhang et al. 2014). Although not detected in race MCCFC in this study, the rating for this gene in the differential set was 1/1⁺, indicating effectiveness. The majority of the association mapping population was resistant to MCCFC, so the lack of detection for this QTL could be indirectly due to the limited phenotypic variation for this race or perhaps the resistance provided by this QTL was masked by other genes that are more prevalent in the population.

In contrast to many of the field and seedling detected QTL detected and discussed above, some QTL were identified only at the seedling stage. Among these, *QSr.umb-2D.2* was detected with races MCCFC, TMRTE, and TPMKC, with PVE values of 5.50%, 6.21%, and 11.87% (Table 3.5). Significant markers were detected around position 56 Mbp. Based upon the ancestral knowledge of this population, it is postulated that this QTL coincides with the resistance gene *Sr6*. This gene is located on chromosome 2D and was originally identified in the Canadian cultivar McMurachy; it has also been documented in winter wheats in the United States (McIntosh et al. 1995; McCallum et al. 2007; Zhang et al. 2014; Mourad et al. 2018). The detection of this QTL across multiple races suggests that *Sr6* may provide effective race-specific resistance to the *Pgt* races evaluated in this study. However, its absence in other races may indicate variability in virulence within the pathogen population, highlighting the importance of combining *Sr6* with additional resistance genes to achieve more durable resistance. This QTL was not identified in the results from the field trials. Previous literature suggests that *Sr6* is less effective at temperatures

greater than 20°C, which could be indicative as to why this QTL was not detected in the field study as rating took place during July when temperatures outside were well above 20°C (Forsyth 1956; Luig 1972; Hewitt et al. 2025).

Finally, *Q_{Sr.umb-1D}* was identified in both the field and seedling evaluations. It was associated with IT in CRM23 with a PVE of 4.66% (Table 3.4) and with races RTHSF and TPMKC with PVEs of 5.70% and 5.05% (Table 3.5), respectively. The marker ‘Ra_c69552_1419’ at position of 8,721,360 bp is the only marker associated with this QTL identified. It is known that the *Sr33* gene resides on distal end of chromosome arm 1DS of wheat (Jones et al. 1990, 1991). This gene confers all-stage resistance for *Pgt* and is effective against Ug99 and related races, though it was found to be ineffective to newer races emerging in Spain back in 2018. This gene originated in *Aegilops tauschii* and was transferred into wheat (Kerber and Dyck 1979; Jones et al. 1990, 1991; Czarnecki and Lukow 1992; Pshenichnikova and Maystrenko 1995; Jin et al. 2007; Olivera et al. 2022b). The physical position of this QTL and the phenotypic data suggest that *Q_{Sr.umb-1D}* may correspond to *Sr33*, although further validation would be needed to confirm this association. Current literature does not describe the deployment of *Sr33* in North American germplasm. One additional consideration is that *Sr33* is known to be linked to *Sr45* on 1DS, which further indicates the importance of this genomic region for stem rust resistance (Marais et al. 1998). Ultimately with only one marker being identified in this region and limited evidence for the presence of *Sr33* being present in germplasm available in North America, further investigation is needed.

Aside from the QTL that have been identified and are likely associated with known/candidate *Sr* genes, additional loci associated with resistance were identified across the field and seedling evaluations. Among the field trials, three QTL were detected across multiple

environments and were associated with multiple field traits, with varying phenotypic variances. *QSr.umb-2A.4* was detected in all three site-years and the pooled dataset, it was associated with severity, IT, and COI. The detection across multiple site-years and PVE values ranging from 5.06–19.40%, suggests this QTL provides stable resistance. *QSr.umb-2D.1* was identified in all site-years and the pooled dataset, it is associated with all three field traits with PVE values up to 11.88%. The identification of the loci across multiple traits and site-years suggest that they may be a reliable source of resistance in the population. Though they do not correspond to well-characterized resistance genes, these loci evidently play a role in the adult plant resistance observed in the field trial evaluations. Suggesting that these loci may be targets for further exploration and incorporation into the breeding program.

Finally, *QSr.umb-3B.2* was identified in CRM24 and WPG24 for IT, and the pooled dataset for IT and COI. PVE values ranged from 2.77–13.23%. The marker identified to be associated with *QSr.umb-3B.2* is located on the long arm of chromosome 3B at 804.2 Mbp. A previous study, evaluating seedling and adult plant resistance to stem rust in a spring wheat population, identified a QTL on 3BL at 802.9 Mbp associated only with seedling resistance (Pradhan et al. 2023). These results suggest this region contains loci associated with stem rust resistance.

Additional QTL were identified in the field trials, which had minor effects and were often observed in limited number of environments and traits. *QSr.umb-1A.2* was identified in both the field and seedling evaluations. In the field, this QTL was associated with IT in the pooled dataset, in the seedling evaluations it was detected in races RTHSF and TPMKC with stronger effects. The marker associated with this QTL in the pooled dataset is ‘AX-94844830’, it has a position of 11,822,296 bp, while at the seedling stage the associated marker is ‘RFL_Contig5637_1008’ at 9,580,143 bp, these markers fall within the 5.5 Mbp window used to determine linkage on

chromosome 1A and if markers belong to the same QTL. A study conducted by Miedaner et al. (2024) evaluated seven populations of winter wheat at the adult plant stage to identify resistance to stem rust. Within one of their populations, they identified a QTL for resistance on chromosome 1A at 7,660,991 bp. While this position does vary slightly from those detected in this study, it does suggest there may be resistance loci on 1AS that could be of value to breeding programs. Other minor field QTLs include *QSr.umb-1A.3*, *QSr.umb-1A.4*, *QSr.umb-2A.2*, *QSr.umb-2A.3*, *QSr.umb-2B.1*, *QSr.umb-2B.2*, *QSr.umb-2B.3*, and *QSr.umb-5A*. Most of these QTL explained less than 5% phenotypic variance and were only detected in one or two site years. These QTL explained smaller proportions of phenotypic variance, their detection across one or more environments suggests they may contribute additively to resistance when combined with other resistance loci.

In the seedling evaluations, two QTL were identified across multiple races. *QSr.umb-6A* was detected in five of the seven *Pgt* races evaluated: QTHSF, RHTSF, RTHSF, TMRTF, and TPMKC. PVE values for this QTL ranged from 4.24% to 10.16%, with the strongest effects observed in RTHSF at 10.16%. *QSr.umb-3B.1* was identified in races QTHSF, TMRTF, and TPMKC, with PVE values ranging from 8.73% to 9.93%. The strongest effects were observed in TMRTF at 9.93%. The detection of these QTL across multiple races highlights their potential utility in conferring resistance to diverse pathogen isolates.

A number of QTL were identified in the seedling evaluations but were detected in only one or two races. *QSr.umb-1A.1* in QTHSF and TMRTF; *QSr.umb-1B.2* in RHTSF; *QSr.umb-2B.4* and *QSr.umb-2B.5* both in TPMKC; *QSr.umb-3A.2* in QTHSF; *QSr.umb-3A.3* and *QSr.umb-5B* in RTHSF and TMRTF; *QSr.umb-6B* in MCCFC and RHTSF; *QSr.umb-7A.1* in MCCFC; *QSr.umb-7A.2* in TMRTF; *QSr.umb-7B.1* in TMRTF; and *QSr.umb-7B.2* in RKQSF. Most of these QTL explained less than 5% of the phenotypic variance. Although many of these loci only conferred

resistance to select races, these loci could potentially be stacked to confer more widespread and durable resistance within breeding material.

Finally, several QTL with unknown chromosomal positions were identified in this study, many of which were detected in the field trials. The LD analysis of the markers involved in these associations suggested that many of the markers were associated with *Qsr.umb-2A.1*, *Qsr.umb-3B.1*, and *Qsr.umb-3D*. Attempts were made to determine the physical location of these markers by BLAST searches in CSv1.0 (IWGSC et al. 2018), which were unsuccessful. These markers were then categorized as unknown. These QTL were detected across multiple environments and were associated with multiple traits. An example of this includes markers ‘AVRIG21480’, ‘AVRIG26841’, ‘AVRIG32301’, ‘AX-158596552’, ‘Excalibur_c25599_359’, ‘Excalibur_c18324-390’, and ‘Kukri_c24446-306’ (Table 3.4). These markers were associated with all site-years and the pooled dataset, plus had an association with all three field traits that were evaluated. Though these QTL had been categorized to be unknown in terms of their positions, the results are indicative that they contribute to resistance within the association mapping population. For future use of these loci, aligning to more recent reference genomes like CSv2.1, may be able to help determine the location of these markers. By determining the position of these markers, this will determine if they are associated with known resistance genes, belong to regions where QTLs identified in this study are located, or represent novel loci.

The QTL stacking analysis provides further insight into the genetic architecture of stem rust resistance in the association mapping population. While resistance generally improved with the accumulation of multiple QTL, the results demonstrated that the contributions provided by these QTL are not equal. Among the QTL evaluated, *Qsr.umb-2A.1* had the largest impact on resistance with lines carrying this QTL consistently performing better than those without it. When this QTL

is used in combination with others, improvement can be seen for all three field traits evaluated. The strong effect that this QTL provides, along with the evidence suggesting the presence of the 2AS/2NS translocation which carries *Sr38*, indicates that this locus is of great importance for the winter wheat breeding program at the University of Manitoba. *Q_{Sr.umb-3D}* also contributed strongly to resistance and was associated with reduced disease levels across all traits, though most notably in IT. The detection of this QTL in both the field and seedling evaluations suggests that it provides more broad-spectrum resistance, furthering support this this QTL corresponds to *Sr24*. *Q_{Sr.umb-1B.1}* displayed a more moderate effect on resistance in comparison to *Q_{Sr.umb-2A.1}* and *Q_{Sr.umb-3D}*, indicating that while it provides resistance it may function better in conjunction with other QTL associated with resistance. In contrast, *Q_{Sr.umb-1D}* alone provide limited resistance, as lines carrying only this QTL showed disease levels like those lacking the evaluated QTL. These results demonstrate that resistance to stem rust in the population is largely influenced through the identification of major QTL. Not only does the presence of these QTL influence the disease resistance, but the combinations that they are in plays a major role too. Based on the results *Q_{Sr.umb-2A.1}* functions as a key driver for resistance in the population, followed by *Q_{Sr.umb-3D}* and *Q_{Sr.umb-1B.1}* which provide moderate effects. *Q_{Sr.umb-1D}* was found to be present widely in the population, though by itself does not provide much resistance calling into question the previous plausibility that suspected *Sr33* at this locus. These findings emphasize that effective resistance breeding should prioritize the selection of key QTL and QTL/gene stacking to maximize the resistance and durability of our cultivars.

3.7 Conclusion

In this study, GWAS identified 16 QTL associated with stem rust resistance at the adult plant stage from field trial evaluations and 22 QTL at the seedling stage. Based on the GWAS results and

comparison with previous literature, it is likely that *Sr6*, *Sr24*, *Sr31*, and *Sr38* are present within the association mapping population. Present findings also suggest a QTL in the region of *Sr33* may have been identified, but further confirmation suggest as marker testing is needed. In addition to the genes postulated to be present in the population, other resistance associated QTL were identified. Though limited in the literature surrounding these QTL, these loci may serve as useful targets for further evaluation and incorporation into breeding material within the winter wheat breeding program at the University of Manitoba. The unknown QTL identified in this study warrant further investigation to determine if these loci associate with already detected QTLs/regions of interest or pose new regions of interest.

The results of this study provide insight into the complex nature of stem rust resistance, showing the influence that major and minor QTL play in resistance. The QTL stacking analysis demonstrated that resistance is largely influenced by the presence of specific QTL and the combinations in which they are found in, which provides further support for the need to stack resistance genes to ensure deployment of durable stem rust resistance in CWRW varieties. Future work should focus on confirming the presence of these putative genes within the population and exploring the influence of the minor QTL identified. This study helps to deepen our understanding of the stem rust resistance present within the winter wheat germplasm present in Canada and demonstrates the value of stacking resistance loci for achieving durable resistance.

Chapter 4: EVALUATION OF GENOMIC SELECTION FOR STEM RUST RESISTANCE IN WESTERN CANADIAN WINTER WHEAT (*Triticum aestivum* L.)

4.1 Abstract

Stem rust, caused by the fungal pathogen *Puccinia graminis* f. sp. *tritici* (*Pgt*), is a globally known, significant and destructive disease of wheat (*Triticum aestivum* L.). Continuous breeding of resistant cultivars is essential to mitigate yield losses and monitor evolving pathogen populations. The objective of this study is to evaluate the suitability of implementing genomic selection (GS) into winter wheat breeding programs by evaluating the predictive accuracy of eight models. These included the parametric models Genomic Best Linear Unbiased Prediction (GBLUP), Ridge Regression (RR), Bayesian models (BayesA, BayesB, and BayesC), and Least Absolute Shrinkage and Selection Operator (LASSO), as well as the semi-parametric model Reproducing Kernel Hilbert Spaces (RKHS) and the machine learning model Random Forest (RF). The population utilized in the study consisted of 326 hard red winter wheats from western Canada and the United States. The population was phenotyped for stem rust resistance in inoculated field trials and seedling assays with a mixture of seven *Pgt* races. Genotypic data were obtained using two high-density SNP chips, a 40K Wheat Barley Infinium array and a 25K Wheat Infinium array. Data were collected on stem rust field severity and infection response. Infection type data was recorded from indoor seedling inoculations with individual races. Model performance was evaluated using ten-fold cross validation, with prediction accuracies reported as Pearson's correlation. Prediction accuracies were high in the evaluation of the field traits with values ranging from 0.743 to 0.810. Severity consistently exhibited the highest prediction accuracy, followed by infection type and COI. Prediction accuracies for the seedling evaluations ranged from 0.528 to 0.772. Among models, RF and Bayesian approaches typically provided the highest accuracies. The results from

this study demonstrate the GS is a promising approach for improving stem rust resistance in winter wheat breeding programs, particularly for field traits.

4.2 Introduction

Wheat (*Triticum aestivum* L.) is one of the most widely grown field crops in Canada with a total of 40.0 million tonnes produced in 2025 (Statistics Canada 2025). Spring wheat and durum (*Triticum turgidum* L.) are the predominant forms grown in Canada, while winter wheat makes up a very small portion of the total production, with most winter wheat grown in Ontario (Statistics Canada 2025). As a major crop both nationally and globally, the threat of yield loss due to disease and environmental factors must be continually evaluated to prevent significant reductions.

Stem rust, caused by *Puccinia graminis* f. sp. *tritici* (*Pgt*) remains a persistent threat to global wheat production, although it has largely been controlled through the incorporation of genetic resistance into many widely grown cultivars. The emergence of highly virulent races, such as Ug99 and its variants, have renewed global interest and efforts to develop cultivars with durable stem rust resistance (Pretorius et al. 2000; Laroche et al. 2019; Aboukhaddour et al. 2020).

Resistance to stem rust can be categorized into all-stage resistance (seedling) and adult plant resistance (APR). Seedling resistance genes are effective through all developmental stages and are often race-specific. APR genes are only effective at the adult plant stage and tend to be non-race-specific (Leonard and Szabo 2005; Ellis et al. 2014; Periyannan et al. 2017). APR genes tend to be less likely to be overcome because they are not specific to individual *Pgt* races, whereas seedling resistance genes are typically race-specific (Laroche et al. 2019). As *Pgt* continues to evolve, ensuring that multiple resistance genes are present in developing lines is critical for maintaining broad-spectrum and durable resistance.

For many breeding programs, traditional phenotypic selection for stem rust resistance requires substantial resources, making it a time-consuming and often an expensive expenditure.

Although it is effective for the development of rust resistant varieties (Ellis et al. 2014), efforts have been made to streamline the selection process. The use of DNA markers has become more common to select for lines with known resistance genes or to aid in pyramiding genes (Ellis et al. 2014). This approach, known as marker-assisted selection (MAS), has aided in making advantageous genetic gains. However, for traits such as stem rust resistance that are often controlled by multiple QTL with varying effects, MAS may be less effective (Collard & Mackill 2008; Rutkoski et al. 2011).

Genomic selection (GS) is an alternative approach that addresses many of the limitations associated with both traditional phenotypic selection and MAS. Rather than focusing on a limited number of markers, GS incorporates information from markers distributed across the entire genome to estimate the genomic estimated breeding values (GEBVs) (Meuwissen et al. 2001). This allows for selection decisions to be made using only the genotypic data, allowing for earlier selection and reducing the need for extensive phenotyping. In turn this may provide savings for breeding programs such as reducing the financial costs associated with phenotyping and reducing the amount of time that may be required to identify lines with superior traits.

The effectiveness of GS is influenced by several factors, including the genetic basis of the trait, the size and composition of the training population, and marker density (Lorenz et al., 2011; Howard et al., 2014). A variety of models are available for GS analyses, these include parametric approaches like GBLUP and the Bayesian models (e.g., BayesA, BayesB, and BayesC), semi-parametric models like reproducing kernel Hilbert spaces (RKHS) and machine learning models like Random Forest (RF) (Heslot et al., 2012; Charmet et al., 2020). These model types differ in how marker effects are estimated and the computational time required to run the models successfully.

The objective of this study was to evaluate the suitability of implementing genomic selection in western Canadian winter wheat breeding programs for stem rust resistance by assessing the prediction accuracy of eight models: GBLUP, Ridge Regression, BayesA, BayesB, BayesC, LASSO, RKHS, and Random Forest.

4.3 Materials and Methods

4.3.1 Plant materials, Phenotypic Data, and Genotypic Data

Details of genotypic and phenotypic collection for this study are described in Chapter 3. A subset of the association mapping population, comprising of 326 lines was used to evaluate the suitability of genomic selection for stem rust resistance.

To recap, the association mapping population underwent the evaluation of its response to inoculation of stem rust in seedling and field trials. In the field trials the population was evaluated for severity, infection type, and coefficient of infection, whereas, at the seedling stage infection type responses were evaluated. Field trials took place over two growing seasons, the first being during the 2022–2023 growing season in Carman, Manitoba and in the following growing season of 2023–2024 in Carman and Winnipeg, Manitoba. Seedling assays were conducted between January to February 2024.

The subset of the population being used for the evaluation of genomic selection (GS) is comprised of individuals that originate from western Canada and from the western United States. Much of the germplasm from the United States was used in the development of current western Canadian germplasm, making this subset of the population much more closely related compared to the full association mapping population.

META-R (Alvarado et al. 2020) was used to calculate the adjusted means for the subset of the population. The best linear unbiased predictions (BLUPs) were estimated for the pooled environmental dataset of the field trial data, the seedling data was converted to a 1–9 scale from the Stakman scale that was used to collect the data (Fatima et al. 2020). The linear ratings of the 1–9 scale were used for the application of GS.

DNA of the population was extracted using the Patricia Warner Wheat and Barley protocol (Pallotta et al. 2003). Genotyping utilized the Illumina Infinium Wheat Barley 40K v1.0 BeadChip (Keeble-Gagnère et al. 2021) and the 25K Wheat Infinium Array from SGS TraitGenetics (Gatersleben, Germany). The same marker set utilized in Chapter 3 are used here for the application of GS, for a total of 43,362 markers.

4.3.2 Genomic Selection Analyses

To conduct the analysis for the suitability of GS with the association mapping population the R package BreedWheat Genomic Selection pipeline (BWGS) version 0.2.1 (Charmet et al. 2020) was used in this study. BWGS is able to impute missing marker data, reduce the number of individuals in the training population and/or the number of markers being utilized, and calculate the genomic estimation of breeding values (GEBV) utilizing 15 different parametric/non-parametric models (Charmet et al. 2020). Genotypic data consisted of all 43,362 SNP markers obtained from the association mapping population, the minor allele frequency was set to 0.05. The mean allele frequency imputation (MNI) function was used to impute missing marker data, the data that was missing was replaced by the frequency of a given marker (Charmet et al. 2020). Phenotypic data were derived from the BLUPs for severity, IT, and COI obtained from the field evaluations. The linear ratings for the seedling evaluations were used.

For the purpose of this study eight models were evaluated for their suitability, six of these models are parametric and include genomic best linear unbiased predictors (GBLUP), ridge regression (RR), least absolute shrinkage and selection operator (LASSO), BayesA, BayesB, and BayesC; the semi-parametric model reproductive kernel Hilbert space (RKHS), and the machine learning model Random Forest (RF).

4.3.2.1 Parametric Models

Six parametric models were evaluated, each making different assumptions about how marker effects are distributed.

GBLUP assumes that the effects of markers are assumed to be evenly distributed across the genome and each marker contributes to genetic variance with small effects that are greater than zero. As part of the model, GBLUP uses a genomic relationship matrix (G matrix) to predict the phenotypes. It does not estimate the individual marker effects, but measures how genetically similar each individual in the population are based on the markers associated with these individuals. Phenotypes are predicted from these relationships (Whittaker et al. 2000; Meuwissen et al. 2001; VanRaden 2008; Heffner et al. 2009; Howard et al. 2022).

Ridge regression, also known as RR-BLUP, performs similarly to GBLUP; however, RR utilizes the markers directly and estimates each of the marker's effects. Like GBLUP it assumes that there is equal variance across all the markers being utilized and shrinks these effects toward zero (Whittaker et al. 2000; Meuwissen et al. 2001; Heffner et al. 2009; Lorenz et al. 2011; Howard et al. 2014).

Least absolute shrinkage and selection operator is a regression method that estimates the effects of markers by minimizing the difference between observed and predicted phenotypes. The

effects of some markers are shrunk towards zero, while others are determined to be exactly zero. LASSO can determine which markers are important to retain and those which have no effects and can be removed (Tibshirani 1996; Usai et al. 2009; Howard et al. 2014, 2022).

Three Bayesian methods were evaluated in this study. Each model has its own set of assumptions about how the effects of markers are distributed. BayesA determines that each marker has their own variance, each marker can have their variance shrunk towards zero independent of another, but some markers maintain larger effects. The effects of each marker follow a scaled t-distribution (Meuwissen et al. 2001; Heffner et al. 2009; Lorenz et al. 2011; Charmet et al. 2020; Howard et al. 2022). BayesB is derived from BayesA, but the variances of the markers are determined differently. In BayesB, a subset of the markers is deemed to have null or very small effects, and the remaining markers have a much larger effect that is different than being zero. BayesB utilizes a scaled t-distribution too for the markers that are identified to have an effect (Meuwissen et al. 2001; Heffner et al. 2009; Lorenz et al. 2011; Charmet et al. 2020; Howard et al. 2022). In BayesC, the assumption is that all SNP effects share a common variance. Similar to BayesB, BayesC assumes that a subset of the markers will have no effect, while the remaining markers contribute to the variation observed (Lorenz et al. 2011; Habier et al. 2011; De Los Campos et al. 2013; Charmet et al. 2020; Howard et al. 2022).

4.3.2.2 Semi-Parametric Models

Reproducing Kernel Hilbert Spaces (RKHS) uses kernel-based methods to model the effects of the markers. The kernel function will convert the marker data into set distances among all the observations (individuals); this then creates a positive definite matrix that allows the data to be used in a linear model framework. This method makes no assumptions about the marker data and

allows for more flexibility in how the genetic effects are determined (Gianola et al. 2006; Howard et al. 2014, 2022).

4.3.2.3 Machine Learning

Random Forest is a machine learning method that builds multiple decision trees and combines their results to make a prediction. The original population is bootstrapped to create multiple training sets; a tree is built from each of these subsets. The prediction from all of the trees are then combined to provide the final predicted outcome (Breiman 2001; Heslot et al. 2012; Reinoso-Peláez et al. 2022).

4.3.3 Cross-validation

The performance of each model was assessed using repeated cross-validation. The dataset was randomly divided into ten folds, nine folds used as the training population and one-fold used as the validation set. Each of the ten folds is iteratively assigned as the validation set. This process was repeated a total of 100 times. Prediction accuracies were calculated as the Pearson correlation coefficient (r) between observed phenotypic values and predicted GEBVs.

Following model evaluation, each model was used to generate genomic estimated breeding values (GEBVs) for all individuals using the full dataset. These predictions were used to rank individuals for potential selection.

4.4 Results

4.5 Phenotypic Distribution and Heritability of Field Traits

A subset of 326 lines from the association mapping population were evaluated for stem rust resistance at both the adult plant stage in field trials and at the seedling stage under controlled

conditions. For the field evaluations, three traits were assessed: severity, infection type, and coefficient of infection.

Heritability estimates for the field traits were high, ranging from 0.92 to 0.95 (Table 4.1). COI exhibited the highest heritability ($H^2 = 0.95$), followed by severity ($H^2 = 0.94$), and infection type ($H^2 = 0.92$). These results indicate that a large proportion of the phenotypic variation observed for these traits is associated with the genetics present in the population rather than the presence of environmental factors.

Table 4.1 Heritability estimates for the three field traits used to evaluate adult plant resistance in the association mapping population. The heritability was calculated from the pooled dataset.

Trait	Heritability ^a
Severity	0.94
Infection Type	0.92
COI	0.95

The distribution for the phenotypic values for each trait is presented in (Figure 4.5.1). Severity and COI exhibited left-skewed distributions, with a large proportion of the lines displaying lower disease levels. Suggesting the presence of resistant individuals present in the population. To contrast, infection type displayed a more prominent bimodal distribution. Indicating the presence of resistant and susceptible individuals residing in the population. Overall, the observed phenotypic variation across the traits suggests that the data is suitable for the application of genomic selection.

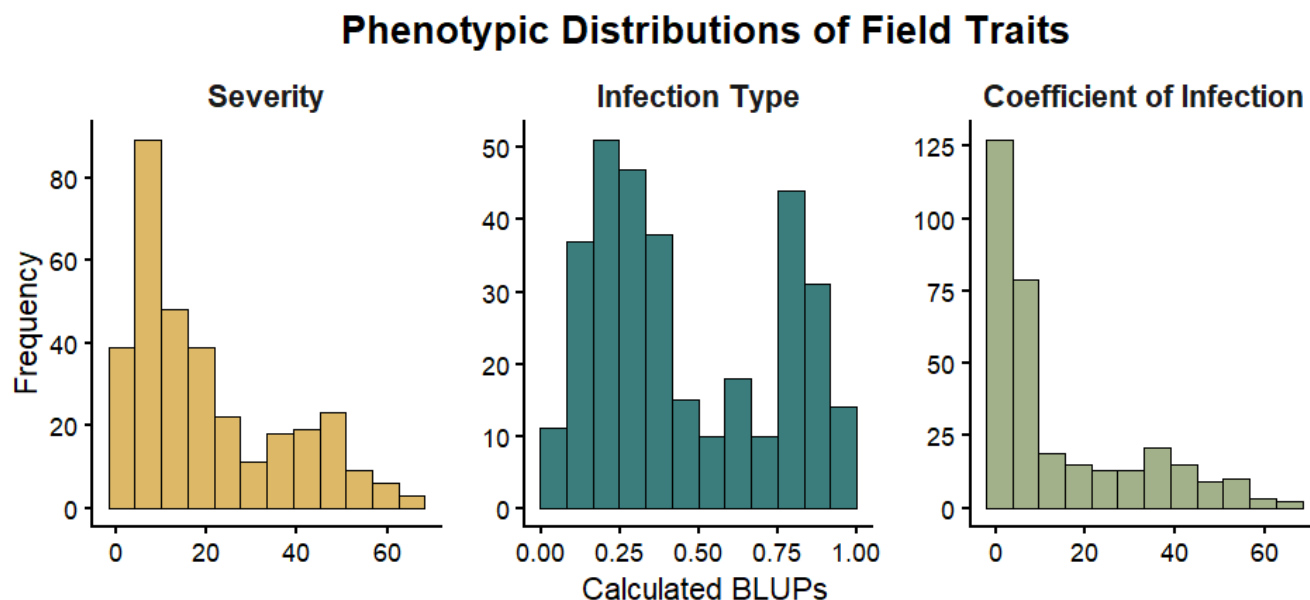


Figure 4.5.1 Distribution of the phenotypic values for the three field traits, stem rust severity, infection type, and coefficient of infection evaluated in the association mapping population ($n = 326$). Values represent BLUP estimates calculated from the pooled environmental dataset.

4.6 Prediction Accuracies for Adult Plant Resistance

Prediction accuracies for the field traits were determined using the calculated BLUP values. Unlike Chapter 3, the data were not transformed using RNOmni and the accuracies varied across the eight models being evaluated (Table 4.2). For severity, the accuracies ranged from 0.784 to 0.810 across the models with BayesB providing the highest prediction accuracy ($r = 0.810$), followed closely by BayesA ($r = 0.807$). For IT, the accuracies ranged from 0.771 to 0.787 with RF providing the highest accuracy ($r = 0.787$). COI displayed accuracies ranging from 0.743 to 0.784 and followed the same trend as IT with RF having the highest prediction accuracy ($r = 0.784$). Overall, genomic selection was most accurate for stem rust severity, followed by IT and COI.

Computational time varied greatly between the eight models evaluated in this study. GBLUP required the least amount of time to evaluate all three traits, requiring only 2.4 hours, while LASSO and RR required approximately 5.6 and 14.8 hours, respectively. In contrast, the

Bayesian models all required around 48 hours to evaluate the traits. Among the Bayesian models, BayesB achieved the highest prediction accuracy and required the longest computational time. For the non-parametric models RF required the longest amount of time to calculate at 145 hours, and RKHS required 16 hours (Figure 4.6.1).

The relationship between computational runtime and prediction accuracy for the field traits is shown in Figure 4.6.1. Prediction accuracies were relatively similar across all models for each trait, despite large differences in computational time. For severity, BayesB achieved the highest prediction accuracy, although several other models, including GBLUP, RKHS, and BayesA, showed comparable performance. A similar trend was observed for infection type and coefficient of infection, where differences in prediction accuracy among models were minimal. In contrast, computational runtime varied considerably among models, ranging from a few hours for GBLUP to over 100 hours for models such as Random Forest. Notably, models with substantially longer runtimes did not consistently result in improved prediction accuracies.

Table 4.2 Accuracy of eight GS models to predict field stem rust data (data pooled over environments).

Severity	GBLUP	RR	BayesA	BayesB	BayesC	LASSO	RKHS	RF
r ^a	0.796	0.788	0.807	0.810	0.798	0.784	0.799	0.788
SD ^b	0.006	0.006	0.007	0.005	0.007	0.010	0.006	0.006
Infection Type								
r	0.778	0.776	0.776	0.783	0.779	0.771	0.780	0.787
SD	0.006	0.006	0.008	0.007	0.006	0.010	0.006	0.007
COI								
r	0.764	0.759	0.764	0.769	0.766	0.743	0.773	0.784
SD	0.007	0.007	0.007	0.009	0.007	0.011	0.007	0.007
Time to run (hours)	2.40	14.76	47.66	49.43	48.04	5.63	16.04	145.20

^aPearson correlation coefficient

^bSD, standard deviation of r among all cross-validation iterations

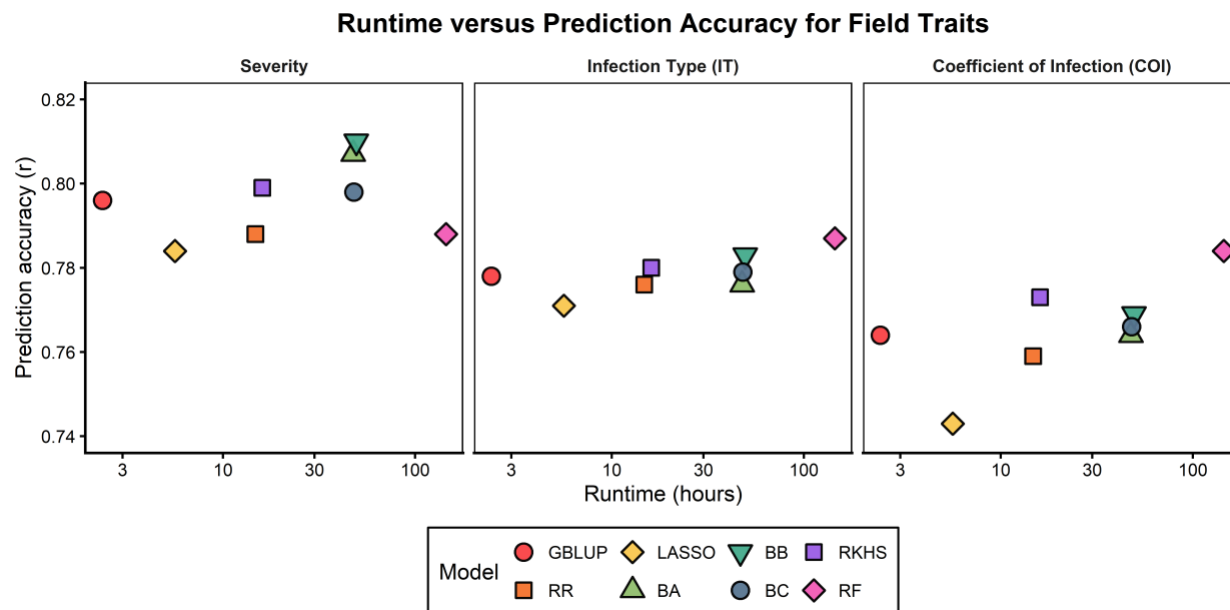


Figure 4.6.1 Relationship between computational runtime and prediction accuracy (r) for genomic selection models evaluated for field traits, including severity, infection type (IT), and coefficient of infection (COI). Each point represents the prediction accuracy of a single model for a specific trait. Runtime (hours) is presented on a $\log_{10}$ scale to account for the wide range in computational time among models. Models are distinguished by colour and shape. Abbreviations: GBLUP: Genomic Best Unbiased Linear Predictions; RR: Ridge Regression; LASSO: Least Absolute Shrinkage and Selection Operator; BA/BB/BC: Bayesian methods; RKHS: Reproducing Kernel Hilbert Spaces (RKHS); and RF: Random Forest.

4.7 Assessment of Predictive Performance and Model Fit

To assess model performance, the relationship between the observed phenotypes and the predicted values were examined using both the cross-validation (CV) predictions (Figure 4.7.1) and the calculated genomic estimated breeding values (GEBVs) derived from the full dataset of the 326 lines evaluated (Figure 4.7.2). BayesB was selected as the best performing parametric model across the field traits.

Under CV, the prediction accuracies, represented as Pearson's correlation (r), were 0.815 severity, 0.789 for IT, and 0.775 for COI. The linear relationships observed across the traits shows that the model can capture the genetic effects for the traits being evaluated. However, a wider spread of data points at higher phenotypic values suggests that the predictions at higher values may

not be as accurate. Since CV uses validation folds that are separate from the training population, these correlations provide estimates for how the model would perform on previously unseen phenotypes.

To contrast, the genomic predications presented in Figure 4.7.2 produced much higher correlation values with severity at 0.953, IT at 0.947, and COI at 0.946. In these plots, the data points cluster closely along the line of best fit, this does such a strong model fit. However, these values are inflated relative to the CV results, as when evaluating the GEBVs it uses the dataset that was utilized for both training and prediction.

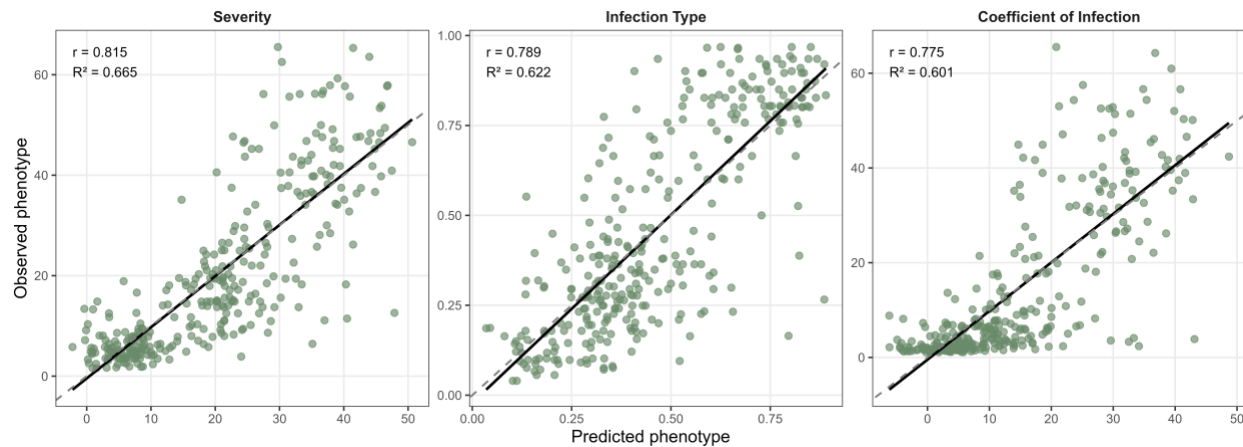


Figure 4.7.1 Observed versus predicted phenotypes for the BayesB model under ten-fold cross-validation for severity, infection type (IT), and coefficient of infection (COI) in the genomic selection subset ($n = 326$). Each point represents an individual genotype, with predicted values obtained from validation folds. Solid lines represent the line of best fit. Pearson's correlation coefficient (r) and coefficient of determination (R^2) are shown for each trait. These plots reflect the predictive ability of the model.

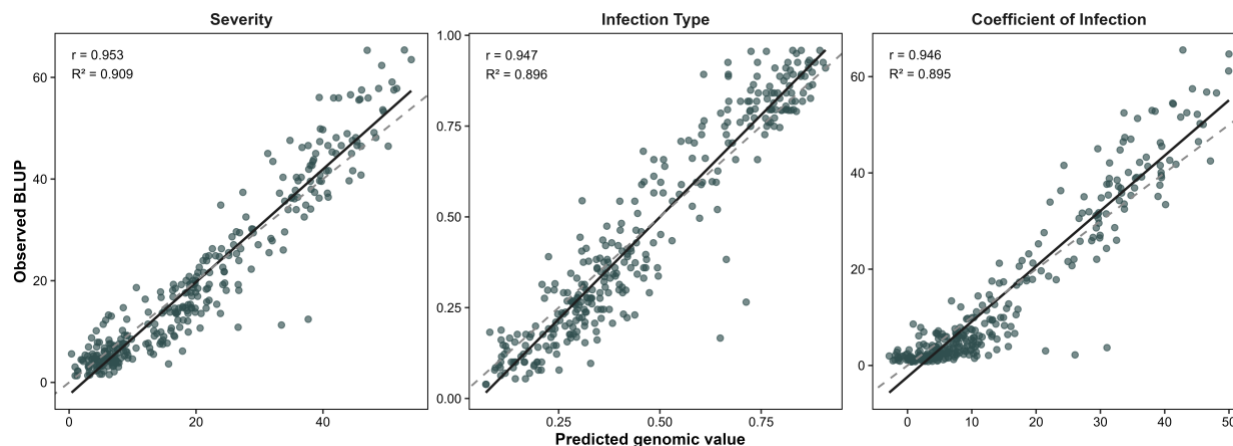


Figure 4.7.2 Observed phenotypes versus genomic estimated breeding values (GEBVs) predicted by the BayesB model using the full dataset for severity, infection type (IT), and coefficient of infection (COI) ($n = 326$). Each point represents an individual genotype. Solid lines represent the line of best fit, and dashed lines indicate the line of equality ($y = x$). Pearson's correlation coefficient (r) and coefficient of determination (R^2) are shown for each trait. These plots represent in-sample model fit and, therefore, show inflated correlations relative to cross-validation results.

4.8 Phenotypic Distribution of Seedling Evaluations

The phenotypic distribution of the seedling infection type responses varied across the seven *Pgt* races being evaluated in this study (Figure 4.8.1). Infection type responses were converted to a linear 1–9 scale, all the races exhibited bimodal distributions.

Large proportions of the population exhibited highly susceptible reactions to races RTHSF, TMRTF, and TPMKC (IT = 8–9). For races RKQSF and RTHSF, there was a more balanced split in the population, with approximately equal proportions of lines displaying resistant and susceptible reactions. Finally, MCCFC and QTHSF displayed a more centralized distribution with a greater proportion of the lines exhibiting resistant reactions.

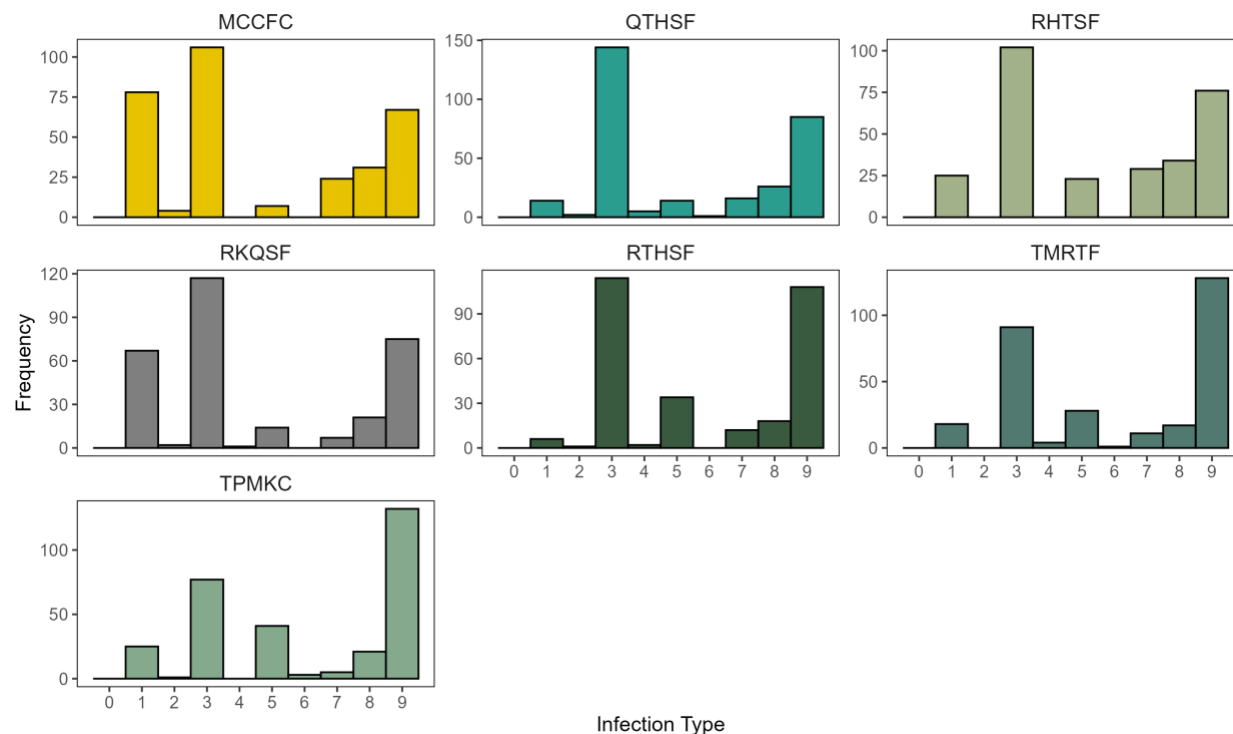


Figure 4.8.1 Phenotypic distributions of seedling infection type responses across the seven Pgt races evaluated in the genomic selection subset ($n = 326$). Histograms represent the frequency of discrete infection type scores (0–9) for each race.

4.9 Prediction Accuracies for Seedling Resistance

The models were evaluated using the seedling assay data (Table 4.3), where prediction accuracies were typically lower than those observed for the field traits. Prediction accuracies varied across the seven races evaluated with r values ranging from 0.530 to 0.772. Models evaluating race QTHSF performed the worst out of all the races being evaluated with prediction accuracies ranging from 0.530 to 0.624.

For most races, prediction accuracies ranging from 0.60 to 0.69 were observed. However, higher prediction accuracies were observed for TMRTF and TPMKC. With these two races, three of the models evaluated had prediction accuracies above 0.700. For TMRTF, the top performing

models were BayesB ($r = 0.748$), LASSO ($r = 0.764$), and RF ($r = 0.758$). Similarly, for TPMKC the results are as follows BayesB ($r = 0.745$), LASSO ($r = 0.758$), and RF ($r = 0.772$).

Computational time required to evaluate the models using the seedling data varied greatly. GBLUP required the least amount of time, with an approximate run time 5.27 hours across all races. LASSO required 12.95 hours, and RR with 34.89 hours, respectively. The Bayesian models required substantially longer runtimes amongst the parametric models that were evaluated. BayesA required 113.69 hours, BayesB with 111.11 hours, and BayesC with 111.74 hours. For the non-parametric models, RKHS required 34.06 hours and RF required the longest run time with 409.98 hours.

The relationship between computational runtime and prediction accuracy for the seedling races is presented in Figure 4.9.1. Prediction accuracies varied across the seven races, with lower accuracies generally observed for QTHSF and higher accuracies for TMRTF and TPMKC. Across all races, differences in prediction accuracy among models were relatively small, with multiple models showing comparable performance within each race. In contrast, computational runtime differed substantially among models, with GBLUP requiring a few hours, Bayesian models needing several days and Random Forest requiring close to 3 weeks to complete evaluations across all races. Despite this large difference in computational demand, more computationally intensive models did not consistently outperform simpler models. These results indicate that model choice had a limited impact on prediction accuracy for seedling resistance, while greatly influencing computational efficiency

Table 4.3 Accuracy of eight GS models to predict seedling IT data for seven *Pgt* races.

Race		GBLUP	RR	BayesA	BayesB	BayesC	LASSO	RKHS	RF
MCCFC	^a Accuracy (r)	0.640	0.643	0.640	0.676	0.641	0.699	0.658	0.669
	^b SD	0.008	0.09	0.009	0.009	0.010	0.014	0.008	0.010
QTHSF	Accuracy (r)	0.53	0.532	0.528	0.589	0.53	0.624	0.546	0.599
	SD	0.013	0.012	0.016	0.012	0.013	0.017	0.013	0.012
RHTSF	Accuracy (r)	0.641	0.651	0.641	0.682	0.643	0.693	0.662	0.709
	SD	0.010	0.009	0.012	0.009	0.009	0.013	0.010	0.008
RKQSF	Accuracy (r)	0.657	0.659	0.656	0.675	0.658	0.666	0.673	0.689
	SD	0.011	0.010	0.012	0.012	0.010	0.013	0.010	0.011
RTHSF	Accuracy (r)	0.634	0.638	0.684	0.692	0.641	0.682	0.656	0.725
	SD	0.013	0.011	0.014	0.012	0.012	0.014	0.011	0.080
TMRTF	Accuracy (r)	0.637	0.650	0.660	0.748	0.645	0.764	0.666	0.758
	SD	0.012	0.010	0.013	0.011	0.011	0.011	0.010	0.009
TPMKC	Accuracy (r)	0.641	0.640	0.690	0.745	0.646	0.758	0.641	0.772
	SD	0.014	0.011	0.017	0.012	0.012	0.011	0.011	0.008
Time to run (hours)		5.27	34.89	113.69	111.11	111.74	12.95	34.06	409.98

^aPearson correlation coefficient^bSD, standard deviation of r among all cross-validation iterations



Figure 4.9.1 Relationship between computational runtime and prediction accuracy (r) for genomic selection models evaluated using seedling resistance data across seven *Pgt* races. Each point represents the prediction accuracy of a single model for an individual race. Runtime (hours) is presented on a log₁₀ scale to account for the wide range in computational time among models. Models are distinguished by colour and shape. Abbreviations: GBLUP: Genomic Best Unbiased Linear Predictions; RR: Ridge Regression; LASSO: Least Absolute Shrinkage and Selection Operator; BA/BB/BC: Bayesian methods; RKHS: Reproducing Kernel Hilbert Spaces (RKHS); and RF: Random Forest.

4.10 Discussion

Technological advancements, such as GS, can have the potential to improve efficiency within breeding programs by reducing the need for phenotyping, as predictions can be made using genotypic data (Heffner et al. 2009; Rutkoski et al. 2011). MAS has become a routine approach in breeding programs to increase genetic gains by using molecular markers to identify lines that possess traits of interest and to aid in the pyramiding of resistance genes (Collard and Mackill 2007; Heffner et al. 2010; Rutkoski et al. 2011; Song et al. 2023). GS can be viewed as an extension of MAS, as it estimates marker effects across the entire genome of an individual and calculates the predicted GEBVs (Meuwissen et al. 2001; Heffner et al. 2009). The objective of this study was to evaluate the performance of multiple GS models and their suitability for predicting stem rust resistance in a subset of the winter wheat association mapping population comprising of 326 lines from western Canada and the United States.

Prediction accuracies for the field traits (severity, IT, and COI) ranged from 0.743 to 0.810 across all models evaluated. Severity consistently exhibited the highest prediction accuracy. Heritability estimates were high, ranging from 0.92 to 0.95, indicating that a large proportion of the phenotypic variation was genetically controlled. These values tend to be slightly higher than what has been identified in other studies.

In contrast, seedling assays produced lower and more variable prediction accuracies, ranging from 0.528 to 0.772. Higher accuracies were observed for races that were more virulent on the population (e.g., TMRTF and TPMKC), while lower accuracies were observed for less virulent races such as QTHSF. The differences in prediction accuracies across races suggests that the predictability of seedling resistance may rely heavily on which *Sr* genes are effective against

each race, and how they are distributed in the population. Whereas the stem rust resistance in the field studies tends to be governed by the presence of multiple genes or loci, this in part may be due to the use of mixed inoculum (i.e., a mixture of seven *Pgt* isolates) in the field trial evaluations.

Several studies have evaluated GS for stem rust resistance in wheat, particularly at the adult plant stage. Rutkoski et al. (2014) evaluated 365 CIMMYT breeding lines and reported prediction accuracies ranging from 0.446 to 0.6198, with a mean broad sense heritability of 0.82. In a subsequent study, Rutkoski et al. (2015) evaluated 374 lines from CIMMYT stem rust screening nurseries. These lines were used to train GS models; a validation population was created from fourteen individuals of the original training population. Two cycles of genomic selection and one cycle of phenotypic selection were carried out over a two-year period, one cycle on the population derived from the fourteen individuals (C_0) and the second cycle on the subsequent population (C_1) derived from the C_0 . The mean heritability across the environments was found to be 0.78 and the mean GS prediction accuracy in C_0 was 0.37 ± 0.11 and C_1 0.69 ± 0.11 . Similarly, Juliana et al. (2017) reported prediction accuracies ranging from 0.312 to 0.65, with heritability estimates of 0.59 and 0.50 across two growing seasons. Finally, Azizinia et al. (2020) reported prediction accuracies for both field (0.49 to 0.50) and seedling (0.43 to 0.46) resistance. These studies tended to evaluate multiple models as was conducted in our study, though there was overlap with the following models of GBLUP, BayesA, BayesB, BayesC, and RKHS.

Across all the traits, differences in prediction accuracy among models were relatively small, particularly for the field traits. In some cases, semi-parametric (RKHS) and machine learning (RF) models performed just as well if not slightly better than the parametric models. This suggests that factors such as computational intensity and the time required to run the models should be considered when selecting a GS model. Less computationally demanding models such a

GBLUP, required a few hours to run, whereas more complex models, such as RF, required days to weeks to run to completion. Increased model complexity and computational time did not inherently result in improved prediction accuracies; this highlights a consideration for breeding programs where computational efficiency may be favoured when prediction accuracy gains are minimal.

The lower prediction accuracies for the seedling resistance compared to the adult plant resistance, could likely be due to the differences in the genetic basis of these traits. In the seedling assays each *Pgt* race was evaluated individually on the population, the results in Chapter 3 of this study suggest that most of the resistance observed in the population were controlled by one or two race-specific *Sr* genes, such as *Sr6*, *Sr24*, *Sr38*, and *Sr31*. These three genes were identified as QTL in response to the two seedling races that had the highest prediction accuracies (TMRTF and TPMKC). In contrast, the field trials exposed the GWAS population to a mixture of the *Pgt* races, so the resistance identified in these field trials likely reflected the combined effects of multiple genes. This would include both major race-specific genes and additional loci identified to be associated with resistance. These results suggest that the polygenic nature of the adult plant resistance may be better suited to GS and why the prediction accuracies were much higher for the field trial traits.

4.11 Conclusion

The results of this study show that there is potential for GS to be implemented for the selection of stem rust resistance in western Canadian winter wheat breeding programs. The prediction accuracies observed for the field trials suggest that GS could be used to identify individuals with adult plant resistance, potentially allowing for a reduction in the amount of traditional phenotyping required. This has important implications for breeding programs, as it may lead to reduced breeding cycle times and provide both financial and time saving advantages. In contrast, prediction

accuracies for seedling resistance were more variable across the *Pgt* races evaluated, reflecting differences in the underlying genetic architecture. The results from this study suggest that GS may be less effective for traits that are controlled by major race-specific genes compared to those that are polygenic. Limitations for the implementation of GS must be considered, including the need for a well-defined and representative training population, having access to this genotypic and phenotypic data, and sufficient computational resources to run the various models and handle large datasets. Overall, GS represents a valuable tool that can complement existing approaches such as field stem rust nurseries and marker-assisted selection (MAS). The integration of GS into winter wheat breeding programs has the potential to improve selection efficiency and accelerate the development of cultivars with durable stem rust resistance.

Chapter 5: GENERAL DISCUSSION

Wheat is Canada's most widely grown field crop and a major driver of the agriculture sector and Canadian economy (Cereals Canada 2025). While spring wheat and durum account for most of the production, winter wheat provides several important agronomic benefits in western Canada, including improved ground cover over fall and winter, reduced soil erosion, better use of early spring moisture, and enhanced weed suppression. Despite these advantages, winter wheat still represents a relatively small proportion of total production.

Stem rust remains a significant threat to wheat production despite decades of successful control using resistant cultivars. While no major epidemics have occurred in Canada since the 1950s, largely due to the deployment of resistant varieties and barberry eradication programs, the continued evolution of *Pgt* and the emergence of highly virulent races globally highlight the importance of maintaining effective and durable resistance. This is particularly important for western Canadian winter wheat, where production is already limited and breeding efforts must focus on both agronomic performance and disease resistance.

The results of this study provide a comprehensive assessment of stem rust resistance in western Canadian winter wheat through the combined use of GWAS and GS. GWAS analysis identified multiple QTL associated with resistance across several chromosomes, including regions corresponding to known *Sr* genes such as *Sr6*, *Sr24*, *Sr31*, *Sr33*, and *Sr38*. These findings support that many previously identified resistance genes are present within this germplasm, while also highlighting additional loci that may contribute to resistance. The detection of QTL across multiple environments and measures of stem rust resistance further supports the stability of several key resistance regions, particularly those located on chromosomes 2A and 3D, which were consistently associated with reduced disease severity, infection type, and coefficient of infection.

Seedling resistance in the population was largely controlled by one or a few major race-specific genes, resulting in variable phenotypic responses across the seven *Pgt* races evaluated. This was reflected in the GWAS results, where race-specific QTL were identified, and in the GS results, where prediction accuracies for seedling resistance were more variable ($r = 0.53\text{--}0.77$). In contrast, adult plant resistance evaluated under field conditions exhibited a more polygenic nature. Multiple QTL with small to moderate effects were identified, and resistance was expressed across environments. This was reflected in the GS results, where prediction accuracies for field traits were higher and more consistent ($r = 0.74\text{--}0.81$). The high heritability estimates observed for these traits further indicate that a large proportion of the phenotypic variation is genetically controlled, making them well suited for genomic prediction. These results demonstrate that GS shows promise for traits with a polygenic nature, as seen with the results from the field trials in this population.

Although differences among GS models were observed, they were generally small. While the Bayesian models, RKHS, and RF often showed higher prediction accuracies, these improvements were minimal and often came with substantially increased computational requirements. In contrast, GBLUP provided comparable prediction accuracies while being significantly more efficient to run. Results from GS demonstrated that breeders will need to consider time and resource constraints when considering using or implementing GS within their programs.

The GS prediction accuracies observed in this study for adult plant resistance were generally higher than those reported in previous studies, which may be attributed to several factors. The relatively high heritability values indicate that the differences in phenotypes observed are due to the genetic differences among the individuals in the association mapping population. Additionally, the association mapping population used in this study may be more closely related

compared to the more diverse wheat populations evaluated in the previous studies, which could improve model performance. The use of a mixed inoculum in the field trials may have also contributed to the expression of multiple resistance mechanisms, further enhancing the effectiveness of GS.

Despite these promising results, several limitations should be considered. The GS training population consisted of a single association mapping panel, and prediction accuracies will decrease when models are applied to unrelated or less closely related populations. In addition, GS was less effective for seedling resistance traits controlled by major genes, reinforcing the need to integrate GS with MAS depending on the trait of interest. Finally, the computational demands of more complex models may limit their practicality for some breeding programs.

From a breeding perspective, the results of this study support the integration of GS into winter wheat breeding programs. GS can be used as a tool for early-generation selection, allowing breeders to identify promising lines prior to extensive phenotypic evaluation. This has the potential to reduce both the time and cost associated with traditional phenotyping and may help accelerate breeding cycles. However, GS should not be viewed as a replacement for existing methods, but rather as a complementary approach. A combined strategy that utilizes GS for polygenic traits such as adult plant resistance, alongside MAS for major resistance genes, is likely to be the most effective approach for developing durable resistance.

Future research should focus on validating GS models across additional populations and environments to assess their robustness and applicability in breeding programs. Evaluating lines that were most recently part of the Western Canadian Winter Wheat Cooperative registration trials could validate the results that were observed in this study. In addition, further work is needed to refine the identification of key QTL and develop breeder-friendly markers that can be readily

implemented in selection programs. Expanding GS to include other important traits, such as yield, winter hardiness, and resistance to additional diseases like leaf and stripe rust and FHB, will also be important for improving overall breeding efficiency.

Overall, this study demonstrates that stem rust resistance in western Canadian winter wheat is controlled by a combination of major genes and minor QTL, with distinct genetic architectures observed at the seedling and adult plant stages. Genomic selection shows strong potential as a tool for improving adult plant resistance and enhancing breeding efficiency. However, its effectiveness is dependent on the underlying genetic architecture of the trait, and its implementation should be carefully integrated with other breeding approaches. As pathogen populations continue to evolve and environmental conditions change, the use of combined genomic and phenotypic strategies will be essential for developing resilient wheat cultivars and ensuring long-term food security.

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APPENDICES

A.1 List of Abbreviations

Abbreviation	Name
AAFC	Agriculture and Agri-Food Canada
AFLP	Amplified Fragment Length Polymorphisms
APR	Adult Plant Resistance
BayesA	Bayesian A
BayesB	Bayesian B
BayesC	Bayesian C
BLAST	Basic Local Alignment Search Tool
BLUEs	Best Linear Unbiased Estimates
BLUPs	Best Linear Unbiased Predictions
bp	base pair
CEHRW	Canada Eastern Hard Red Winter
CESRW	Canada Eastern Soft Red Winter
CEWW	Canada Eastern White Winter
CIM	composite interval mapping
cM	centi-Morgan
COI	Coefficient of Infection
CV	Coefficient of variation
CWRW	Canada Western Red Winter
DNA	Deoxyribonucleic acid
FDR	False discovery rate
FHB	Fusarium head blight
GAPIT	Genome Association and Prediction Integrated Tool
GBLUP	Genomic Best Linear Unbiased Predictors
GEBV	Genomic estimated breeding value
GS	Genomic Selection
GWAS	Genome-wide Association Study
IT	Infection Type
IT	Infection Type
LASSO	Least Absolute Shrinkage and Selection Operator
LD	Linkage disequilibrium
LEA	Landscape and Ecological Association
LeRDC	Lethbridge Research and Development Centre
MAB	Marker-assisted back-cross
MAF	Minor allele frequency
MAI	Marker-assisted introgression
MAS	Marker-assisted selection

Abbreviation	Name
Mbp	Megabase pairs
META-R	Multi-Environment Trait Analysis
MLM	Mixed linear model
MR	Moderately resistant
MS	Moderately susceptible
MTA	Marker-trait association
PCA	Principal Component Analysis
PCR	Polymerase Chain Reaction
<i>Pgt</i>	<i>Puccinia graminis</i> f.sp. <i>tritici</i>
PVE	Phenotypic variation explained
QQ	Quantile-quantile
QTL	Quantitative Trait Loci
R	Resistant
RCBD	Randomized Complete Block Design
RF	Random Forest
RFLP	Restriction fragment length polymorphisms
RKHS	Reproducing kernel Hilbert spaces
RNOmni	Rank-Based Inverse Normal Transformation
RR	Ridge Regression
S	Susceptible
SIM	simple interval mapping
sNMF	Sparse nonnegative matrix factorization
SNP	Single Nucleotide Polymorphism
SSR	Simple Sequence Repeat
STS	Sequence Tagged Sites
TASSEL	Trait Analysis by a SSoCiation, Evolution and Linkage

A.2 Supplemental Figures

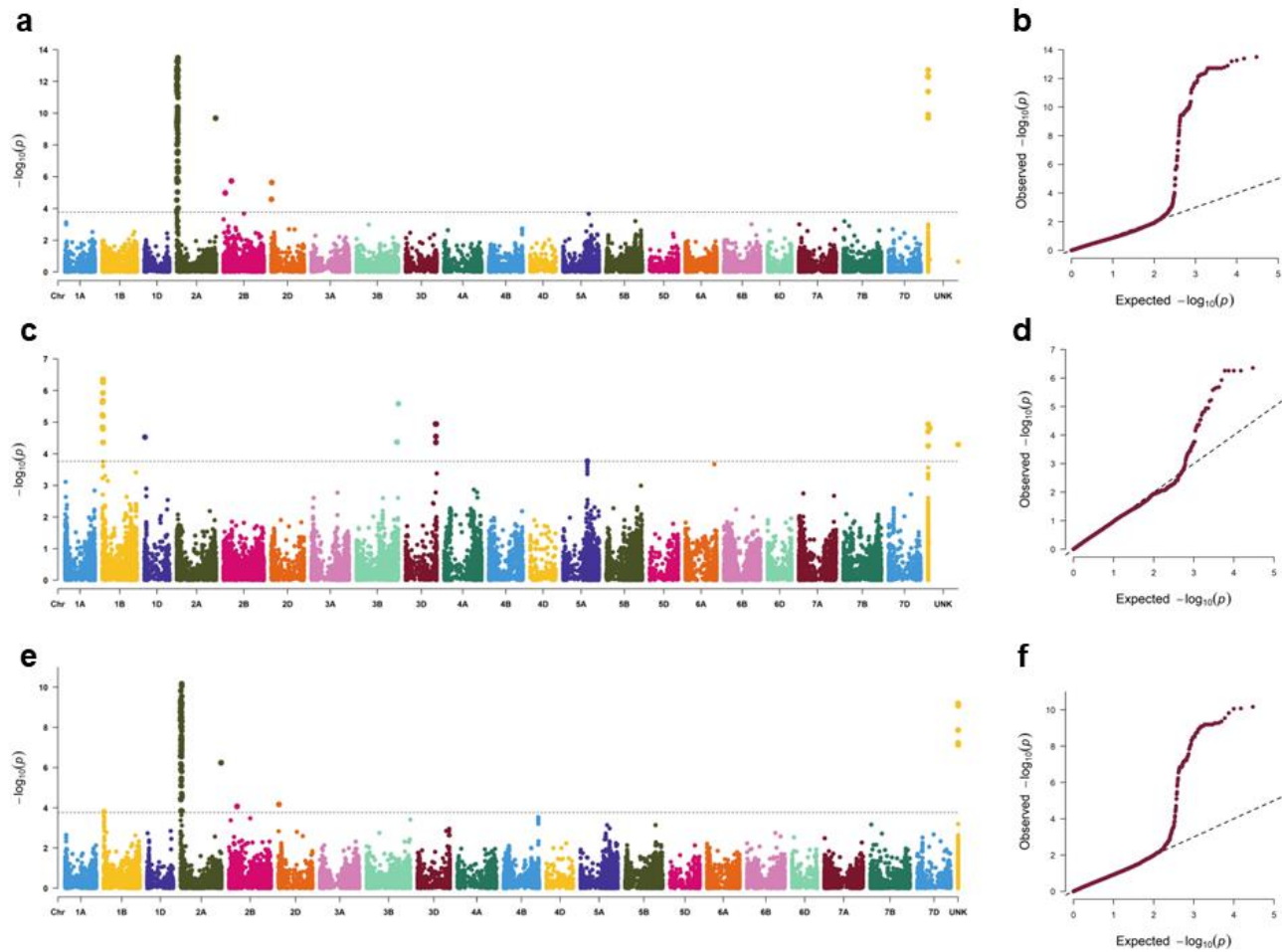


Figure S1 Manhattan and quantile-quantile (QQ) plots produced from the genome-wide association analysis of Carman 2023. On the Manhattan plots the y-axis indicates the $-\log_{10}(p)$ and the x-axis indicates the chromosomal position of the markers. The dashed line indicates the false discovery rate threshold (FDR) of $-\log_{10}(p) = 3.76$. The QQ plots illustrate the observed $-\log_{10}(p)$ against the expected $-\log_{10}(p)$. A total of 29,766 markers were used. Severity is represented in panels a-b, infection type in c-d, and coefficient of infection in e-f.

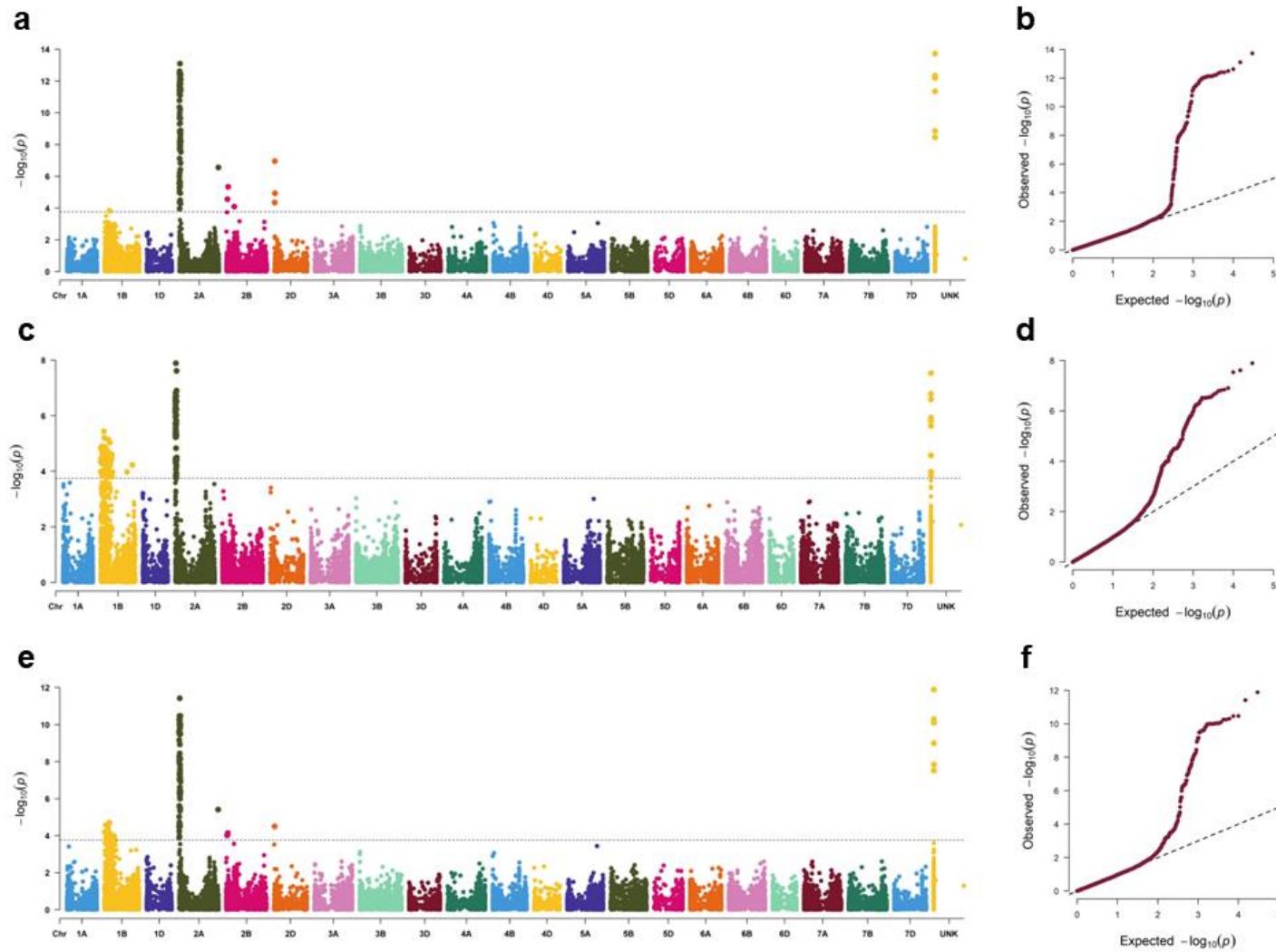


Figure S2 Manhattan and quantile-quantile (QQ) plots produced from the genome-wide association analysis of Carman 2024. On the Manhattan plots the y-axis indicates the $-\log_{10}(p)$ and the x-axis indicates the chromosomal position of the markers. The dashed line indicates the false discovery rate threshold (FDR) of $-\log_{10}(p) = 3.76$. The QQ plots illustrate the observed $-\log_{10}(p)$ against the expected $-\log_{10}(p)$. A total of 29,766 markers were used. Severity is represented in panels a-b, infection type in c-d, and coefficient of infection in e-f.

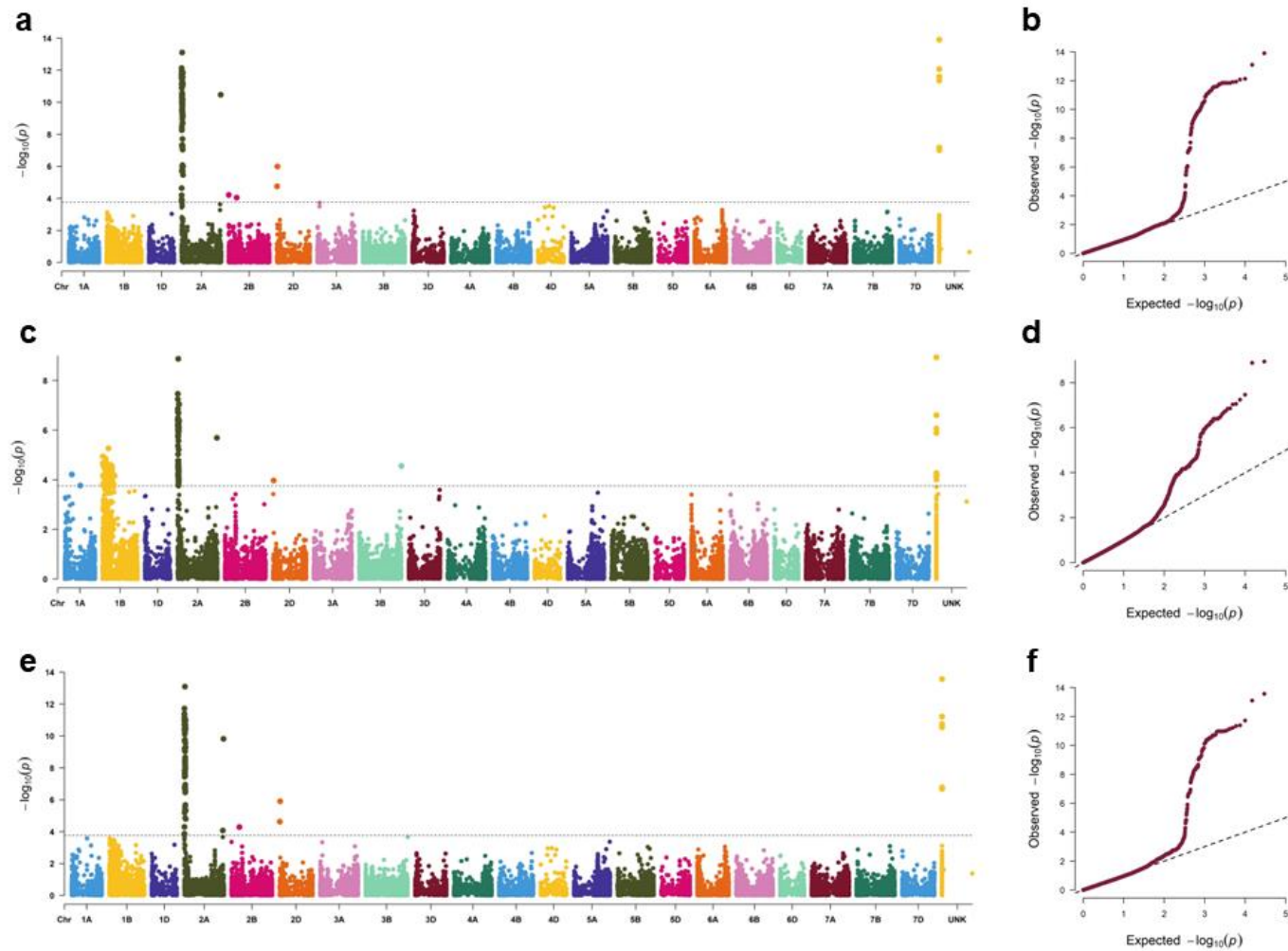


Figure S3 Manhattan and quantile-quantile (QQ) plots produced from the genome-wide association analysis of Winnipeg 2024. On the Manhattan plots the y-axis indicates the $-\log_{10}(p)$ and the x-axis indicates the chromosomal position of the markers. The dashed line indicates the false discovery rate threshold (FDR) of $-\log_{10}(p) = 3.76$. The QQ plots illustrate the observed $-\log_{10}(p)$ against the expected $-\log_{10}(p)$. A total of 29,766 markers were used. Severity is represented in panels a-b, infection type in c-d, and coefficient of infection in e-f

A3. Raw Phenotypic Field Data

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	226	1	134	40	S
CRM24	730	2	134	40	S
WPG24	266	1	134	30	S
CRM23	32	1	22A*13	20	MS
CRM23	474	2	22A*13	15	MS
CRM24	62	1	22A*13	1	R
WPG23	48	1	22A*13	20	R
WPG23	393	2	22A*13	15	R
WPG24	102	1	22A*13	5	MR
WPG24	585	2	22A*13	5	MR-MS
CRM24	237	1	2504_82_51	5	R
CRM24	710	2	2504_82_51	20	R
WPG24	316	1	2504_82_51	10	R-MR
WPG24	441	2	2504_82_51	10	R-MR
CRM23	154	1	32C*17	10	MS
CRM23	390	2	32C*17	40	MS-S
CRM24	828	2	32C*17	0	0
CRM24	71	1	32C*17	5	R
WPG23	95	1	32C*17	10	MR-MS
WPG23	532	2	32C*17	5	R
WPG24	237	1	32C*17	5	R
WPG24	492	2	32C*17	5	R-MR
CRM23	153	1	43I*18	1	MR
CRM23	435	2	43I*18	1	MS
CRM24	633	2	43I*18	0	0
CRM24	284	1	43I*18	5	R
WPG23	438	2	43I*18	5	MR
WPG23	251	1	43I*18	15	R
WPG24	197	1	43I*18	5	R
WPG24	712	2	43I*18	5	R
CRM24	225	1	79TK070_1064	40	MS
WPG24	827	2	79TK070_1064	80	S
CRM23	86	1	AAC_ELEVATE	25	R
CRM23	512	2	AAC_ELEVATE	5	R
CRM24	99	1	AAC_ELEVATE	5	R
CRM24	766	2	AAC_ELEVATE	10	R
WPG23	243	1	AAC_ELEVATE	5	R
WPG23	419	2	AAC_ELEVATE	6	R-MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	132	1	AAC_ELEVATE	20	MR
WPG24	798	2	AAC_ELEVATE	20	R-MR
CRM23	18	1	AAC_GATEWAY	5	R
CRM23	436	2	AAC_GATEWAY	5	R
CRM24	866	2	AAC_GATEWAY	10	MR
CRM24	352	1	AAC_GATEWAY	0	0
WPG23	181	1	AAC_GATEWAY	10	R
WPG23	422	2	AAC_GATEWAY	5	R
WPG24	269	1	AAC_GATEWAY	10	R-MR
WPG24	636	2	AAC_GATEWAY	20	R-MR
CRM23	62	1	AAC_GOLDRUSH	10	R
CRM23	437	2	AAC_GOLDRUSH	20	R-MR
CRM24	437	1	AAC_GOLDRUSH	10	R
CRM24	592	2	AAC_GOLDRUSH	10	R
WPG23	322	1	AAC_GOLDRUSH	10	R
WPG23	408	2	AAC_GOLDRUSH	10	R
WPG24	340	1	AAC_GOLDRUSH	20	R-MR
CRM24	97	1	AAC_ICEFIELD	0	0
CRM24	713	2	AAC_ICEFIELD	0	0
WPG23	42	1	AAC_ICEFIELD	1	R
WPG23	594	2	AAC_ICEFIELD	1	R
WPG24	370	1	AAC_ICEFIELD	0	0
WPG24	525	2	AAC_ICEFIELD	0	0
CRM23	243	1	AAC_NETWORK	0	0
CRM23	440	2	AAC_NETWORK	5	R
CRM24	863	2	AAC_NETWORK	0	0
CRM24	95	1	AAC_NETWORK	5	R
WPG23	40	1	AAC_NETWORK	10	R
WPG23	345	2	AAC_NETWORK	5	R
WPG24	99	1	AAC_NETWORK	0	0
CRM23	53	1	AAC_VORTEX	5	MR
CRM23	481	2	AAC_VORTEX	5	MS
CRM24	351	1	AAC_VORTEX	0	0
CRM24	624	2	AAC_VORTEX	0	0
WPG23	310	1	AAC_VORTEX	15	R
WPG23	470	2	AAC_VORTEX	5	R
WPG24	172	1	AAC_VORTEX	5	MR
WPG24	751	2	AAC_VORTEX	5	R
CRM23	170	1	AAC_WILDFIRE	40	MS
CRM23	362	2	AAC_WILDFIRE	35	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	292	1	AAC_WILDFIRE	60	S
CRM24	878	2	AAC_WILDFIRE	70	S
WPG23	232	1	AAC_WILDFIRE	25	MR
WPG23	417	2	AAC_WILDFIRE	20	MR-MS
WPG24	94	1	AAC_WILDFIRE	40	S
WPG24	666	2	AAC_WILDFIRE	65	S
CRM23	118	1	ABILENE	30	MR
CRM23	351	2	ABILENE	10	R
CRM24	356	1	ABILENE	0	0
CRM24	790	2	ABILENE	0	0
WPG23	179	1	ABILENE	5	R
WPG23	332	2	ABILENE	5	R
WPG24	28	1	ABILENE	15	MR
WPG24	729	2	ABILENE	10	MR
CRM24	386	1	AC_BELLATRIX	40	MS
CRM24	800	2	AC_BELLATRIX	20	MS
WPG24	427	1	AC_BELLATRIX	40	MS
WPG24	583	2	AC_BELLATRIX	60	S
CRM24	456	2	AC_MORLEY	25	MS-S
CRM24	89	1	AC_MORLEY	70	S
WPG24	281	1	AC_MORLEY	45	S
WPG24	611	2	AC_MORLEY	65	S
CRM23	163	1	AC_READYMADE	40	MS
CRM23	328	2	AC_READYMADE	40	MS
CRM24	694	2	AC_READYMADE	20	MR
CRM24	24	1	AC_READYMADE	30	MR-MS
WPG23	315	1	AC_READYMADE	30	MR-MS
WPG23	587	2	AC_READYMADE	25	MR-MS
WPG24	351	1	AC_READYMADE	5	MR-MS
WPG24	578	2	AC_READYMADE	35	MR-MS
CRM24	55	1	AC_SAMPSON	20	MS
CRM24	834	2	AC_SAMPSON	45	MS-S
WPG24	489	2	AC_SAMPSON	20	MR-MS
WPG24	407	1	AC_SAMPSON	30	MS-S
CRM23	338	2	AC_TEMPEST	50	MS-S
CRM23	165	1	AC_TEMPEST	60	S
CRM24	492	2	AC_TEMPEST	30	MS-S
CRM24	384	1	AC_TEMPEST	30	S
WPG23	215	1	AC_TEMPEST	15	MR
WPG23	636	2	AC_TEMPEST	20	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	476	2	AC_TEMPEST	65	MS-S
WPG24	402	1	AC_TEMPEST	45	S
CRM24	166	1	ACCIPITER	0	0
CRM24	814	2	ACCIPITER	10	R
WPG23	545	2	ACCIPITER	0	0
WPG23	259	1	ACCIPITER	5	R
WPG24	663	2	ACCIPITER	10	R
WPG24	137	1	ACCIPITER	10	R-MR
CRM24	809	2	AL_BIDUM_114_PI372331	40	MS-S
CRM24	153	1	AL_BIDUM_114_PI372331	40	S
WPG24	149	1	AL_BIDUM_114_PI372331	25	S
WPG24	746	2	AL_BIDUM_114_PI372331	65	S
CRM24	25	1	AL_BIDUM_114_PI518782	60	S
CRM24	651	2	AL_BIDUM_114_PI518782	40	S
WPG24	141	1	AL_BIDUM_114_PI518782	10	MS-S
WPG24	804	2	AL_BIDUM_114_PI518782	50	S
CRM23	171	1	ALABASKAJA	60	MS-S
CRM23	265	2	ALABASKAJA	60	S
CRM24	291	1	ALABASKAJA	60	S
CRM24	468	2	ALABASKAJA	50	S
WPG23	17	1	ALABASKAJA	20	MS
WPG23	431	2	ALABASKAJA	25	MS
WPG24	448	2	ALABASKAJA	50	MS-S
WPG24	303	1	ALABASKAJA	60	S
CRM23	374	2	ALBIDUM_11	20	MS
CRM23	3	1	ALBIDUM_11	40	S
CRM24	74	1	ALBIDUM_11	25	MS-S
CRM24	692	2	ALBIDUM_11	70	S
WPG23	266	1	ALBIDUM_11	5	MS
WPG23	390	2	ALBIDUM_11	35	MS
WPG24	87	1	ALBIDUM_11	25	S
WPG24	588	2	ALBIDUM_11	60	S
CRM23	342	2	AL'BIDUM_114_PI372331	40	MS
CRM23	11	1	AL'BIDUM_114_PI372331	80	MS-S
WPG23	210	1	AL'BIDUM_114_PI372331	25	MR
WPG23	616	2	AL'BIDUM_114_PI372331	11	MR-MS
WPG23	58	1	AL'BIDUM_114_PI518782	20	MS
WPG23	588	2	AL'BIDUM_114_PI518782	10	MS
CRM23	181	1	ALBIDUM_12	60	MS
CRM23	274	2	ALBIDUM_12	5	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	41	1	ALBIDUM_12	50	S
CRM24	479	2	ALBIDUM_12	40	S
WPG23	294	1	ALBIDUM_12	16	MR-MS
WPG23	365	2	ALBIDUM_12	5	MR-MS
WPG24	51	1	ALBIDUM_12	40	S
WPG24	810	2	ALBIDUM_12	75	S
CRM24	29	1	ALICE	10	R
WPG23	211	1	ALICE	10	R
WPG23	485	2	ALICE	5	R
WPG24	456	2	ALICE	30	MR
CRM24	368	1	ALLIANCE	25	MR
CRM24	547	2	ALLIANCE	5	R-MR
WPG23	296	1	ALLIANCE	15	R
WPG23	597	2	ALLIANCE	5	R
WPG24	294	1	ALLIANCE	25	MR
WPG24	486	2	ALLIANCE	30	MS
CRM23	143	1	ARAPAHOE	5	MR
CRM23	297	2	ARAPAHOE	5	R
CRM24	318	1	ARAPAHOE	0	0
CRM24	829	2	ARAPAHOE	0	0
WPG23	306	1	ARAPAHOE	5	R
WPG23	436	2	ARAPAHOE	5	R
WPG24	376	1	ARAPAHOE	10	R
WPG24	770	2	ARAPAHOE	20	R-MR
CRM24	584	2	ARCHER	15	MR
CRM24	245	1	ARCHER	20	MS
WPG23	96	1	ARCHER	5	R
WPG23	363	2	ARCHER	20	R
WPG24	40	1	ARCHER	20	MS
WPG24	501	2	ARCHER	40	MS-S
CRM24	288	1	ARINALRFOR	25	MR
CRM24	823	2	ARINALRFOR	40	MS
CRM24	178	1	AUGUSTA	60	S
CRM24	664	2	AUGUSTA	70	S
WPG23	172	1	AUGUSTA	5	MR
WPG23	544	2	AUGUSTA	20	MS
WPG24	7	1	AUGUSTA	40	S
CRM23	226	1	BENEFIT	40	MS
CRM23	317	2	BENEFIT	35	MS
CRM24	441	2	BENEFIT	40	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	159	1	BENEFIT	10	R
WPG23	420	2	BENEFIT	15	MR
WPG23	286	1	BENEFIT	15	MR-MS
WPG24	249	1	BENEFIT	40	MR
WPG24	623	2	BENEFIT	10	R
CRM24	413	1	BEZOSTAJA_1	40	S
CRM24	487	2	BEZOSTAJA_1	40	S
WPG23	484	2	BEZOSTAJA_1	5	MR-MS
WPG23	94	1	BEZOSTAJA_1	5	MS
WPG24	72	1	BEZOSTAJA_1	40	S
WPG24	655	2	BEZOSTAJA_1	50	S
CRM24	193	1	BEZOSTAYA	15	MS-S
CRM24	583	2	BEZOSTAYA	25	MS-S
WPG24	31	1	BEZOSTAYA	25	MS-S
WPG24	473	2	BEZOSTAYA	50	MS-S
CRM23	373	2	BIGHORN_P1601204	35	MR
CRM23	133	1	BIGHORN_P1601204	15	MS
WPG23	38	1	BIGHORN_P1601204	10	MR
WPG23	644	2	BIGHORN_P1601204	15	MR
CRM24	415	1	BIGHORN_PI601204	30	MS
CRM24	840	2	BIGHORN_PI601204	40	MS
WPG24	590	2	BIGHORN_PI601204	30	MR
WPG24	371	1	BIGHORN_PI601204	35	MS
CRM24	307	1	BIGHORN_W240	30	MS
CRM24	739	2	BIGHORN_W240	30	MS
WPG23	107	1	BIGHORN_W240	15	R
WPG23	452	2	BIGHORN_W240	11	R-MR
WPG24	528	2	BIGHORN_W240	30	MR
CRM24	689	2	BLIZZARD	70	S
WPG24	377	1	BLIZZARD	80	S
WPG24	791	2	BLIZZARD	60	S
CRM23	167	1	BROADVIEW	40	R
CRM23	422	2	BROADVIEW	10	R
CRM24	118	1	BROADVIEW	10	R
CRM24	603	2	BROADVIEW	10	R
WPG23	241	1	BROADVIEW	0	0
WPG23	391	2	BROADVIEW	5	R
WPG24	388	1	BROADVIEW	20	MR
WPG24	675	2	BROADVIEW	10	R-MR
CRM23	162	1	BROME	30	MR-MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	433	2	BROME	40	MR-MS
CRM24	622	2	BROME	15	MS-S
CRM24	383	1	BROME	40	S
WPG23	67	1	BROME	20	MS
WPG23	558	2	BROME	15	MS
WPG24	612	2	BROME	30	MS-S
WPG24	69	1	BROME	15	S
CRM24	705	2	BUSSARD	20	S
CRM24	400	1	CALEDONIA	30	MS
CRM24	795	2	CALEDONIA	35	MS
WPG24	164	1	CALEDONIA	40	MS
WPG24	487	2	CALEDONIA	60	MS-S
CRM24	131	1	CAMELOT	40	MS
CRM24	858	2	CAMELOT	20	MS-S
WPG24	541	2	CAMELOT	10	MS-S
CRM24	132	1	CAPO	5	MR
CRM24	675	2	CAPO	10	MR
WPG24	398	1	CAPO	5	MS-S
WPG24	575	2	CAPO	5	R
CRM23	264	2	CARNAVAL	25	MR
CRM23	35	1	CARNAVAL	10	R
CRM24	75	1	CARNAVAL	5	R
CRM24	582	2	CARNAVAL	10	R
WPG23	187	1	CARNAVAL	0	0
WPG23	554	2	CARNAVAL	5	R
WPG24	880	2	CARNAVAL	20	MR
WPG24	273	1	CARNAVAL	15	R
CRM23	486	2	CDC_BUTEO	35	MS
CRM23	142	1	CDC_BUTEO	50	MS-S
CRM24	719	2	CDC_BUTEO	20	MR-MS
WPG23	203	1	CDC_BUTEO	10	MS
WPG23	607	2	CDC_BUTEO	5	MS
WPG24	284	1	CDC_BUTEO	25	MS-S
WPG24	574	2	CDC_BUTEO	25	MS-S
CRM24	188	1	CDC_CHASE	0	0
CRM24	476	2	CDC_CHASE	5	R
WPG24	534	2	CDC_CHASE	10	R
WPG24	361	1	CDC_CHASE	10	R-MR
CRM23	273	2	CDC_CLAIR	25	MR-MS
CRM23	247	1	CDC_CLAIR	20	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	218	1	CDC_CLAIR	30	MS
CRM24	557	2	CDC_CLAIR	20	MS-S
WPG23	547	2	CDC_CLAIR	0	0
WPG23	15	1	CDC_CLAIR	5	MS
WPG24	302	1	CDC_CLAIR	20	MS-S
WPG24	563	2	CDC_CLAIR	30	MS-S
CRM23	259	2	CDC_FALCON	30	MR
CRM23	84	1	CDC_FALCON	70	R-MR
CRM24	370	1	CDC_FALCON	10	R
CRM24	505	2	CDC_FALCON	5	R
WPG23	557	2	CDC_FALCON	0	0
WPG23	208	1	CDC_FALCON	1	R
WPG24	134	1	CDC_FALCON	10	MR
WPG24	753	2	CDC_FALCON	25	MR
CRM23	41	1	CDC_HARRIER	30	MS
CRM23	319	2	CDC_HARRIER	20	MS
CRM24	72	1	CDC_HARRIER	30	MR
CRM24	504	2	CDC_HARRIER	30	MS
WPG23	113	1	CDC_HARRIER	5	MR
WPG23	445	2	CDC_HARRIER	6	MR-MS
WPG24	565	2	CDC_HARRIER	30	MS
WPG24	332	1	CDC_HARRIER	20	MS-S
CRM24	372	1	CDC_KESTREL	25	MR
CRM24	570	2	CDC_KESTREL	20	MS
WPG24	571	2	CDC_KESTREL	20	MR
WPG24	314	1	CDC_KESTREL	30	MS-S
CRM23	318	2	CDC_OSPNEY	30	MS
CRM23	182	1	CDC_OSPNEY	100	MS-S
CRM24	381	1	CDC_OSPNEY	20	MR
CRM24	666	2	CDC_OSPNEY	30	MS-S
WPG23	150	1	CDC_OSPNEY	25	MR
WPG23	348	2	CDC_OSPNEY	5	MR
WPG24	560	2	CDC_OSPNEY	30	MS-S
WPG24	392	1	CDC_OSPNEY	60	S
CRM23	195	1	CDC_PTARMIGAN	70	S
CRM23	324	2	CDC_PTARMIGAN	60	S
CRM24	232	1	CDC_PTARMIGAN	70	S
CRM24	850	2	CDC_PTARMIGAN	80	S
WPG23	204	1	CDC_PTARMIGAN	30	MS
WPG23	572	2	CDC_PTARMIGAN	50	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	335	1	CDC_PTARMIGAN	70	S
WPG24	624	2	CDC_PTARMIGAN	60	S
CRM23	255	1	CDC_RAPTOR	10	R
CRM23	385	2	CDC_RAPTOR	5	R
CRM24	496	2	CDC_RAPTOR	10	MR
CRM24	182	1	CDC_RAPTOR	5	R
WPG23	84	1	CDC_RAPTOR	10	R
WPG23	518	2	CDC_RAPTOR	5	R
WPG24	338	1	CDC_RAPTOR	20	MR
WPG24	745	2	CDC_RAPTOR	30	MS
WPG24	553	2	CHAE_RAE	70	S
CRM24	125	1	CHAMPLAIN	40	S
WPG23	277	1	CHAMPLAIN	25	MS
WPG23	606	2	CHAMPLAIN	15	MS-S
WPG24	628	2	CHAMPLAIN	30	MS-S
WPG24	194	1	CHAMPLAIN	50	S
CRM23	289	2	CHEYENNE	50	MR-MS
CRM23	116	1	CHEYENNE	35	S
CRM24	780	2	CHEYENNE	30	MS-S
CRM24	271	1	CHEYENNE	75	S
WPG23	613	2	CHEYENNE	15	MS
WPG23	39	1	CHEYENNE	30	MS-S
WPG24	442	2	CHEYENNE	50	MS-S
WPG24	333	1	CHEYENNE	25	S
CRM24	221	1	COMANCHE	20	MR
CRM24	686	2	COMANCHE	50	S
WPG23	598	2	COMANCHE	10	MR
WPG23	25	1	COMANCHE	15	MR-MS
WPG24	511	2	COMANCHE	45	MS-S
WPG24	119	1	COMANCHE	30	S
CRM23	341	2	CULVER	5	MS
CRM23	70	1	CULVER	15	R
CRM24	139	1	CULVER	0	0
CRM24	489	2	CULVER	5	MS
WPG23	501	2	CULVER	0	0
WPG23	88	1	CULVER	15	MR
WPG24	368	1	CULVER	0	0
WPG24	833	2	CULVER	10	R-MR
CRM23	83	1	DH_LETH_14_3_23	65	MS-S
CRM23	337	2	DH_LETH_14_3_23	60	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	201	1	DH_LETH_14_3_23	70	MS
CRM24	549	2	DH_LETH_14_3_23	35	MS-S
WPG23	320	1	DH_LETH_14_3_23	5	MR
WPG23	565	2	DH_LETH_14_3_23	11	MR-MS
WPG24	815	2	DH_LETH_14_3_23	45	MS-S
CRM24	76	1	DNESTROVSKAJA_25	20	MS
WPG23	283	1	DNESTROVSKAJA_25	5	MS
WPG23	467	2	DNESTROVSKAJA_25	5	MS
WPG24	444	2	DNESTROVSKAJA_25	50	MS-S
WPG24	359	1	DNESTROVSKAJA_25	80	S
CRM24	252	1	ELKHORN	40	S
CRM24	637	2	ELKHORN	50	S
WPG24	196	1	ELKHORN	30	MS-S
WPG24	654	2	ELKHORN	70	S
CRM23	395	2	EMERSON	20	MR-MS
CRM23	44	1	EMERSON	10	MS
CRM24	792	2	EMERSON	0	0
CRM24	410	1	EMERSON	10	MR
WPG23	162	1	EMERSON	10	MR
WPG23	596	2	EMERSON	1	MR
WPG24	474	2	EMERSON	20	MR-MS
WPG24	54	1	EMERSON	10	R
CRM23	211	1	ERHARDT	5	MS
CRM23	466	2	ERHARDT	5	R
CRM24	46	1	ERHARDT	10	MS
CRM24	536	2	ERHARDT	5	MS
WPG23	321	1	ERHARDT	15	MR
WPG23	383	2	ERHARDT	10	R
WPG24	872	2	ERHARDT	1	MS-S
WPG24	410	1	ERHARDT	5	R
CRM24	670	2	F_216_68	10	MR
CRM24	283	1	F_216_68	10	R
WPG24	438	1	F_216_68	30	MR
WPG24	708	2	F_216_68	30	MR
CRM23	103	1	FHB_148	20	MR
CRM23	387	2	FHB_148	20	MR-MS
CRM24	94	1	FHB_148	30	S
CRM24	720	2	FHB_148	45	S
WPG23	180	1	FHB_148	5	MR
WPG23	480	2	FHB_148	5	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	15	1	FHB_148	30	MS-S
CRM23	60	1	FLOURISH	20	MR
CRM23	497	2	FLOURISH	10	R
CRM24	4	1	FLOURISH	10	R
CRM24	538	2	FLOURISH	10	R
WPG23	56	1	FLOURISH	10	R
WPG23	537	2	FLOURISH	5	R
WPG24	650	2	FLOURISH	20	MR
WPG24	367	1	FLOURISH	20	R-MR
CRM23	129	1	FRANKENMEUTH	30	MS
CRM23	343	2	FRANKENMEUTH	90	MS-S
CRM24	880	2	FRANKENMEUTH	60	S
WPG23	200	1	FRANKENMEUTH	10	MR
WPG23	450	2	FRANKENMEUTH	6	MS-S
WPG24	86	1	FRANKENMEUTH	40	S
CRM23	74	1	FREDERICK	10	R
CRM23	448	2	FREDERICK	10	R
CRM24	157	1	FREDERICK	0	0
CRM24	846	2	FREDERICK	0	0
WPG23	63	1	FREDERICK	5	R
WPG23	378	2	FREDERICK	10	R
WPG24	679	2	FREDERICK	0	0
WPG24	112	1	FREDERICK	5	MR
CRM24	34	1	FREDRICK	20	MS-S
CRM24	716	2	FREDRICK	35	MS-S
WPG23	442	2	FREDRICK	5	MR
WPG23	99	1	FREDRICK	25	MR-MS
WPG24	221	1	FREDRICK	30	MS-S
WPG24	707	2	FREDRICK	70	S
CRM23	333	2	FREEDOM	0	0
CRM23	1	1	FREEDOM	30	MR
CRM24	541	2	FREEDOM	0	0
WPG23	242	1	FREEDOM	10	R
WPG23	564	2	FREEDOM	10	R
WPG24	723	2	FREEDOM	5	R-MR
CRM24	49	1	FRONTENAC	20	MS
CRM24	596	2	FRONTENAC	10	MS-S
WPG23	522	2	FRONTENAC	5	MR
WPG23	248	1	FRONTENAC	10	MS
WPG24	68	1	FRONTENAC	25	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	747	2	FRONTENAC	50	S
CRM24	755	2	GAINES	35	MS-S
CRM24	241	1	GAINES	40	S
WPG24	91	1	GAINES	45	MS-S
WPG24	477	2	GAINES	70	S
CRM24	534	2	GALLUS	20	MS-S
CRM24	330	1	GALLUS	30	S
WPG24	329	1	GALLUS	65	S
WPG24	801	2	GALLUS	60	S
CRM24	255	1	GO	50	MS-S
CRM24	871	2	GO	50	S
WPG24	232	1	GO	25	MS-S
WPG24	773	2	GO	75	S
CRM23	105	1	HANOVER	10	MR
CRM23	414	2	HANOVER	10	MR
CRM24	864	2	HANOVER	40	MS
CRM24	135	1	HANOVER	30	MS-S
WPG23	410	2	HANOVER	6	MR-MS
WPG23	282	1	HANOVER	20	R
WPG24	643	2	HANOVER	40	S
CRM23	253	1	HARDING	5	MS
CRM23	418	2	HARDING	20	MS
CRM24	196	1	HARDING	10	MR
CRM24	746	2	HARDING	10	MR
WPG23	560	2	HARDING	0	0
WPG23	278	1	HARDING	20	MR
WPG24	431	1	HARDING	10	R-MR
WPG24	693	2	HARDING	15	R-MR
CRM24	540	2	HARUS	40	MS-S
WPG23	589	2	HARUS	15	MR
WPG23	89	1	HARUS	40	MS-S
WPG24	279	1	HARUS	45	S
WPG24	727	2	HARUS	70	S
CRM24	98	1	HARVARD	20	MS-S
CRM24	848	2	HARVARD	30	MS-S
WPG24	379	1	HARVARD	30	MS-S
WPG24	797	2	HARVARD	50	S
CRM24	56	1	HOLME	15	MS-S
CRM24	700	2	HOLME	50	S
WPG24	81	1	HOLME	30	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	79	1	HONG_MANG_MAI	30	MS
CRM24	779	2	HY_301_HRW	10	MR
WPG24	49	1	HY_301_HRW	30	MR
WPG24	573	2	HY_301_HRW	15	R-MR
CRM24	167	1	IWA8609583	40	MS
CRM24	498	2	IWA8609583	60	S
WPG24	131	1	IWA8609583	25	MS
WPG24	475	2	IWA8609583	70	S
WPG24	835	2	IWA8610620	25	MR-MS
CRM24	552	2	JAGGER	25	MR
WPG24	721	2	JAGGER	20	MS
WPG24	216	1	JAGGER	1	MS-S
CRM24	551	2	JB_ASANO	25	MS-S
CRM24	189	1	JB_ASANO	30	S
WPG24	420	1	JB_ASANO	10	MS-S
CRM23	378	2	JERRY	5	MR
CRM23	31	1	JERRY	30	MS
CRM24	338	1	JERRY	10	MR
CRM24	701	2	JERRY	5	MS
WPG23	221	1	JERRY	10	R
WPG23	392	2	JERRY	5	R
WPG24	292	1	JERRY	10	MR-MS
WPG24	711	2	JERRY	45	MS-S
CRM24	371	1	JOSEF	15	MS-S
CRM24	836	2	JOSEF	40	S
WPG24	365	1	JOSEF	5	S
WPG24	786	2	JOSEF	35	S
CRM23	109	1	JUDITH	40	MR-MS
CRM23	358	2	JUDITH	25	MR-MS
CRM24	69	1	JUDITH	10	MS
CRM24	797	2	JUDITH	20	MS
WPG23	372	2	JUDITH	6	MR-MS
WPG23	146	1	JUDITH	5	MS
WPG24	230	1	JUDITH	25	MS
WPG24	459	2	JUDITH	45	MS-S
CRM23	314	2	KELDIN	40	MS
CRM23	176	1	KELDIN	80	MS-S
CRM24	200	1	KELDIN	30	MS-S
CRM24	697	2	KELDIN	35	S
WPG23	528	2	KELDIN	15	MR-MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	10	1	KELDIN	30	MS
WPG24	285	1	KELDIN	35	S
WPG24	817	2	KELDIN	70	S
CRM23	306	2	KHARKOV_22MC	40	MS
CRM23	219	1	KHARKOV_22MC	40	MS-S
CRM24	763	2	KHARKOV_22MC	40	MS-S
CRM24	363	1	KHARKOV_22MC	70	S
WPG23	262	1	KHARKOV_22MC	20	MS
WPG23	625	2	KHARKOV_22MC	25	MS-S
WPG24	383	1	KHARKOV_22MC	70	S
WPG24	577	2	KHARKOV_22MC	70	S
CRM24	830	2	KONYA_2002	20	MS
CRM24	236	1	KONYA_2002	15	MS-S
WPG23	54	1	KONYA_2002	10	MR
WPG23	552	2	KONYA_2002	5	MR
WPG24	267	1	KONYA_2002	40	S
WPG24	877	2	KONYA_2002	35	S
CRM24	490	2	KRASNODARSKAJA_33	40	MS-S
CRM24	312	1	KRASNODARSKAJA_33	30	S
WPG23	235	1	KRASNODARSKAJA_33	10	MR
WPG23	327	2	KRASNODARSKAJA_33	5	MS
WPG24	393	1	KRASNODARSKAJA_33	30	S
WPG24	548	2	KRASNODARSKAJA_33	40	S
CRM24	233	1	KRASNODARSKAYA_99	10	R
CRM24	791	2	KRASNODARSKAYA_99	5	R
WPG24	82	1	KRASNODARSKAYA_99	20	MR
WPG24	540	2	KRASNODARSKAYA_99	5	R
CRM24	239	1	LAI_YANG_QIU	35	MS
CRM24	644	2	LAI_YANG_QIU	40	MS-S
CRM23	114	1	LEXINGTON	25	MS
CRM23	467	2	LEXINGTON	5	MS
CRM24	179	1	LEXINGTON	5	R
CRM24	744	2	LEXINGTON	5	S
WPG23	105	1	LEXINGTON	1	MR
WPG23	599	2	LEXINGTON	1	MS
WPG24	572	2	LEXINGTON	1	MS-S
WPG24	223	1	LEXINGTON	5	S
CRM24	148	1	M808_AAFC	40	S
CRM24	820	2	M808_AAFC	70	S
WPG23	33	1	M808_AAFC	35	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	571	2	M808_AAFC	25	MS
WPG24	586	2	M808_AAFC	30	MS-S
WPG24	180	1	M808_AAFC	40	S
CRM23	288	2	M808_JIC	35	MS
CRM23	199	1	M808_JIC	60	S
CRM24	43	1	M808_JIC	45	MS-S
CRM24	610	2	M808_JIC	30	S
WPG23	199	1	M808_JIC	20	MS
WPG23	386	2	M808_JIC	11	MS-S
WPG24	313	1	M808_JIC	80	S
WPG24	704	2	M808_JIC	50	S
CRM23	445	2	MCCLINTOCK	10	MR
CRM23	101	1	MCCLINTOCK	1	R
CRM24	831	2	MCCLINTOCK	0	0
CRM24	60	1	MCCLINTOCK	5	R
WPG23	74	1	MCCLINTOCK	20	MR
WPG23	350	2	MCCLINTOCK	5	R
WPG24	349	1	MCCLINTOCK	0	0
WPG24	713	2	MCCLINTOCK	10	R
CRM24	530	2	MCGUIRE	15	MS
CRM24	107	1	MCGUIRE	10	R-MR
WPG24	21	1	MCGUIRE	15	MS
WPG24	503	2	MCGUIRE	30	MS
CRM23	403	2	MIKON	60	MS-S
CRM23	175	1	MIKON	65	S
CRM24	343	1	MIKON	40	S
CRM24	798	2	MIKON	40	S
WPG23	120	1	MIKON	15	MS-S
WPG23	360	2	MIKON	25	MS-S
WPG24	37	1	MIKON	70	S
WPG24	762	2	MIKON	85	S
CRM24	339	1	MINTURKI	60	S
CRM24	629	2	MINTURKI	30	S
WPG24	104	1	MINTURKI	20	MS-S
WPG24	829	2	MINTURKI	40	MS-S
CRM24	405	1	MIR_JUB_50	60	S
CRM24	519	2	MIR_JUB_50	30	S
WPG24	840	2	MIR_JUB_50	65	S
CRM23	91	1	MIRAS	40	MS
CRM23	431	2	MIRAS	50	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	467	2	MIRAS	30	MS
CRM24	80	1	MIRAS	15	MS-S
WPG23	561	2	MIRAS	10	MR
WPG23	68	1	MIRAS	10	MS
WPG24	235	1	MIRAS	25	MS
WPG24	697	2	MIRAS	45	S
CRM23	502	2	MIRONOVSKAJA_808	40	MS-S
CRM23	197	1	MIRONOVSKAJA_808	40	S
CRM24	136	1	MIRONOVSKAJA_808	45	S
CRM24	737	2	MIRONOVSKAJA_808	60	S
WPG23	57	1	MIRONOVSKAJA_808	10	MS
WPG23	502	2	MIRONOVSKAJA_808	15	MS
WPG24	640	2	MIRONOVSKAJA_808	25	MS-S
WPG24	418	1	MIRONOVSKAJA_808	70	S
CRM23	202	1	MOATS	50	MS
CRM23	312	2	MOATS	30	MS
CRM24	485	2	MOATS	0	0
CRM24	244	1	MOATS	5	R
WPG23	576	2	MOATS	1	MR
WPG23	87	1	MOATS	5	MS
WPG24	113	1	MOATS	5	R
WPG24	559	2	MOATS	5	R
CRM23	8	1	MORGAN	30	MS
CRM23	496	2	MORGAN	20	MS
CRM24	399	1	MORGAN	50	MS
CRM24	753	2	MORGAN	40	MS
WPG23	183	1	MORGAN	0	0
WPG23	569	2	MORGAN	10	R
WPG24	95	1	MORGAN	20	MS-S
WPG24	518	2	MORGAN	40	S
CRM24	162	1	MV_22_77	20	MS
CRM24	775	2	MV_22_77	50	MS-S
WPG24	344	1	MV_22_77	30	MS-S
WPG24	695	2	MV_22_77	70	S
CRM24	443	2	NANG_RIM_NO_17	80	S
WPG24	356	1	NANG_RIM_NO_17	70	S
CRM24	160	1	NARIDANA	30	S
CRM24	695	2	NARIDANA	40	S
WPG24	641	2	NARIDANA	10	MR
CRM23	240	1	ND_NOREEN	10	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	275	2	ND_NOREEN	30	MR-MS
CRM24	440	1	ND_NOREEN	10	MR
CRM24	870	2	ND_NOREEN	10	MR
WPG23	387	2	ND_NOREEN	5	MR
WPG23	303	1	ND_NOREEN	5	R
WPG24	41	1	ND_NOREEN	10	R
WPG24	874	2	ND_NOREEN	10	R-MR
CRM24	147	1	NEELEY	70	MS
CRM24	581	2	NEELEY	25	MS
WPG24	777	2	NEELEY	80	S
CRM23	408	2	NEKOTA	20	MR
CRM23	136	1	NEKOTA	55	MS-S
CRM24	380	1	NEKOTA	20	MR
CRM24	602	2	NEKOTA	10	MS-S
WPG23	617	2	NEKOTA	10	MR
WPG23	7	1	NEKOTA	5	MS
WPG24	217	1	NEKOTA	20	MS
CRM23	90	1	NORSTAR_CITR17735	40	S
CRM23	396	2	NORSTAR_CITR17735	50	S
CRM24	51	1	NORSTAR_CITR17735	40	S
CRM24	875	2	NORSTAR_CITR17735	75	S
WPG23	491	2	NORSTAR_CITR17735	10	MS
WPG23	41	1	NORSTAR_CITR17735	50	MS-S
WPG24	401	1	NORSTAR_CITR17735	35	S
WPG24	680	2	NORSTAR_CITR17735	55	S
CRM23	98	1	NORSTAR_CN1730	30	MS
CRM23	292	2	NORSTAR_CN1730	40	MS
CRM24	277	1	NORSTAR_CN1730	70	S
CRM24	879	2	NORSTAR_CN1730	80	S
WPG23	218	1	NORSTAR_CN1730	20	MR
WPG23	584	2	NORSTAR_CN1730	25	MR-MS
WPG24	227	1	NORSTAR_CN1730	25	S
WPG24	821	2	NORSTAR_CN1730	75	S
CRM23	72	1	NORSTAR_OTTAWA	65	MS
CRM23	266	2	NORSTAR_OTTAWA	70	MS-S
CRM24	9	1	NORSTAR_OTTAWA	50	S
CRM24	495	2	NORSTAR_OTTAWA	65	S
WPG23	620	2	NORSTAR_OTTAWA	5	MS
WPG23	21	1	NORSTAR_OTTAWA	30	MS-S
WPG24	126	1	NORSTAR_OTTAWA	50	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	856	2	NORSTAR_OTTAWA	50	S
CRM23	301	2	NORWIN	40	MR-MS
CRM23	194	1	NORWIN	10	MS
CRM24	662	2	NORWIN	35	MS
CRM24	412	1	NORWIN	30	MS-S
WPG23	195	1	NORWIN	5	R
WPG23	369	2	NORWIN	10	R
WPG24	782	2	NORWIN	30	MS
WPG24	124	1	NORWIN	35	MS-S
CRM24	191	1	NUPLAINS	10	R
CRM24	613	2	NUPLAINS	10	R
WPG24	199	1	NUPLAINS	20	MR
CRM23	164	1	NUSKY	20	MS
CRM23	478	2	NUSKY	20	MS
CRM24	26	1	NUSKY	30	MS-S
CRM24	618	2	NUSKY	20	MS-S
WPG23	144	1	NUSKY	25	MR
WPG23	610	2	NUSKY	10	MR
WPG24	254	1	NUSKY	30	MS
WPG24	460	2	NUSKY	40	MS-S
CRM24	287	1	NUWEST	10	MS
CRM24	711	2	NUWEST	10	MS-S
WPG24	222	1	NUWEST	5	MS-S
WPG24	682	2	NUWEST	30	MS-S
CRM24	238	1	OAC_CONSTELLATION	60	MS
CRM24	500	2	OAC_CONSTELLATION	60	S
WPG24	143	1	OAC_CONSTELLATION	20	MS-S
WPG24	510	2	OAC_CONSTELLATION	70	S
CRM23	311	2	ODESSKAJA_51	25	MR-MS
CRM23	130	1	ODESSKAJA_51	40	MS
CRM24	714	2	ODESSKAJA_51	30	MS-S
CRM24	391	1	ODESSKAJA_51	50	S
WPG23	90	1	ODESSKAJA_51	15	MS
WPG23	531	2	ODESSKAJA_51	5	R
WPG24	46	1	ODESSKAJA_51	50	S
WPG24	776	2	ODESSKAJA_51	90	S
CRM24	290	1	ORO	50	S
CRM24	857	2	ORO	50	S
WPG23	492	2	ORO	11	MR-MS
WPG23	185	1	ORO	15	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	645	2	ORO	20	MS-S
WPG24	16	1	ORO	30	S
CRM23	149	1	OVERLAND	60	MS
CRM23	281	2	OVERLAND	55	S
CRM24	575	2	OVERLAND	30	MS-S
CRM24	207	1	OVERLAND	40	S
WPG23	123	1	OVERLAND	10	MR
WPG23	645	2	OVERLAND	10	MR
WPG24	490	2	OVERLAND	35	MS
WPG24	116	1	OVERLAND	30	S
CRM24	424	1	PATRAS	20	MS-S
CRM24	653	2	PATRAS	25	S
WPG23	202	1	PATRAS	10	MS
WPG23	358	2	PATRAS	5	MS
WPG24	419	1	PATRAS	20	MS-S
CRM23	460	2	PAUL	20	MS
CRM23	239	1	PAUL	25	S
CRM24	853	2	PAUL	30	MS
CRM24	439	1	PAUL	50	S
WPG23	98	1	PAUL	15	MS-S
WPG23	496	2	PAUL	5	R-MR
WPG24	208	1	PAUL	25	MS-S
WPG24	591	2	PAUL	50	S
CRM23	347	2	PEREGRINE	10	MR-MS
CRM23	188	1	PEREGRINE	5	MS
CRM24	324	1	PEREGRINE	0	0
CRM24	668	2	PEREGRINE	5	MR
WPG23	145	1	PEREGRINE	10	R
WPG23	578	2	PEREGRINE	1	R
WPG24	133	1	PEREGRINE	10	R
WPG24	638	2	PEREGRINE	5	R
CRM24	674	2	PERLO	25	MS-S
CRM24	40	1	PERLO	80	S
WPG24	130	1	PERLO	70	S
CRM24	639	2	PLAINSMAN_V	20	MR
CRM24	332	1	PLAINSMAN_V	10	R
WPG24	6	1	PLAINSMAN_V	0	0
WPG24	767	2	PLAINSMAN_V	20	MR-MS
WPG23	69	1	POTENZIAL	0	0
WPG23	466	2	POTENZIAL	0	0

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	607	2	PRIESLEY	1	MS
WPG24	426	1	PRIESLEY	1	MR
WPG24	550	2	PRIESLEY	5	MS
CRM24	545	2	PRINCETON	20	MS-S
CRM24	375	1	PRINCETON	25	S
WPG24	382	1	PRINCETON	20	MS-S
WPG24	875	2	PRINCETON	35	S
CRM23	51	1	PRIVILEG	1	MR
CRM23	371	2	PRIVILEG	5	MR
CRM24	150	1	PRIVILEG	0	0
CRM24	587	2	PRIVILEG	0	0
WPG23	412	2	PRIVILEG	0	0
WPG23	263	1	PRIVILEG	10	R
WPG24	502	2	PRIVILEG	5	R-MR
CRM24	396	1	PRO_81	5	MS
CRM24	481	2	PRO_81	5	MS
WPG24	173	1	PRO_81	55	MS
CRM24	17	1	PROWERS_99	5	R
CRM24	722	2	PROWERS_99	10	R
WPG24	258	1	PROWERS_99	25	MR
CRM23	68	1	RADIANT	60	MS
CRM23	451	2	RADIANT	40	S
CRM24	21	1	RADIANT	55	S
CRM24	521	2	RADIANT	30	S
WPG23	121	1	RADIANT	20	MS
WPG23	503	2	RADIANT	20	MS
WPG24	11	1	RADIANT	25	MS-S
WPG24	479	2	RADIANT	70	S
CRM24	305	1	RCATL33	60	MS-S
CRM24	756	2	RCATL33	30	MS-S
WPG24	297	1	RCATL33	70	S
WPG24	527	2	RCATL33	50	S
CRM24	507	2	RENAN	5	MR
CRM24	209	1	RENAN	50	MS
WPG23	112	1	RENAN	0	0
WPG23	568	2	RENAN	5	MR
WPG24	162	1	RENAN	1	MR
WPG24	465	2	RENAN	5	R
CRM24	211	1	RIDA	70	MS
CRM24	778	2	RIDA	70	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	287	1	RIDA	30	MR
WPG23	591	2	RIDA	25	MR-MS
WPG24	380	1	RIDA	70	S
WPG24	470	2	RIDA	70	S
CRM23	321	2	ROUGH RIDER	5	MR
CRM23	37	1	ROUGH RIDER	5	MS
CRM24	427	1	ROUGH RIDER	10	R
CRM24	821	2	ROUGH RIDER	20	R
WPG23	160	1	ROUGH RIDER	20	R
WPG23	385	2	ROUGH RIDER	10	R
WPG24	123	1	ROUGH RIDER	1	MS
WPG24	646	2	ROUGH RIDER	20	R-MR
CRM23	23	1	RUBY	70	MR-MS
CRM23	329	2	RUBY	45	MR-MS
CRM24	145	1	RUBY	15	MS-S
CRM24	679	2	RUBY	50	S
WPG23	136	1	RUBY	5	MR
WPG23	351	2	RUBY	10	MR
WPG24	150	1	RUBY	25	S
WPG24	853	2	RUBY	45	S
CRM24	185	1	RUMELI	0	0
CRM24	873	2	RUMELI	0	0
WPG23	481	2	RUMELI	0	0
WPG23	155	1	RUMELI	5	R
WPG24	256	1	RUMELI	10	MR
WPG24	806	2	RUMELI	55	S
CRM24	258	1	SADABOZU	20	MR
WPG24	472	2	SADABOZU	25	MS
CRM24	469	2	SEU_SEUN_12	40	S
CRM23	262	2	SEWARD	15	MR-MS
CRM23	209	1	SEWARD	30	MS
CRM24	862	2	SEWARD	20	MS
CRM24	397	1	SEWARD	25	MS-S
WPG23	216	1	SEWARD	15	R
WPG23	402	2	SEWARD	25	R-MR
WPG24	118	1	SEWARD	30	MS
WPG24	820	2	SEWARD	40	MS-S
CRM23	119	1	SKJALDAR	30	MS
CRM23	426	2	SKJALDAR	40	MS
CRM24	92	1	SKJALDAR	50	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	817	2	SKJALDAR	60	S
WPG23	506	2	SKJALDAR	20	MS
WPG23	130	1	SKJALDAR	20	R-MR
WPG24	129	1	SKJALDAR	20	MS-S
WPG24	549	2	SKJALDAR	60	S
CRM24	44	1	STRELINSKAJA_MESTNAJA	60	S
CRM24	816	2	STRELINSKAJA_MESTNAJA	70	S
WPG23	49	1	STRELINSKAJA_MESTNAJA	45	MS
WPG23	602	2	STRELINSKAJA_MESTNAJA	40	MS-S
WPG24	417	1	STRELINSKAJA_MESTNAJA	65	S
WPG24	450	2	STRELINSKAJA_MESTNAJA	65	S
CRM24	667	2	SU_KAE_NO_169	40	MS-S
WPG23	30	1	SU_KAE_NO_169	10	MR
WPG23	490	2	SU_KAE_NO_169	10	MR
CRM23	303	2	SUNDANCE	35	MS
CRM23	78	1	SUNDANCE	80	MS-S
CRM24	251	1	SUNDANCE	65	S
CRM24	454	2	SUNDANCE	45	S
WPG23	628	2	SUNDANCE	10	MR-MS
WPG23	97	1	SUNDANCE	10	MS
WPG24	315	1	SUNDANCE	80	S
WPG24	722	2	SUNDANCE	45	S
CRM23	112	1	SUNRISE	10	MR
CRM23	327	2	SUNRISE	5	MR
CRM24	635	2	SUNRISE	0	0
CRM24	217	1	SUNRISE	10	MR
WPG23	364	2	SUNRISE	5	MR
WPG23	267	1	SUNRISE	10	R
WPG24	64	1	SUNRISE	10	MR
WPG24	728	2	SUNRISE	5	R
CRM23	97	1	SWAINSON	5	MR
CRM23	382	2	SWAINSON	5	MR
CRM24	636	2	SWAINSON	0	0
CRM24	137	1	SWAINSON	5	R
WPG23	567	2	SWAINSON	10	R
WPG23	37	1	SWAINSON	20	R-MR
WPG24	692	2	SWAINSON	0	0
WPG24	394	1	SWAINSON	10	R
CRM23	368	2	THUNDERBIRD	30	MR
CRM23	87	1	THUNDERBIRD	40	R-MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	389	1	THUNDERBIRD	0	0
CRM24	735	2	THUNDERBIRD	0	0
WPG23	541	2	THUNDERBIRD	0	0
WPG23	80	1	THUNDERBIRD	10	MR
WPG24	539	2	THUNDERBIRD	5	MR
WPG24	309	1	THUNDERBIRD	10	R-MR
CRM24	326	1	TIBER	60	S
CRM24	523	2	TIBER	40	S
WPG24	536	2	TIBER	30	MS-S
WPG24	352	1	TIBER	40	S
CRM24	90	1	U9_26_32	30	MS
CRM24	590	2	U9_26_32	5	S
WPG24	811	2	U9_26_32	70	S
CRM23	201	1	ULIANOVKA	80	MS-S
CRM23	404	2	ULIANOVKA	45	S
CRM24	203	1	ULIANOVKA	65	S
CRM24	604	2	ULIANOVKA	50	S
WPG23	152	1	ULIANOVKA	5	MS
WPG23	648	2	ULIANOVKA	15	MS
WPG24	291	1	ULIANOVKA	40	S
WPG24	523	2	ULIANOVKA	40	S
CRM23	425	2	UM1174	20	MR
CRM23	236	1	UM1174	35	MR-MS
CRM24	835	2	UM1174	30	MS
CRM24	333	1	UM1174	5	R
WPG23	476	2	UM1174	5	MR
WPG23	77	1	UM1174	5	MS
WPG24	538	2	UM1174	10	MR
CRM23	196	1	UM5116	30	MR-MS
CRM23	484	2	UM5116	0.5	R
CRM24	249	1	UM5116	10	MS
CRM24	543	2	UM5116	5	MS-S
WPG23	228	1	UM5116	25	R
WPG23	612	2	UM5116	10	R
WPG24	535	2	UM5116	10	MR-MS
CRM23	88	1	UM6233	30	MR
CRM23	409	2	UM6233	20	MR-MS
CRM24	650	2	UM6233	20	MS
CRM24	204	1	UM6233	1	R
WPG23	261	1	UM6233	15	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	586	2	UM6233	5	R
WPG24	330	1	UM6233	40	MR
WPG24	625	2	UM6233	20	R-MR
CRM23	394	2	VAKKA	40	MS
CRM23	76	1	VAKKA	70	MS-S
CRM24	278	1	VAKKA	65	S
CRM24	806	2	VAKKA	60	S
WPG23	252	1	VAKKA	20	MR
WPG23	499	2	VAKKA	10	MS
WPG24	507	2	VAKKA	40	MS-S
WPG24	327	1	VAKKA	70	S
CRM24	322	1	VONA	0	0
CRM23	430	2	W214	60	MR-MS
CRM23	29	1	W214	40	MS-S
CRM24	704	2	W214	40	MS
CRM24	300	1	W214	65	S
WPG23	590	2	W214	30	MR-MS
WPG23	201	1	W214	25	MS-S
WPG24	20	1	W214	35	S
WPG24	604	2	W214	50	S
CRM23	49	1	W238	5	MR
CRM23	276	2	W238	10	R
CRM24	328	1	W238	0	0
CRM24	707	2	W238	10	R
WPG23	429	2	W238	0	0
WPG23	103	1	W238	5	R
WPG24	533	2	W238	10	MR
WPG24	44	1	W238	10	R
CRM24	15	1	W279	30	MS
CRM24	860	2	W279	10	MS
WPG24	139	1	W279	10	MS-S
WPG24	698	2	W279	35	MS-S
CRM24	220	1	W286	25	MS
CRM24	856	2	W286	25	MS
WPG24	18	1	W286	15	MS-S
WPG24	819	2	W286	70	S
CRM24	28	1	W297	20	MR
CRM24	573	2	W297	10	MS
WPG24	423	1	W297	10	MR
WPG24	468	2	W297	25	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	567	2	W301	30	MS-S
CRM24	231	1	W301	60	S
WPG24	635	2	W301	40	MS
WPG24	30	1	W301	40	S
CRM23	5	1	W304	30	MS
CRM23	290	2	W304	40	MS
CRM24	302	1	W304	40	MS
CRM24	673	2	W304	70	S
WPG23	328	2	W304	10	MR-MS
WPG23	26	1	W304	20	MS
WPG24	39	1	W304	30	MS-S
WPG24	831	2	W304	70	S
CRM24	223	1	W308	0	0
CRM24	632	2	W308	0	0
WPG24	594	2	W308	0	0
WPG24	229	1	W308	5	MR
CRM24	192	1	W317	5	R
CRM24	699	2	W317	10	R
WPG23	163	1	W317	0	0
WPG23	647	2	W317	10	R
WPG24	220	1	W317	20	MR-MS
WPG24	671	2	W317	10	R-MR
CRM24	597	2	W319	0	0
CRM24	138	1	W319	5	R
WPG24	98	1	W319	15	MR
WPG24	709	2	W319	10	MR
CRM24	206	1	W321	40	MS
CRM24	516	2	W321	30	MS
WPG24	156	1	W321	25	MS
WPG24	677	2	W321	40	MS-S
CRM24	113	1	W343	20	MS
CRM24	452	2	W343	10	MS-S
WPG24	52	1	W343	20	MS-S
WPG24	724	2	W343	50	S
CRM23	99	1	W349	70	MS-S
CRM23	420	2	W349	60	MS-S
CRM24	340	1	W349	50	S
CRM24	680	2	W349	60	S
WPG23	257	1	W349	30	MS
WPG23	510	2	W349	10	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	326	1	W349	50	S
WPG24	605	2	W349	60	S
CRM23	144	1	W350	40	MS
CRM23	458	2	W350	40	MS
CRM24	1	1	W350	40	S
CRM24	808	2	W350	60	S
WPG23	563	2	W350	25	MS
WPG23	12	1	W350	25	MS-S
WPG24	241	1	W350	25	MS
WPG24	566	2	W350	40	MS-S
CRM24	696	2	W358	25	MS-S
CRM24	419	1	W358	40	S
WPG24	386	1	W358	45	S
WPG24	520	2	W358	50	S
CRM23	232	1	W360	30	MS
CRM23	364	2	W360	40	MS
CRM24	91	1	W360	80	S
CRM24	483	2	W360	60	S
WPG23	295	1	W360	15	MR-MS
WPG23	539	2	W360	202	MR-MS
WPG24	253	1	W360	70	S
WPG24	733	2	W360	80	S
CRM24	8	1	W361	60	S
CRM24	642	2	W361	85	S
WPG24	634	2	W361	40	MS-S
WPG24	390	1	W361	80	S
CRM23	383	2	W371	20	MS
CRM23	223	1	W371	30	S
CRM24	598	2	W371	5	MS-S
CRM24	349	1	W371	40	S
WPG23	246	1	W371	30	MR
WPG23	472	2	W371	5	MS
WPG24	67	1	W371	30	MS
WPG24	706	2	W371	30	MS-S
CRM24	240	1	W372	60	S
CRM24	767	2	W372	65	S
WPG24	109	1	W372	35	S
WPG24	454	2	W372	70	S
CRM24	171	1	W378	0	0
CRM24	718	2	W378	0	0

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	312	1	W378	0	0
WPG24	498	2	W378	5	R
CRM24	615	2	W380	30	MS-S
CRM24	270	1	W380	70	S
WPG24	77	1	W380	20	MS-S
CRM23	186	1	W383	70	MS-S
CRM23	443	2	W383	70	MS-S
CRM24	544	2	W383	50	MS-S
CRM24	23	1	W383	70	S
WPG23	193	1	W383	15	MR
WPG23	468	2	W383	15	MR-MS
WPG24	726	2	W383	70	S
CRM24	394	1	W384	70	S
CRM24	867	2	W384	80	S
WPG24	207	1	W384	40	S
WPG24	544	2	W384	30	S
CRM23	285	2	W385	60	MS-S
CRM23	71	1	W385	70	S
CRM24	684	2	W385	50	MS-S
CRM24	360	1	W385	65	S
WPG23	154	1	W385	35	MR-MS
WPG23	449	2	W385	15	MS
WPG24	385	1	W385	50	S
WPG24	736	2	W385	75	S
CRM23	349	2	W386	50	MS-S
CRM23	238	1	W386	30	S
CRM24	727	2	W386	40	MS-S
CRM24	52	1	W386	55	S
WPG23	115	1	W386	5	MS
WPG23	326	2	W386	10	MS
WPG24	626	2	W386	30	MS-S
WPG24	45	1	W386	40	S
CRM24	358	1	W388	50	MS
CRM24	685	2	W388	65	S
WPG23	395	2	W388	25	MR-MS
WPG23	126	1	W388	30	MS
WPG24	613	2	W388	50	MS-S
WPG24	304	1	W388	75	S
CRM24	253	1	W389	70	S
CRM24	764	2	W389	30	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	62	1	W389	25	MR-MS
WPG23	643	2	W389	25	MR-MS
WPG24	515	2	W389	25	MS-S
WPG24	422	1	W389	70	S
CRM24	395	1	W390	60	S
CRM24	445	2	W390	50	S
WPG24	185	1	W390	60	S
WPG24	803	2	W390	70	S
CRM23	401	2	W391	50	MR-MS
CRM23	45	1	W391	90	MS-S
CRM24	425	1	W391	40	MS
CRM24	772	2	W391	60	S
WPG23	281	1	W391	10	MR
WPG23	521	2	W391	6	MR-MS
WPG24	387	1	W391	70	S
WPG24	719	2	W391	50	S
CRM24	109	1	W392	15	MS-S
CRM24	683	2	W392	70	S
WPG24	163	1	W392	30	MS
WPG24	705	2	W392	50	MS
CRM23	174	1	W393	50	MS
CRM23	302	2	W393	45	MS-S
CRM24	382	1	W393	60	S
CRM24	553	2	W393	55	S
WPG23	231	1	W393	45	MS
WPG23	454	2	W393	20	MS
WPG24	262	1	W393	65	S
WPG24	844	2	W393	60	S
CRM23	205	1	W397	60	MS
CRM23	352	2	W397	35	MS
CRM24	140	1	W397	60	MS
CRM24	448	2	W397	60	S
WPG23	117	1	W397	20	MR
WPG23	407	2	W397	6	MR-MS
WPG24	174	1	W397	30	MS-S
WPG24	796	2	W397	55	S
CRM23	472	2	W398	40	MS
CRM23	134	1	W398	90	MS-S
CRM24	359	1	W398	30	MS
CRM24	470	2	W398	30	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	72	1	W398	10	MR-MS
WPG23	430	2	W398	20	MS
WPG24	458	2	W398	50	MS-S
WPG24	311	1	W398	50	S
CRM24	586	2	W399	10	MS
CRM24	388	1	W399	10	R
WPG23	329	2	W399	1	MR
WPG23	254	1	W399	1	MS
WPG24	347	1	W399	1	MS
WPG24	656	2	W399	5	MS-S
CRM23	191	1	W400	60	MS
CRM23	367	2	W400	50	MS
CRM24	526	2	W400	40	MS-S
CRM24	301	1	W400	45	S
WPG23	302	1	W400	10	MS
WPG23	630	2	W400	30	MS
WPG24	389	1	W400	40	MS
WPG24	832	2	W400	65	S
CRM23	207	1	W401	30	MR
CRM23	388	2	W401	40	MR
CRM24	73	1	W401	1	R
CRM24	453	2	W401	20	R
WPG23	236	1	W401	15	R
WPG23	579	2	W401	5	R
WPG24	206	1	W401	30	MR
WPG24	610	2	W401	10	MR-MS
CRM23	251	1	W404	40	MS
CRM23	375	2	W404	30	MS
CRM24	336	1	W404	35	MS
CRM24	579	2	W404	40	S
WPG23	421	2	W404	25	MR-MS
WPG23	19	1	W404	30	MS
WPG24	463	2	W404	35	MS-S
WPG24	128	1	W404	45	S
CRM23	348	2	W405	45	MS
CRM23	56	1	W405	90	MS-S
CRM24	789	2	W405	25	MS
CRM24	264	1	W405	60	S
WPG23	196	1	W405	30	MR
WPG23	511	2	W405	15	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	140	1	W405	30	MS
WPG24	445	2	W405	40	MS-S
CRM24	357	1	W406	0	0
CRM24	849	2	W406	10	R
WPG24	299	1	W406	10	MR
WPG24	858	2	W406	5	R
CRM24	174	1	W407	10	R
CRM24	741	2	W407	5	R
WPG24	567	2	W407	20	MR
WPG24	66	1	W407	10	R-MR
CRM23	213	1	W408	10	MR
CRM23	361	2	W408	10	MR-MS
CRM24	156	1	W408	10	R
CRM24	641	2	W408	20	R
WPG23	47	1	W408	0	0
WPG23	457	2	W408	5	R
WPG24	282	1	W408	0	0
WPG24	644	2	W408	5	R
CRM23	67	1	W409	20	MR
CRM23	323	2	W409	15	MR
CRM24	577	2	W409	20	MR
CRM24	285	1	W409	5	R
WPG23	147	1	W409	10	R
WPG23	500	2	W409	5	R
WPG24	366	1	W409	10	R
WPG24	794	2	W409	25	R-MR
CRM24	42	1	W410	30	MS-S
CRM24	515	2	W410	35	MS-S
WPG24	457	2	W410	60	MS-S
WPG24	268	1	W410	35	S
CRM24	5	1	W411	10	R
CRM24	759	2	W411	20	R
WPG23	415	2	W411	5	MR
WPG23	81	1	W411	5	R
WPG24	239	1	W411	20	R-MR
WPG24	754	2	W411	10	R-MR
CRM24	33	1	W412	15	MR
CRM24	801	2	W412	10	R
WPG24	391	1	W412	5	R
WPG24	652	2	W412	10	R-MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	84	1	W413	60	S
CRM24	616	2	W413	40	S
WPG23	230	1	W413	25	MR-MS
WPG23	553	2	W413	20	MR-MS
WPG24	506	2	W413	65	MS-S
WPG24	339	1	W413	40	S
CRM23	427	2	W415	5	MR
CRM23	132	1	W415	20	MS
CRM24	183	1	W415	0	0
CRM24	640	2	W415	5	S
WPG23	269	1	W415	10	R
WPG23	633	2	W415	5	R
WPG24	245	1	W415	0	0
WPG24	530	2	W415	5	R
CRM24	254	1	W416	5	R
CRM24	742	2	W416	10	R
WPG24	107	1	W416	10	R
WPG24	771	2	W416	10	R
CRM23	151	1	W417	30	MS
CRM23	355	2	W417	40	MS
CRM24	104	1	W417	50	S
CRM24	627	2	W417	30	S
WPG23	209	1	W417	35	MR-MS
WPG23	333	2	W417	20	MS
WPG24	167	1	W417	30	MS-S
WPG24	742	2	W417	40	MS-S
CRM23	256	1	W418	1	R
CRM23	298	2	W418	5	R
CRM24	329	1	W418	0	0
CRM24	558	2	W418	0	0
WPG23	624	2	W418	0	0
WPG23	198	1	W418	5	MR
WPG24	3	1	W418	10	MR
WPG24	665	2	W418	5	R
CRM23	92	1	W419	60	MS
CRM23	293	2	W419	35	MS
CRM24	854	2	W419	30	MS-S
CRM24	6	1	W419	50	S
WPG23	91	1	W419	30	MR-MS
WPG23	379	2	W419	40	MR-MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	76	1	W419	50	S
WPG24	813	2	W419	65	S
CRM23	376	2	W420	25	MR-MS
CRM23	82	1	W420	70	MS
CRM24	10	1	W420	20	MS-S
CRM24	825	2	W420	30	MS-S
WPG23	55	1	W420	30	MR
WPG23	384	2	W420	15	MR
WPG24	631	2	W420	20	MS
WPG24	224	1	W420	35	MS-S
CRM23	250	1	W421	60	MS
CRM23	482	2	W421	30	MS-S
CRM24	57	1	W421	20	MS
CRM24	706	2	W421	30	MS
WPG23	167	1	W421	15	MR
WPG23	534	2	W421	15	MR
WPG24	272	1	W421	35	MS-S
WPG24	749	2	W421	40	MS-S
CRM24	529	2	W424	20	MS
CRM24	308	1	W424	40	S
WPG24	373	1	W424	65	S
WPG24	569	2	W424	70	S
CRM23	2	1	W426	10	R
CRM23	410	2	W426	20	R
CRM24	273	1	W426	20	MR
WPG23	164	1	W426	5	R
WPG23	446	2	W426	5	R
WPG24	32	1	W426	10	MR
WPG24	812	2	W426	40	MS-S
CRM24	168	1	W427	20	MR
CRM24	609	2	W427	5	R
WPG24	96	1	W427	20	MR
WPG24	755	2	W427	1	MS-S
CRM23	185	1	W428	40	MR
CRM23	407	2	W428	30	R
CRM24	417	1	W428	20	R
CRM24	580	2	W428	10	R-MR
WPG23	245	1	W428	20	R
WPG23	614	2	W428	5	R
WPG24	310	1	W428	30	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	855	2	W428	35	MR
CRM23	155	1	W429	1	MR
CRM23	258	2	W429	10	R-MR
CRM24	837	2	W429	20	MR
CRM24	350	1	W429	10	R
WPG23	336	2	W429	1	MR
WPG23	300	1	W429	20	R
WPG24	5	1	W429	10	MR
WPG24	545	2	W429	10	R
CRM24	88	1	W431	5	R
CRM24	847	2	W431	10	R-MR
WPG24	190	1	W431	25	MR
WPG24	809	2	W431	25	MR
CRM23	189	1	W432	30	MR
CRM23	291	2	W432	25	MR
CRM24	765	2	W432	10	R
CRM24	199	1	W432	20	R-MR
WPG23	76	1	W432	5	R
WPG23	374	2	W432	5	R
WPG24	264	1	W432	25	R-MR
WPG24	512	2	W432	10	R-MR
CRM23	399	2	W433	40	MR-MS
CRM23	190	1	W433	50	S
CRM24	671	2	W433	40	MS-S
CRM24	234	1	W433	30	R
WPG23	214	1	W433	30	MR
WPG23	456	2	W433	15	MR-MS
WPG24	551	2	W433	30	MS
WPG24	23	1	W433	35	MS-S
CRM23	106	1	W435	30	MR
CRM23	500	2	W435	30	R
CRM24	222	1	W435	25	MR
CRM24	614	2	W435	10	R
WPG23	271	1	W435	10	R
WPG23	366	2	W435	5	R
WPG24	244	1	W435	15	MR
WPG24	622	2	W435	20	R
CRM24	733	2	W436	10	MR
CRM24	122	1	W436	5	R
WPG24	231	1	W436	5	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	480	2	W436	10	R
CRM24	367	1	W437	10	R
CRM24	447	2	W437	10	R
WPG24	300	1	W437	10	MR
WPG24	800	2	W437	20	R-MR
CRM23	184	1	W438	30	MR
CRM23	485	2	W438	30	MR
CRM24	734	2	W438	10	MR
CRM24	213	1	W438	10	R
WPG23	175	1	W438	5	R
WPG23	595	2	W438	1	R
WPG24	26	1	W438	10	R
WPG24	669	2	W438	20	R-MR
CRM23	441	2	W439	30	MR
CRM23	96	1	W439	50	R-MR
CRM24	294	1	W439	5	R
CRM24	796	2	W439	10	R
WPG23	92	1	W439	15	R
WPG23	546	2	W439	5	R
WPG24	334	1	W439	20	MR-MS
WPG24	766	2	W439	30	MS
CRM24	729	2	W440	0	0
CRM24	101	1	W440	5	R
WPG24	694	2	W440	0	0
WPG24	355	1	W440	5	MR
CRM23	63	1	W441	5	MS
CRM23	411	2	W441	10	MS
CRM24	119	1	W441	0	0
CRM24	548	2	W441	0	0
WPG23	239	1	W441	5	R
WPG23	461	2	W441	5	R
WPG24	686	2	W441	0	0
WPG24	177	1	W441	5	MR
CRM24	407	1	W442	40	MS-S
CRM24	565	2	W442	50	S
WPG24	289	1	W442	65	S
WPG24	667	2	W442	60	S
CRM24	83	1	W443	10	R
CRM24	623	2	W443	5	R
WPG24	90	1	W443	30	R-MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	731	2	W443	10	R-MR
CRM23	127	1	W444	20	MS
CRM23	506	2	W444	25	MS
CRM24	403	1	W444	40	MS
CRM24	626	2	W444	20	MS
WPG23	618	2	W444	30	MR-MS
WPG23	129	1	W444	15	MS
WPG24	306	1	W444	30	MS-S
WPG24	469	2	W444	40	MS-S
CRM24	464	2	W446	10	MS-S
CRM24	422	1	W446	25	S
WPG24	214	1	W446	30	S
WPG24	818	2	W446	50	S
CRM24	96	1	W447	0	0
CRM24	771	2	W447	0	0
WPG24	59	1	W447	10	MR
WPG24	750	2	W447	20	R-MR
CRM23	384	2	W448	5	MR
CRM23	120	1	W448	40	R-MR
CRM24	757	2	W448	10	MR
CRM24	198	1	W448	5	R
WPG23	519	2	W448	0	0
WPG23	35	1	W448	25	R
WPG24	478	2	W448	30	MR
WPG24	85	1	W448	10	R-MR
CRM24	65	1	W449	0	0
CRM24	472	2	W449	20	MR
WPG24	283	1	W449	20	R-MR
WPG24	467	2	W449	10	R-MR
CRM24	411	1	W450	10	MR
CRM24	612	2	W450	10	R
WPG24	158	1	W450	10	MR
WPG24	609	2	W450	5	R-MR
CRM23	272	2	W451	50	MS
CRM23	9	1	W451	40	MS-S
CRM24	22	1	W451	50	S
CRM24	442	2	W451	65	S
WPG23	603	2	W451	15	MR
WPG23	319	1	W451	40	R-MR
WPG24	320	1	W451	40	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	868	2	W451	60	MS-S
CRM23	61	1	W452	25	MR
CRM23	267	2	W452	25	MR-MS
CRM24	762	2	W452	30	MR
CRM24	433	1	W452	5	R
WPG23	238	1	W452	15	R
WPG23	632	2	W452	5	R
WPG24	261	1	W452	10	MR
WPG24	589	2	W452	10	R
CRM23	215	1	W453	30	MR
CRM23	444	2	W453	15	MS
CRM24	205	1	W453	20	MR
CRM24	708	2	W453	20	MS
WPG23	52	1	W453	10	MR
WPG23	361	2	W453	5	MS
WPG24	159	1	W453	1	MR
CRM23	178	1	W455	1	MS
CRM23	475	2	W455	10	R
CRM24	269	1	W455	0	0
CRM24	751	2	W455	0	0
WPG23	516	2	W455	5	MR
WPG23	233	1	W455	5	R
WPG24	209	1	W455	0	0
WPG24	526	2	W455	1	R
CRM23	123	1	W456	5	MR
CRM23	322	2	W456	10	MR
CRM24	782	2	W456	0	0
CRM24	177	1	W456	5	R
WPG23	601	2	W456	0	0
WPG23	184	1	W456	10	R
WPG24	57	1	W456	10	R
WPG24	581	2	W456	10	R
CRM23	158	1	W457	10	MR
CRM23	340	2	W457	20	MR
CRM24	281	1	W457	15	R
CRM24	682	2	W457	20	R-MR
WPG23	128	1	W457	10	MR
WPG23	640	2	W457	5	R
WPG24	278	1	W457	10	R
WPG24	674	2	W457	5	R-MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	444	2	W458	0	0
CRM24	418	1	W458	10	R
WPG24	240	1	W458	0	0
WPG24	672	2	W458	5	R
CRM24	725	2	W460	5	MR
CRM24	197	1	W460	1	R
WPG24	878	2	W460	25	MR
WPG24	169	1	W460	20	MR-MS
CRM24	317	1	W461	0	0
CRM24	578	2	W461	5	MS
WPG24	246	1	W461	0	0
WPG24	500	2	W461	10	MR-MS
CRM24	542	2	W462	0	0
CRM24	142	1	W462	10	R
WPG23	44	1	W462	10	MR
WPG23	325	2	W462	5	R
WPG24	360	1	W462	10	MR
WPG24	451	2	W462	25	MR
CRM23	111	1	W463	10	MR
CRM23	350	2	W463	25	R-MR
CRM24	282	1	W463	0	0
CRM24	721	2	W463	0	0
WPG23	23	1	W463	0	0
WPG23	459	2	W463	0	0
WPG24	295	1	W463	20	MR
WPG24	859	2	W463	10	R
CRM24	248	1	W465	5	R
CRM24	648	2	W465	5	R
WPG24	83	1	W465	20	MR
WPG24	830	2	W465	25	MR
CRM23	415	2	W466	5	MR
CRM23	30	1	W466	5	R
CRM24	30	1	W466	0	0
CRM24	752	2	W466	0	0
WPG23	570	2	W466	0	0
WPG23	297	1	W466	10	R
WPG24	55	1	W466	5	R
WPG24	521	2	W466	5	R
CRM24	279	1	W467	5	R
CRM24	688	2	W467	10	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	730	2	W467	5	R
WPG24	436	1	W467	20	R-MR
CRM24	31	1	W469	5	R
CRM24	572	2	W469	10	R
WPG24	439	1	W469	20	R-MR
WPG24	464	2	W469	10	R-MR
CRM23	206	1	W471	10	MR
CRM23	462	2	W471	5	R-MR
CRM24	112	1	W471	0	0
CRM24	621	2	W471	0	0
WPG23	631	2	W471	0	0
WPG23	318	1	W471	5	R
WPG24	243	1	W471	5	R
WPG24	764	2	W471	5	R
CRM23	115	1	W472	5	MR
CRM23	263	2	W472	1	MR
CRM24	647	2	W472	0	0
CRM24	274	1	W472	5	R
WPG23	311	1	W472	0	0
WPG23	377	2	W472	0	0
WPG24	452	2	W472	10	R
CRM24	508	2	W473	0	0
CRM24	103	1	W473	5	R
WPG24	428	1	W473	0	0
WPG24	846	2	W473	0	0
CRM24	315	1	W475	0	0
CRM24	465	2	W475	10	MR
WPG24	781	2	W475	30	MR-MS
WPG24	307	1	W475	5	R-MR
CRM24	45	1	W476	0	0
CRM24	509	2	W476	0	0
WPG23	170	1	W476	0	0
WPG23	375	2	W476	0	0
WPG24	792	2	W476	0	0
WPG24	328	1	W476	5	R
CRM23	228	1	W477	20	MS
CRM23	300	2	W477	10	MS
CRM24	522	2	W477	5	MS-S
CRM24	151	1	W477	5	S
WPG23	250	1	W477	5	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	535	2	W477	5	MR
WPG24	274	1	W477	5	MS-S
WPG24	795	2	W477	25	MS-S
CRM23	117	1	W479	60	MS
CRM23	446	2	W479	40	MS
CRM24	170	1	W479	30	MS
CRM24	859	2	W479	35	S
WPG23	536	2	W479	20	MR-MS
WPG23	324	1	W479	15	MS-S
WPG24	318	1	W479	45	S
WPG24	768	2	W479	70	S
CRM23	248	1	W481	55	MS-S
CRM23	480	2	W481	25	MS-S
CRM24	493	2	W481	20	MS
CRM24	431	1	W481	25	MS-S
WPG23	237	1	W481	5	MR
WPG23	409	2	W481	5	R
WPG24	865	2	W481	40	MS-S
WPG24	437	1	W481	60	S
CRM23	169	1	W482	20	MR
CRM23	511	2	W482	5	MR
CRM24	376	1	W482	10	R
CRM24	698	2	W482	10	R
WPG23	291	1	W482	5	MR
WPG23	486	2	W482	10	R
WPG24	182	1	W482	5	R
WPG24	522	2	W482	10	R
CRM24	114	1	W483	40	S
CRM24	466	2	W483	40	S
WPG24	822	2	W483	55	MS-S
WPG24	179	1	W483	50	S
CRM23	246	1	W484	5	MS
CRM23	360	2	W484	5	MS
CRM24	7	1	W484	0	0
CRM24	471	2	W484	5	MS
WPG23	135	1	W484	10	MR
WPG23	551	2	W484	1	R
WPG24	153	1	W484	0	0
WPG24	710	2	W484	1	MR
CRM23	315	2	W485	40	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	69	1	W485	70	S
CRM24	803	2	W485	30	MS-S
CRM24	266	1	W485	60	S
WPG23	349	2	W485	11	MR-MS
WPG23	27	1	W485	30	MS
WPG24	226	1	W485	30	S
WPG24	651	2	W485	70	S
CRM24	106	1	W486	40	MS
CRM24	681	2	W486	50	MS-S
WPG24	424	1	W486	50	MS-S
WPG24	668	2	W486	50	S
CRM23	284	2	W487	35	MS
CRM23	168	1	W487	40	MS-S
CRM24	868	2	W487	60	MS-S
CRM24	38	1	W487	70	S
WPG23	487	2	W487	15	MR-MS
WPG23	11	1	W487	20	MS
WPG24	265	1	W487	30	MS-S
WPG24	557	2	W487	60	S
CRM23	495	2	W488	0	0
CRM23	121	1	W488	1	MR
CRM24	247	1	W488	0	0
CRM24	512	2	W488	5	R
WPG23	31	1	W488	0	0
WPG23	397	2	W488	0	0
WPG24	554	2	W488	0	0
CRM23	39	1	W489	40	MS
CRM23	372	2	W489	40	MS
CRM24	486	2	W489	20	MS-S
CRM24	272	1	W489	65	S
WPG23	111	1	W489	15	MR
WPG23	482	2	W489	6	MR-MS
WPG24	110	1	W489	15	MS-S
WPG24	688	2	W489	50	S
CRM24	432	1	W490	50	S
CRM24	620	2	W490	70	S
WPG24	165	1	W490	25	MS-S
WPG24	700	2	W490	60	S
CRM24	47	1	W492	45	S
CRM24	761	2	W492	40	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	42	1	W492	30	S
WPG24	784	2	W492	75	S
CRM24	463	2	W494	10	MR
CRM24	161	1	W494	5	R
WPG23	176	1	W494	10	R
WPG23	458	2	W494	5	R
WPG24	188	1	W494	20	MR
WPG24	752	2	W494	10	R
CRM23	470	2	W496	30	MS
CRM23	15	1	W496	60	MS-S
CRM24	377	1	W496	30	MS
CRM24	588	2	W496	40	MS-S
WPG23	346	2	W496	20	MR
WPG23	169	1	W496	25	MR-MS
WPG24	186	1	W496	40	MS-S
WPG24	555	2	W496	25	MS-S
CRM23	233	1	W497	1	MR
CRM23	393	2	W497	15	MS
CRM24	628	2	W497	0	0
CRM24	13	1	W497	5	MS
WPG23	22	1	W497	10	MR
WPG23	582	2	W497	5	R
WPG24	111	1	W497	10	MS
WPG24	873	2	W497	5	MS
CRM23	353	2	W498	40	MS
CRM23	52	1	W498	50	S
CRM24	691	2	W498	35	MS
CRM24	149	1	W498	20	MS-S
WPG23	309	1	W498	10	MR
WPG23	337	2	W498	10	MR
WPG24	568	2	W498	60	MS-S
WPG24	146	1	W498	25	S
CRM23	94	1	W499	35	MS
CRM23	287	2	W499	40	MS
CRM24	787	2	W499	30	MS
CRM24	54	1	W499	25	MS-S
WPG23	403	2	W499	35	MR-MS
WPG23	4	1	W499	70	MS-S
WPG24	440	1	W499	40	MS-S
WPG24	718	2	W499	40	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	237	1	W500	50	MR-MS
CRM23	268	2	W500	50	MS
CRM24	361	1	W500	40	MS-S
CRM24	826	2	W500	40	MS-S
WPG23	227	1	W500	15	MR
WPG23	504	2	W500	30	MR-MS
WPG24	248	1	W500	60	S
WPG24	596	2	W500	50	S
CRM23	19	1	W501	65	MS-S
CRM23	295	2	W501	45	MS-S
CRM24	347	1	W501	30	MS-S
CRM24	754	2	W501	20	MS-S
WPG23	222	1	W501	15	MR
WPG23	574	2	W501	15	MR-MS
WPG24	183	1	W501	35	MS
WPG24	670	2	W501	40	MS-S
CRM23	359	2	W502	10	MR-MS
CRM23	27	1	W502	35	MS
CRM24	655	2	W502	10	MR
CRM24	181	1	W502	15	MS-S
WPG23	161	1	W502	15	MR
WPG23	331	2	W502	10	R
WPG24	532	2	W502	20	MR-MS
WPG24	121	1	W502	25	MS
CRM23	365	2	W504	25	R
CRM23	75	1	W504	50	R-MR
CRM24	102	1	W504	20	MR
CRM24	503	2	W504	20	MS
WPG23	32	1	W504	20	R
WPG23	523	2	W504	5	R
WPG24	494	2	W504	15	MR-MS
WPG24	53	1	W504	20	R-MR
CRM23	40	1	W505	70	MR-MS
CRM23	294	2	W505	45	MS-S
CRM24	434	1	W505	40	S
CRM24	559	2	W505	30	S
WPG23	284	1	W505	10	MR
WPG23	543	2	W505	15	MS
WPG24	852	2	W505	50	MS-S
WPG24	212	1	W505	30	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	12	1	W506	20	MR
CRM23	389	2	W506	1	MS
CRM24	173	1	W506	0	0
CRM24	638	2	W506	0	0
WPG23	308	1	W506	20	R
WPG23	629	2	W506	5	R
WPG24	808	2	W506	15	MR
WPG24	346	1	W506	5	R-MR
CRM24	657	2	W507	0	0
CRM24	316	1	W507	5	R
WPG23	626	2	W507	0	0
WPG23	168	1	W507	5	R
WPG24	114	1	W507	0	0
WPG24	714	2	W507	0	0
CRM24	428	1	W508	30	MS-S
CRM24	652	2	W508	30	MS-S
WPG23	335	2	W508	6	MR-MS
WPG23	141	1	W508	10	MS
WPG24	92	1	W508	35	MS
WPG24	837	2	W508	50	MS-S
CRM23	50	1	W509	10	MR
CRM23	296	2	W509	30	MR
CRM24	619	2	W509	0	0
CRM24	306	1	W509	5	R
WPG23	224	1	W509	20	R
WPG23	622	2	W509	10	R
WPG24	60	1	W509	20	MR
CRM23	160	1	W510	10	R
CRM23	320	2	W510	30	R
CRM24	421	1	W510	5	R
CRM24	514	2	W510	5	R-MR
WPG23	61	1	W510	5	MS
WPG23	605	2	W510	5	R
WPG24	399	1	W510	0	0
WPG24	823	2	W510	10	R-MR
CRM23	398	2	W511	50	MS
CRM23	122	1	W511	40	S
CRM24	392	1	W511	40	MS
CRM24	838	2	W511	30	MS-S
WPG23	219	1	W511	35	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	646	2	W511	25	MR-MS
WPG24	514	2	W511	30	MS-S
CRM23	128	1	W513	10	R
CRM23	464	2	W513	1	R
CRM24	461	2	W513	0	0
CRM24	215	1	W513	5	R
WPG23	71	1	W513	20	MR
WPG23	514	2	W513	10	R
WPG24	205	1	W513	20	MR
CRM23	108	1	W514	1	MR
CRM23	494	2	W514	10	R
CRM24	152	1	W514	5	R
CRM24	724	2	W514	10	R
WPG23	171	1	W514	10	R
WPG23	566	2	W514	5	R
WPG24	462	2	W514	10	MR
WPG24	38	1	W514	10	R
CRM23	177	1	W515	0	0
CRM23	381	2	W515	1	MR
CRM24	811	2	W515	0	0
CRM24	58	1	W515	5	R
WPG23	305	1	W515	10	R
WPG23	498	2	W515	15	R-MR
WPG24	157	1	W515	1	R
WPG24	826	2	W515	5	R
CRM23	214	1	W516	1	MS
CRM23	400	2	W516	5	MS
CRM24	32	1	W516	10	R
CRM24	455	2	W516	10	R
WPG23	538	2	W516	0	0
WPG23	132	1	W516	10	R
WPG24	276	1	W516	5	R
WPG24	720	2	W516	10	R
CRM23	204	1	W517	5	MR
CRM23	369	2	W517	1	R
CRM24	430	1	W517	0	0
CRM24	462	2	W517	5	R
WPG23	604	2	W517	0	0
WPG23	118	1	W517	10	MR
WPG24	683	2	W517	5	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	288	1	W517	5	R-MR
CRM23	17	1	W518	20	MR
CRM23	261	2	W518	5	MR
CRM24	599	2	W518	0	0
CRM24	309	1	W518	10	R
WPG23	156	1	W518	10	R
WPG23	424	2	W518	10	R-MR
WPG24	738	2	W518	0	0
WPG24	8	1	W518	5	R
CRM23	252	1	W519	10	MS
CRM23	257	2	W519	5	MS
CRM24	250	1	W519	5	R
CRM24	672	2	W519	5	R
WPG23	60	1	W519	5	MR
WPG23	473	2	W519	5	MR
WPG24	429	1	W519	1	MR
WPG24	816	2	W519	5	R
CRM23	339	2	W520	0	0
CRM23	34	1	W520	0.5	R
CRM24	48	1	W520	0	0
CRM24	566	2	W520	0	0
WPG23	525	2	W520	0	0
WPG23	272	1	W520	5	MR
WPG24	587	2	W520	0	0
WPG24	308	1	W520	5	R-MR
CRM23	102	1	W521	10	MS
CRM23	280	2	W521	1	MS
CRM24	345	1	W521	0	0
CRM24	539	2	W521	0	0
WPG23	178	1	W521	10	R
WPG23	505	2	W521	5	R
WPG24	845	2	W521	15	MS
WPG24	341	1	W521	5	R
CRM23	200	1	W522	1	MR
CRM23	447	2	W522	5	R
CRM24	491	2	W522	0	0
CRM24	298	1	W522	5	R
WPG23	515	2	W522	0	0
WPG23	6	1	W522	5	R
WPG24	201	1	W522	10	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	620	2	W522	5	R
CRM23	336	2	W524	0	0
CRM23	249	1	W524	5	R
CRM24	59	1	W524	0	0
CRM24	537	2	W524	5	R
WPG23	585	2	W524	0	0
WPG23	9	1	W524	1	R
WPG24	785	2	W524	0	0
WPG24	204	1	W524	10	R
CRM23	354	2	W525	25	MR
CRM23	43	1	W525	10	R
CRM24	416	1	W525	10	R
CRM24	845	2	W525	10	R
WPG23	253	1	W525	10	R
WPG23	600	2	W525	5	R
WPG24	374	1	W525	20	R-MR
WPG24	734	2	W525	20	R-MR
CRM23	139	1	W527	20	MS
CRM23	366	2	W527	5	MS
CRM24	214	1	W527	0	0
CRM24	502	2	W527	1	MS
WPG23	478	2	W527	0	0
WPG23	28	1	W527	5	R
WPG24	576	2	W527	0	0
WPG24	122	1	W527	10	R
CRM23	145	1	W528	0	0
CRM23	449	2	W528	0	0
CRM24	409	1	W528	0	0
CRM24	819	2	W528	0	0
WPG23	593	2	W528	0	0
WPG23	137	1	W528	10	MS
WPG24	250	1	W528	0	0
WPG24	564	2	W528	0	0
CRM23	225	1	W529	70	MS
CRM23	442	2	W529	60	MS-S
CRM24	569	2	W529	25	MS-S
CRM24	163	1	W529	30	S
WPG23	142	1	W529	20	MR-MS
WPG23	475	2	W529	35	MS-S
WPG24	36	1	W529	15	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	716	2	W529	70	S
CRM23	254	1	W531	1	MS
CRM23	499	2	W531	1	MS
CRM24	373	1	W531	0	0
CRM24	736	2	W531	0	0
WPG23	307	1	W531	15	R
WPG23	451	2	W531	10	R
WPG24	421	1	W531	0	0
WPG24	556	2	W531	10	R
CRM23	309	2	W532	1	MS
CRM23	28	1	W532	85	MS-S
CRM24	874	2	W532	5	MR
CRM24	210	1	W532	5	MS
WPG23	78	1	W532	20	MR
WPG23	400	2	W532	10	R
WPG24	191	1	W532	5	R
WPG24	529	2	W532	10	R
CRM23	344	2	W533	1	MR
CRM23	242	1	W533	5	R
CRM24	527	2	W533	1	MS
CRM24	268	1	W533	5	R
WPG23	18	1	W533	5	R
WPG23	460	2	W533	1	R
WPG24	864	2	W533	0	0
WPG24	138	1	W533	1	MS
CRM24	414	1	W534	0	0
CRM24	785	2	W534	0	0
WPG23	323	1	W534	0	0
WPG23	548	2	W534	1	R
WPG24	621	2	W534	10	R
WPG24	120	1	W534	10	R-MR
CRM23	126	1	W535	5	MS
CRM23	363	2	W535	1	MS
CRM24	804	2	W535	0	0
CRM24	243	1	W535	5	R
WPG23	530	2	W535	0	0
WPG23	194	1	W535	1	R
WPG24	29	1	W535	10	MR
WPG24	847	2	W535	5	R
CRM23	93	1	W536	1	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	334	2	W536	5	MR-MS
CRM24	224	1	W536	0	0
CRM24	546	2	W536	5	R
WPG23	479	2	W536	5	R
WPG23	274	1	W536	10	R-MR
WPG24	415	1	W536	0	0
WPG24	649	2	W536	5	R
CRM23	131	1	W537	5	MR
CRM23	386	2	W537	5	R
CRM24	77	1	W537	0	0
CRM24	869	2	W537	0	0
WPG23	14	1	W537	10	MR
WPG23	367	2	W537	5	R
WPG24	48	1	W537	10	MS
WPG24	824	2	W537	5	R
CRM23	439	2	W538	10	MR
CRM23	6	1	W538	10	R
CRM24	276	1	W538	15	MR
CRM24	484	2	W538	10	MR
WPG23	220	1	W538	5	R
WPG23	362	2	W538	5	R
WPG24	202	1	W538	20	MR
WPG24	869	2	W538	25	MR
CRM23	208	1	W539	20	MR
CRM23	468	2	W539	5	MR-MS
CRM24	61	1	W539	0	0
CRM24	677	2	W539	0	0
WPG23	108	1	W539	0.5	R
WPG23	342	2	W539	0.5	R
WPG24	101	1	W539	0	0
WPG24	793	2	W539	5	MR
CRM24	533	2	W540	10	MS
CRM24	212	1	W540	20	R-MR
WPG23	488	2	W540	5	MR
WPG23	279	1	W540	20	MS-S
WPG24	485	2	W540	10	MR-MS
WPG24	430	1	W540	5	MS-S
CRM23	152	1	W541	40	MS
CRM23	402	2	W541	50	S
CRM24	154	1	W541	20	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	656	2	W541	30	MS
WPG23	166	1	W541	20	MR
WPG23	418	2	W541	20	MR
WPG24	115	1	W541	50	MS-S
WPG24	443	2	W541	60	MS-S
CRM23	33	1	W542	1	R
CRM23	279	2	W542	1	R
CRM24	143	1	W542	0	0
CRM24	760	2	W542	0	0
WPG23	70	1	W542	1	R
WPG23	441	2	W542	1	R
WPG24	105	1	W542	0	0
WPG24	870	2	W542	0	0
CRM23	270	2	W543	30	MR
CRM23	180	1	W543	30	MS
CRM24	19	1	W543	0	0
CRM24	693	2	W543	10	MS
WPG23	217	1	W543	5	R
WPG23	356	2	W543	5	R
WPG24	73	1	W543	20	MR
WPG24	787	2	W543	25	MR
CRM23	46	1	W544	15	R
CRM23	469	2	W544	5	R
CRM24	70	1	W544	5	MS
CRM24	643	2	W544	10	S
WPG23	611	2	W544	0	0
WPG23	82	1	W544	20	R
WPG24	484	2	W544	10	MR-MS
WPG24	353	1	W544	10	MS-S
CRM23	48	1	W545	5	MR
CRM23	493	2	W545	5	MR-MS
CRM24	295	1	W545	5	R
CRM24	630	2	W545	10	R
WPG23	276	1	W545	20	R
WPG23	334	2	W545	5	R
WPG24	10	1	W545	1	MS
WPG24	691	2	W545	1	MS
CRM24	242	1	W546	5	MS
CRM24	478	2	W546	5	MS-S
WPG24	570	2	W546	0	0

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	211	1	W546	1	S
CRM23	179	1	W547	20	R
CRM23	428	2	W547	10	R
CRM24	146	1	W547	0	0
CRM24	475	2	W547	10	R
WPG23	29	1	W547	10	MR
WPG23	634	2	W547	25	R
WPG24	259	1	W547	0	0
WPG24	466	2	W547	5	R
CRM24	379	1	W548	0	0
CRM24	827	2	W548	0	0
WPG23	1	1	W548	1	R
WPG23	608	2	W548	5	R
WPG24	331	1	W548	0	0
WPG24	676	2	W548	0	0
CRM23	221	1	W549	5	MS
CRM23	277	2	W549	1	MS
CRM24	257	1	W549	10	R
CRM24	709	2	W549	5	R
WPG23	406	2	W549	1	MS
WPG23	65	1	W549	5	R
WPG24	756	2	W549	1	MS
WPG24	425	1	W549	1	R-MR
CRM23	36	1	W550	5	MR
CRM23	424	2	W550	1	MS
CRM24	510	2	W550	0	0
CRM24	362	1	W550	10	R
WPG23	59	1	W550	10	R
WPG23	627	2	W550	5	R
WPG24	203	1	W550	1	MS
WPG24	482	2	W550	5	R
CRM23	391	2	W551	5	MS
CRM23	20	1	W551	20	MS-S
CRM24	304	1	W551	20	MR-MS
CRM24	576	2	W551	1	MS-S
WPG23	223	1	W551	1	MS
WPG23	524	2	W551	1	R
WPG24	657	2	W551	1	MS-S
WPG24	263	1	W551	10	R-MR
CRM23	42	1	W552	25	MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	498	2	W552	10	MR-MS
CRM24	165	1	W552	20	MS
CRM24	594	2	W552	10	R
WPG23	43	1	W552	30	R
WPG23	426	2	W552	15	R
WPG24	225	1	W552	10	MR-MS
WPG24	735	2	W552	10	R-MR
CRM23	13	1	W553	10	MS
CRM23	453	2	W553	10	MS-S
CRM24	665	2	W553	5	MS
CRM24	14	1	W553	5	MS-S
WPG23	20	1	W553	10	MR
WPG23	370	2	W553	5	R
WPG24	867	2	W553	5	MS-S
CRM23	159	1	W554	20	MR
CRM23	487	2	W554	5	MR
CRM24	571	2	W554	20	MR
CRM24	261	1	W554	10	R
WPG23	314	1	W554	10	MR
WPG23	357	2	W554	5	R
WPG24	433	1	W554	20	MR-MS
WPG24	597	2	W554	10	R-MR
CRM23	156	1	W555	5	MR
CRM23	370	2	W555	1	MS
CRM24	703	2	W555	0	0
CRM24	260	1	W555	5	R
WPG23	264	1	W555	15	R
WPG23	471	2	W555	10	R
WPG24	778	2	W555	10	MR-MS
WPG24	260	1	W555	5	R
CRM24	129	1	W556	0	0
CRM24	563	2	W556	0	0
WPG23	256	1	W556	5	R
WPG23	437	2	W556	1	R
WPG24	432	1	W556	0	0
WPG24	658	2	W556	0	0
CRM23	224	1	W557	20	MR
CRM23	335	2	W557	40	MS
CRM24	53	1	W557	30	MS
CRM24	649	2	W557	5	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	64	1	W557	10	MR
WPG23	339	2	W557	5	R
WPG24	547	2	W557	30	MS
WPG24	400	1	W557	5	R
CRM23	138	1	W558	50	MS
CRM23	501	2	W558	45	MS
CRM24	194	1	W558	40	S
CRM24	625	2	W558	40	S
WPG23	173	1	W558	10	MR
WPG23	388	2	W558	20	MR
WPG24	236	1	W558	30	MS-S
WPG24	838	2	W558	40	S
CRM23	24	1	W559	50	MS
CRM23	504	2	W559	30	S
CRM24	180	1	W559	20	MS
CRM24	532	2	W559	5	MS-S
WPG23	299	1	W559	25	MR
WPG23	542	2	W559	20	MR
WPG24	151	1	W559	20	MS
WPG24	660	2	W559	10	MS-S
CRM23	25	1	W560	30	R
CRM23	283	2	W560	20	R-MR
CRM24	126	1	W560	5	R
WPG23	642	2	W560	0	0
WPG23	100	1	W560	10	R
WPG24	403	1	W560	0	0
CRM24	385	1	W561	0	0
CRM24	768	2	W561	20	R
WPG23	580	2	W561	0	0
WPG23	186	1	W561	5	R
WPG24	213	1	W561	20	MR
WPG24	453	2	W561	30	MR
CRM24	187	1	W562	5	R
CRM24	449	2	W562	20	R-MR
WPG23	289	1	W562	0	0
WPG23	508	2	W562	0	0
WPG24	546	2	W562	20	MR-MS
WPG24	97	1	W562	25	R-MR
CRM24	334	1	W563	0	0
CRM24	777	2	W563	0	0

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	434	1	W563	0	0
WPG24	455	2	W563	5	R
CRM24	426	1	W566	0	0
CRM24	678	2	W566	0	0
WPG23	124	1	W566	5	R
WPG23	509	2	W566	5	R
WPG24	409	1	W566	0	0
WPG24	760	2	W566	0	0
CRM24	303	1	W567	0	0
CRM24	555	2	W567	0	0
WPG23	83	1	W567	0	0
WPG23	621	2	W567	5	R
WPG24	689	2	W567	0	0
WPG24	252	1	W567	5	R-MR
CRM23	454	2	W568	0	0
CRM23	95	1	W568	5	MR
CRM24	337	1	W568	0	0
CRM24	497	2	W568	0	0
WPG23	36	1	W568	10	R
WPG23	549	2	W568	5	R
WPG24	375	1	W568	0	0
WPG24	601	2	W568	0	0
CRM23	135	1	W570	20	MR
CRM23	332	2	W570	5	MR
CRM24	296	1	W570	10	R
CRM24	786	2	W570	20	R
WPG23	50	1	W570	10	R
WPG23	405	2	W570	10	R
WPG24	369	1	W570	10	R
WPG24	495	2	W570	20	R-MR
CRM23	173	1	W571	60	R-MR
CRM23	488	2	W571	15	R-MR
CRM24	824	2	W571	0	0
CRM24	280	1	W571	10	R
WPG23	469	2	W571	5	MR
WPG23	190	1	W571	10	R
WPG24	247	1	W571	25	MS
WPG24	662	2	W571	15	R-MR
CRM23	405	2	W572	15	R
CRM23	113	1	W572	40	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	788	2	W572	0	0
CRM24	311	1	W572	5	R
WPG23	104	1	W572	5	R
WPG23	520	2	W572	5	R
WPG24	857	2	W572	5	R
WPG24	195	1	W572	10	R-MR
CRM23	79	1	W573	10	R
CRM23	510	2	W573	10	R
CRM24	310	1	W573	0	0
CRM24	810	2	W573	0	0
WPG23	592	2	W573	10	R
WPG23	174	1	W573	15	R-MR
WPG24	404	1	W573	0	0
WPG24	552	2	W573	5	R
CRM23	210	1	W574	1	MR
CRM23	304	2	W574	5	MR
CRM24	313	1	W574	0	0
CRM24	458	2	W574	5	R
WPG23	101	1	W574	10	R
WPG23	562	2	W574	10	R
WPG24	411	1	W574	0	0
WPG24	744	2	W574	0	0
CRM24	323	1	W575	0	0
CRM24	687	2	W575	0	0
WPG23	497	2	W575	0	0
WPG23	207	1	W575	5	R
WPG24	322	1	W575	10	R
WPG24	582	2	W575	5	R
CRM23	141	1	W580	10	MS
CRM23	260	2	W580	5	MS-S
CRM24	851	2	W580	5	MR
CRM24	35	1	W580	5	MS
WPG23	474	2	W580	1	MS
WPG23	46	1	W580	25	R
WPG24	24	1	W580	1	MS
WPG24	642	2	W580	5	MS-S
CRM23	58	1	W581	10	MR
CRM23	331	2	W581	1	MR-MS
CRM24	520	2	W581	1	MR
CRM24	195	1	W581	1	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	615	2	W581	1	R
WPG23	125	1	W581	15	R-MR
WPG24	271	1	W581	15	MS
WPG24	775	2	W581	10	R
CRM23	421	2	W582	1	MS
CRM23	66	1	W582	10	R
CRM24	774	2	W582	1	MR
CRM24	378	1	W582	5	R
WPG23	85	1	W582	10	MR
WPG23	330	2	W582	5	R
WPG24	659	2	W582	0	0
WPG24	100	1	W582	5	MR
CRM24	169	1	W584	1	MS
CRM24	517	2	W584	5	R
WPG23	317	1	W584	1	R
WPG23	513	2	W584	5	R
WPG24	305	1	W584	5	R
WPG24	715	2	W584	5	R
CRM23	140	1	W585	1	MS
CRM23	434	2	W585	1	R
CRM24	186	1	W585	0	0
CRM24	805	2	W585	0	0
WPG23	292	1	W585	10	R
WPG23	423	2	W585	5	R
WPG24	286	1	W585	5	MR
WPG24	684	2	W585	5	R-MR
CRM23	489	2	W586	0	0
CRM23	220	1	W586	5	MR
CRM24	176	1	W586	0	0
CRM24	531	2	W586	0	0
WPG23	159	1	W586	0	0
WPG23	550	2	W586	0	0
WPG24	319	1	W586	0	0
WPG24	681	2	W586	0	0
CRM24	374	1	W587	20	MS-S
CRM24	645	2	W587	10	MS-S
WPG23	477	2	W587	0	0
WPG23	8	1	W587	5	MS
WPG24	348	1	W587	10	MS
WPG24	508	2	W587	10	R-MR

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	230	1	W588	0	0
CRM23	416	2	W588	10	R
CRM24	110	1	W588	0	0
CRM24	813	2	W588	0	0
WPG23	495	2	W588	0	0
WPG23	177	1	W588	5	R
WPG24	395	1	W588	0	0
WPG24	842	2	W588	0	0
CRM24	799	2	W589	30	MS-S
CRM24	208	1	W589	1	R
WPG23	189	1	W589	10	MR
WPG23	381	2	W589	15	MR
WPG24	218	1	W589	25	MS-S
WPG24	758	2	W589	10	R-MR
CRM24	164	1	W590	10	MR
WPG24	595	2	W590	15	MS-S
WPG24	242	1	W590	20	S
CRM24	229	1	W591	5	R
CRM24	876	2	W591	5	R
WPG24	160	1	W591	10	R
WPG24	488	2	W591	10	R
CRM23	150	1	W592	10	R
CRM23	429	2	W592	5	R
CRM24	319	1	W592	0	0
CRM24	506	2	W592	0	0
WPG23	493	2	W592	5	MR
WPG23	265	1	W592	20	R
WPG24	637	2	W592	0	0
WPG24	397	1	W592	10	R
CRM24	39	1	W593	20	MR
CRM24	748	2	W593	20	R
WPG23	66	1	W593	20	MR
WPG23	355	2	W593	5	MR
WPG24	350	1	W593	10	MR
WPG24	825	2	W593	20	R-MR
CRM23	217	1	W594	5	R
CRM23	508	2	W594	5	R
CRM24	128	1	W594	10	R
CRM24	877	2	W594	5	R-MR
WPG23	301	1	W594	10	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	581	2	W594	1	R
WPG24	200	1	W594	5	R
WPG24	807	2	W594	10	R-MR
CRM23	241	1	W595	20	MR
CRM23	406	2	W595	5	R
CRM24	402	1	W595	5	R
CRM24	474	2	W595	5	R
WPG23	270	1	W595	15	R
WPG23	347	2	W595	5	R
WPG24	861	2	W595	0	0
WPG24	78	1	W595	5	MR
CRM23	455	2	W596	1	MR
CRM23	55	1	W596	1	MS
CRM24	50	1	W596	0	0
CRM24	842	2	W596	0	0
WPG23	533	2	W596	0	0
WPG23	148	1	W596	20	R
WPG24	219	1	W596	0	0
WPG24	648	2	W596	0	0
CRM24	815	2	W597	0	0
CRM24	184	1	W597	1	R
WPG24	74	1	W597	10	R
WPG24	509	2	W597	5	R
CRM23	21	1	W598	1	MS
CRM23	491	2	W598	0.5	R
CRM24	404	1	W598	0	0
CRM24	712	2	W598	0	0
WPG23	559	2	W598	0	0
WPG23	157	1	W598	10	MR
WPG24	614	2	W598	0	0
WPG24	34	1	W598	10	R
CRM23	392	2	W599	0	0
CRM23	166	1	W599	1	R
CRM24	259	1	W599	0	0
CRM24	852	2	W599	0	0
WPG23	226	1	W599	0	0
WPG23	340	2	W599	0	0
WPG24	416	1	W599	0	0
WPG24	579	2	W599	0	0
CRM23	313	2	W600	5	MR-MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	4	1	W600	5	MS
CRM24	190	1	W600	0	0
CRM24	843	2	W600	1	MS
WPG23	507	2	W600	0	0
WPG23	45	1	W600	5	R
WPG24	516	2	W600	0	0
WPG24	275	1	W600	1	MS
CRM23	222	1	W601	10	MR
CRM23	417	2	W601	50	MS
CRM24	121	1	W601	0	0
CRM24	560	2	W601	0	0
WPG23	131	1	W601	20	R
WPG23	382	2	W601	5	R
WPG24	653	2	W601	5	R
WPG24	189	1	W601	1	R-MR
CRM23	14	1	W602	5	MR
CRM23	505	2	W602	5	R
CRM24	86	1	W602	0	0
CRM24	723	2	W602	5	MR
WPG23	197	1	W602	0	0
WPG23	639	2	W602	0	0
WPG24	251	1	W602	0	0
WPG24	725	2	W602	0	0
CRM23	216	1	W603	1	MS
CRM23	412	2	W603	5	MS
CRM24	365	1	W603	5	MS
CRM24	717	2	W603	5	MS
WPG23	583	2	W603	6	MR-MS
WPG23	106	1	W603	10	R
WPG24	743	2	W603	5	MR-MS
WPG24	290	1	W603	10	S
CRM23	183	1	W604	40	MR-MS
CRM23	457	2	W604	5	R
CRM24	235	1	W604	20	R
CRM24	861	2	W604	5	R
WPG23	555	2	W604	5	MS
WPG23	116	1	W604	5	R
WPG24	1	1	W604	5	MR
WPG24	524	2	W604	5	R
CRM23	85	1	W605	94	MS-S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	471	2	W605	70	MS-S
CRM24	63	1	W605	50	S
CRM24	488	2	W605	60	S
WPG23	540	2	W605	20	MR
WPG23	109	1	W605	30	MR-MS
WPG24	14	1	W605	30	S
WPG24	828	2	W605	50	S
CRM24	158	1	W606	0	0
CRM24	676	2	W606	0	0
WPG23	489	2	W606	0	0
WPG23	139	1	W606	5	R
WPG24	198	1	W606	10	R
WPG24	447	2	W606	10	R
CRM23	80	1	W607	5	MR
CRM23	380	2	W607	1	MS
CRM24	773	2	W607	5	MS-S
CRM24	155	1	W607	5	R
WPG23	140	1	W607	10	R
WPG23	401	2	W607	10	R
WPG24	58	1	W607	0	0
WPG24	879	2	W607	1	MS-S
CRM24	556	2	W608	1	MR
CRM24	286	1	W608	5	MS
WPG23	414	2	W608	6	MR-MS
WPG23	234	1	W608	10	R
WPG24	396	1	W608	0	0
WPG24	788	2	W608	20	R-MR
CRM24	321	1	W609	0	0
WPG23	165	1	W609	10	R
WPG23	404	2	W609	5	R
WPG24	740	2	W609	10	MR
WPG24	323	1	W609	20	R
CRM23	278	2	W610	10	MR
CRM23	137	1	W610	80	R-MR
CRM24	175	1	W610	0	0
CRM24	750	2	W610	10	MR
WPG23	151	1	W610	15	MR
WPG23	453	2	W610	5	R
WPG24	33	1	W610	5	MR-MS
WPG24	602	2	W610	5	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	263	1	W611	30	MR
CRM24	832	2	W611	20	R-MR
WPG23	371	2	W611	1	MR
WPG23	244	1	W611	10	R
WPG24	176	1	W611	20	MR
WPG24	841	2	W611	30	MR
CRM23	229	1	W612	1	MS
CRM23	490	2	W612	5	MS
CRM24	460	2	W612	1	MR
CRM24	289	1	W612	5	R
WPG23	192	1	W612	10	R
WPG23	338	2	W612	1	R
WPG24	178	1	W612	10	R
WPG24	732	2	W612	5	R-MR
CRM23	187	1	W613	5	MR-MS
CRM23	325	2	W613	5	MR-MS
CRM24	528	2	W613	5	MS
CRM24	130	1	W613	1	R
WPG23	494	2	W613	0	0
WPG23	312	1	W613	5	R
WPG24	193	1	W613	1	MR-MS
WPG24	483	2	W613	1	MS-S
CRM23	104	1	W614	0	0
CRM23	450	2	W614	1	MS
CRM24	127	1	W614	0	0
CRM24	715	2	W614	0	0
WPG23	268	1	W614	5	MR
WPG23	428	2	W614	10	R
WPG24	774	2	W614	0	0
WPG24	184	1	W614	10	R
CRM23	245	1	W615	30	MR
CRM23	316	2	W615	30	MR
CRM24	738	2	W615	10	MR
CRM24	398	1	W615	20	R
WPG23	399	2	W615	5	MR
WPG23	93	1	W615	5	R
WPG24	170	1	W615	10	MR
WPG24	513	2	W615	20	MR
CRM24	133	1	W616	0	0
CRM24	844	2	W616	5	MS

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	394	2	W616	0	0
WPG23	24	1	W616	1	MS
WPG24	848	2	W616	0	0
WPG24	381	1	W616	5	MS
CRM23	212	1	W617	5	MS
CRM23	452	2	W617	5	MS
CRM24	561	2	W617	0	0
CRM24	438	1	W617	5	MS
WPG23	13	1	W617	5	R
WPG23	344	2	W617	5	R
WPG24	127	1	W617	5	MR-MS
WPG24	757	2	W617	30	S
CRM23	459	2	W618	1	MR-MS
CRM23	244	1	W618	10	MS
CRM24	342	1	W618	5	MR
CRM24	769	2	W618	15	MS
WPG23	260	1	W618	20	MR
WPG23	455	2	W618	5	MR
WPG24	354	1	W618	10	R
WPG24	862	2	W618	10	R
CRM23	73	1	W619	5	MS
CRM23	476	2	W619	5	MS
CRM24	12	1	W619	5	MS
CRM24	457	2	W619	5	R
WPG23	138	1	W619	0	0
WPG23	575	2	W619	0	0
WPG24	93	1	W619	5	R
WPG24	678	2	W619	5	R
CRM23	269	2	W620	50	MR-MS
CRM23	110	1	W620	5	MS
CRM24	116	1	W620	10	MR
CRM24	812	2	W620	20	MR
WPG23	149	1	W620	5	MR-MS
WPG23	411	2	W620	5	MR-MS
WPG24	362	1	W620	25	MS
WPG24	615	2	W620	10	MS
CRM23	16	1	W621	15	MR
CRM23	286	2	W621	70	R-MR
CRM24	124	1	W621	10	R
CRM24	654	2	W621	10	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	304	1	W621	10	R
WPG23	641	2	W621	15	R
WPG24	145	1	W621	10	MR
WPG24	772	2	W621	10	R
CRM23	124	1	W622	0	0
CRM23	307	2	W622	0	0
CRM24	230	1	W622	0	0
CRM24	525	2	W622	0	0
WPG23	443	2	W622	0	0
WPG23	34	1	W622	10	MR
WPG24	4	1	W622	0	0
WPG24	805	2	W622	0	0
CRM24	348	1	W623	25	MS
CRM24	482	2	W623	35	MS
WPG23	609	2	W623	40	MR-MS
WPG23	240	1	W623	10	MS
WPG24	70	1	W623	25	MS-S
WPG24	789	2	W623	50	MS-S
CRM23	234	1	W624	5	R
CRM23	473	2	W624	5	R
CRM24	784	2	W624	0	0
CRM24	120	1	W624	5	R
WPG23	86	1	W624	0	0
WPG23	389	2	W624	5	R
WPG24	154	1	W624	1	R
WPG24	685	2	W624	5	R
CRM23	107	1	W625	1	MS
CRM23	477	2	W625	5	R
CRM24	740	2	W625	0	0
CRM24	20	1	W625	5	R
WPG23	290	1	W625	5	R
WPG23	343	2	W625	1	R
WPG24	336	1	W625	0	0
WPG24	748	2	W625	0	0
CRM23	38	1	W626	15	MR
CRM23	461	2	W626	1	MR
CRM24	353	1	W626	0	0
CRM24	865	2	W626	0	0
WPG23	206	1	W626	10	R
WPG23	432	2	W626	5	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	89	1	W626	5	R
WPG24	561	2	W626	5	R
CRM23	147	1	W627	5	MS
CRM23	465	2	W627	1	MS
CRM24	81	1	W627	0	0
CRM24	745	2	W627	5	MS-S
WPG23	273	1	W627	5	MR
WPG23	635	2	W627	5	R
WPG24	192	1	W627	5	R-MR
WPG24	607	2	W627	5	R-MR
CRM23	59	1	W628	5	MR
CRM23	479	2	W628	1	MR-MS
CRM24	320	1	W628	0	0
CRM24	631	2	W628	0	0
WPG23	229	1	W628	5	MR
WPG23	447	2	W628	10	R
WPG24	35	1	W628	0	0
WPG24	703	2	W628	0	0
CRM23	161	1	W629	10	MR
CRM23	456	2	W629	5	MS
CRM24	355	1	W629	0	0
CRM24	574	2	W629	1	MS
WPG23	73	1	W629	10	MR
WPG23	462	2	W629	5	MR
WPG24	187	1	W629	0	0
WPG24	863	2	W629	1	MS
CRM23	227	1	W630	5	MR
CRM23	326	2	W630	1	R
CRM24	219	1	W630	0	0
CRM24	833	2	W630	5	MR-MS
WPG23	3	1	W630	5	MR
WPG23	637	2	W630	5	R
WPG24	543	2	W630	0	0
WPG24	277	1	W630	1	MS
CRM23	65	1	W631	35	MR
CRM23	483	2	W631	10	R-MR
CRM24	501	2	W631	30	MR
CRM24	115	1	W631	20	R
WPG23	2	1	W631	5	R
WPG23	341	2	W631	5	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	839	2	W631	25	MR
WPG24	270	1	W631	10	R-MR
CRM23	54	1	W632	10	R
CRM23	432	2	W632	1	R
CRM24	262	1	W632	0	0
CRM24	608	2	W632	0	0
WPG23	517	2	W632	0	0
WPG23	212	1	W632	15	R
WPG24	321	1	W632	5	R
WPG24	647	2	W632	5	R
CRM23	125	1	W633	20	MR
CRM23	282	2	W633	10	MR
CRM24	364	1	W633	10	MR
CRM24	663	2	W633	20	MR
WPG23	188	1	W633	5	MR
WPG23	353	2	W633	10	R
WPG24	168	1	W633	20	MR
WPG24	633	2	W633	25	MR
CRM23	89	1	W634	10	MS
CRM23	492	2	W634	5	R
CRM24	408	1	W634	0	0
CRM24	690	2	W634	0	0
WPG23	573	2	W634	5	MR
WPG23	255	1	W634	20	R
WPG24	790	2	W634	1	MS
WPG24	337	1	W634	10	R-MR
CRM23	146	1	W635	5	MR
CRM23	509	2	W635	5	MS
CRM24	67	1	W635	1	R
CRM24	781	2	W635	5	R-MR
WPG23	288	1	W635	10	MR
WPG23	380	2	W635	1	R
WPG24	632	2	W635	0	0
WPG24	106	1	W635	5	R
CRM23	377	2	W636	1	MR
CRM23	231	1	W636	5	MS
CRM24	393	1	W636	0	0
CRM24	646	2	W636	0	0
WPG23	413	2	W636	5	MR
WPG23	182	1	W636	5	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG24	144	1	W636	5	R
WPG24	608	2	W636	5	R
CRM23	57	1	W637	5	R
CRM23	310	2	W637	15	R-MR
CRM24	354	1	W637	1	R
CRM24	611	2	W637	5	R
WPG23	293	1	W637	10	R
WPG23	398	2	W637	5	R
WPG24	358	1	W637	5	R
WPG24	860	2	W637	10	R
CRM24	265	1	W638	0	0
CRM24	732	2	W638	0	0
WPG23	205	1	W638	5	R
WPG23	368	2	W638	1	R
WPG24	412	1	W638	0	0
WPG24	799	2	W638	10	R
CRM23	22	1	W639	25	MS
CRM23	345	2	W639	1	MS
CRM24	702	2	W639	0	0
CRM24	117	1	W639	5	R
WPG23	75	1	W639	10	MR
WPG23	638	2	W639	10	R
WPG24	505	2	W639	1	MS-S
WPG24	136	1	W639	5	R
CRM23	193	1	W640	10	MR-MS
CRM23	271	2	W640	5	MS
CRM24	585	2	W640	1	MS
CRM24	64	1	W640	5	R
WPG23	527	2	W640	0	0
WPG23	213	1	W640	10	R
WPG24	406	1	W640	1	MS
WPG24	717	2	W640	1	MS-S
CRM23	100	1	W641	1	MR
CRM23	308	2	W641	1	R
CRM24	82	1	W641	0	0
CRM24	661	2	W641	0	0
WPG23	158	1	W641	5	R
WPG23	512	2	W641	10	R
WPG24	673	2	W641	0	0
WPG24	71	1	W641	5	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM24	327	1	W642	0	0
CRM24	872	2	W642	0	0
WPG23	191	1	W642	5	MR
WPG23	448	2	W642	5	R-MR
WPG24	405	1	W642	0	0
WPG24	702	2	W642	0	0
CRM24	172	1	W643	0	0
CRM24	818	2	W643	0	0
WPG23	247	1	W643	10	R
WPG23	556	2	W643	5	R
WPG24	876	2	W643	0	0
WPG24	257	1	W643	5	R
CRM23	397	2	W644	30	MR
CRM23	64	1	W644	30	R
CRM24	387	1	W644	5	R
CRM24	511	2	W644	5	R
WPG23	5	1	W644	10	MR
WPG23	396	2	W644	10	R
WPG24	531	2	W644	10	R
WPG24	147	1	W644	5	R-MR
CRM23	148	1	W645	1	MR
CRM23	330	2	W645	1	R
CRM24	423	1	W645	0	0
CRM24	807	2	W645	0	0
WPG23	313	1	W645	5	MS
WPG23	425	2	W645	10	R
WPG24	364	1	W645	0	0
WPG24	761	2	W645	0	0
CRM23	77	1	W646	50	R
CRM23	413	2	W646	10	R-MR
CRM24	793	2	W646	0	0
CRM24	366	1	W646	30	MS
WPG23	16	1	W646	20	MR
WPG23	433	2	W646	5	MR
WPG24	384	1	W646	20	MR
WPG24	497	2	W646	5	R-MR
CRM23	198	1	W647	0	0
CRM23	356	2	W647	0	0
CRM24	794	2	W647	0	0
CRM24	228	1	W647	1	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	53	1	W647	25	MR
WPG23	440	2	W647	1	R
WPG24	661	2	W647	0	0
CRM23	419	2	W648	0	0
CRM23	10	1	W648	1	R
CRM24	78	1	W648	0	0
CRM24	513	2	W648	0	0
WPG23	258	1	W648	10	MR
WPG23	526	2	W648	10	R
WPG24	298	1	W648	0	0
WPG24	639	2	W648	5	R
CRM23	438	2	W649	1	MR
CRM23	47	1	W649	5	R
CRM24	227	1	W649	0	0
CRM24	459	2	W649	1	MS
WPG23	110	1	W649	0	0
WPG23	373	2	W649	5	R
WPG24	871	2	W649	0	0
WPG24	228	1	W649	10	R
CRM23	235	1	W650	10	MR
CRM23	503	2	W650	25	MR
CRM24	634	2	W650	0	0
CRM24	66	1	W650	10	R
WPG23	444	2	W650	1	MR
WPG23	280	1	W650	10	R
WPG24	84	1	W650	10	R
WPG24	701	2	W650	20	R-MR
CRM23	81	1	W651	40	MR
CRM23	305	2	W651	35	MR
CRM24	839	2	W651	30	MR
CRM24	275	1	W651	20	R
WPG23	122	1	W651	25	MR
WPG23	416	2	W651	15	R-MR
WPG24	75	1	W651	10	MR-MS
WPG24	562	2	W651	20	R-MR
CRM23	26	1	WAHOO	40	MR
CRM23	463	2	WAHOO	40	MR
CRM24	451	2	WAHOO	10	MR
CRM24	346	1	WAHOO	50	MS
WPG23	316	1	WAHOO	10	R

Environment	Plot	Rep	Genotype	Severity	Infection Type
WPG23	439	2	WAHOO	5	R
WPG24	293	1	WAHOO	35	MS
WPG24	769	2	WAHOO	40	MS-S
CRM24	429	1	WALDE	20	MS
CRM24	743	2	WALDE	50	MS
WPG24	765	2	WALDE	35	MR-MS
WPG24	103	1	WALDE	30	MS
CRM24	123	1	WASATCH	20	MS-S
CRM24	450	2	WASATCH	45	MS-S
WPG23	114	1	WASATCH	15	MS
WPG23	577	2	WASATCH	20	MS-S
WPG24	481	2	WASATCH	50	MS-S
CRM24	314	1	WENTWORTH	0	0
CRM24	728	2	WENTWORTH	20	R
WPG24	866	2	WENTWORTH	20	MR
WPG24	343	1	WENTWORTH	1	R
CRM24	660	2	WESLEY	5	MR
CRM24	299	1	WESLEY	10	R
WPG23	102	1	WESLEY	10	R
WPG23	359	2	WESLEY	10	R
WPG24	664	2	WESLEY	5	MS
WPG24	363	1	WESLEY	50	S
CRM24	144	1	WESTMONT	30	MS
CRM24	568	2	WESTMONT	35	MS
WPG23	298	1	WESTMONT	15	MR
WPG23	465	2	WESTMONT	15	MR-MS
WPG24	25	1	WESTMONT	20	MS-S
WPG24	593	2	WESTMONT	25	MS-S
CRM23	379	2	WILLOW_CREEK	10	MR
CRM23	172	1	WILLOW_CREEK	30	MS
CRM24	111	1	WILLOW_CREEK	60	S
CRM24	591	2	WILLOW_CREEK	50	S
WPG23	275	1	WILLOW_CREEK	50	MR-MS
WPG23	619	2	WILLOW_CREEK	25	MR-MS
WPG24	148	1	WILLOW_CREEK	20	S
WPG24	802	2	WILLOW_CREEK	60	S
CRM24	499	2	WINALTA	30	MS-S
CRM24	36	1	WINALTA	30	S
WPG24	627	2	WINALTA	45	MS-S
WPG24	357	1	WINALTA	60	S

Environment	Plot	Rep	Genotype	Severity	Infection Type
CRM23	192	1	WINDSTAR	1	MR
CRM23	507	2	WINDSTAR	10	R
CRM24	731	2	WINDSTAR	10	R
WPG23	79	1	WINDSTAR	10	R
WPG23	434	2	WINDSTAR	1	R
WPG24	584	2	WINDSTAR	10	MR-MS
WPG24	142	1	WINDSTAR	5	R-MR
CRM24	93	1	WINOKA	20	R
CRM24	802	2	WINOKA	20	R
WPG23	354	2	WINOKA	10	MR
WPG23	249	1	WINOKA	20	R
WPG24	43	1	WINOKA	15	MR
WPG24	542	2	WINOKA	10	R
CRM24	331	1	YOGO	50	S
CRM24	524	2	YOGO	35	S
WPG24	50	1	YOGO	55	S
CRM24	293	1	YORKSTAR	55	S
CRM24	606	2	YORKSTAR	80	S
WPG23	127	1	YORKSTAR	25	MS
WPG23	376	2	YORKSTAR	35	MS
WPG24	779	2	YORKSTAR	75	S

A4. Seedling Assay Phenotypic Data

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
NORWIN	0	0;	2+	3	0;	4	34
AC READYMADE	3 4	4	4	4	4	43	4
CDC KESTREL	1 1-	1	1-1	11-	22+	43	34
W214	3 4	3	2	22+	11+	43	43
W238	0	2 3	22+	1-;	11+	1 1+	;
THUNDERBIRD	; 1-	-	-	-	-	-	1
BIGHORN	0	;1	34	4	;1	43	43
CDC CLAIR	0	4	3	43	43	1;	0;
CDC OSPREY	3 4	3 4	-	4	4	-	4
W279	0	0	4	1;	0;	4	4
AC TEMPEST	4	4	43	4	43	4	34
AC BELLATRIX	4 3	1 2	22+	22+	11-	34	43
W301	3 4	1 1-	22+	4	22+	-	4
W304	4 3	4	4	4	43	4	43
RUBY	4 3	3 4	-	1;	43	43	4
W317	1;	1 1-	11-	1	11-	11+	1+1
W319	1 1-	1-	11-	1	1	11+	1-;
W321	4 3	3 4	4	-	-	4	4
CDC HARRIER	;1-	3 3+	-	1-	33+	4	4
CDC FALCON	1-;	1-	11-	11-	11-	11-	2+2
RADIANT	2	4	-	43	4	4	43+
CDC RAPTOR	1 1-	1-	1-;	11-	11-	11-	2+2
W343	4 3	3 3+	34	34	-	-	-
W349	4 3	3 4	4	4	4	4	4
W350	4	3 4	4	4	4	4	4
CDC PTARMIGAN	4 3	4	43	34	43	43	4
W358	3	3 4	43	4	43	4	43
W360	4	4	4	4	4	34	43
W361	3 4	4	3	4	4	4	4
W371	3 4	4	4	4	43	43	43
W372	4	4 3	4	4	43	43	4
MCCLINTOCK	1;	1 1+	43	1;	1;	22-	33-
W378	0;	1 1+	4	11-	1;	2	4
W380	4	4	4	4	43	43	43
W383	3 4	4	4	4	4	43	43
W384	4 3	4 3	4	34	43	4	4
W385	4	4 3	22-	43	43	33+	4

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
W386	4	4	-	4	4	4	43
W388	3 4	4	4	4	4	4	4
W389	3 4	4	4	4	43	43	4
W390	4 3	4	34	4	43	4	4
W391	-	4	43	34	43	4	4
W392	4	4?	34	34	4	4	43
W393	4	4 3	4	4	4	4	4
W397	4	4	4	4	4	4	4
W398	4	4	33+-	4	4	4	4
W399	4	4 3	4	34	4	4	4
W400	4 3	4	4	4	4	4	4
W401	1 1-	2 2-	1-;	11-	22-	3+4	34
W404	3 4	3 4	22+	4	4	4	34
W405	4	4 3	34	43	-	4	34
W406	1-;	1-	11-	0	11-	1+1	2-2
W407	1-	1	1	1	1	1+1	22+
W408	1-;	1 1+	1-	11-	11+	43	22+
W409	1-;	1;	1-	1-	2	33+	34
W411	0;	1-1	1;	1-	1-1	1+1	11+
W412	4	4	-	4	4	34	34
W413	2	4 3	4	4	4	4	-
CDC BUTEO	4	1 1-	11+	22-	2	-	34
W415	3 4	4	-	4	4	0;	4
W416	4 3	2 2+	22-	3-3	22+	4	4
W417	4 3	2 1-	11+	2	11+	43	4
W418	0	1	11-	1	11+	0;	0;
W419	4	4	43	4	4	43	43
W420	3 4	1	-	22+	1+	-	-
W421	4	1 1+	11-	11+	22+	34	4
W424	1;	-	-	1	1	22-	23-
BROADVIEW	1-;	1	11-	1	1+1	11+	2-2
W426	0;	1	11-	1	1	1	11+
W427	1-;	1 1-	1;	1	1-1	11-	1+1
W428	1 1-	1-;	11-	11-	1	11+	1+1
W429	1;	1-	1-;	-	11-	11+	11-
W431	;1-	1 1-	1-;	1-	1	1	11+
W432	1-	-	-	;1	11-	11+	1-1
W433	3 4	4	-	4	4	34	4
FLOURISH	1;	1 1-	11+	1	11-	22+	2-2
W435	1 1+	1 1-	11+	1	1	11+	22+

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
W437	1;	1-;	1-;	11-	11-	11+	-
W438	1-	1	3	11-	22-	22+	1-1
W439	1 1+	1 1-	4	1	1	2	2+2
W440	3	4 3	34	4	3-3	4	-
W441	4	4	4	4	34	4	4
W442	4	4	1-	-	4	4	34
W443	1 1-	1-1	43	1	1	11-	11-
W444	3	4 3	43	34	4	34	4
W446	3 4	4	11+	34	34	34	43
W447	11+	1-1	1+1	11-	1-1	11-	11-
W448	1	1 1-	1+1	1	11-	11-	11-
W449	1-;	1-	;1-	1	11+	11+	11+
W450	-	1	34	0;	1-1	1-	11-
W451	4	2 2+	1-	34	22+	43	4
W452	1 1-	1 1-	0	11-	11-	22-	22-
W453	1	1 1+	-	1-;	43	43	-
EMERSON	4 3	3 4	34	4	4	4	4
W455	1	1 1-	43	1	11-	11+	22+
W456	1 1+	1 1-	43	1	11-	2	22+
W457	0	1+	34	1	22-	0;	1;
W458	1 1-	1 1-	33+	0	11-	11+	2+2
W461	3 4	4 3	2	2-2	33+	43	4
W462	1;	1-;	1-	0;	11-	1	11+
W463	1	1 1-	1-1	1	11-	11+	22-
W465	1-	1 1-	-	1	11-	22+	22-
W466	1;	1 1+	2+	0;	1-;	22+	2+2
W467	0;	1-;	0;	0;	1-;	11+	1+1
KELDIN	3 4	-	11+	34	4	4	4
W469	1-;	1 1-	11-	1	11-	-	1+1
W471	1	1-	-	-	-	-	2+2
W472	1-;	-	0	11-	4	0	34
W473	1-	1 1-	1	0	-	1	1-1
W475	1+ 1	2 1	33+	11+	22+	3-3	43
W476	3 4	4	34	22+	34	4	4
W477	0;	4	1	33-	34	0	0;
AAC GATEWAY	1;	1-;	1-;	1	11-	1-;	11-
W479	3 4	4	33+	4	4	4	4
W481	3 4	4	34	4	4	4	4
W482	0;	1 1+	1-	11-	22+	0;	1;
W483	3 4	4 3	4	4	4	4	4

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
W484	4	3 4	-	43	4	4	4
W485	4	4	4	4	4	43	4
W486	4	4 3	4	4	4	4	43
W487	4	4	0	4	4	4	4
W488	1+ 1	1- 1+	0	0;	1-;	11+	11+
W489	4	-	4	4	4	4	4
W490	3 4	4	22+	4	4	4	4
W492	3	4 3	-	4	4	4	4
W494	1	1 1-	-	1	11-	11+	11+
AAC ELEVATE	1;	1	0	0	1	-	11-
W496	3 4	4 3	43	4	4	4	43
W497	0	3 4	4	0;	43	1+;	4sp
W498	3 4	4	43	4	43	4	4
W499	4	4 3	34	4	4	4	43
W500	3	3 4	43	43	4	4	4
W501	4	4	43	43	4	4	43
W502	;1-	2+	11-	1-	3-3	4	43
MOATS	1 1-	1 1+	3-3	11-	1-;	2	22+
W504	1 1-	2 2+	1	11-	11-	23-	23-
W505	4	2	-	34	4	4	33-
W506	4	;1-	34	-	43	4	4
W507	1-;	0	1-;	0	1	11-	11-
W508	4	3 4	34	4	4	4	4
W509	1 1+	4	0;	11+	-	3-	-
W510	1;	1 1-	11-	0	11-	1-1	22-
W511	3 4	4 3	34	4	4	4	4
AAC WILDFIRE	4	4 3	4	4	4	4	4
W513	0	0;	0	0	1-;	11+	1+1
W514	0	1+;	-	0;	1	-	43
W515	1-;	;1-	22+	1	1-;	22+	1+1
W516	0	0	0	0;	4	4	34
W517	1;	1	2	0;	0;1-	11+	1;
W518	1 1-	1 1-	0	11-	1;	11-	11-
W519	3	3+	11+	-	4;	4	4
W520	0;	0	1-	1-	11-	1-1	22-
W521	1;	1;	2+	11-	2sp	4	34
W522	1-;	1-	0	1-	1;	1-1	1+1
SUNRISE	2	1;	34	11-	1;	2+2	2+2
W524	1-;	1	1-;	0	11-	11-	11+
W525	1	1 1-	11-	1	11+	1	11-

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
AAC GOLDRUSH	0	0	-	11+	-	-	11-
W527	4	3 4	4	4	4	4	4
W528	0	1-	0;	0	22+	1-;	1;
W529	4	4	34	34	4	4	4
AAC ICEFIELD	1 1+	1	33-	0;	1	3	43
W531	0	3+	0	0;	22+	0;	4
W532	3	-	-	-	22+	4	-
W533	0	2 2+	0	0;	1+;	4	33+
W534	0;	1-;	-	1	1-1	1-;	1-
W535	0	0;	0;	0	-	4	4
W536	0;	3 4	0	0;	34	-	43
W537	1-;	0;	1	0	11-	0	1+1
W538	1-;	1-1	1-;	1	11-	-	-
W539	1 1+	2 2+	-	11+	11-	22+	2+2
W540	3	2	-	4	4	4	4
W541	4	4 3	3	4	4	4	4
W542	0	1 1-	34	1;	11-	1;	0;
W543	1-;	1 1-	1	1	11-	1+1	1+1
W544	3	3 4	4	-	-	-	4
W545	3 4	-	33+	-	34	3	34
W546	0;	2	1-;	1-;	22+	11+	4
W547	0	1-	1	0;	1-;	1-1	11-
W548	0	1-	-	0;	-	11+	11+
W549	3	2 2+	33+	34	4	4	4
W550	3	3	43	-	-	4	4
W551	0;	1 1+	11+	2	2;	43	4
W552	1-;	2 2+	11-	11-	22+	34	43
W553	4	3	4	1-	4	4	4
W554	1-	-	1-	11-	11-	11+	11-
W555	4	3 4	-	34	4	3-3	-
W556	1	1 1-	34	1-;	11-	2+2	2+2
W557	1;	1-	1-	2	-	4sp	4
W558	4	4 3	4	34	43	43	4
W559	3 4	3 4	34	4	43	4	4
W560	1-;	1-	1-;	11-	1+-	1-1	1+1
W561	1-	1 1-	1-;	1;	22-	1	1
W562	1-	1	11-	11+	11-	1+1	1+1
W563	0;	1 1+	11-	0;	1;	22+	2+2
BROME	4	3 4	3-3	11+	4	4	4
SWAINSON	1;	1-1	11+	1;	1+-	22+	2+2

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
W566	1-	1 1+	3-3	0;	1;	11+	1+1
W567	3	4 3	43	11-	22+	4	34
W568	1-	1	3-3	0;	11-	11-	1+1
AAC NETWORK	3	1 1-	34	;1	34	-	3-3
W570	1-	1	11-	1-	11-	1-	1-1
W571	1 1-	1	11-	1-	22-	11+	1-
W572	0;	1-;	0;	0;	;	11-	1;
W573	0;	1 1-	1-;	0;	1;	1;	0;
W574	1-	1 1-	34	0;	22-	22+	2+2
W575	0;	1 1-	11+	1-1	11-	11-	2+2
W580	4	4 3	34	4	33+	4	4
W581	4	4 3	34	4	4	4	-
W582	4	4	43	3	4	34	34
AAC VORTEX	3	3 4	22+	2	3-3	4	2+2
W584	4	4 3	43	3-	3-	4	34
W585	3	4 3	43	43	34	4	4
W586	1 1-	1	3-3	1-;	11-	1+1	1+1
W587	;1-	4 3	1-	1-	34	4	1+1
W588	1	1 1-	3	11-	1;	11+	1+1
W589	3	4 3	22+	4	33+	34	4
W590	2	1	11-	1-1	-	0;	34
W591	0;	1-1	0;	0;	1;	0;	1;
W592	0;	1-	0	1-;	1-1	0	1;
W593	1-	1 1-	0;	11-	11+	1+1	11-
W594	0;	0	1-	0;	1-;	0	1+1
W595	0;	1-	1-1	0;	1;	11-	1+1
W596	1 1+	4 3	43	1;	4	4	-
W597	1	1 1-	1;	11-	11-	1-	1+1
W598	1;	1 1+	34	0	-	-	2?
W599	0	1	22+	0;	1-;	11-	11+
W600	1 1-	1-	1;	1	11-	11+	22+
W601	4	4	4	3	4	4	4
W602	4	3 4	3+4	-	34	4	4
W603	0;	1 1-	34	-	0;	43	43
W604	1	1 1-	11-	1	11+	1-	1+1
W605	3 4	4 3	4	4	4	4	4
W606	3	1	22+	0	4	4	4
W607	3	3 4	11+	2;	-	4	43
W608	4	4	34	4	34	4	43
W609	1-;	1	43	1-	1-1	11+	11+

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
W610	1-;	4 3	1-;	1-	3-3	33+	4
W611	0;	1 1-	11+	0	11+	1-	1
W612	0	-	33+	0	13	-	4
W613	4	3 4	4	1	4	4	4
W614	0	4 3	34	34	4	-	0;
W615	1;	1 1-	11-	1	1-1	1-	1+1
W616	4 3	4	43	0	4	4	4
W617	0;	1 1+	0;	0;	22-	34	4
W618	3	4	4	-	34	4	34
W619	1	4	43	4	33+	4	2+2
W620	1;	4	11-	11-	3+4	43	43
W621	1	1 1+	11-	11-	22+	22+	2+2
W622	0	1 1-	0;	0	1;	0;	0;
W623	4	2 1	4	0	3-3	4	4
W624	1-	1	11-	1-;	11-	1	2+2
W625	0;	1;	43	11-	11-	0;	0;
W626	0	1	1	01-	22+	4	34
W627	0	4	43	1-;	4	1-;	0;
W628	0	4	-	0	-	-	0
W629	4	4 3	34	0	34	4	4
W630	3	3	34	1;	4	4	3+4
W631	2	1 1+	4	11-	22+	22-	2+2
W632	0;	1-;	22+	0;	1-;	11-	11-
W633	1	1 1-	33+	0;	11+	11+	1+1
W634	0	4	3-3	1-	33+	4	4
W635	0	3 4	43	34	4	34	2+2
W636	1-	3 4	34	4	43	33-	1+1
W637	1-;	1	11-	11-	11+	2	1+1
W638	3	1	1	11-	11-	4	34
W639	4	4	22+	4	4	4	-
W640	3	4 3	4	34	4	11-	43
W641	0	1	34	0;	1-;	1	11-
W642	0;	1	1;	0;	0;	11-	1
W643	1 1-	1-	11-	1-;	1-;	1+2	11+
W644	1	1 1-	11-	0	11+	11+	11+
W645	1-;	1 1-	1	0;	11-	1-;	2+2
W646	1;	1	-	0	11+	11+	1+1
W647	1-	1-	11-	0;	11-	1+	1+1
W648	0;	1;	0	1	1;	4	-
W649	0;	4;	3	34	4	22-	4

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
W650	1;	1 1-	11+	1-	22-	22+	22+
W651	1+	1 2	22+	11-	11+	1-;	2+2
ND NOREEN	0	3	22+	1-;	3-3	4	0;
ARINALRFOR	1	1	11+	22+	33+	1;	34
JAGGER	3	4	-	22+	33+	34	3-3
JULIUS	3	3 4	2	34	4	4	43+
M808 (AAFC)	4	1 1+	11+	3-	22+	4	4
M808 (JIC)	4	1	11+	11+	3-3	43	4
ALBIDUM 11	3 4	4 3	4	4	43	4	43
ALBIDUM 12	3	4	33+	34	43	4	4
BERNSTEIN	4	4	34	3+	4	22+	43
HEREWARD	1 1-	3 4	11-	34	-	11-	43
KRASNODARSKAYA 99	0;	1-1	1-;	1-;	1-;	43	11-
MEISTER	3	4	43	-	3-3	43	4
MIKON	3 4	1	4	4	34	34	4
MIRAS	3	1 1-	11-	1-1	1-1	43	4
NARIDANA	-	-	4	-	11+	4	4
PATRAS	3 4	3 4	33+	4	43	43	33+
PERLO	3	3 4	34	4	4	4	43
POTENZIAL	2	3	-	-	3	34	4
PRIVILEG	3	2	34	1-	3-3	34	34
RIDA	3	3 4	33+	4	43	4	4
SOISSONS	0;	1-1	11-	1	11-	;1-	;
VITUS	1 2	-	33+	4	34	4	43
UM1174	1;	1 1-	1+	1	34	22+	4
UM6233	2 2+	0;	33+	11+	22+	33-	34
43I*18	0	1	0	4	11+	2+2	1+1
FHB 148	1 1+	3 4	-	4	34	22+	4
CALEDONIA	1 2	3 4	2+	4	43	4	43
HANOVER	3	3	33-	4	34	4	4
FREEDOM	1-;	0	33+	-	-	1-;	11-
22A*13	0	3	0	0;	22+	4	4
32C*17	0;	3 4	-	0	43	33+	;1
PEREGRINE	0;	3	3	0	43	4	4
AC_MORLEY	2	4 3	43	3	4	4	4
AC_SAMPSON	0;	1	22+	4	2+	4	4
GALLUS	4	3	33+	4	33+	43	4
HARVARD	3 4	3 4	22+	4	3+4	43	4
HY_301_HRW	1-;	1-1	1-;	0	11-	0;	1-
PRIESLEY	3 4	1	34	34	43	4	4

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
PRINCETON	3	1	33+	0	4	4	4
PRO_81	4	1 1+	33-	4	43	4	4
WENTWORTH	1-	1-;	1-;	1-	11-	11-	11-
ACCIPITER	1-;	1 1-	1-;	1;	11-	1+2	1+1
CDC CHASE	0;	1-;	1-;	0;	1-	11+	1+1
OAC CONSTELLATION	3 4	3 4	22+	4	43	4	4
WINALTA	3 4	1	3-	4	43	4	4
KHARKOV 22MC	4	4	34	4	43	4	4
SUNDANCE	4	4	4	4	43	4	4
NORSTAR_CN1730	4	3 4	4	4	4	4	4
ALABASKAJA	4	3-	34	43	43	4	4
ULIANOVKA	3 4	-	22+	43	33+	4	4
HARUS	3 4	3	22+	-	34	4	4
MIR. JUB. 50	3 4	1 1-	11+	11+	22-	34	4
FREDRICK	0;	1-	1-;	1	11-	11-	11-
SKJALDAR	1	1	11-	4	-	-	0
HOLME	4	3	4	4	43	-	4
WALDE	3	4	3-3	34	-	34	-
STRELINSKAJA MESTNAJA	4	4 3	4	-	4	4	4
ALICE	1-;	1	11-	0	11+	22-	2+2
ALLIANCE	3 4	2	22+	0;	1	4	4
ARAPAHOE	0	1	1	0	11-	0;	0
BIGHORN	-	0	11+	-	-	4	4
CAMELOT	0;	3	1-	0	43	11-	1
CULVER	0	1	33+	2	12-	12-	0;
ELKHORN	0	3 4	22+	4	43	1-;	0;
ERHARDT	0	0	1	0	0;	4	43
HARDING	0;	1 1+	1	0	12	0;	0;
JERRY	0	-	4	0;	4	0;	0
JUDITH	0	3	4	4	4	22-	0
MCGUIRE	1-;	3 4	11-	1	4	11+	2+2
MORGAN	0	3	33+	3-3	4	1;	0
NEELEY	4 3	1 1+	3-	2	-	4	4
NEKOTA	0	1 1+	11-	-	43	4	43
NUPLAINS	1-;	1	11-	1-	3-3	11-	2+2
NUSKY	0	2+	4	4	4	11+	0;
NUWEST	0	4	34	4	-	11+	0
OVERLAND	3	1	1	0	1	-	4
PAUL	3 4	3	4	0	4	34	4
PROWERS 99	1-;	1 1+	11-	0	22+	12-	11-

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
SEWARD	0	3 4	34	34	43	1;	0;
TIBER	4	3-3	4	4	4	4	4
WAHOO	0;	1	22+	22+	2	34	1;
WESLEY	0	4 3	11-	1;	-	1+;	0;
WILLOW CREEK	3 4	4	22+	4	43	4	4
WINDSTAR	0	1 1-	-	11-	11-	1-;	0
ABILENE	1-;	1-	1-	11-	11-	11-	1+1
ARCHER	0	1	1-;	11+	4	0	0
AUGUSTA	3 4	0 1-	22+	34	33+	4	4
BEZOSTAJA 1	3	3	34	4	4	4	4
CHEYENNE	3	4	34	34	4	4	4
COMANCHE	4 3	0	33-	22+	34	4	43
GAINES	3	4	4	4	34	34	3+
MINTURKI	3 4	4 3	43	33+	4	43	4
MIRONOVSKAJA 808	3 4	1 1-	11-	11+	2+2	4	4
NORSTAR_CITR17735	3 4	3	4	4	4	4	4
ORO	3	0	43	4	4	43	43
PLAINSMAN V	1-;	1	11-	1	2+2	11-	2+2
RENAN	4	1	1	11-	2	4	4
ROUGH RIDER	0	0	11+	1	-	4	4
VONA	1-;	1	1;	0	1+1	22+	43
WASATCH	3 4	1 1-	4	11+	43	4	43
WESTMONT	3 4	1+ 1	22-	11+	22+	43	4
WINOKA	0;	1 1-	43	4	34	11-	0;
YORKSTAR	4	3 4	22+	4	43	4	33-
VAKKA	3	3 4	43	4	43	4	43
BUSSARD	3	1	34	4	33+	4	4
YOGO	2 2+	4 3	4	4	43	34	4
AL'BIDUM-114_PI372331	2+	4 3	1	4	33+	4	43
AL'BIDUM-114_PI518782	2	3 4	2	4	43	4	4
SADABOZU	3 4	4 3	34	4	43	43	43
NANG RIM NO. 17	3 4	4 3	4	4	43	4	4
SABINA	1 1-	1 1+	1;	4	43	1;	4
SEU SEUN 12	3	1	2	2-2	-	4	43
SU KAE NO. 169	3 4	3 4	22+	34	34	34	43
F 216-68	4	4	4	4	4	4	4
KRASNODARSKAJA 33	4 3	4	33+	4	4	4	4
ODESSKAJA 51	3	4	34	4	4	43	4
DNESTROVSKAJA 25	4 3	3 4	34	2	4	4	4
I/122	2	4	1	4	4	33-	4

NAME	MCCFC	QTHSF	RHTSF	RKQSF	RTHSF	TMRTF	TPMKC
79TK070-1064	3 4	4 3	34	1	33+	3	-
MV-22-77	3	2 2+	11+	2	3-3	4	4
2504/82-51	1	1	1-;	1;	2+2	2+2	-
HONG MANG MAI	3 4	4	34	-	4	43	4
134	3	4	34	1	43	43	43
U9-26-32	1	3 4	22+	4	-	43	4
RUMELI	1	2 2+	11-	11-	22+	2+2	34
KONYA 2002	1 2	4	11-	4	4	1+1	4
BEZOSTAYA	4 3	4 3	22+	4	4	43	4
FRONTENAC	4	4	3	1;	34	4	4
BENEFIT	3 4	3 4	-	-	-	43	-
CARNAVAL	1;	1-;	1;	0;	1+1	1	11+
LEXINGTON	3	-	34	-	34	4	4
CHAMPLAIN	4 3	4 3	34	4	4	34	4
NORSTAR_OTTAWA	3 4	4 3	34	4	4	4	4
DH_LETH.14-3-23	3 4	4	1	1	4	43	43
LAI YANG QIU	3	4	3	11-	43	-	4
CAPO	1	4	3	1;	2+2	-	4
JOSEF	2	4	34	4	4	4	34
UM5116	1 1+	1-	3	0	2+2	2+2	1
FRANKENMEUTH	4	4	22+	0	34	43	4
RCATL33	1 1+	3 4	1	4	4	-	34
FREDERICK	2	4 3	3	1-1	4	4	34
CHINESE SPRING	3 4	4	3	4	4	4	34

A5. List of Genotypes used for Genomic Selection

Genotypes									
22A*13	CULVER	SWAINSON	W392	W441	W499	W542	W586	W623	WINOKA
32C*17	EMERSON	THUNDERBIRD	W393	W444	W500	W543	W587	W624	YOGO
43I*18	ERHARDT	TIBER	W397	W446	W501	W544	W588	W625	
AAC_Coldfront	FLOURISH	UM1174	W398	W448	W502	W545	W589	W626	
AAC_ELEVATE	GAINES	UM5116	W399	W449	W504	W546	W590	W627	
AAC_GATEWAY	HARDING	UM6233	W400	W451	W505	W547	W591	W628	
AAC_GOLDRUSH	JERRY	VONA	W401	W452	W506	W548	W592	W629	
AAC_ICEFIELD	JUDITH	W214	W405	W453	W508	W549	W593	W630	
AAC_NETWORK	KELDIN	W238	W406	W455	W509	W550	W594	W631	
AAC_VORTEX	MCCLINTOCK	W279	W407	W456	W510	W551	W595	W632	
AAC_WILDFIRE	MCGUIRE	W286	W408	W457	W513	W552	W596	W633	
ABILENE	MINTURKI	W297	W409	W458	W514	W553	W597	W634	
AC_BELLATRIX	MOATS	W301	W411	W460	W515	W554	W598	W635	
AC_TEMPEST	MORGAN	W304	W412	W462	W516	W555	W599	W636	
ACCIPITER	ND_NOREEN	W308	W413	W463	W517	W556	W600	W637	
ALICE	NEELEY	W317	W415	W466	W518	W557	W602	W638	
ALLIANCE	NEKOTA	W319	W416	W467	W519	W558	W603	W639	
ARAPAHOE	NORSTAR_CITR17735	W321	W417	W471	W520	W559	W604	W640	
ARCHER	NORSTAR_CN1730	W343	W418	W472	W521	W560	W605	W641	
BIGHORN_PI601204	NORSTAR_OTTAWA	W349	W419	W473	W522	W561	W606	W642	
BIGHORN_W240	NORWIN	W350	W420	W475	W524	W562	W607	W643	
BLIZZARD	NUPLAINS	W358	W421	W476	W525	W563	W608	W644	
BROADVIEW	NUSKY	W360	W424	W477	W527	W566	W609	W645	
BROME	NUWEST	W361	W426	W479	W528	W567	W610	W646	
CAMELOT	ORO	W371	W427	W481	W529	W568	W611	W647	
CDC_BUTEO	OVERLAND	W372	W428	W482	W531	W570	W612	W648	
CDC_CHASE	PAUL	W378	W429	W483	W532	W571	W613	W649	
CDC_CLAIR	PEREGRINE	W380	W431	W484	W533	W572	W614	W650	
CDC_FALCON	PLAINSMAN_V	W383	W432	W485	W534	W573	W615	W651	
CDC_HARRIER	PROWERS_99	W384	W433	W486	W535	W574	W616	WAHOO	
CDC_KESTREL	RADIANT	W385	W435	W487	W536	W575	W617	WASATCH	
CDC_OSPREY	ROUGH RIDER	W386	W436	W488	W537	W580	W618	WESLEY	
CDC_PTARMIGAN	RUBY	W388	W437	W489	W538	W581	W619	WESTMONT	
CDC_RAPTOR	SEWARD	W389	W438	W494	W539	W582	W620	WILLOW_CREEK	
CHEYENNE	SUNDANCE	W390	W439	W496	W540	W584	W621	WINALTA	
COMANCHE	SUNRISE	W391	W440	W498	W541	W585	W622	WINDSTAR	