PRE-BREEDING TO COMBINE DIVERSE DISEASE RESISTANCE AND AGROTYPE GENES IN HARD WINTER WHEAT

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MASTER OF SCIENCE

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ABSTRACT

The NDSU hard winter wheat (HWW) breeding program aims to develop improved varieties that are better equipped to cope with the environmental and disease challenges of the northern prairies. To achieve this, it is necessary to continually acquire and employ useful disease resistance and adaptation genes from other sources of HWW and hard spring wheat (HSW) germplasm. The first study pursued the transfer of the FHB resistance QTL *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* (obtained from HSW PI 277012) through continued backcrosses into the HWW cultivar, ND Noreen, utilizing previously derived cross segregates and markers. A second study used modified single seed descent (SSD) inbreeding with phenotypic and marker selection steps to combine the FHB resistance QTL, *Fhb1*, and *Qfhs.ifa-5A.1* (from HSW CM82036), with good winter hardiness, semi-dwarf plant height, and resistance to leaf, stem, and stripe rust.

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DEDICATION

I would like to dedicate this thesis to my parents: Bhoj Raj Adhikari and Rama Devi Adhikari who taught me the principles and practices of agriculture since my childhood and whose efforts brought me in the North Dakota State University, Fargo USA from a small town of Nepal.

TABLE OF CONTENTS

ABSTRACT iii

ACKNOWLEDGMENTS iv

DEDICATION v

LIST OF TABLES ix

LIST OF FIGURES xi

LIST OF APPENDIX TABLES xii

CHAPTER I. GENERAL SUMMARY OF THE STUDY 1

CHAPTER II. LITERATURE REVIEW 2

Introduction 2

Economic Importance 2

Types of Wheat 3

Winter Wheat in the United States 3

Diseases of Wheat 5

Wheat Rusts 5

Wheat Stem Rust 6

Wheat Stripe Rust 8

Wheat Leaf Rust 9

Fusarium Head Blight 12

Infection and Symptoms of Fusarium Head Blight 13

Types of Resistance 14

Mechanism of FHB Resistance 15

FHB Management Strategies 17

Sources of FHB Resistance 18

FHB Resistance QTL in Wheat Breeding 20

Major Effect FHB Resistance QTL on Chromosome 5A 23

Wheat Breeding for FHB Resistance 25

References 27

CHAPTER III. TRANSFER OF FHB RESISTANCE QTL *QFHB.RWG-5A.1* AND *QFHB.RWG-5A.2* TO HARD RED WINTER WHEAT 34

Abstract 34

Introduction 35

Material and Methods 37

Study Outline 37

Marker Analyses and Background Selection 39

Screening for Type II Resistance (Greenhouse) 41

Greenhouse FHB Trial 2 - Evaluation of Nine $F_{2:3}$ Lines That Were Established From the F_2 of Four Crosses 20M1 B_1F_1 Plants 41

Results and Discussion 42

Identification of SNP Markers Suitable for Haplotyping and Marker-Assisted Selection 42

Chromosome 5A SNP Haplotypes of the Parents and Backcross Progeny 44

Screening of the B_1F_1 for Type II Resistance in Greenhouse FHB Trial 1. 47

Marker Results Pertaining to Selections Included in Second Greenhouse FHB Trial 48

Background Selection 50

Greenhouse FHB Resistance Trial 2: Evaluation of Selected 19M13 and 20M1 Progenies 51

Conclusion 55

References 56

CHAPTER IV. PRE-BREEDING WITH KEY RESISTANCE AND AGROTYPE GENES TO IMPROVE HARD WINTER WHEAT GERMPLASM 58

Abstract 58

Introduction 58
Material and Methods 61
Plant Material and General Outline 61
Unreplicated Check Plot Yield Trial 62
Phenotypic Measurements and Selection 64
Data Analysis 65
Marker Analyses 65
Results and Discussion 66
Unreplicated Check Plot Yield Trial 66
Marker Screening 67
Conclusion 70
References 71
APPENDIX 74

LIST OF TABLES

Table		Page
1.	Summary of FHB resistance QTL/genes discussed in the study	26
2.	Parents, control genotypes, and B_1F_1 plants that were used for DNA analyses and FHB type II resistance tests	40
3.	Summary of polymorphic SNP data obtained with respect to 5AS region I within which *Qfhb.rwg-5A.1* is believed to occur and 5AL region III within which *Qfhb.rwg-5A.2* is believed to occur. The polymorphisms were seen with regard to the parents, F_1, and F_2 (CP46-C) populations that were studied are shown. The genotypes with PI 277012/GP80-derived alleles are indicated in grey	43
4.	SNP haplotypes with respect to the chromosome 5AS critical region (believed to harbor the *Qfhb.rwg.5A.1* QTL). Data are shown of the parents and B_1F_1 populations ND Noreen/F_2: 46CP-6 (112 plants) and ND Noreen/F_2: 46CP-9 (19 plants). The PI 277012-derived allele at each locus is colored orange	45
5.	SNP haplotypes with respect to the chromosome 5AL critical region that is believed to harbor *Qfhb.rwg.5A.2*. Data are shown for the parents and B_1F_1 of crosses ND Noreen/F_2: 46CP-6 (112 plants) and ND Noreen/F_2: 46CP-9 (19 plants). The PI 277012-derived allele at each locus is colored orange	46
6.	F_2-derived F_3 lines that were obtained from four selected B_1F_2 plants of cross 20M1 and parental controls that were evaluated in the second greenhouse FHB resistance trial. “R” indicates that the marker allele associated with the resistance QTL was detected whereas “r” indicates the presence of the susceptibility-associated marker allele	49
7.	Analysis of variance of the parents and nine bulked lines (Table 6) that were selected from cross 20M1 based on the presence of markers associated with *Qfhb.rwg-5A.1* and *Qfhb.rwg.5A.2*	52
8.	Average infection severities of the parents and nine introgression lines believed to carry resistance QTL from PI 277012 that was tested in the final FHB greenhouse trial	53
9.	Hard red winter wheat parents used for initiating the study	63
10.	Crosses made among eight winter wheat parents	64
11.	Agronomic and phenotypic data pertaining to nine entries that were selected in an un-replicated yield trial (field)	67
12.	Summary of the observed range and mean values of all the lines, selected lines, and checks for traits measured in the field trial at Casselton (2020)	67

13. Summary of lines selected for inbred line development 69

14. Summary of marker screening results for the selected F_5 lines 70

LIST OF FIGURES

Figure — Page

1. Current and previous crosses have been made in an attempt to transfer FHB resistance QTL *Qfhb.rwg-5A.1* (symbol R1) and *Qfhb.rwg-5A.2* (symbol R2) from HRS wheat donor lines RWG21 and GP80 to winter wheat. The F1:2 19M13 served as starting material for the present study 38

2. Distribution of Fusarium head blight infection severity in $B_1F_{1:2}$ families in the first greenhouse trial. The average infection severities of the parental controls are shown with arrows..... 48

3. Agarose gel showing Barc186 marker polymorphism among parents and 20M1 B_1F_1:2 plants. The smaller band around 100 bp is associated with the Qfhb.rwg-5A.1 resistance. The lanes containing 20M1-12-3, 20M1 12-12, 20M1-23-6 (homozygous), 20M1-28-10 (heterozygous), 20M1-58-8, 20M1 58-10, 20M1-58-18, 20M1-58-27, and 20M1-58-32, (homozygous) lines are shown 50

4. Agarose gel showing Gpw2136 marker polymorphisms of parents and 20M1 $B_1F_{1:2}$-plants. The smaller band 1 (around 180 bp) is associated with the *Qfhb.rwg-5A.2* resistance. The lanes containing 20M1-12-3 (homozygous), 20M1-12-12 (heterozygous), 20M1-23-6,20M1-28-10, 20M1-58-8, 20M1-58-10, 20M1-58-18, 20M1-58-27, and 20M1-58-32, (homozygous) are shown..... 50

5. (a) Histogram of the actual data, and (b) a normalized distribution fitted on the data and showing the percentage of background recovery (horizontal axis) for the B_1F_1 20M1 plants used in the study..... 51

6. Layout of the check-plot field trial at Casselton, 2020. There were 25 blocks (with the two outmost blocks shown here). Each block consisted of twelve 5 ft rows where two adjacent rows constituted a plot. The grey shaded plots show the positions of the check plots. Check plots were planted to the variety Ideal 63

7. Outline of the inbreeding and selection scheme that was employed. 64

LIST OF APPENDIX TABLES

Table — Page

A1. Polymorphic chromosome 5A SNP loci that were used in the study and their map locations 74

A2. Summary of polymorphic chromosome 5A SNP data obtained with respect to the parents, F_1, and F_2 (CP46-C) populations that were studied. The PI 277012-derived chromosome regions are indicated in dark grey. The light gray map regions derive from non-PI 277012 sources 76

A3. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen. Polymorphism for 21 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.1* region are shown. Alleles in orange color derive from PI 277012/GP80. Light grey regions do not derive from PI 277012 suggesting that CP46C-6 was a heterozygote having two different lengths of introgressed PI 277012 chromatin 78

A4. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen. Polymorphism for 15 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.2* region are shown. Alleles in orange color derive from PI 277012/GP80. The non-polymorphic region (grey) suggests that loci 59300 and 27941 may be incorrectly mapped. 85

A5. Percentage of ND Noreen background recovered for each chromosome and the number of polymorphic SNPs that were involved. 89

A6. Evaluation of parents and $B_1F_{1:2}$ families for FHB resistance in the first greenhouse trial 93

A7. Agronomic traits and yield data obtained in an un-replicated yield trial that was planted at Casselton in 2020 96

A8. Marker screening results for all the F_5 plants that were analyzed in the study 102

A9. Disease severity of parents and selected 20M1 progenies across replications at greenhouse 107

CHAPTER I. GENERAL SUMMARY OF THE STUDY

The NDSU hard winter wheat (HWW) breeding program aims to develop improved varieties that are better equipped to cope with the environmental and disease challenges of the northern prairies. Annually, significant production losses occur to winterkill, Fusarium head blight (FHB), wheat rust diseases, bacterial leaf streak, tan spot, and Septoria nodorum blotch. Recently, stripe rust infections have become more regular, making it necessary to breed for resistance to this devastating disease as well. Broadening the available genetic diversity of the breeding population is a significant component of the total breeding effort. To achieve this, it is necessary to continually acquire and employ useful disease resistance and adaptation genes from other sources of HWW and hard spring wheat (HSW) germplasm. Newly introgressed genes often occur singly in highly related, lower-yielding winter wheat genetic backgrounds and need to be systematically combined into more diverse and more productive combinations that will impart multi-pathogen resistance. This project had two components to it. The first study pursued the transfer of the FHB resistance QTL *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* (obtained from HSW PI 277012) through continued backcrosses into the HWW cultivar, ND Noreen, utilizing previously derived cross segregates and markers. A second study used modified single seed descent (SSD) inbreeding with phenotypic and marker selection steps to combine the FHB resistance QTL, *Fhb1,* and *Qfhs.ifa-5A.1* (from HSW CM82036), with good winter hardiness, semi-dwarf plant height, and resistance to leaf, stem, and stripe rust.

CHAPTER II. LITERATURE REVIEW

Introduction

Wheat (*Triticum spp*.) is a major crop and primary food grain in the United States (Paulsen and Shroyer, 2008). Wheat is also one of the world's leading food crops and is grown in more than 122 countries (FAOSTAT, 2020). It ranks third, after maize and rice, as the most-produced cereal in the world, contributing around 700 million metric tons annually and being grown on more than 215 million hectares of land (Golan et al., 2015). Being a staple food crop, wheat provides about 20 percent of the global human calorie intake (Peng et al., 2011). The United States accounts for seven percent of total global wheat production and wheat is the third largest crop in the United States in terms of planted area, production and gross farm income after corn and soybeans (USDA-ERS, 2020).

Economic Importance

Globally, the United States is the fourth largest producer of wheat, producing 1.884 billion bushels of winter, spring and durum wheat in 2018/19 (USDA-ERS 2020). Wheat is also a significant contributor to North Dakota's economy. The state consistently ranks first in the Unites States in durum and spring wheat production. 3.1 million hectares of wheat were grown in North Dakota in 2018 with an average yield of 3,201 kg/ha (North Dakota Wheat Commission https://www.ndwheat.com/growers/chartsandstats. The average yield of winter wheat in the United States was 2.94 tons/ha in 2018 and 3.40 tons/ha in 2019. U.S. hard winter wheat production totaled 18.3 million metric tons in 2018 and 23.1 million metric tons in 2019, accounting for more than 40% of U.S. total wheat production (Hard Red Winter Wheat Regional Quality Survey, 2019).

Types of Wheat

Wheat varieties grown in the United States have either a winter or a spring growth habit. Winter wheat is sown in the fall and harvested in the summer because it needs to be exposed to low temperatures for flowering and to produce seeds. Spring wheat and durum wheat are planted in spring and harvested in late summer or fall. Depending on grain color and hardness, US-grown wheat is classified into five major classes: hard red winter (HRW), hard red spring (HRS), soft red winter (SRW), white, and durum wheat. Each class has different end-uses. HRW and HRS are mainly used for the manufacture of products requiring high protein flour. SRW and white wheat are used to make products requiring low protein flour, such as cakes, cookies, noodles and white crusted bread, while durum wheat is mainly used in the production of pasta. These kinds of wheat are grown in different regions of the US: HRW is mainly grown in the Great Plains (northern Texas through Montana). HRS is grown in the northern plains (North Dakota, Montana, Minnesota, and South Dakota). SRW is grown in the States along the Mississippi River and in the Eastern States. White wheat (winter and spring) is grown in Washington, Oregon, Idaho, Michigan and New York. Durum wheat is cultivated in North Dakota and Montana (USDA-ERS, 2020).

Winter Wheat in the United States

There is a distinct difference between spring and winter wheat, although the vegetative characteristics of these two types of wheat are very similar. Winter wheat can withstand freezing temperatures for extended periods of time during the early vegetative stage and requires exposure to freezing or near freezing temperatures to trigger the reproductive stage. In other words, if winter wheat does not pass through a period of cold temperatures, then it does not produce seed. Two things that are needed for winter wheat to perform optimally and produce

good yields are cold acclimatization and vernalization (https://extension.sdstate.edu/what-makes-winter-wheat-winter-wheat). In order to vernalize, winter temperatures usually need to be under eight degrees Celsius for an extended period. The harsh winters of North Dakota can result in winter kill and crop losses when it gets excessively cold. Survivability can be improved by planting genetically cold tolerant cultivars and adopting appropriate cultural practices such as minimum tillage.

Winter wheat accounts for 60-80 per cent of the total United States wheat production (USDA-ERS, 2020). The main growing region for winter wheat in the United States is the Great Plains with Kansas producing by far the most winter wheat (Plains Grains Inc., 2017). In the period 2010-2017, Kansas planted an average of 3.3 million hectares of winter wheat each year and produced a total of 9.25 million metric tons. Winter wheat is far less popular in North Dakota due to the harsh winters. Annual production is highly variable and depends primarily on fall planting conditions, winter kill, spring moisture availability and flooding. From 2010 to 2017, 132 thousand hectares (average) of winter wheat have been planted annually in North Dakota to yield 427 thousand metric tons (average). More recently (2018 and 2019), hard-red winter wheat production in North Dakota amounted to only 0.08 million metric tons and 0.10 million metric tons respectively. The average yield of winter wheat in North Dakota from 2010 to 2017 was 3092.2 kg/ha, higher than that of Kansas, which had an average yield of 2890.6 kg/ha. The average yield of winter wheat in the U.S. was 2.94 tons/ha in 2018 and 3.40 tons/ha in 2019. The overall production of hard winter wheat in the U.S. was 18.3 million metric tons in 2018 and 23.1 million metric tons in 2019 which accounts for more than 40% of the total wheat production in the U.S. (Hard Red Winter Wheat Regional Quality Survey, 2019).

Diseases of Wheat

Diseases of wheat in North Dakota include leaf rust *(Puccinia triticina* Eriks.) (*Pt*), stripe rust (*Puccinia striiformis* f. sp. *tritici*) (*Pst*), stem rust (*Puccinia graminis* f. sp. *tritici*) (*Pgt*), tan spot (*Pyrenophora tritici-repentis*), Septoria nodorum blotch (*Parastagonospora nodorum*), bacterial leaf streak (*Xanthomonas translucens* pv. *undulosa*) and head blight/scab (*Fusarium graminearum*) (Boyd 2005; Kolmer; 2005 Hodson, 2011). Other recently emerged or lesser diseases, such as wheat blast (*Magnaporthe oryzae* pathotype *Triticum*) and spot blotch (*Bipolaris sorokiniana*), respectively, also threaten grain production (Figueroa et al., 2018). All these diseases hamper the ability of the crop to achieve its actual yield potential (Curtis, 2002). Cultural and chemical means of disease control are often inadequate as well as financially and environmentally costly. Resistance breeding is the most cost-effective way to protect a crop against disease and is often integrated with chemical and cultural measures to maximize protection.

Wheat Rusts

Rusts are among the most harmful diseases of cereals and have coexisted and evolved with these crops throughout their domestication (Pretorius et al., 1984). The plasticity of cereal rusts is a key feature of great importance in relation to their distribution and epidemiology. According to Law et al. (1967), it is believed that, prior to the establishment of cereal crops, rusts were present on grasses and that they became adapted to cereals as these came into being. *Pucciniales* are binding parasites that cause rust disease in many species of plants. One of the main problems with rust resistance breeding is that the pathogen's virulence genes can mutate quickly, making race-specific resistance genes of wheat useless (McIntosh, 1988). In all wheat-growing regions, rust caused periodic serious epidemics, the occurrence of which depended on

environmental conditions, pathotype virulence, cultivar susceptibility, and time of onset of disease (Roelfs et al., 1992; Boyd, 2005).

Wheat Stem Rust

Stem or black rust of wheat is a serious disease of wheat in many areas of the world (Singh et al., 2008). It is known to cause severe devastation, reducing an apparently healthy-looking wheat crop to broken stems and shriveled grains in less than one month (Singh et al., 2008). *Pgt* attacks not only wheat but also barley and some grasses, such as wild barley grass (Park, 2007). Because of its great economic importance and its widespread occurrence throughout the world, *Pgt* is the most extensively studied (Anikster and Wahl, 1979; Roelfs, 1985). *Pgt* is an obligate biotroph, which is heteroecious in alternating between telial and aecial hosts, and macrocyclic with five spore stages that differ in morphology and function (Singh et al., 2008). *Pgt* is characterised by the presence of uredinia, commonly occurring on the leaf sheaths of the wheat plant, but also on true stem tissues, leaves, spikes, glumes, and awns. On leaves, *Pgt* pustules develop mostly on the underside but may penetrate and produce limited sporulation on the upper side. Masses of urediniospores produced in the pustules are brownish-red in color and are easily dislodged from plants. Towards the end of the growing season, uredinia convert from dark brown to black telia, thus the disease is also called black rust (Singh et al, 2008). The minimum, optimum, and maximum temperatures for the germination of urediniospores are 20 degrees Celsius, 15 –24 degrees Celsius, and 30 degrees Celsius; and for sporulation 50, 30, and 40 degrees Celsius (Roelfs et al., 1992), respectively. Wheat, barley, triticale, and a few related species are the primary hosts for *Pgt*. There are many species of *Berberis* and less commonly *Mahonia* that are susceptible to *P. graminis* (Roelfs, 1985) but *B.*

vulgaris is the most important alternate host. The alternate hosts can provide a source of inoculum in the form of aeciospores (Singh et al., 2008).

To reduce the threat posed by *Pgt* in the US, inoculum sources have generally been reduced by the removal of common barberry near wheat fields (Singh et al., 2008). Kolmer (2005) reported that the eradication of barberry reduced the evolution of new pathotypes in North America and reduced the initial source of inoculum in this region. Numerous stem rust resistance (*Sr*) genes in wheat have been described and cataloged (McIntosh et al., 1995). There are 50 established (named) genes that provide resistance to stem rust and another 22 genes that do not yet have established names (USDA-ARS Cereal Disease Lab, 2017).

Sr24 offers resistance to most races of stem rust, including the virulent race Ug99 (TTKSK). Incidents of virulence on this major resistance gene have been reported in South Africa (Mago et al., 2005) and India (Bhardwaj, 1990). *Sr24* is not effective against a more recent variant of UG99, designated TTKST. *Sr24* resides on the 3DL chromosome of the rust-resistant hexaploid wheat, Agent (Smith et al., 1968). *Sr24* is also completely associated with *Lr24* (McIntosh 1976).

Stem rust resistance gene *Sr39* provides resistance to all currently known pathotypes of *Pgt* including Ug99 (TTKSK) and its variants TTKST and TTTSK, which are virulent on *Sr24* and *Sr36* respectively, two frequently deployed resistance genes. Sr*39* was transferred to the hexaploid wheat cultivar “Marquis” from *T. speltoides* (Kerber and Dyke, 1990). The gene is located on a translocated segment of *T. speltoides* chromosome 2S to wheat chromosome 2B. The translocated segment carries the seedling stem rust resistance gene *Sr39* and an adult-plant hypersensitive leaf rust resistance gene *Lr35*.

Wheat Stripe Rust

Yellow or stripe rust is another destructive wheat disease in many parts of the world, particularly North Africa, and central and West Asia. It has caused recurrent, severe crop losses since the dawn of agriculture (ICARDA, 2011).

Pst infects green tissues of cereal crops and grasses from early growth stages to maturity of the plant (Chen, 2005). On susceptible adult plants, the fungus develops tiny, yellow to orange-colored pustules in long, narrow stripes on leaves and leaf sheaths, and also infects glumes and awns (Line, 2002). The interaction between the environment, host, and the pathogen itself dictates urediniospores germination. The percentage of urediniospores that germinate decreases with increasing exposure to sunshine. For the survival and development of *Pst* on wheat, optimum temperatures and adequate precipitation are important. Rapilly (1979) also reported that continuous humidity (dew formation) on plant surfaces was required for at least three hours. Uredinia strips or necrosis formed by plants following the elongation of stem and occurrence of different levels of chlorosis and necrosis. The severity of disease development depends on the host plant resistance, humidity, and temperature (Chen, 2005), and plant resistance symptoms may include reduced sporulation, chlorosis, and necrosis. Jin et al. (2010) and Zhao et al. (2013) reported that sexual recombination is of primary importance to *Pst* variability, especially in areas where wheat and susceptible barberry (*Berberis* spp) coexist. Therefore, it is important to eradicate volunteer wheat plants and barberries as part of the overall attempt to control stripe rust. Yield lost to stripe rust infection can range from 10% in high-resistance lines to 70% in low-resistance lines. It has been estimated that 5.47 million tons of wheat are lost to stripe rust infection each year, resulting in an economic loss of $979 million (Beddow, 2015). Fungicide use is an important control strategy in some areas (Hodson, 2011). In

Western Europe, chemicals are the main strategy for stripe rust control, although efforts are underway to reduce dependence on these compounds. Fungicides may be an important control option when severe rust epidemics occur, but their use results in significantly higher production costs for farmers (Hodson, 2011).

Yr17 is classified as a seedling (all-stage) resistance gene conferring a low infection type. Seedlings with *Yr17* frequently had intermediate to high infection types when inoculated with isolates that caused little or no disease on adult plants of the same wheat lines. A long chromosomal fragment that contains three rust-resistance genes was translocated from the short arm of *T. ventricosum* 2N to bread wheat chromosome 2AS (Bariana et al., 1993). This segment includes three disease resistance genes: *Lr37, Yr17,* and *Sr38* conferring resistance to leaf rust, stripe rust, and stem rust respectively. The resistance gene *Yr17* is used by many breeding programs to develop resistant cultivars.

Wheat Leaf Rust

Leaf rust is the most common and widely distributed rust disease of wheat. The fungus is heteroecious, and therefore requires a telial/uredinial host (usually wheat) and an alternate (pycnial/aecial) host (*Thalictrum speciosissimum* or *Isopyrum fumaroides*) to complete the entire life cycle (Bolton et al., 2008).

Leaf rust occurs more regularly and is more widely distributed than *Pgt* or *Pst*. The damage caused by leaf rust depends on the plant growth stage at the time of infection. The pathogen primarily attacks the leaf blades, although it can also infect the leaf sheath and glumes in highly susceptible cultivars. If infection occurs early, it can cause extensive yield losses; 60–70% infection on the flag leaf at spike emergence may cause grain yield losses of more than 30%, whereas the same infection level at the soft dough stage may result in as little as 7% loss.

Yield loss can be severe (more than 50%) if infection occurs at an earlier stage (Huerta-Espino et al., 2011). The direct damage due to leaf rust tends to be less than that caused by stem rust or stripe rust, however, due to its more frequent and widespread occurrence, it is believed to cause higher overall global losses (Huerta-Espino et al., 2011). Like all rust diseases, damage due to leaf rust becomes more severe when large areas are sown to a single variety or closely related cultivars (Samborski, 1985). Yield losses in winter wheat can be severe (Samborski, 1985). According to Roelfs (1978) epidemics have occurred more frequently in winter wheat in some parts of the USA; for example, losses were estimated at more than three million tons (monetary value of over $350 million) from 2000 to 2004 (Huerta-Espino et al., 2011).

Weather conditions, particularly temperature and moisture, determine the survival of the uredinia stage of *Pt* between seasons and wheat crops (Eversmeyer and Kramer, 1994). Infection can occur at around 20°C with dew periods of three hours or less, but longer dew periods are required for more infections (Roelfs et al., 1992). *Pt* uses voluntary, susceptible wheat plants and alternate hosts as a green bridge for survival between crop cycles. *Triticum aestivum* is the primary host of *Pt*; it generally poses a lesser threat to *T. turgidum* L. except in the Mediterranean, Middle East, Ethiopia, and India, where durum wheat is more widely cultivated. *Puccinia triticina* f. sp. *secalis* which is a pathogen of rye does not attack wheat (Roelfs et al., 1992). *Pt* needs to live with a host from season to season. Several species of *Thalictrum*, *Anchusas, Clematis*, and *Isopyrum* can serve as alternate hosts creating opportunity for sexual breeding in *Pt* (Roelfs et al., 1992). This phase is important as it allows the fungus to recombine virulence, avirulence, and other genetic characteristics (Roelfs et al. 1992) and evolve into more virulent pathotypes. The eradication of the alternative hosts, therefore, helps to reduce the threat posed by *Pt*.

The employment of host resistance genes is the most effective and useful approach to protect wheat against *Pt.* Many cultivars that are resistant to prevailing pathotypes have been developed in different production areas. Seventy-seven leaf rust resistance genes have been discovered and named (USDA-ARS Cereal Disease Lab, 2017). These cataloged genes are of diverse origin: while many have been discovered in common wheat, others have been introgressed from the direct ancestors and more distant wild relatives of common wheat.

Lr46 is a slow rusting gene. This gene does not invoke a race-specific, hypersensitive response but instead provides partial or minor gene resistance to infection. It can delay the infection process in adult plants and thereby limit the development of symptoms by a broad range of leaf rust races. The gene was first discovered on chromosome 1B in the cultivar "Pavon 76." (Singh et al., 1998). When compared to controls without the gene, the latency time of infected mature plants was considerably shorter in plants with *Lr46*. *Lr46* confers resistance in the same way that *Lr34* does, although with a smaller effect (Martinez et al., 2001).

Dyck et al. (1966) discovered *Lr34*, a pleiotropic, broad-spectrum, quantitative, slow-rusting, and race-insensitive leaf rust resistance gene, in Canada (Kolmer et al. 2008; Lagudah et al. 2009; Singh et al.2011; Risk et al.2013) *Lr34* confers a moderate level of resistance, which is most visible on adult plant flag leaves during grain filling (Krattinger et al., 2009). *Lr34* is found on the short arm of wheat chromosome 7D and is related to the *Yr18* adult plant stripe rust resistance gene and the *Pm38* powdery mildew resistance gene. It is also linked to *Ltn1*, which causes leaf tip necrosis (LTN) on the flag leaf at the adult stage.

Lr68 is another adult plant resistance (APR) gene that confers slow rusting resistance to wheat leaf rust. The gene, formerly known as *LrP*, was discovered in CIMMYT's spring wheat "Parula." The gene is located on Parula's chromosome 7BL. *Lr68* is most likely derived from the

Brazilian wheat cultivar "Frontana," which also appears in the pedigree of ‘Parula’ and other CIMMYT wheat (Herrera-Foessel et al., 2012).

Lr56 is a hypersensitive response leaf rust resistance gene introgressed into hexaploid wheat from *Aegilops sharonensis*. The gene provides protection in the seedling stage. This gene occurs in wheat chromosome 6A (Marais et al., 2006).

Fusarium Head Blight

Fusarium head blight (FHB), also commonly known as scab, is a devastating fungal disease of wheat and barley around the world, causing significant economic losses in many countries including the United States (Nganje, 2004), Canada (Gilbert and Tekauz, 2000), China (Bai and Shaner, 2004) and the Netherlands (Snijders, 1990). FHB not only reduces wheat yields but also causes economic losses due to mycotoxin accumulation in the grain. In the United States, total direct and secondary economic losses due to FHB in wheat and barley were estimated at $7.7 billion between 1993 and 2001 (Nganje et al. 2004). Losses were particularly serious for North Dakota, which suffered close to 45 percent of the total US losses during this period. FHB is of great concern to wheat producers because of its yield-reducing capability as well as the threat posed by mycotoxin contamination (mostly deoxynivalenol or DON) of food and feed grain (McMullen et al., 1997; Dweba et al., 2017; ElDoliefy et al., 2020 Ghimire et al., 2021). Smith (1884) first described the disease symptoms and morphological characteristics of *Fusisporium culmorum* (now *Fusarium culmorum*) causing FHB. Epidemics were first reported in the United Kingdom in 1884 (Hao et al., 2020). FHB was first reported in the states of Indiana, Delaware, and Ohio in the 1890s (Chester, 1890; Arthur, 1891).

Infection and Symptoms of Fusarium Head Blight

Many *Fusarium* species cause FHB, but the fungal pathogen *Fusarium graminearum* Schwabe [teleomorph *Gibberella zeae* (Schweinitz) Petch] is the world's leading causal pathogen (Bai and Shaner, 1994). FHB occurs all over the world, particularly in wheat-growing areas with high humidity, heavy rainfall and adequate moisture during the critical disease development period. Infection is favored by high relative humidity (>90 percent) and moderately warm temperatures between 15 and 30 degree Celsius. Infected plant debris is important for the overwintering of the fungus (Atanasoff, 1920; McMullen et al., 1997) and provides a source of spores for infection of a new crop. Wheat and barley are susceptible to head infection beginning with flowering and extending through the soft dough stage of kernel development. Spores land on exposed anthers and grow into the developing kernels, glumes, and other parts of the head causing damage to the seeds. Infected spikelets usually show premature bleaching and discoloration (Schmale III and Bergstrom, 2003). The disease results in the accumulation of light pink spores on the rachis and glumes of the individual spikes. Later in the season, blue-black spherical bodies may appear on the surface of the affected spikelets. As symptoms progress, the fungus colonizes the developing grain, which causes it to shrink from the inside (McMullen et al., 1997; Schmale III and Bergstrom, 2003). FHB infection will eventually reduce yield, deteriorate quality, cause grain shriveling, and mycotoxin contamination.

Deoxynivalenol (DON) is the most common mycotoxin that is produced and poses a serious threat to animal and human health. The allowable level of DON is below 1 ppm for finished, human consumed wheat products such as flour and for all the animal feed it is 5 to 10 ppm (FDA, 2010). Type B trichothecenes (such as DON) are fungal virulence factors that are acutely phytotoxic and have been positively associated with FHB disease severity (O'Donnell et

al., 2000; Lemmens et al., 2005; Buerstmayr et al., 2019). Cowger and Arellano (2010) found that due to late infection and rainfall immediately after anthesis, an asymptomatic wheat field with a lower incidence of visually infected grain may also produce higher DON content seed.

Types of Resistance

Schroeder and Christensen (1963) reported two types of resistance to FHB in wheat: resistance to the initial infection (type I resistance) and resistance to disease spread in the heads (type II resistance). Different inoculation and screening approaches are used to measure the two types of FHB resistance. In the type I resistance studies, both sides of each selected spike (50% of florets shedding pollen) are spray-inoculated with a suspension containing 12,000-14,000 conidial spores per ml of dH_2O, either early in the morning or in the evening. The percentage of diseased spikes (FHB incidence) is determined 14 days after spray inoculation. Time of inoculation, time of assessment, and inoculum concentration are critical for the identification of type I resistance to FHB (Bai and Shaner, 2004). If the inoculation is carried out before the anthers are extruded from the florets, susceptible cultivars can easily escape the disease and appear to have type I resistance (Bai and Shaner, 2004). Also, if the evaluation time is too late, or the inoculum concentration is too high, then different levels of type I resistance are difficult to distinguish between different cultivars and may be confused with type II resistance (Bai and Shaner, 2004).

Point inoculation is used to quantify resistance to the spread of the disease (type II). A *Fusarium graminearum* suspension consisting of approximately 50,000 conidial spores per 1ml dH_2O is injected into a single floret. The site is the third or fourth spikelet from the tip when the spike is in 50% anthesis (50% of the flowers in the spike are shedding pollen). Type II resistance is measured as the percentage of diseased spikelets at 20-22 days after point

inoculation, depending on weather conditions. Type II resistance assessment is not as ambiguous and difficult to quantify as type I resistance, and type II resistance has been widely identified in wheat cultivars around the world.

Mesterhazy (1995) proposed that there are five types of active resistance. In addition to the conventional type I and type II FHB resistance, three additional types of resistance were postulated. Resistance to DON accumulation is referred to as resistance of type III. Miller et al. (1985) have shown that resistant wheat cultivars have relatively less DON than susceptible wheat cultivars under the same pathogen pressure and environmental conditions. There are two main reasons for the low concentration of DON in resistant cultivars: factors that inhibit DON accumulation and factors that promote DON degradation. Either or both may be active in resistant cultivars. Miller and Arnison (1986) have shown that the FHB resistant cultivar "Frontana" degraded more DON than the susceptible cultivar "Casavant". Tolerance, defined as type IV resistance by Mesterhazy (1995), is perceived as the absence of significant differences in yield between susceptible and tolerant plants when both exhibit the primary FHB symptoms. Resistance to kernel infection, measured as the percentage of infected kernels and referred to as type V resistance, is difficult to measure because resistance types I and II also reduce the level of kernel infection, thus confounding the actual measurement of type V resistance (Shaner, 2002; Bai and Shaner, 2004).

Mechanism of FHB Resistance

Because of their complexity and diversity, the mechanisms of FHB resistance are still unknown (Xiao et al., 2013). In general, morphological and physiological resistance mechanisms can be distinguished (Gilsinger et al., 2005).

Morphological tolerance, also known as avoidance, helps cultivars to avoid infection by the fungus, resulting in a low incidence of disease. Morphological resistance results from cultivar characteristics such as height, awnedness, and flower opening time during anthesis. Hiltona et al. (1999) studied the negative relationship between the resistance of cultivars and the height of the tiller, and two-year field results showed that taller winter wheat had less FHB symptom severity because the heads of taller cultivars are further away from the crop debris that serves as the inoculum source. Mesterhazy (1995) found that cultivars with awns were more susceptible to FHB than awn-less cultivars under natural field infection. Gilsinger (2005) demonstrated that shorter flowering time reduced the risk of FHB infection and identified four SSR markers associated with this trait.

Physiological resistance is usually based on biochemical pathways that can inhibit pathogen infection (Gilsinger et al., 2005). In order to better understand the resistance factors associated with FHB, it is necessary to study the role of defense response genes, also known as the pathogenesis-related (PR) genes (Pritsch et al. 2001). Defense response genes encode proteins such as PR-1 (unknown); PR-1, 3-glucanase; PR-3 (chitinase); PR-4 (acid chitinase) and PR-5 (thaumatin-like protein) (Linthorst and Loon, 1991). Pritsch et al. (2000) studied transcripts of defense genes expressed during infection with *F. graminearum* and found that except for PR-4 and PR-5, other defense response genes accumulated in both resilient and susceptible cultivars. Based on the induction timing of these defense response genes it was concluded that they may be associated with *F. graminearum* infection (Pritsch et al., 2000). Further observations have shown that direct contact with pathogens is not necessary for the induction of defense response genes in both resistant and susceptible plants (Pritsch et al. 2001). The Li and Yen (2008) study concluded that resistance to FHB in wheat is not associated with any PR gene based on their

observations and previous research. However, according to Xiao et al. (2013), PR 5 and PR 14 were critical to *Fhb1*-controlled FHB resistance. Earlier and increased accumulation of PR 5 transcripts were also observed in the Pritsch et al. (2000) study.

It has also been found that FHB resistance is associated with defense signaling pathways, including the jasmonic (JA), ethylene (ET), salicylic acid (SA), calcium ions, phosphatic acid (PA), and reactive oxygen species pathways (ROS) (Ding et al., 2011; Gottwald et al., 2012; and Xiao et al., 2013). Although conflicting outcomes were obtained in these studies, JA signaling has been investigated in almost all these studies. The JA pathway is normally involved in plant protection against necrotrophic pathogens, whereas the ET pathway regulates plant defense against biotrophic pathogens (Gottwald et al., 2012). *F. graminearum* is, however, considered a hemi-biotrophic pathogen with a short biotrophic phase before the necrotrophic phase (Jansen et al., 2005).

FHB Management Strategies

FHB management strategies include a variety of approaches, such as chemical, cultural, biological control, and the use of resistant cultivars (Pirgozliev et al., 2002). Singly, none of these strategies is effective enough, however, combinations of these strategies provide for reliable control (Gilbert et al., 2013).

Chemical control of FHB has been extensively studied. Paul et al. (2008) investigated the efficacy of triazole-based fungicides for control of FHB and DON accumulation in wheat and identified the efficacy of fungicides containing prothioconazole, metconazole, and tebuconazole + prothioconzaole for control of FHB and DON. Tebuconazole + prothioconazole was the most effective fungicide combination for reducing the FHB index, while metconazole was the most effective fungicide for reducing DON (Paul et al., 2008). Another group of fungicides,

strobilurins, has also been shown to control FHB disease. Azoxystrobin significantly reduced the accumulation of FHB and DON, while it was much less effective than metconazole in the Pirgozliev experiment (2002). Fungicide cannot stop the growth of the FHB fungus once it has penetrated the structure of the plant. Since FHB is a floral infective disease, the plants are most vulnerable during anthesis (Yoshida et al., 2012). The success of fungicide application, therefore, hinges on precise timing, dosage and methods of application (Ackermann et al., 2013).

Field management strategies, such as soil tillage and crop rotation have a significant impact on the level of FHB infection and crop quality. Soil tillage may affect the location and amount of previous crop residues, such as wheat straw and corn stalks, which is a natural material for the pathogen to colonize for overwintering. DillMacky and Jones (2000) recorded the highest incidence and severity of FHB if wheat followed corn in a crop rotation and the lowest if wheat followed soybean. If reduced tillage utilized a previously infected wheat or cornfield, there will be a significant increase in severity of disease and accumulation of DON in wheat (Koch et al., 2005; Pereyra and DillMacky, 2008). However, Koch et al. (2005) indicated that compared to resistance in cultivars and crop rotation, soil tillage is less important in FHB control. A good field management strategy combined with the use of resistant wheat cultivars and fungicide application provides a potent and good strategy to reduce DON contamination under high disease risk conditions (Koch et al., 2005).

Sources of FHB Resistance

The use of resistant cultivars is more effective and economic in the control of FHB and mycotoxin accumulation compared to the other management strategies (Gilbert et al., 2000). The germplasm currently used for resistance breeding can be divided into three groups based on regions of origin and wheat types (Gilbert et al., 2000; Bai et al., 2004). The first group includes

spring wheat from Asia, including the Chinese cultivar ‘Sumai 3’and its derivatives 'Ning', and 'Wangshuibai' (Lin et al., 2004; Lin et al., 2006), plus the Japanese cultivars 'Nobeoka Bozu' (Mesterhazy, 1995), 'Shinchunaga' (Bai et al., 2001) and 'Nyu Bai' (Liu et al., 2003). All these materials, especially Sumai 3 and its Ning derivatives, have been widely used in wheat breeding programs. The second group includes Brasilian spring wheat cultivars 'Frontana' (Steiner et al., 2004) and 'Encruzilhada' (Bai et al., 2004). The third group consists of the winter wheat cultivars 'Praag8' and 'Novokrumka' (Snijders 1990). In addition to these three categories, US cultivars, such as ‘Ernie' (McKendry et al. 1995), 'Truman' (McKendry et al., 2005), and 'Goldfield' (Gilsinger et al., 2005), are moderately resistant to FHB and have also been used in some U.S. breeding programs.

Since the sources of resistance to FHB are limited, alien chromosome introgressions are another good option to increase resistance levels and broaden the genetic base (Cai et al., 2005; Zeng et al., 2013). Alien chromosome segments with resistance genes, but without apparent deleterious linkage drag, can be transferred to adapted wheat via translocations (Cai et al., 2005). FHB resistance sources have been reported in many grass species including species of the genus *Thinopyrum* such as *elongatum, ponticum, intermedium,* and *distichum* (Cai et al., 2008). Alien gene transfer attempts typically involve cytogenetic approaches collectively known as chromosome engineering to identify the target alien chromosome, develop addition, substitution, translocation, or recombinant lines by crossing and backcrossing with adapted common wheat (Bai et al., 2018). A large-effect FHB resistance locus has been discovered and transferred from *Thinopyrum elongatum* (Guo et al., 2015). This gene was named *Fhb7,* occurs on the long arm of *Thinopyrum elongatum* chromosome 7E, confers type II resistance, reduces FHB severity of spike infection by up to 85%, and also substantially decreases mycotoxin in blighted kernels

(Kuzmanović et al., 2019). The genes *Fhb3* and *Fhb6* were respectively transferred to common wheat from *Leymus racemosus* (Qi et al., 2008) and *Elymus tsukushiensis* (Cainong et al., 2015). *Thinopyrum distichum* has also been reported to show strong resistance to FHB (Chen et al., 2001).

FHB Resistance QTL in Wheat Breeding

Quantitative trait locus (QTL) mapping has been extensively used in FHB research. Waldron et al., (1999) mapped a significant QTL for FHB resistance, *Fhb1* on 3BS, from the 'Sumai3' and 'Stoa' 14 populations. The latter result was later confirmed by several studies (Anderson et al., 2001; Zhou et al., 2002), and *Fhb1* was fine mapped in two populations within a 1.27 cM interval and 6.05 cM interval (Cuthbert et al., 2006). This QTL was also found in several other resistance sources from China, such as 'Ning7840' (Bai et al., 1999; Zhou et al., 2002), 'Huapei57-2' (Bourdoncle et al., 2003), and Ning894037 (Shen et al., 2003), and is currently considered to provide the strongest type II FHB resistance (Bai et al., 2004).

FHB resistance is a quantitative trait controlled by many QTL, each of which provides only partial resistance and shows strong environmental interaction (Bai and Shaner, 2004). In different studies from North America, South America, Asia, and Europe, nearly 500 QTL associated with FHB resistance types I - IV have been mapped on all 21 chromosomes of hexaploid wheat (Buerstmayr et al., 2019). Despite being detected in individual studies, only a few of these QTL are stable across several studies and could be successfully employed in breeding programs (Buerstmayr et al., 2009). This inconsistency can be explained in part by the nature of the plant material used, such as the genetic background, the magnitude of the difference in resistance between the parents and the sources of resistance used; the pathogen species used in the study, such as *F. graminearum* or *F. culmorum*; the type of resistance being evaluated, such

as type I or type II; and the variation in techniques used (Kolb et al., 2001). Seven major QTL have been formally assigned gene symbols, namely *Fhb1, Fhb2, Fhb3, Fhb4, Fhb5, Fhb6, and Fhb7,* where QTL *Fhb3, Fhb6, and Fhb7* are derived from alien species (Bai et al., 2018).

Fhb1, derived from 'Sumai3' is the most stable of all the type II resistance QTL and was mapped to the short arm of chromosome 3B (Cuthbert et al., 2006; Su et al., 2019; Waldron et al., 1999). Formerly referred to as *Qfhs.ndsu-3BS, Fhb1* was mapped as a single Mendelian locus between two STS markers spanning a genetic distance of 1.2 cM (Liu et al., 2006). The critical region was further reduced to 0.08 cM between STS markers STS3B-355 and STS3B-334 (Liu et al., 2008). Due to its large effect and stability across a wide range of genetic backgrounds and environments, *Fhb1* has by far been the most widely used QTL for resistance type II in most wheat breeding programs and has been targeted for fine mapping and map-based cloning of underlying genes (Rawat et al., 2016; Schweiger et al., 2016). A candidate gene in the QTL region, PFT, was identified and thought to be *Fhb1* (Rawat et al., 2016). However, further studies to characterize PFT in 348 wheat accessions only partially confirmed its role in FHB resistance, since the same gene also existed in susceptible wheat accessions (He et al., 2018). Two recent studies that attempted map-based cloning of *Fhb1* have identified a histidine-rich calcium-binding gene called 'His' (syn: TaHRC) that is thought to confer FHB resistance (Li et al., 2019; Su et al., 2019). Other QTL have also been identified in Chinese landraces. In addition to the major QTL on 3BS found among recombinant inbred lines (RILs) of the cross, Ning7840/Clark; Zhou et al (2002) detected two further small QTL on chromosomes 2BL and 2AS. QTL was also reported on chromosomes 5AS (Buerstmayr et al., 2002; Somers et al., 2003) and 6BS (Anderson et al., 2001; Yang et al., 2003) and were repeatedly identified in

several studies (Cuthbert et al., 2007). The QTL on chromosome 6BS was later named *Fhb2* and is flanked by two SSR markers, *gwm133* and *gwm644* (Cuthbert et al., 2007).

FHB resistance genes, *Fhb2, Fhb4,* and *Fhb5* have been fine-mapped, and closely linked markers have been identified (Cuthbert et al., 2007; Liu et al. 2010; Steiner et al., 2019b; Xue et al., 2010; Xue et al., 2011). *Fhb2,* which primarily confers type II resistance, is found on chromosome 6B short arm and has been verified in multiple studies (Bai et al., 2018; Buerstmayr et al., 2009; Cuthbert et al., 2007). *Fhb4* and *Fhb5,* which are found on chromosomes 4B and 5A, respectively, confer type I resistance to FHB and were discovered in 'Wangshuibai' (Lin et al., 2006; Xue et al., 2010; Xue et al., 2011*). Fhb2,* as well as *Fhb4* and *Fhb5,* which were discovered in Asian spring wheat, have also been found in resistance sources from other geographic origins and have been shown to provide varying levels of resistance to FHB (Bai et al., 2018; Buerstmayr et al., 2009). Depending on the resistance sources used, the latter three genes have been linked to type I or type II resistance (Bai et al., 2018).

Resistance genes *Fhb3, Fhb6,* and *Fhb7,* are derived from wild wheat relatives. The type II resistance gene *Fhb3* was discovered in the tetraploid species *Leymus racemosus* and successfully transferred to chromosome 7A of hexaploid wheat using cytogenetic manipulation techniques (Qi et al., 2008). *Fhb6* was transferred to chromosome 1A (using homoeologous recombination) from the distant wild relative, *Elymus tsukushiensis,* and it also confers type II resistance (Cainong et al., 2015). *Fhb7* was derived from wheatgrass *Thinopyrum ponticum* chromosome 7el2 and confers FHB type II resistance (Shen et al. 2004; Shen and Ohm, 2007). Wang et al. (2020) recently cloned and characterized *Fhb7* and concluded that the gene encodes glutathione S-transferase (GST) based on the genome assembly of *Thinopyrum elongatum.*

Major Effect FHB Resistance QTL on Chromosome 5A

Qfhs.ifa-5A is a strong-effect FHB resistance QTL that occurs on chromosome arm 5AS and provides type I FHB resistance by reducing initial infection (Buerstmayr et al., 2003). *Qfhs.ifa-5A*, to a lesser extent, also confers resistance of type II (Schweiger et al., 2013). *Qfhs.ifa-5A* was discovered in Sumai-3 and its derivatives and reported to explain about 20% of the phenotypic variation observed by Buerstmayr et al. (2003). *Qfhs.ifa-5A* is flanked by markers *Xgwm293* and *Xgwm156*. The markers *Xgwm293, Xgwm304a, Xgwm1057, Xbarc186, Xbarc117*, and *Xbarc56* appeared to be closely or fully linked to the centromere within that interval. Buerstmayr et al. (2018) used doubled haploid (DH) and random inbred line (RIL) populations for more accurate mapping and reduced the critical region to a 1.6cM stretch flanked by *Xbarc196* and *Xwmc805*. Among the markers tested, *Xcfa2250,* and *Xgpg503* mapped closest to the centromere with a genetic distance of 0.9cM between *Xcfa2250* and *Xbarc186* in the NI-RIL map containing seven loci. A recombination-independent radiation hybrid mapping (RH mapping) technique was also used to improve resolution in the peri-centromeric region of 5AS (Buerstmayr et al., 2018). The map resolution obtained through RH mapping for the interval *Qfhs.ifa-5A* was 389-fold better compared to the genetic map and 66 loci were found over a distance of 3500.3cR in the same interval (1cR equals ~0,77Mb for 5AS; Huchriede et al., 1999).

Chu et al. (2011) reported the presence of another two major resistance QTL (*Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2*, respectively) on chromosome arms 5AS and 5AL of the germplasm line PI 277012 (hexaploid spring wheat). The type II resistance of PI 277012 was like the resistance in 'Sumai3'. *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* explained up to 20 and 32% of the variation in FHB severity, respectively (Chu et al., 2011). In addition, the two QTL strongly

reduced the percentage of Fusarium damaged kernels (FDK) and DON accumulation in seeds (Chu et al., 2011). *Qfhb.rwg-5A.1* was mapped within a range of 40.8cM flanked by *Xcfa2104* and *Xgwm617* with a peak at the *Xbarc40* marker on the 5AS. *Qfhb.rwg-5A.2* was located within a 40.4cM interval flanked by markers *Xwmc470* and *Xbarc48* and peaked at marker *Xcfd39* (Chu et al., 2011). There is a clear overlap of the chromosome regions that border *Qfhs.ifa-5A*, *Qfhb.rwg-5A.1*, and *Fhb5* in the published genetic maps of Buerstmayr et al. (2003, 2009, and 2018), Chu et al. (2011), Somers et al. (2004), and Sourdille et al. (2004). Chu et al. (2011) also concluded that *Qfhs.ifa-5A* and *Qfhb.rwg-5A.1* might be the same locus or different alleles of the same locus. The exact relationship between these three 5AS QTL remains unclear.

Qfhb.rwg-5A.2 represented a novel FHB resistance QTL in wheat (Chu et al., 2011). PI 277012 has a *T. timopheevi* accession in its ancestry which is the likely source of the FHB resistance QTL. PI 277012 is non-free threshing (has tough glumes) due to it having the recessive allele, *q*, at the domestication locus *Q*, which was first isolated by Simons et al. (2006). The *Q* gene is located on 5AL and mapped approximately 5.4cM from *Xcfd39*, the marker locus closest to the *Qfhb.rwg-5A.2* peak (Chu et al., 2011). In a more recent study, the *Q* gene was mapped at 8.5cM from *Xcfa39* using the same PI 277012-derived DH population (Zhao, 2017). One of the DH lines from the mapping population (DH#80 or GP80) retained both *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* but had acquired the *Q*-allele and therefore has free-threshing like ordinary wheat (Chu et al., 2011).

Chu et al. (2011) provided closely linked molecular markers for *Qfhb.rwg-5A.2* that occurred in the *Xwmc470-Xbarc48* interval. Zhao (2017) narrowed the region containing *Qfhb.rwg-5A.2* to a genomic region of 0.4cM and developed three CAPS markers (M2375, M2620, and M2781) associated with *Qfhb.rwg-5A.2* that are believed to be more precise.

Wheat Breeding for FHB Resistance

The goal of plant breeding is to develop new cultivars with improved yield, pest resistance, and processing quality. Towards this end-use is made of new and existing genetic variation, careful testing, and selection (Asins, 2002). By using traditional breeding strategies, mainly based on new cross combinations and repeated tests, breeders have improved FHB resistance under natural and artificial epidemic environments (Buerstmayr et al., 2009). However, as a quantitative trait, FHB resistance is complex, and breeding for FHB resistance is complicated by genetic factors of the host and of the pathogen, genotype by environment interaction, undesirable agronomic traits, and phenotyping difficulties (Buerstmayr et al., 2009; Rudd et al., 2001).

Marker-assisted selection (MAS) has been applied to facilitate traditional breeding, but Miedaner et al. (2006) cautioned that MAS combined with phenotypic selection would be the best approach to utilize the quantitative variation of disease resistance. Gene introgression and pyramiding, as traditional breeding approaches, usually introduce genes from different gene pools into adapted lines to augment the resistance. Applying molecular markers could assist in identifying the exact genes integrated into the breeding lines while reducing the cycle length of selection processes and reducing labor and cost with the improvement of technologies (Miedaner et al., 2006; Shen et al., 2003). With the rapid development of next-generation sequencing, genotyping-by-sequencing (GBS) that does not require preliminary sequence information (Deschamps et al., 2012) has significant potential benefits for plant breeding. It is valuable for species with large and complex genome sequence information and limited public resources, especially for wheat genetic studies which are restricted by its large genome size (17 giga base pairs) and high repetitive sequence content (around 80%) (Benchley et al., 2012). Poland et al.

(2012) utilized GBS to develop a high-density genetic map with 20,000 SNPs in wheat. Genomic selection (GS) is a potentially useful tool for plant breeding, making it possible to increase disease resistance, identify low impact QTL for disease-resistance, and to improve the disease resistant germplasm within fewer cycles compared to MAS (Miedaner et al., 2006). Rutkoski et al. (2012) confirmed several advantages of GS for FHB resistance in wheat breeding. However, the applications of GBS and GS are still limited, and more research is needed to develop additional markers and prediction models for FHB resistance.

To provide for easy referencing, all the FHB resistance QTL that are mentioned in the study are summarized in Table 1.

Table 1. Summary of FHB resistance QTL/genes discussed in the study.

QTL	Chr. Location	Source	References
Qfhs.ifa-5A	5AS	Sumai-3 and derivatives	Buerstmayr et al., 2002; Somers et al., 2003 Xue et al., 2011
Qfhb.rwg.5A.1	5AS	PI 277012	Chu et al., 2011
Qfhb.rwg.5A.2	5AL	PI 277012	Chu et al., 2011
Fhb1	3BS	Sumai-3, Ning 7840, Wangshuibai	Bai et al., 1999; Waldron et al., 1999; Buerstmayr et al., 2002, 2003; Cuthbert et al., 2006
Fhb2	6BS	Sumai-3, Ning 8026, Ning 894037, Blackbird	Waldron et al., 1999; Anderson et al., 2001, Cuthbert et al., 2007
Fhb3	7A	*Leymus racemosus*	Qi et al., 2008
Fhb4	4B	Wangshuibai	Lin 2006; Jia et al., 2018
Fhb5	5A	Wangshuibai	Lin 2006; Jia et al., 2018
Fhb6	1A	*Elymus tsukushiensis*	Cainong et al., 2015
Fhb7	$7el_2$/7BL Tr	*Thinopyrum ponticum*	Shen et al., 2004; Shen and Ohm, 2007; Wang et al., 2020

References

Anderson, J.A., R.W. Stack, S. Liu, B.L. Waldron, A.D. Fjeld, et al. 2001. DNA markers for Fusarium head blight resistance QTLs in two wheat populations. Theor. Appl. Genet. 102: 1164–1168.

Anikster, Y., and I. Wahl. 1979. Coevolution of rust fungi on Gramineae and Liliaceae and their hosts. Ann. Rev. Phytopathol. 17: 367-403.

Atanasoff, D. 1920. Fusarium-blight (Scab) of wheat and other cereals. J. Agric. Res. 20: 1–32.

Bai G., and G. Shaner. 1994. Scab of wheat: Prospects for control. Plant Dis. 78: 760–766.

Bai, G., and G. Shaner. 2004. Management and resistance in wheat and barley to Fusarium head blight. Annu. Rev. Phytopathol. 42: 135–161.

Bai, G., F.L. Kolb, G. Shaner, and L.L. Domier. 1999. Amplified fragment length polymorphism markers linked to a major quantitative trait locus controlling scab resistance in wheat. Phytopathology. 89: 343–348.

Bai, G., Z. Su, and J. Cai. 2018. Wheat resistance to Fusarium head blight. Can. J. Plant Pathol. 40: 336–346.

Bariana H.S., and R. A. McIntosh. 1993. Cytogenetic studies in wheat. XV. Location of rust resistance genes in VPM1 and their genetic linkage with other disease resistance genes in chromosome 2A. Genome. 36:476-82.

Bernardo, A.N., R.L. Bowden, M.N. Rouse, M.S. Newcomb, D.S. Marshall, et al. 2013. Validation of molecular markers for new stem rust resistance genes in U.S. hard winter wheat. Crop Sci. 53: 755–764.

Bhardwaj S.C., S.K. Nyar, M. Prashar, J. Kumar, M. K. Menon, and S.B. Singh. 1990. A pathotype of *Puccinia graminis* f. sp. *tritici* on *Sr24* in India. Cereal Rusts and Powdery Mildews Bulletin. 18:35-38.

Boyd, L. 2005. Can Robigus defeat an old enemy? – Yellow rust of wheat. The Journal of Agricultural Science, 143: 233-243.

Buerstmayr, H., B. Steiner, L. Hartl, M. Griesser, N. Angerer, et al. 2003. Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. II. Resistance to fungal penetration and spread. Theor. Appl. Genet. 107: 503–508.

Buerstmayr, H., T. Ban, and J.A. Anderson. 2009. QTL mapping and marker-assisted selection for Fusarium head blight resistance in wheat: A review. Plant Breed. 128: 1–26.

Cai, X., S. Xu, R. Oliver, Q. Zhang, R. Stack, S. Zhong, et al. (2008). Alien introgression of FHB resistance in wheat challenges and strategies. In: Appels R, Eastwood R, Lagudah E, Langridge P, Mackay M, McIntyre L, and Sharp P, editors. Proceedings of the 11th

International Wheat Genet Symposium; Aug 24–29; Brisbane, Qld, Australia: Sydney Univ Press, Sydney (Australia).

Cainong, J.C., W.W. Bockus, Y. Feng, P. Chen, L. Qi, et al. 2015. Chromosome engineering, mapping, and transferring of resistance to Fusarium head blight disease from *Elymus tsukushiensis* into wheat. Theor. Appl. Genet. 128: 1019–1027.

Catalog of Gene Symbols. Komugi- Wheat genetic resources database. https://shigen.nig.ac.jp/wheat/komugi/genes/symbolClassList.jsp (accessed 13th March 2020)

Catanzariti, A.M., P.N. Dodds, J.G. Ellis, and B.J. Staskawicz. 2010. The interaction of avirulence and resistance gene products in flax rust disease - Providing advances in rust research. Can. J. Plant Pathol. 32: 11–19.

Chen, Q., F. Eudes, R.L. Conner, R. Graf, A. Comeau, et al. 2001. Molecular cytogenetic analysis of a durum wheat x *Thinopyrum distichum* hybrid used as a new source of resistance to Fusarium head blight in the greenhouse. Plant Breed. 120: 375–380.

Chu, C., Z. Niu, S. Zhong, S. Chao, T.L. Friesen, et al. 2011. Identification and molecular mapping of two QTLs with major effects for resistance to Fusarium head blight in wheat. Theor. Appl. Genet. 123: 1107–1119.

Collard, B.C.Y., and D.J. Mackill. 2008. Marker-assisted selection: An approach for precision plant breeding in the twenty-first century. Philos. Trans. R. Soc. B Biol. Sci. 363: 557–572.

Collard, B.C.Y., M.Z.Z. Jahufer, J.B. Brouwer, and E.C.K. Pang. 2005. An introduction to markers, quantitative trait loci (QTL) mapping, and marker-assisted selection for crop improvement: The basic concepts. Euphytica 142: 169–196.

Curtis, B.C. 2002 Wheat in the World. In: Curtis, B.C., Rajaram, S. and Macpherson, H.G., Eds., Bread Wheat Improvement and Production, Plant Production and Protection Series 30, FAO, Roma, 1-18.

Cuthbert, P.A., D.J. Somers, and A. Brulé-Babel. 2007. Mapping of *Fhb2* on chromosome 6BS: A gene controlling Fusarium head blight field resistance in bread wheat (*Triticum aestivum* L.). Theor. Appl. Genet. 114: 429–437.

Cuthbert, P.A., D.J. Somers, J. Thomas, S. Cloutier, and A. Brulé-Babel. 2006. Fine mapping *Fhb1*, a major gene controlling fusarium head blight resistance in bread wheat (*Triticum aestivum* L.). Theor. Appl. Genet. 112: 1465–1472.

Dyck, P.L., D.J. Samborsk, and R.G. Anderson. 1966. Inheritance of adult plant leaf rust resistance derived from common wheat varieties Exchange and Frontana. Can. J. Genet. Cytol. 8: 665–671.

Figueroa, M., K. Hammond-Kosack, and P. S. Solomon. 2018. A review of wheat disease – a field perspective. Molecular Wheat Pathology 19: 1523-1536.

Flor, H.H. 1971. Current status of the gene-for-gene concept. North: 275–296.

Gervais, L., F. Dedryver, J.Y. Morlais, V. Bodusseau, S. Negre, et al. 2003. Mapping of quantitative trait loci for field resistance to Fusarium head blight in European winter wheat. Theor. Appl. Genet. 106: 961–970.

Golan, G., A. Oksenberg, and Peleg, Z. 2015. Genetic evidence for differential selection of grain and embryo weight during wheat evolution under domestication. Journal of Experimental Botany 66:5703-5711.

Guo, J., X. Zhang, Y. Hou, J. Cai, X. Shen, et al. 2015. High-density mapping of the major FHB resistance gene *Fhb7* derived from *Thinopyrum ponticum* and its pyramiding with *Fhb1* by marker-assisted selection. Theor. Appl. Genet. 128: 2301–2316.

Guo, P.G., G.H. Bai, and G.E. Shaner. 2003. AFLP and STS tagging of a major QTL for Fusarium head blight resistance in wheat. Theor. Appl. Genet. 106: 1011–1017.

Herrera-Foessel S.A., R.P. Singh, J. Huerta-Espino, G.M. Rosewarne, S.K. Periyannan, L. Viccar, V. Calvo-Salazar, C. Lan, and E.S. Lagudah. 2012. *Lr68***:** a new gene conferring slow rusting resistance to leaf rust in wheat. Theoretical and Applied Genetics, 124:1475-1486.

Hodson, D.P., J. Grønbech-Hansen, P. Lassen, Y. Alemayehu, J. Arista, K. Sonder, P. Kosina, P. Moncada, K. Nazari, R. F. Park, Z. A. Pretorius, L. J. Szabo, T. Fetch, and Y. Jin. 2011. Tracking the wheat rust pathogens. Proceedings of BGRI 2011 Technical Workshop, St. Paul, Minnesota: pp3-13.

Hooker, A.L. 1967. The genetics and expression of resistance in plants to rusts of the genus Puccinia. Annu. Rev. Phytopathol. 5:163-78.

Kerber E.R., and P. L. Dyke. 1990. Transfer to hexaploid wheat of linked genes for adult-plant leaf rust and seedling stem rust resistance from amphiploid of *Aegiolops speltoides* **x** *Triticum monoccum***.** Genome. 33:530-537.

Kolmer, J. 2005. Tracking wheat rust on a continental scale. Current Opinion Plant Biol.4:441-449.

Kolmer, J.A. 2003. Postulation of leaf rust resistance genes in selected soft red winter wheats. Crop Sci. 43: 1266–1274.

Kolmer, J.A., R.P. Singh, D.F. Garvin, L. Viccars, H.M. William, J. Huerta-Espino, et al. 2008. Analysis of the rust resistance region in wheat germplasm. Crop Sci. 48: 1841-1852.

Kolmer, J.A., Y. Jin, and D.L. Long. 2007. Wheat leaf and stem rust in the United States. Aust. J. Agric. Res. 58: 631–638.

Kou, Y., and S. Wang. 2010. Broad-spectrum and durability: understanding of quantitative disease resistance. Curr. Opin. Plant Biol. 13: 181–185.

Kuzmanović, L., G. Mandalà, S. Tundo, R. Ciorba, M. Frangella, et al. 2019. Equipping Durum Wheat—*Thinopyrum ponticum* Recombinant Lines with a *Thinopyrum elongatum* major QTL for resistance to Fusarium diseases through a cytogenetic strategy. Front. Plant Sci. 10: 1–17.

Law, C.N., and R. Johnson. 1967. A Genetic study of leaf rust of wheat. Can. Jo. Gen. Cytol. 805-822.

Lemmens, M., U. Scholz, F. Berthiller, C. Dall'Asta, A. Koutnik, et al. 2005. The ability to detoxify the mycotoxin deoxynivalenol colocalizes with a major quantitative trait locus for fusarium head blight resistance in wheat. Mol. Plant-Microbe Interact. 18: 1318–1324.

Mago R., H.S. Bariana, I.S. Dundas, W. Spielmeyer, G.J. Lawrence, A.J. Pryor, and J.G. Ellis. 2005. Development of PCR markers for the selection of wheat stem rust resistance genes *Sr24* and *Sr26* in diverse wheat germplasm. Theoretical and Applied Genetics. 111:496-504.

Mardi, M., L. Pazouki, H. Delavar, M.B. Kazemi, B. Ghareyazie, et al. 2006. QTL analysis of resistance to Fusarium head blight in wheat using a 'Frontana'-derived population. Plant Breed. 125: 313–317.

Martínez F, R.E. Niks, R.P. Singh, and D. Rubiales. 2001. Characterization of *Lr46*, a gene conferring partial resistance to wheat leaf rust. Hereditas. 135:111-114.

McIntosh, R. 1988. The role of specific genes in breeding for durable stem rust resistance in wheat and triticale. In: N.W. Simmonds, S. Rajaram, editors. Breeding strategies for resistance to rusts of wheat. CIMMYT. Mexico. P. 1-2.

Mcintosh, R.A., C. R. Wellings, and R. F. Park. 1995. an Atlas of resistance genes. CSIRO Publication, Australia.

McMullen, M., G. Bergstrom, E. De Wolf, R. Dill-Macky, D. Hershman, et al. 2012. Fusarium head blight disease cycle, symptoms, and impact on grain yield and quality frequency and magnitude of epidemics since 1997. Plant Dis. 96: 1712–1728.

Mcmullen, M., R. Jones, D. Gallenberg, and S. America. 1997. Scab of Wheat and Barley: A re-emerging disease of devastating impact. Plant Dis. 81.

Mesterhazy, A. 1995. Types and components of resistance to Fusarium head blight of wheat. Plant breeding, 114: 377-386.

Mesterházy, Á., T. Bartók, C.G. Mirocha and R. Komoroczy. 1999. Nature of wheat resistance to Fusarium head blight and the role of deoxynivalenol for breeding. Plant breeding, 118: 97-110.

Nganje, W.E., S. Kaitibie, W.W. Wilson, L.F. Leistritz, and D.A. Bangsund. 2004. Economic impacts of fusarium head blight in wheat and barley: 1993-2001.

Oelke, L.M., and J.A. Kolmer. 2004. Characterization of leaf rust resistance in hard red spring wheat cultivars. Plant Dis. 88: 1127–1133.

Park, R.F. 2007. Stem rust of wheat in Australia. Australian Journal of Agricultural Research 6: 558-566

Paul, P.A., P.E. Lipps, D.E. Hershman, M.P. McMullen, M.A. Draper, et al. 2008. Efficacy of triazole-based fungicides for Fusarium head blight and deoxynivalenol control in wheat: A multivariate meta-analysis. Phytopathology 98: 999–1011.

Paulsen, G.M., and J.P. Shroyer. 2008. The early history of wheat improvement in the great plains. Agron. J. 100.

Peng, J.H., D. Sun, and E. Nevo. 2011. Domestication evolution, genetics and genomics in wheat. Mol. Breeding 28, 281-301.

Peterson, P. D. 2001. Stem rust of wheat: exploring the concepts. In P.D. Peterson, editor. Stem rust of wheat: from ancient enemy to modern foe. The American Phytopathological Society. St. Paul, MN. P.1-2

Plains Grains Inc. 2020. Hard red winter wheat regional quality survey report 2019. https://www.plainsgrains.org/wp-content/uploads/2019/11/Plains-Grains-2019-Winter-Wheat-Report-Web.pdf (accessed: 15th May 2020).

Pretorius, Z.A., R.D. Wilcoxson, D. L. Long, and J.F. Schafer. 1984. Detecting the wheat leaf resistance gene *Lr13* in seedlings. Plant disease 68: 585-586.

Ren, J., Z. Wang, Z. Du, M. Che, Y. Zhang, et al. 2019. Detection and validation of a novel major QTL for resistance to Fusarium head blight from *Triticum aestivum* in the terminal region of chromosome 7DL. Theor. Appl. Genet. 132: 241–255.

Risk, J.M., L.L. Selter, H. Chauhan, S.G. Krattinger, J. Kumlehn, et al. 2013. The wheat *Lr34* gene provides resistance against multiple fungal pathogens in barley. Plant Biotech. J. 11: 847–854.

Roelfs, A.P., R.P. Singh, and E.E. Saari. 1992. Rust diseases of wheat: concepts and methods of disease management. CIMMYT, Mexico, 80.

Rudd, J.C., R. D. Horsley, A.L. McKendry, and E. M. Elias. 2001. Host plant resistance genes for Fusarium head blight. Crop Science, 41: 620-627.

Schmale III, D.G. and G.C. Bergstrom. 2003. Fusarium head blight in wheat. The Plant Health Instructor. doi:10.1094/PHI-I-2003-0612-01.

Sharma, I. 2012. Disease in wheat crops- an introduction. In: I. Sharma, editor. Disease resistance in wheat. CABI Wallingford Oxfordshire, UK, p. 1.

Singh, R.P., A. Mujeeb-Kazi, and J. Huerta-Espino. 1998. *Lr46*: A Gene Conferring Slow-Rusting Resistance to Leaf Rust in Wheat. Phytopathology. 88:890-894.

Singh, R.P., D.P. Hodson, J. Huerta-Espino, S. Bhavani, et al. 2011. The Emergence of Ug99 races of the stem rust fungus is a threat to world wheat production. Annu. Rev. Phytopathol. 49: 465–481.

Singh, R.P., D.P. Hodson, J. Huerta-Espino, Y. Jin, P. Njau, R. Wanyera, et al. 2008. Will stem rust destroy the world's wheat crop? Adv. Agron. 98: 272-309.

Singh, R.P., D.P. Hodson, J. Huerta-Espino, Y. Jin, S. Bhavani, P. Njau, et al. 2011. The emergence of Ug99 races of the stem rust fungus is a threat to world. Annu. Rev. Phytopathol. 49:465-81.

Smith E.L., A.M. Schlehuber, H. C. Young Jr, and L.H. Edwards. 1968. Registration of agent wheat. Crop Sci. 8:511-512.

Spolti, P., E.M. Del Ponte, Y. Dong, J.A. Cummings, and G.C. Bergstrom. 2014. Triazole sensitivity in a contemporary population of *Fusarium graminearum* from New York wheat and competitiveness of a tebuconazole-resistant isolate. Plant Dis. 98: 607–613.

Sthapit, J., M. Newcomb, J.M. Bonman, X. Chen, and D.R. See. 2014. Genetic diversity for stripe rust resistance in wheat landraces and identification of accessions with resistance to stem rust and stripe rust. Crop Sci. 54: 2131–2139.

Tanksley, S.D. 1983. Molecular markers in plant breeding. Plant Mol. Biol. Report. 1: 3–8.

USDA Agriculture Research Service. wheat sector at a glance. https://www.ers.usda.gov/topics/crops/wheat/wheat-sector-at-a-glance/ (accessed 12th March 2020)

USDA-ARS Cereal Disease Lab. 2017. Resistance Genes. St. Paul, MN. https://www.ars.usda.gov/midwest-area/4stpaul/cereal-disease lab/docs/resistance genes/resistance-genes/. Accessed on 11/03/2020.

Waldron, B.L., B. Moreno-Sevilla, J.A. Anderson, R.W. Stack, and R.C. Frohberg. 1999. RFLP mapping of QTL for Fusarium head blight resistance in wheat. Crop Sci. 39: 805–811.

Wiese, M.V. 1987. Infectious diseases – disease caused by bacteria and mycoplasmas. In: M.V. Wiese, editor. Compendium of wheat disease. Second Edition. The American Phytopathological Society. St Paul, MN, USA. Pg. 16-41.

Xue, S., F. Xu, M. Tang, Y. Zhou, G. Li, et al. 2011. Precise mapping *Fhb5*, a major QTL conditioning resistance to Fusarium infection in bread wheat (*Triticum aestivum* L.). Theor. Appl. Genet. 123: 1055–1063.

Xue, S., G. Li, H. Jia, F. Xu, F. Lin, et al. 2010. Fine mapping *Fhb4*, a major QTL conditioning resistance to Fusarium infection in bread wheat (*Triticum aestivum* L.). Theor. Appl. Genet. 121: 147–156.

Yang, J., G. Bai, and G.E. Shaner. 2005. Novel quantitative trait loci (QTL) for Fusarium head blight resistance in wheat cultivar 'Chokwang'. Theor. Appl. Genet. 111: 1571–1579.

Young, N.D. 1999. A cautiously optimistic vision for marker-assisted breeding. Mol. Breed. 5: 505–510.

Zhang, L., P. Luo, Z. Ren, and H. Zhang. 2011. Controlling Fusarium head blight of wheat (*Triticum aestivum* L.) with genetics. Adv. Bio.

Zhang, X., G. Bai, W. Bockus, X. Ji, and H. Pan. 2012. Quantitative trait loci for Fusarium head blight resistance in U.S. hard winter wheat cultivar ‘Heyne’. Crop Sci. 52: 1187–1194.

CHAPTER III. TRANSFER OF FHB RESISTANCE QTL *QFHB.RWG-5A.1* AND *QFHB.RWG-5A.2* TO HARD RED WINTER WHEAT

Abstract

The fungal disease Fusarium head blight (FHB caused by *Fusarium graminearum* Schwabe) poses a major threat to winter wheat in North Dakota. Most currently grown winter wheat cultivars are susceptible to highly susceptible to the disease. Many diverse minor resistance genes with varying effectiveness have been described and mapped in recent literature and these can be introduced and applied in breeding programs. This study aimed to transfer the FHB resistance QTL *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* from the spring wheat GP80 to hard red winter wheat. In a previous analysis of the cross: GP80/Novus-4// 19CP29/3/ND Noreen, single nucleotide polymorphisms (SNPs) and simple sequence repeat (SSR) markers were used to select two winter habit derivatives (named CP46C-6 and CP46C-9), each believed to be homozygous for *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2*. CP46C-6 and CP46C-9 were then backcrossed to ND Noreen. The respective parents and B_1F_1 were genotyped with the Illumina 90K SNP platform and the genotype data were analyzed to identify SNP haplotypes that corresponded to GP80 chromosome 5A segments retained in CP46C-6 and CP46C-9. The average ND Noreen background recovered across all chromosomes was also estimated for each B_1F_1. All parents and B_1F_1 were then evaluated for greenhouse FHB Type II resistance and the most promising plants (based on marker predictions, FHB resistance, and greenhouse agrotype) were identified. In a further attempt to confirm the transfer of FHB resistance, nine selections that were believed to be homozygous for different combinations of *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* were compared in a second greenhouse FHB trial. The data suggested that resistance is comparable to that of GP80

occurs among the introgressions; however, ND Noreen was found to have a significant level of native resistance which confounded interpretation of the results.

Introduction

Fusarium head blight (FHB) of cereals such as wheat and barley put the safety and security of food and feed products derived from these crops at risk. The disease is widespread and can result in significant losses in grain yield and quality. Aside from direct yield losses, Fusarium mycotoxin contamination of the crop is a major problem (Buerstmayr et al., 2014). Yield and quality reductions in wheat and barley resulted in a $3 billion loss in the United States during the 1990s (Windels, 2000). The cultivation of resistant varieties is critical to the integrated management of the disease. In wheat, FHB resistance is a truly quantitative trait that is governed by polygenes and modulated by the environment. Breeding productive cultivars with enhanced FHB resistance is thus not trivial and requires substantial investment (Buerstmayr et al., 2013).

Major improvement in genetic resistance is achieved through breeding and selection. Information collected by Buerstmayr et al. (2009) and Buerstmayr et al. (2019) showed that around 500 QTL have been reported and of these 104 (20%) were described as a major QTL. *Fhb1* (originally designated *Qfhs.ndsu-3BS*) on chromosome 3BS of Sumai 3 and lines descended from it has been shown to reduce FHB symptoms by an average of 20%–25% across different genetic backgrounds (Anderson, 2007). *Fhb1* confers type II resistance (Anderson et al., 2001; Buerstmayr et al., 2002). The gene is thought to be involved in the conversion of deoxynivalenol (DON) to the less toxic DON-3-O-glycoside (Lemmens et al., 2005). Sumai 3 and its derivatives have been the most frequently used source material for FHB resistance breeding (Buerstmayr et al., 2009).

Chromosome 5A has also been shown to harbor valuable resistance genes, with over 14 FHB resistance QTL having been mapped to it (Cai et al., 2016). PI 277012 was identified as a novel source of very useful resistance that occurs on chromosome arms 5AS and 5AL. PI 277012 was developed at the Estacion Experimental de Aula Dei, Zaragoza, Spain, and has the pedigree: 'Extremo Sur'/ 'Argelino'//*T. timopheevii* (Chu et al., 2011). SSR markers were used to map the PI 277012 resistance genes in a doubled haploid population derived from a PI 277012/Grandin cross. The two significant QTL associated with chromosome 5A (in PI 277012) were named *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2*, respectively. The two loci explained up to 20% and 32% of the variation in FHB severity, respectively. *Qfhb.rwg-5A.1* was located between the markers *Xcfa2104* and *Xgwm617* on 5AS. The QTL *Qfhb.rwg.5A.2* was mapped between SSR markers *Xwmc470* and *Xbarc48.* The latter QTL showed a weak correlation with plant height. Both QTL demonstrated significant type I and type II resistance, as well as resistance to DON accumulation (Chu et al., 2011). Genetic maps of Buerstmayr et al. (2003, 2009, and 2018), Chu et al. (2011) showed that the chromosomes areas harboring *Qfhs.ifa-5A*, *Qfhb.rwg-5A.1*, and *Fhb5* overlap. However, the precise relative locations of these 5AS QTL are unknown, and it is unclear if they are different loci or alleles of the same locus.

The NDSU HRWW breeding program obtained a hard-red spring wheat line, RWG21 (pedigree = Russ 2*/PI 277012); believed to contain both *Qfhb.rwg-5A.1* (5AS) and *Qfhb.rwg-5A.2* (5AL), from the USDA-ARS, Fargo. Following crosses with winter wheat, the line Novus-4 (= RWG21/Jerry) was selected from a large population of doubled haploids and single seed descent progenies. Novus-4 has a winter growth habit, is intermediate in terms of winter hardiness, and has moderate FHB resistance; however, it only carries *Qfhb.rwg-5A.1* and does not have *Qfhb.rwg-5A.2* (Tao, 2019). Results obtained by Tao (2019) showed that the source line

RWG21 did not contain *Qfhb.rwg-5A.2* either. In order to transfer *Qfhb.rwg-5A.2*, a new transfer attempt had to be initiated by utilizing GP80 as the donor source and producing the cross: GP80/Novus-4//19CP29/3/ND Noreen (Ganaparthi 2021). In the absence of reliable markers for both genes, Ganaparthi (2021) relied on SNP haplotyping of the parents and progeny to derive a haplotype map of GP80 chromosome 5A. Suitably polymorphic SNP markers were then identified to characterize segregates with different lengths of PI 277012 derived chromatin within the critical donor chromosome regions. The same populations were tested for FHB resistance and winter habit segregates with increased resistance were derived. The most promising segregates were backcrossed to ND Noreen. The present study was done to characterize the backcross progenies that were produced. Reliable markers were still not available for this follow-up study which therefore continued to employ SNP haplotyping for selection purposes and for genome-wide SNP comparisons to assess the recovery of the ND Noreen genetic background in the B_1F_1 progeny. Finally, a second greenhouse FHB trial was conducted to confirm the transfer of either or both of *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* through the first backcross.

Material and Methods

Study Outline

This study is a continuation of previous attempts (initiated by Tao (2019) and Ganaparthi (2020)) to introgress resistance QTL *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* from HSW into HRW. Utilizing segregates from Ganaparthi's study that was believed to harbor both genes, a backcross to ND Noreen (named cross 20M1) was made and analyzed. Figure 1 outlines the crosses and backcrosses utilized in this study. Tao (2019) produced the germplasm line Novus-4 which has

Qfhb.rwg-5A.1 whereas Ganaparthi (2020) produced the $F_{1:2}$ 19M13 that served as the starting material for the present study.

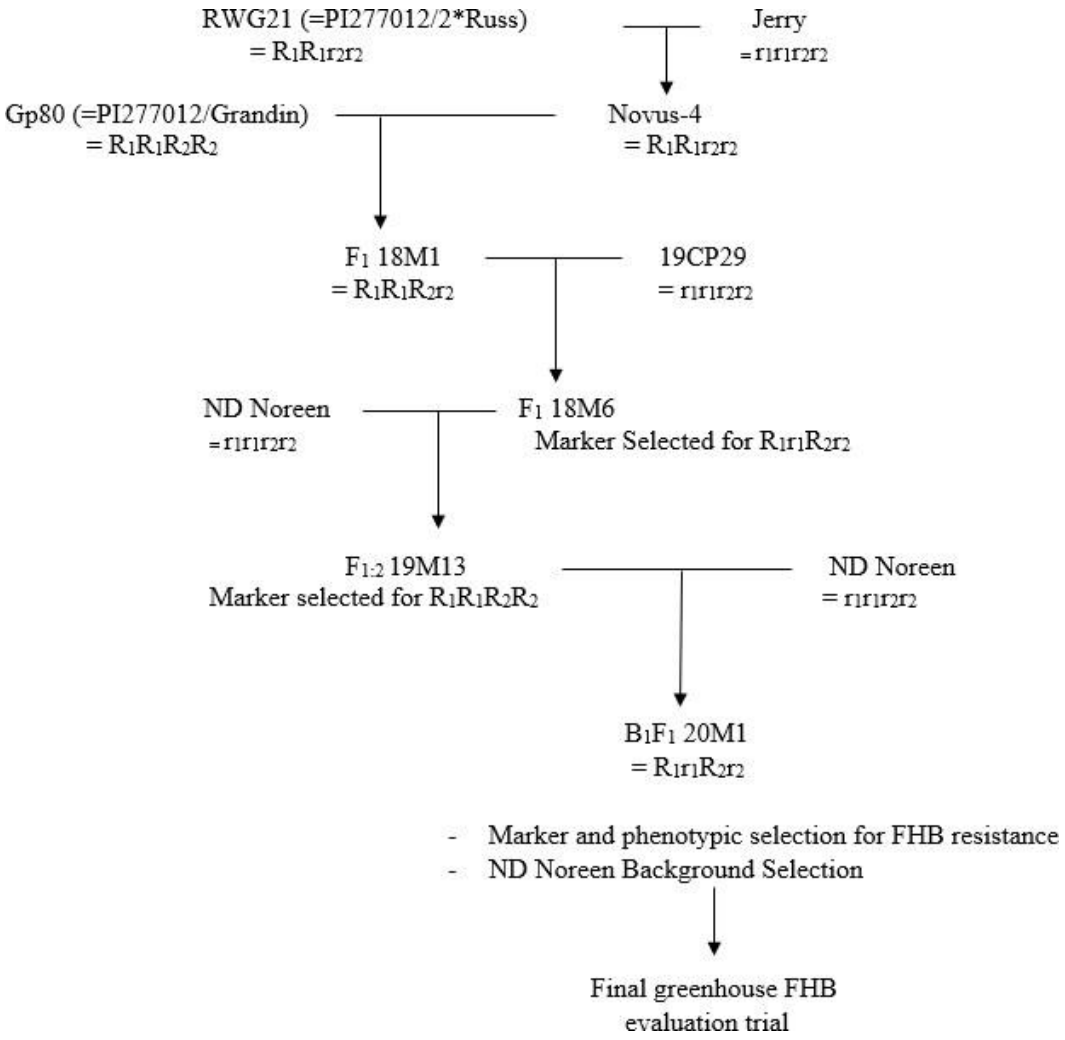


Figure 1. Current and previous crosses have been made in an attempt to transfer FHB resistance QTL *Qfhb.rwg-5A.1* (symbol R1) and *Qfhb.rwg-5A.2* (symbol R2) from HRS wheat donor lines RWG21 and GP80 to winter wheat. The F1:2 19M13 served as starting material for the present study.

Two F_2 19M13 plants (named CP46C-6 and CP46C-9), each believed to be homozygous for *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2*, were selected with the help of SSR markers that occurred within the SNP haplotypes selected by Ganaparthi (2020) and backcrossed to the winter wheat ND Noreen to produce two B_1F_1:20M1 (= GP80/Novus-4// 19CP29/3/ 2* ND Noreen)

populations consisting of 112 seeds (20M1A) and 20 seeds (20M1B), respectively. One hundred and thirty-one surviving B_1F_1 seedlings were planted with appropriate parental controls (Table 2) 5to do marker analyses and FHB resistance testing. The B_1F_1 plants were grown in 6" plastic pots in an un-replicated greenhouse trial layout with two replicates of each control. Leaf samples were cut on the B_1F_1 and parental controls which were then used for DNA extraction and genotyping.

Marker Analyses and Background Selection

F_2 plants from the cross 19M13 (= GP80/ Novus-4//19CP29/3/ND Noreen) and progenitors GP80, Novus-4, 19CP29 and ND Noreen were initially tested with the simple sequence repeat (SSR) markers Barc186 and Gwm304 (linked to *Qfhb.rwg-5A.1*; Chu et al., 2011 and Tao 2019) and Gpw2136, Gpw2181, and Gpw2172 linked to *Qfhb.rwg-5A.2* (Tao 2019) by Ganaparthi (2020) to confirm the presence of useful marker polymorphisms and the likely presence of *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2*. Two such plants were found to be homozygous for the targeted marker loci (Ganaparthi 2020, Personal Communication), and were named CP46C-6 and CP46C-9 respectively and were used for making the backcross to ND Noreen.

The study of Ganaparthi (2020) identified SNP haplotypes within the chromosome regions that were reported to harbor the respective QTL peaks and were suitably polymorphic to the originating germplasm (PI 277012 and GP80) and the parents used to produce cross 19M13. In this study, the parents and progeny listed in Table 2 were utilized for SNP analyses employing the Illumina Infinium iSelect 90K SNP array. DNA extraction and SNP analyses were performed by the USDA-ARS Bioscience Research Lab in Fargo, North Dakota, USA (https://www.ars.usda.gov/plains-area/fargo-nd/etsarc). SNPs were clustered using the manual

option of Genome Studio 2.0 with the polyploid clustering module (https://www.illumina.com/techniques/arrays/array-data-analysis-experimental-design/genomestudio.html). SNPs with a GenTrain score of more than 90% were selected and exported to MS-Excel. The 90K consensus map of Wang et al. (2014) was consulted as an aid to finding the chromosome locations, deriving SNP haplotypes of individual genotypes, and estimating the proportion of genetic background recovered during backcrossing. The same set of DNA samples was analyzed (Winter wheat breeding program laboratory, Plant Sciences Department, Loftsgard Hall) for polymorphism to SSR loci that occur in the same chromosome region as *Qfhb.rwg-5A-1* (*Xbarc186*) and *Qfhb.rwg-5A.2 (Xgpw2136)*. For doing the SSR marker analyses, seedling leaves were cut, and DNA was extracted following a modification of the Triticarte Pty. Ltd (http://www.triticarte.com.au/) protocol. Quality and concentration of extracted DNA were checked using agarose gel electrophoresis and staining with ethidium bromide. DNA concentration was adjusted to 10 ng/µl before using it in polymerase chain reactions (PCRs). The marker primer sequences and PCR conditions for the markers that were employed are available on the Grain genes website (http://www.wheat.pw.usda.gov).

Table 2. Parents, control genotypes, and B_1F_1 plants that were used for DNA analyses and FHB type II resistance tests.

Test/control genotypes	**DNA analyses**	**FHB resistance**
GP80	Yes	Yes
Novus-4	Yes	Yes
19CP29	Yes	Yes
ND Noreen	Yes	Yes
F_1: 18M6 GP80/Novus-4//19CP29	Yes[1]	No
F_2: 19M13 plant A (alias CP46C-6)	Yes[1]	No
F_2: 19M13 plant B (alias CP46C-9)	Yes[1]	No
112 B_1F_1: 20M1(A) plants = CP46C-6/ND Noreen	Yes	Yes
20 B_1F_1: 20M1(B) plants = CP46C-9/ND Noreen	Yes	Yes

[1] From stored, freeze-dried leaves.

Screening for Type II Resistance (Greenhouse)

In the first greenhouse FHB trial, the B_1F_1 genotypes and controls in Table 2 were evaluated in an un-replicated experiment. In the second greenhouse FHB trial, the entries given in Table 6 were evaluated in a replicated trial. The second greenhouse trial was done to confirm the transfer of the complete FHB resistance to winter wheat. In both experiments, the plants were vernalized at 4 ^{o}C for 65 days and moved to a greenhouse. Up to three spikes per plant were tagged with the date when flowering and inoculated with freshly prepared FHB spores. The disease severity of each infected spike was recorded 21 days after inoculation. The single spikelet injection method was used to inoculate a single central spikelet per spike during anthesis (Stack, 1989). A mixture of *Fusarium graminearum* isolates (Fg 8_13, Fg 10_124_1, Fg 10_135_5, and Fg 13_79) was obtained from Dr. S. Zhong (Department of Plant Pathology, North Dakota State University) was used as inoculum. A 10 µl-droplet containing the isolate mixture (spore concentration approx. 100,000 conidia per ml) was injected into the spike's middle floret. Spikes that had been inoculated were immediately covered with a moist plastic bag and left for 48-72 hours. After the initial inoculation, the greenhouse temperature was increased to 72–76^{o}F. At 21-24 days after inoculation, infection severity was determined by manually counting the total number of spikelets and the number of infected spikelets per spike.

Greenhouse FHB Trial 2 - Evaluation of Nine $F_{2:3}$ Lines That Were Established from the F_2 of Four Crosses 20M1 B_1F_1 Plants

An attempt was made to confirm the presence of *Qfhb.rwg-5A-1* and *Qfhb.rwg-5A.2* in the selected progenies. Four B_1F_1 plants were selected based on overall phenotype and apparent FHB resistance in the first FHB trial and F_2 plants within each family were screened with *Xbarc186* and *Xgpw2136* to identify likely homozygotes for the two resistance QTL. F_2 plants

that were likely homozygotes for the resistance QTL were identified (markers) and harvested separately and are listed in Table 6. A greenhouse trial to measure Type II FHB resistance was then conducted. This took the form of a randomized block experiment with 12 entries planted in six replications. Five seeds of an entry planted in a 6-inch pot constituted one replication.

Results and Discussion

Identification of SNP Markers Suitable for Haplotyping and Marker-Assisted Selection

SNP loci that have previously been mapped to chromosome 5A (Wang et al., 2014) were manually edited in Genome Studio 2.0 and the data of polymorphic markers were exported to Excel. Two hundred and sixty polymorphic SNPs were identified on chromosome 5A in the region between 15.5 cM and 148.3 cM (Addendum Table A1). However, many of the markers occurred in close clusters which reduced their ability to distinguish among chromosome regions.

The 5A map of doubled haploid line GP80 (Addendum Table A2) spans the region between 8.12 cM to 148 cM and consists of three regions namely regions I and III (PI 277012-derived), and region II (Grandin-derived) (Ganaparthi, 2020). Region I is the 5AS region believed to harbor QTL *Qfhb.rwg-5A.1*. GP80 and Novus-4 share PI 277012-derived SNP alleles on 5AS that would suggest that these areas are associated with *Qfhb.rwg-5A.1*. The suitably polymorphic (for haplotype mapping) SNPs and polymorphisms detected within this region (19.9 cM to 38.7 cM) are shown in Table 3 and Addendum Table A3. The mapping results of Chu et al. (2011), SSR marker results of Tao (2019), and Ganaparthi (2020) suggested that *Qfhb.rwg-5A.2* occurs in region III, PI 277012-derived 5AL distal region of GP80 which also harbors the SSR marker loci *Xgpw2136, Xgpw2172, Xgwm179,* and *Xgwm126*. Fifteen SNP markers occurred in this region, 13 of which unambiguously detected the GP80 polymorphism

(Addendum Table A4). The results obtained for these polymorphic SNP loci are summarized in Table 3 and occurred within the 104.8 – 117.6 cM region of 5AL.

Table 3. Summary of polymorphic SNP data obtained with respect to 5AS region I within which *Qfhb.rwg-5A.1* is believed to occur and 5AL region III within which *Qfhb.rwg-5A.2* is believed to occur. The polymorphisms were seen with regard to the parents, F_1, and F_2 (CP46-C) populations that were studied are shown. The genotypes with PI 277012/GP80-derived alleles are indicated in grey.

Marker cluster[a]	Map Distance (cM)	GP80[b]	Novus-4[c]	19CP29	18M6(F_1)	ND Noreen	F_2 CP46C-6	F_2 CP46C-9
	5AS region							
8	19.89	AA	AA	BB	AB	BB	AA	AA
9	23.4	BB	BB	AA	AB	AA	AB	BB
10	25.2 - 25.6	AA	AA	BB	AB	AA	AA	AA
11	26.5	BB	BB	AA	AB	AA	BB	BB
12	35.3 - 35.9	BB	BB	AA	AB	AA	AB	BB
13	36.5 - 36.8	AA	AA	BB	AB	BB	AB	AA
14	38.7	AA	AA	BB	AB	BB	AA	AA
	5AL region							
60	104.8	BB	AA	AA	AB	AA	BB	BB
61	105.3 - 105.9	AA	BB	BB	AB	BB	AA	AA
62	109.4	BB	BB	AA	AB	AA	BB	BB
63	111.2	AA	AA	BB	AB	BB	AA	AA
64	113.1	AA	AA	BB	AB	BB	AA	AA
65	113.14	AA	BB	BB	AB	BB	AA	AA
66	114.5 - 114.9	BB	AA	BB	BB	AA	BB	BB
67	115.2 - 115.8	BB	AA	BB	BB	BB	BB	BB
68	116.0	AA	BB	BB	AB	AA	AA	AA
69	117.6	BB	BB	AA	AB	AA	BB	BB
70	117.6	BB	AA	AA	AB	AA	BB	BB

[a] The SNP loci within each cluster are listed in the Addendum (Table A2)
[b] GP80 map was obtained from Ganaparthi (2020).
[c] Novus-4 map was provided by Marais (2021 – Personal Communication)

Chromosome 5A SNP Haplotypes of the Parents and Backcross Progeny

SNP haplotypes (at 21 loci) for the chromosome 5AS critical marker region (19.9 cM to 38.7cM) in the backcross parents ND Noreen, and F_2 plants 46C-6 and 46C-9, are summarized in Table 4. The corresponding haplotype data for the two B_1F_1 populations are also shown in Table 4, following the parental and control data, and show heterozygous F_1 genotypes as expected. Within the ND Noreen/CP46C-6 B_1F_1 population there were two major 5AS haplotypes containing PI 277012 chromatin. Haplotype I included 52 plants whereas haplotype II occurred in 60 plants. One of the haplotype II plants lacked the PI 277012 allele at SNP locus 79581 (Table 4) which was more likely to be the result of an inaccurate SNP call than a double crossover. Haplotype I extended over the total 19.9 cM to 38.7 cM regions whereas in haplotype II recombination had occurred. In haplotype II, PI 277012 chromatin in the 35.4 cM to 36.9 cM region was replaced with non-PI 277012 chromatin. In the case of the second B_1F_1 population (ND Noreen/CP46C-9) only haplotype I (19 plants) was found. Regarding the 5AL critical region, only one haplotype (III) was found in both B_1F_1 ND Noreen/CP46C-6 and ND Noreen/CP46C-9 populations (Table 5). The haplotype spanned the region from 104.9 cM to 117.7 cM.

Table 4. SNP haplotypes with respect to the chromosome 5AS critical region (believed to harbor the *Qfhb.rwg.5A.1* QTL). Data are shown of the parents and B_1F_1 populations ND Noreen/F_2: 46CP-6 (112 plants) and ND Noreen/F_2: 46CP-9 (19 plants). The PI 277012-derived allele at each locus is colored orange.

	Markers			**33441**	**52121**	**66040**	**8504**	**9821**	**34969**	**39861**	**56402**	**70992**	**79257**	**80757**	**71078**	**78836**	**76028**	**78834**	**12084**	**48151**	**48152**	**76999**	**79581**	**43006**
	Map distance (cM)	**No. of plants**	**Average FHB infection**	**19.9**	**19.9**	**19.9**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**35.4**	**35.4**	**35.9**	**35.9**	**36.6**	**36.6**	**36.6**	**36.6**	**36.9**	**38.7**
ND Noreen			0.28	BB	AA	BB	AA	AA	AA	BB	BB	BB	BB	BB	BB	AA	BB	AA	BB	BB	BB	BB	AA	BB
46CP-6 (F_2)				AA	BB	AA	BB	BB	BB	AA	AA	AA	AA	AA	AB	AB	AB	AB	AB	AB	AB	AB	AB	AA
											ND Noreen/46CP-6 progeny													
Haplotype I[a]	52		0.23	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
Haplotype II[b]	59		0.21	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
Haplotype II[c]	1		0.19	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AB	AB
ND Noreen			0.28	BB	AA	BB	AA	AA	AA	BB	BB	BB	BB	BB	BB	AA	BB	AA	BB	BB	BB	BB	AA	BB
46CP-9(F_2)				AA	BB	AA	BB	BB	BB	AA	AA	AA	AA	AA	AA	BB	AA	BB	AA	AA	AA	AA	BB	AA
											ND Noreen/46C-9 progeny													
Haplotype I[a]	19		0.24	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB

[a] Heterozygotes for the complete 5AS haplotype. [b] Heterozygotes for a smaller, recombined 5AS haplotype; [c] Believed to be the same haplotype as II but with SNP 79581 wrongly called.

Table 5. SNP haplotypes with respect to the chromosome 5AL critical region that is believed to harbor *Qfhb.rwg.5A.2*. Data are shown for the parents and B_1F_1 of crosses ND Noreen/F_2: 46CP-6 (112 plants) and ND Noreen/F_2: 46CP-9 (19 plants). The PI 277012-derived allele at each locus is colored orange.

Markers			**76979**	**12226**	**48788**	**61152**	**15035**	**58284**	**33039**	**80888**	**7052**	**11676**	**66080**	**10029**	**11245**
Map distance (cM)	**No. of plants**	**Average FHB infection**	**104.9**	**105.4**	**106.0**	**109.4**	**111.2**	**113.1**	**113.1**	**113.1**	**114.5**	**115.0**	**115.0**	**117.7**	**117.7**
ND Noreen		0.28	AA	BB	AA	AA	BB	BB	BB	BB	AA	BB	AA	AA	AA
46CP-6 (F_2)			BB	AA	BB	BB	AA	AA	AA	AA	BB	AA	BB	BB	BB
			ND Noreen/46CP-6 progeny												
Haplotype III	112	0.22	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
ND Noreen		0.28	AA	BB	AA	AA	BB	BB	BB	BB	AA	BB	AA	AA	AA
46CP-9 (F_2)			BB	AA	BB	BB	AA	AA	AA	AA	BB	AA	BB	BB	BB
			ND Noreen/46CP-9 progeny												
Haplotype III	19	0.24	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB

Note: The complete data for both populations are shown in Table A4 (Addendum)

Screening of the B_1F_1 for Type II Resistance in Greenhouse FHB Trial 1.

One hundred and thirty-one B_1F_1 plants were screened for FHB resistance with 112 plants derived from the cross between CP46C-6 and ND Noreen and 20 from the cross between CP46C-9 and ND Noreen. An average of three spikes per plant were inoculated with freshly prepared inoculum. The FHB infection severities of single plants ranged from 7% to 68% (Figure 2). The detailed data are listed in Addendum (Table A6). The average infection percentages of the controls were 11% for GP 80, 19% for Novus-4 (has *Qfhb.rwg-5A.1*), 28% for ND Noreen, and 57% for cross parent 19CP29 (known to be very susceptible). Parent GP80 was derived from PI 277012 and harbors both *Qfhb.rwg-5A.1* and *Qfhb.rwg-5A.2* and was therefore expected to be the most resistant. The average infection percentages of the two sets of 112 and 19 B_1F_1 plants were respectively 22% and 24%. Thirty-three of the 131 plants tested had infection severities less than or equal to that of GP80 (Addendum Table A6). Among the 112 plants derived from the ND Noreen/F_2: 46CP-6 cross, those with haplotype I, (52 plants) had an average infection percentage of 23%; whereas those with haplotype II (60 plants) had an average infection percentage of 21%. Within the ND Noreen/F_2: 46CP-9 group (19 plants), only haplotype I occurred, and the average infection percentage amounted to 23% (Table 5). Thus, it seems unlikely (but the possibility cannot be ruled out) that a resistance QTL occurs within the 5AS region for which haplotypes I and II differ.

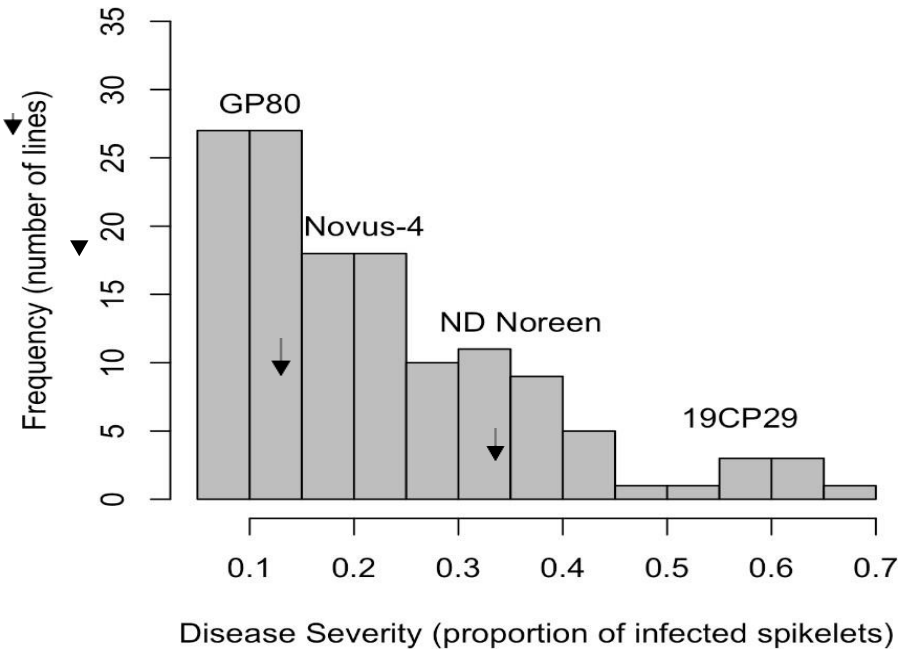


Figure 2. Distribution of Fusarium head blight infection severity in $B_1F_{1:2}$ families in the first greenhouse trial. The average infection severities of the parental controls are shown with arrows.

Marker Results Pertaining to Selections Included in Second Greenhouse FHB Trial

Among the SSR markers linked to *Qfhb.rwg-5A.1* (Barc186, Gwm304, and Gwm239) and *Qfhb.rwg-5A.2* (Gpw2136, Gpw2172, and Gpw2181), markers Barc186 and Gpw2136 showed useful polymorphisms (Ganaparthi, 2020). The latter two SSR markers were therefore amplified on selected F_2 20M1 plants and control genotypes and separated on agarose gels (Winter Wheat project laboratory). The Barc186 results are shown in Fig. 3. With respect to Barc186, lines GP80 and Novus-4, are believed to have *Qfhb.rwg-5A.1*, whereas 19CP29 does not. The results obtained when evaluating (agarose) the test plants with Gpw2136 are shown in Fig. 4. The test panel was also tested for the same two markers by the Genotyping Center, Fargo,employing a sequencer-based detection method and those results are shown in Table 6. In 2021, Dr. Jason Fiedler (Genotyping Center, Fargo – Personal Communication) developed a kompetitive allele-specific (KASP) marker named

5AL-8.0K which is based on a SNP polymorphism at the QTL peak in the study of Chu et al. (2011). This marker appears to provideaccurate (but parent-dependent) detection of *Qfhb.rwg-5A.2* and was amplified in most of the lines (summarized in Table 6).

Table 6. F_2-derived F_3 lines that were obtained from four selected B_1F_2 plants of cross 20M1 and parental controls that were evaluated in the second greenhouse FHB resistance trial. "R" indicates that the marker allele associated with the resistance QTL was detected whereas "r" indicates the presence of the susceptibility-associated marker allele.

	Qfhb.rwg-5A.1		***Qfhb.rwg-5A.2***			
	Barc186 80018[2]	**Barc186[3] Agarose**	**Fhb 5AL[4] 5AL-8.0K**	**Fhb 5AL[4] 5AL-8.0K**	**Gpw2136[2] 182 bp**	**Gpw2136[3] Agarose**
GP80	RR	RR	RR	RR	RR	RR
ND Noreen	rr	rr	rr	rr	rr	Rr
19CP29	rr	rr	rr	rr	rr	Rr
$B_1F_{2:3}$ 20M1(A)-12 (= CP65-3)[1]	rr	rr	Not tested	RR	Not tested	RR
$B_1F_{2:3}$ 20M1(A)-12 (= CP65-12)[1]	rr	rr	Not tested	Rr	Not tested	Rr
$B_1F_{2:3}$ 20M1(A)-23 (= CP67-6)[1]	RR	RR	Not tested	RR	Not tested	RR
$B_1F_{2:3}$ 20M1(A)-28 (= CP68-10)[1]	Rr	Rr	Not tested	RR	Not tested	RR
$B_1F_{2:3}$ 20M1(A)-58-8[1]	rr	rr	RR	RR	RR	RR
$B_1F_{2:3}$ 20M1(A)-58-10[1]	RR	RR	RR	RR	RR	RR
$B_1F_{2:3}$ 20M1(A)-58-18[1]	RR	RR	RR	RR	RR	RR
$B_1F_{2:3}$ 20M1(A)-58-27[1]	RR	RR	RR	RR	RR	RR
$B_1F_{2:3}$ 20M1(A)-58-32[1]	rr	rr	RR	RR	RR	RR

[1] Pedigree = GP80/Novus-4//19CP29/3/2* ND Noreen
[2] Automated sequencer-based amplification and detection of the SSR marker done by the Genotyping Center, Fargo, ND
[3] Laboratory-based amplification and visualization on agarose.
[4] KASP marker derived by Dr. Jason Fiedler (Genotyping Center, Fargo, ND) which is based on a SNP polymorphism at the *Qfhb.rwg-5A.2* QTL peak (Fiedler, Personal Communication). The marker test was done two times using different DNA samples.

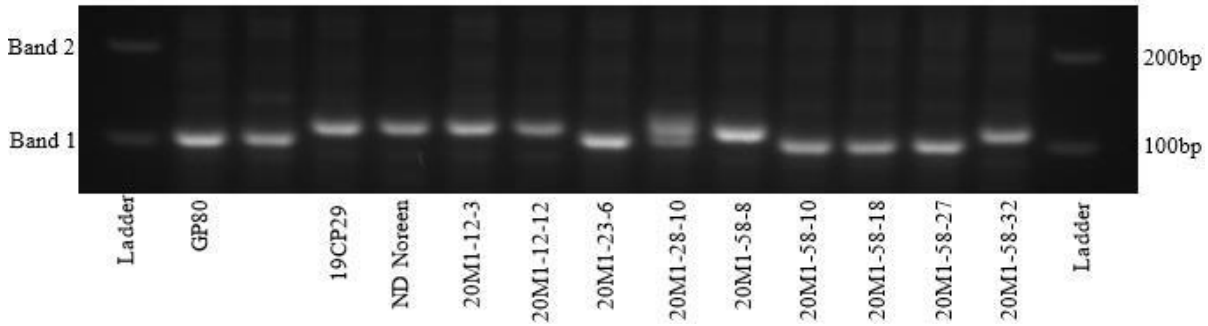


Figure 3. Agarose gel showing Barc186 marker polymorphism among parents and 20M1 B_1F_1:2 plants. The smaller band around 100 bp is associated with the Qfhb.rwg-5A.1 resistance. The lanes containing 20M1-12-3, 20M1-12-12, 20M1-23-6 (homozygous), 20M1-28-10 (heterozygous), 20M1-58-8, 20M1-58-10, 20M1-58-18, 20M1-58-27, and 20M1-58-32, (homozygous) lines are shown.

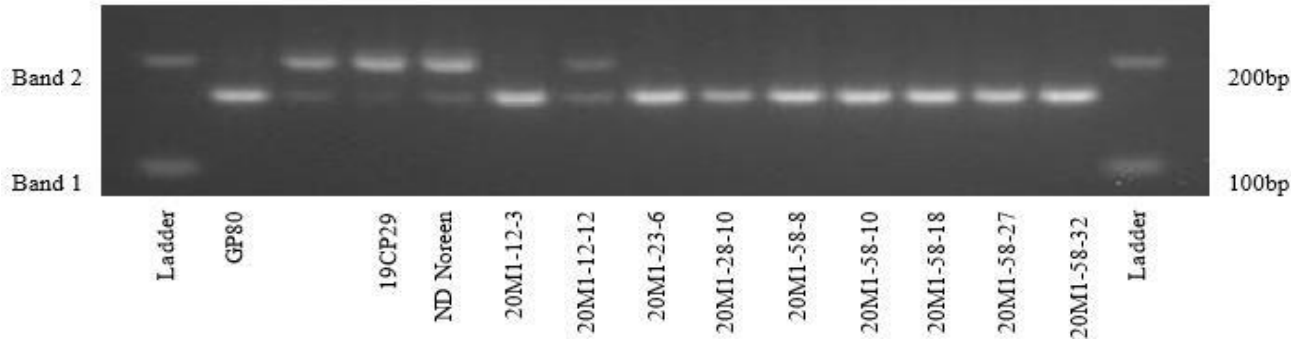
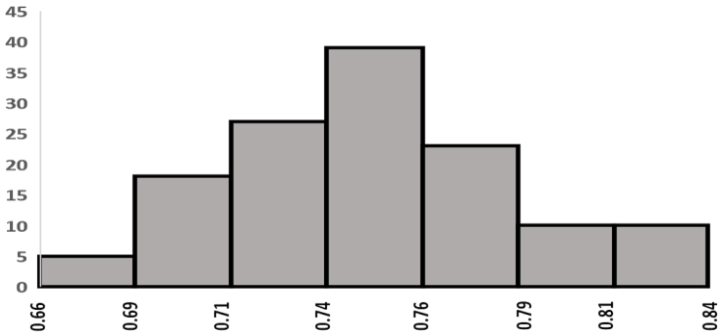


Figure 4. Agarose gel showing Gpw2136 marker polymorphisms of parents and 20M1 $B_1F_{1:2}$-plants. The smaller band 1 (around 180 bp) is associated with the *Qfhb.rwg-5A.2* resistance. The lanes containing 20M1-12-3 (homozygous), 20M1-12-12 (heterozygous), 20M1-23-6,20M1-28-10, 20M1-58-8, 20M1-58-10, 20M1-58-18, 20M1-58-27, and 20M1-58-32, (homozygous) are shown.

Background Selection

Polymorphic SNPs were selected from all the chromosomes and the total ND Noreen background recovery in all the chromosomes was calculated. The percentage of the whole-genome background of the lines varied from 66% to 83% with a mean of 75% (Fig. 5a). On an individual chromosome basis, the background recovery percentages ranged from 73% to 79% with an average of 75%. The detailed background recovery data of all the lines are shown in (Addendum Table A5). Assuming a random distribution, a normalized distribution (Fig. 5b) was derived using the mean and standard deviation of the overall genome recovery of each line.



% of background selection

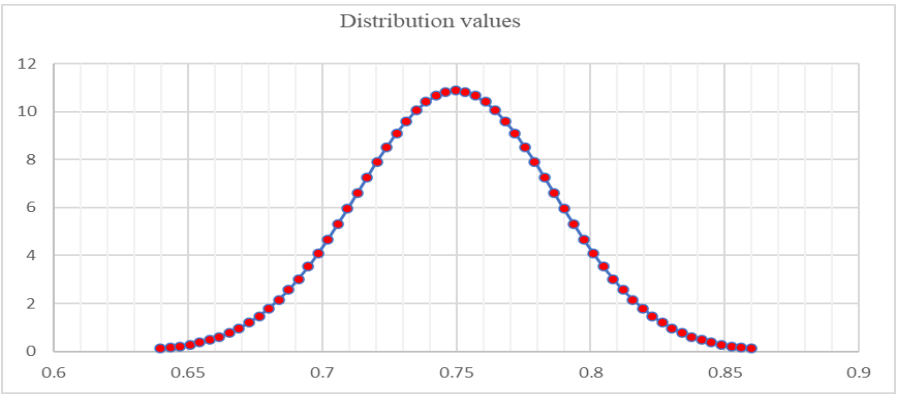


Figure 5. (a) Histogram of the actual data, and (b) a normalized distribution fitted on the data and showing the percentage of background recovery (horizontal axis) for the B_1F_1 20M1 plants used in the study.

Greenhouse FHB Resistance Trial 2: Evaluation of Selected 19M13 and 20M1 Progenies

Twelve entries (Table 6) were tested for FHB resistance in the greenhouse. An average of three spikes per test plant were inoculated with the freshly prepared inoculum. The average FHB infection severity of individual plants ranged from 9% to 86%. The detailed data are provided in Addendum (Table A9). An analysis of variance (ANOVA) was done, and its outcome is summarized in Table 7. Strong, significant differences were observed between the entries and a comparison of the entry means and significance metrics are given in Table 8.

Table 7. Analysis of variance of the parents and nine bulked lines (Table 6) that were selected from cross 20M1 based on the presence of markers associated with *Qfhb.rwg-5A.1* and *Qfhb.rwg.5A.2*.

SOV	DF	SS	MSS	F Value
Model	28	45.97576786	1.64199171	263.38**
Error	997	6.21560238	0.00623431	
Corrected Total	1025	52.19137024		

SOV	DF	SS	MSS	F Value
Replications	5	0.13490058	0.02698012	4.33
Plants (pot)	1	0.00066950	0.00066950	0.11
Entries	11	8.29509374	0.75409943	120.96**
Plants (pot)*Entries	11	0.11794745	0.01072250	1.72

SOV = source of variance, D F= degrees of freedom, SS = sum of squares, and MS = mean squares. ** represents significance at the 5% level of significance

Table 8. Average infection severities of the parents and nine introgression lines believed to carry resistance QTL from PI 277012 that was tested in the final FHB greenhouse trial

	Infection		*Qfhb.rwg-5A.1*		*Qfhb.rwg-5A.2*		ND Noreen
Entries	**Mean[1]**	**Significant differences[2]**	**Markers**	**Haplotype[2]**	**Markers**	**Haplotype**	**Background[4]**
19CP29	0.8638	A	-		-		
20M1_28-10	0.1606	B	Hetero	I	+	III	0.70
ND Noreen	0.1582	B	-		-		
GP80	0.1249	C	+		+		
20M1-58-18	0.1234	C	+	II	+	III	0.75
20M1-58-27	0.1223	CD	+	II	+	III	0.75
20M1-58-8	0.1221	CD	-	II	+	III	0.75
20M1-58-10	0.1204	CD	+	II	+	III	0.75
20M1_23-6	0.1048	CDE	+	II	+	III	0.74
20M1_12-12	0.1024	CDE	-	I	Hetero	III	0.82
20M1_12-3	0.09876	CE	-	I	+	III	0.82
20M1-58-32	0.091	E	-	II	+	III	0.75

[1] Means arranged from higher to lower order of disease severi**ty**

[2] The 5AS arm haplotypes were described in Table 4. Compared to the full haplotype I, haplotype II has a reduced presence of GP80-derived chromatin (a result of crossover).

[3] The 5AL haplotype is described in Table 5.

[4] The ND Noreen background recovery was based on the corresponding B_1F_1 genotype.

The average FHB infection of entries across replications ranged from 9% to 86% in 20M1-58-32 and 19CP29 respectively (Addendum Table A9). Among the parents, GP80 was the most resistant and 19CP29 was the most susceptible. ND Noreen was moderately resistant and is believed to have 'background' or 'native' FHB resistance. Among the selected progenies, line 20M1-58-32 had the lowest infection percentage. Each of the nine selected lines expressed the markers that are associated with *Qfhb.rwg-5A.2*. A new marker, Fhb 5AL, was developed from a SNP associated with the QTL peak and is probably the most reliable of the three marker loci. Thus, judged by the levels of resistance in the selections and the presence of the critical Fhb 5AL allele, it appears likely that *Qfhb.rwg-5A.2* had been transferred. From Tables 3 and 8 it appears that four of the nine lines did not have the critical marker allele of the *Xbarc186* locus. When the presence/absence of the critical Xbarc186 allele is correlated with the size of the haplotype (I or II); the most likely explanation of the results of Table 8 is that *Xbarc186* is located within the PI 277012 chromatin that is common to haplotypes I and II. If this is the case, then the haplotype plus marker locus must have been retained in 20M1-58-18, -27, -10, 20M1-28-10 (heterozygous), and 20M1-23-6. However, both the haplotype and *Xbarc186* are absent from segregates 20M1-58-8, -32, 20M1-12-12, and 20M1-12-3 (segregation from the F_1 heterozygote). The infection percentages of the selections vary within a narrow range and the three selections with the least symptoms appear to also lack the PI 277012 haplotype. Thus, it seems possible that the significant level of background resistance contributed by ND Noreen could mask the effect of *Qfhb.rwg-5A.1*. However, it is also possible that *Qfhb.rwg-5A.1* has been lost during transfer. The map distance between the Barc186 marker and *Qfhb.rwg-5A.1* is not clear from previous studies. Barc186 and Barc180 were earlier suggested to be a suitable flanking marker pair with which to predict the presence of *Qfhs.ifa-5A* which occurs in the same

general chromosome area as *Qfhb.rwg-5A.1*. Since Barc180 was not suitably polymorphic for use in the present study, only Barc186 could be used. In view of the above it is uncertain whether *Qfhb.rwg-5A.1* occurs in/among the nine lines or was earlier on separated from the marker through a crossover. If this has happened the gene can be acquired from the winter wheat Novus-4 to which it was transferred in an earlier study.

Conclusion

From genotyping results, two hundred and sixty SNPs were identified that were polymorphic with respect to the area on PI277012 chromosome 5A that harbors the targeted resistance genes and the same region in the recipient genotypes. Ganaparthi (2020) distinguished three regions on the GP80 chromosome 5A map, namely, regions I and III (PI277012-derived), and region II (Grandin-derived). Region I is the 5AS region believed to harbor QTL *Qfhb.rwg-5A.1* and region III is located on 5AL and is believed to harbor QTL *Qfhb.rwg-5A.2*.

In this study, the parents, backcross populations, and controls were subjected to 90K SNP genotyping. The GP80 chromosome 5A map was used as the reference map. The 5AS region (19.89 to 38.7 cM) that showed polymorphism identical to GP80 and PI299012 was regarded as the most likely location of *Qfhb.rwg.5A.1*. The 5AL polymorphic region that most likely harbored *Qfhb.rwg-5A.2.* occurred within 104.8cM to 117.6cM. Two previously identified, but not fully tested markers, Barc186 and Gwm2136 were used together with the two SNP haplotypes to select and confirm segregates during backcrossing. Finally, nine $B_1F_{2:3}$ families homozygous for one or both resistance genes were selected, and their FHB resistance compared to that of GP80 in a greenhouse trial. The data suggested that the lines had resistance similar to that of GP80. The material constitutes a valuable resource for ongoing selection and pure line

development and will also be employed in crossing blocks to introduce the resistance in a broader range of winter wheat germplasm.

References

Anderson, J.A. 2007. Marker-assisted selection for Fusarium head blight resistance in wheat. Int. J. Food Microbiol. 119: 51–53.

Anderson, J.A., R.W. Stack, S. Liu, B.L. Waldron, A.D. Fjeld, et al. 2001. DNA markers for *Fusarium* head blight resistance QTLs in two wheat populations. Theor. Appl. Genet. 102: 1164–1168.

Buerstmayr, H., B. Steiner, L. Hartl, M. Griesser, N. Angerer, D. Lengauer, T. Miedaner, B. Schneider, and M. Lemmens. 2003. Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. II. Resistance to fungal penetration and spread. Theor. Appl. Genet. 107:503-508.

Buerstmayr, H., M. Lemmens, L. Hartl, L. Doldi, B. Steiner, M. Stierschneider, and P. Ruckenbauer. 2002. Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. I. Resistance to fungal spread (Type II resistance). Theor. Appl. Genet. 104:84-91.

Buerstmayr, H., T. Ban, and J.A. Anderson. 2009. QTL mapping and marker-assisted selection for Fusarium blight resistance in wheat: A review. Plant Breed. 128: 1–26.

Buerstmayr, M., B. Steiner, and H. Buerstmayr. 2019. Breeding for Fusarium head blight resistance in wheat—Progress and challenges. Plant Breed. (November): 1–26.

Buerstmayr, M., B. Steiner, C. Wagner, P. Schwarz, K. Brugger, D. Barabaschi, A. Volante, G. Vale, L. Cattivelli, and H. Buerstmayr. 2018. High-resolution mapping of the pericentromeric region on wheat chromosome arm 5AS harbouring the Fusarium head blight resistance QTL *Qfhs.ifa-5A*. Plant Biotechnol. J. 16:1046-1056.

Cai, J., S. Wang, T. Li, G. Zhang, and G. Bai. 2016. Multiple minor QTLs are responsible for Fusarium Head Blight resistance in Chinese wheat landrace Haiyanzhong. PLoS ONE 11

Chu, C., Z. Niu, S. Zhong, S. Chao, T.L. Friesen, et al. 2011. Identification and molecular mapping of two QTLs with major effects for resistance to Fusarium head blight in wheat. Theor. Appl. Genet. 123: 1107–1119.

Ganaparthi, Venkata Rao, 2020. Transfer of FHB resistance genes into hard red winter wheat North Dakota State University. ProQuest Dissertations & Theses Global. doi:28255914.

Lemmens, M., U. Scholz, F. Berthiller, C. Dall'Asta, A. Koutnik, R. Schuhmacher, G. Adam, H. Buerstmayr, A. Mesterhazy, R. Krska, P. Ruckenbauer .2005. The ability to detoxify the mycotoxin deoxynivalenol colocalizes with a major quantitative trait locus for Fusarium head blight resistance in wheat. Mol Plant-Microbe Interact 18:1318–1324

Tao, H. 2019. Transfer of Fusarium head blight resistance to hard red winter wheat. North Dakota State University. ProQuest Dissertations & Theses Global. doi: 2305192408.

CHAPTER IV. PRE-BREEDING WITH KEY RESISTANCE AND AGROTYPE GENES TO IMPROVE HARD WINTER WHEAT GERMPLASM

Abstract

Winterkill and diseases such as Fusarium head blight (FHB), wheat rusts (leaf rust, stripe rust, and stem rust), bacterial leaf streak, tan spot, and Septoria nodorum blotch cause significant production losses in winter wheat in North Dakota. To improve the FHB resistance of winter wheat, FHB resistance QTL *Fhb1* and *Qfhs.ifa-5A.1* were previously transferred from spring wheat (CM82036). However, the newly acquired genes appeared to be associated with lower yields. Pre-breeding was therefore done with the purpose to develop semi-dwarf inbred lines that are FHB resistant, winter-hardy, high-yielding, and also have a significant leaf, stem, and stripe rust resistance. Eight cross combinations were produced utilizing eight winter wheat genotypes that exhibit one or more of the desired traits. Greenhouse-based single seed descent (SSD) inbreeding with phenotypic selection steps were used to expedite line development. F_3-derived F_4 populations were evaluated for grain yield in an un-replicated field trial. Four single plants (spikes) were selected on phenotype from each of the nine highest yielding families and threshed individually. Five seeds from each selected spike were used for marker-based selection. One hundred and forty F_5-derived inbred lines F_5 with favorable (marker-predicted) resistance gene combinations were established for continued yield testing.

Introduction

Fusarium head blight (FHB) is a serious threat to the production and use of wheat throughout the world. The most cost-effective strategy of controlling the disease is to cultivate genetically resistant cultivars (Buerstmayr et al., 2002); however, breeding for resistance is complicated by quantitative inheritance, which demands the manipulation of numerous minor

effect genes (Bai and Shaner, 1994). Wheat germplasm from Asia, Europe, and South America has been found to possess useful levels of resistance to FHB. Most of the resistance is Asian in origin, including 'Sumai3' and its derivatives (Anderson et al., 2001; Buerstmayr et al., 2003), and 'Wangshiubai' (Ma et al., 2006). Numerous Asian sources contain the major effect QTL, *Fhb1* (Liu et al., 2008), which accounted for approximately 60% of phenotypic variation in FHB resistance in the study of Waldron et al. (1999). Frontana, a Brazilian spring wheat, is another significant source of resistance to FHB (Schröder and Christiansen, 1963).

There are five types of FHB resistance: type I, which is resistance to initial infection; type II, which is resistance to spread within a spike (Schröder and Christensen, 1963); type III, which is resistance to kernel damage; type IV, which is resistance to DON accumulation (Lemmens et al., 2005); and type V, which is tolerance (Mesterházy, 1995). Anderson et al. (2001) and Buerstmayr et al. (2003) suggested that *Fhb1* confers type II resistance. Inbreeding, *Fhb1* is frequently used in combination with the complementing resistance QTL, *Qfhs.ifa-5A.* Both *Fhb1 (Qfhs.ndsu-3BS)* and *Qfhs.ifa-5A* occur in CM82036; are widely used resistance QTL and have been estimated to explain 29-60% (Buerstmayr et al., 2003; Waldron et al., 1999) and 20% (Buerstmayr et al., 2003) of the phenotypic variance for disease severity, respectively. These two QTL's have already been incorporated into the NDSU HRWW breeding program lines.

Stem rust resistant hard red winter wheat cultivars of the great plains and soft red winter wheat mostly carry one or more of *Sr6, Sr24, Sr31, Sr36, SrTmp,* and the resistance associated with the 1AL.1RS translocation. Resistant spring wheat cultivars are mainly resistant with seedling resistance genes *Sr6, Sr9b, Sr11,* and *Sr17* and adult plant resistance gene, *Sr2.* The outbreak of the stem rust race Ug99 (later designated TTKSK) created an enormous threat to U.S. wheat production. Since then different Ug99 derivative races were identified showing

virulence to various widely used stem rust resistance genes (Mcintosh et al., 1995; Singh et al., 2011).

Lr46 contributes horizontal resistance to leaf rust and it is an adult plant resistance (APR)gene. This gene does not provide the host plant with complete immunity against a set of leaf rustraces instead it can delay the infection process and act against a broad spectrum of races. It was first described in cultivar "Pavon 76" and is located on chromosome 1B (Singh et al., 1998). Thetype of resistance conferred by *Lr46* is like that of *Lr34* but with smaller effect (Martinez et al., 2001). *Lr34* is a pleiotropic, broad-spectrum, quantitatively inherited, slow-rusting gene discovered in Canada by Dyck et al. (1966) (In: Kolmer et al., 2008; Lagudah et al., 2009; Singhet al., 2011; Risk et al., 2013). *Lr34* confers a moderate level of resistance, which is most visibleon the flag leaf of adult plants during the grain filling stage. It is located on the short arm of wheat chromosome 7D and is closely related to the adult plant stripe rust resistance gene, *Yr18*, and the powdery mildew resistance gene, *Pm38* (Krattinger et al., 2009).

Lr 68 is another APR gene conferring slow rusting resistance to wheat leaf rust. The gene is located in chromosome 7BL of Parula. The likely origin of *Lr68* is the Brazilian wheat cultivar "Frontana" which also appears in the pedigree of Parula and various other CIMMYT wheats (Herrera-Foessel et al., 2012). *Lr56* is a major gene that gives all-stage leaf rust resistance and was intogressed into hexaploid wheat from *Aegilops sharonensis*. This gene occurs in wheat chromosome 6A (Marais et al., 2006). *Yr17* is classified as a seedling (all-stage) resistance gene. It occurs on a long chromosomal fragment which was translocated from *Triticum ventricosum* chromosome arm 2NS to bread wheat chromosome arm 2AS (Bariana et al., 1993). This translocation harbors three disease resistance genes: *Lr37*, *Yr17* and *Sr38* conferring resistance to leaf rust, stripe rust and stem rust, respectively. The resistance gene *Yr17* has been used by

many breeding programs to develop resistant cultivars. *Sr24* offers resistance to most races of stem rust, including the virulent race Ug99 (TTKSK). Incidents of virulence to this major resistance gene has been reported in South Africa (Mago et al., 2005) and India (Bhardwaj, 1990). *Sr24* resides on the 3DL chromosome arm of the rust- resistant hexaploid wheat, Agent (Smith et al., 1968). Stem rust resistance gene *Sr39* provides resistance to all currently known pathotypes of *Puccinia graminis* f. sp. *tritici* (*Pgt*) including Ug99 (TTKSK) and its variants TTKST and TTTSK. The latter races are respectively virulent on two frequently deployed resistance genes *Sr24* and *Sr36*. *Sr39* was transferred to the hexaploid wheat cultivar "Marquis" from *T. speltoides* (Kerber and Dyke, 1990). The gene is located on a translocated segment of *T. speltoides* chromosome 2S to wheat chromosome 2B.

This study aimed to produce new, diverse inbred lines that are high in yield and possess *Fhb1* in association with combinations of additional and agronomically useful resistance genes.This will help increase the available FHB resistance in the breeding population and help to fullyintegrate it with resistance to other prevailing diseases such as the leaf, stem and stripe rust, bacterial leaf streak, tan spot, and Septoria nodorum blotch.

Material and Methods

Plant Material and General Outline

A detailed description of eight winter wheat lines/cultivars that were used as parents is provided in Table 10. Eight crosses (Table 10) were made among the parents and the F_1 were planted for seed increase. The F_2 were used in the winter of 2019/20 to initiate single seed descent inbreeding and selection as outlined in Fig. 7. The F_2 was grown in plastic trays (four seeds planted in each of 24 cups per tray). The 192 F_2 plants per cross were vernalized and the seedlings were infected with mixed leaf rust and stem rust spores. The most severely infected

seedlings were removed, and the remaining plants were raised to maturity when plants that were too tall were also discarded. Two F_3 seeds per selection were replanted (February 2020) in big pots to safeguard against drying out in the warmer summer greenhouse. Three lineages (two seeds per lineage) were planted in each pot. F_3 plants were selected based on height and productivity.

Unreplicated Check Plot Yield Trial

In September 2020, 100 of the best $F_{3:4}$ lines representing all eight crosses (Table 10) were selected in the greenhouse based on phenotype, height, and fertility and planted in the field as an unreplicated check plot yield trial. The seeds were planted into soy stubble on 20th September 2020 at the NDSU research farm at Casselton. Plots were planted at a density of 1 to 1.2 million seeds per acre.

The planted area covered12.5 ft x 257 ft (3.81 m x 78.33 m) and consisted of twenty-five 12.5ft x 5 ft (3.81m x1.52 m) blocks. Each block consisted of six two-row plots. The plot length was 5 ft (1.52 m). A single check variety (Ideal) was planted in the outer (border) plots (Fig. 6). Nitrogenous fertilizer (Urea) was applied by the seed farm at a rate of 260 lbs urea/acre. The herbicide Wolverine advanced (Bayer Crop Science) was applied at 1.7 pints/acre on the 26th of May 2021to control weeds. Grasshoppers became problematic later in the season and were aerially sprayed with Lambda Cy at the rate of 3.2 oz/ acre on the 14th of June 2021. The plots were evaluated for winter survival, agronomic performance, and disease resistance and were harvested in total.

Table 9. Hard red winter wheat parents used for initiating the study.

Parent	Traits[1,2]	Pedigree	Resistance genes[3]
1	T; CH	CM82036/Jerry//Jerry-*Lr56*	*Lr34*; *Lr56*; 1B1R
2	SD; CH; W	Broadview/SD07W083-4	*Fhb1*; *Qfhs.ifa-5A; Lr34*; *Lr46*; *Yr17*; *tsn1*
3	TSD; NH	Radiant/RCATL33//Ideal	*Sr24*; unknown FHB resistance
4	T; CH	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo	*Fhb1; Lr46*; 1B1R; *Yr17*
5	T; CH	Norstar-*Fhb1*, *Sr39*	*Fhb1*; *Sr39/Lr35*; *Lr34*; *Lr46*; *Lr68*; 1B.1R
6	SD; MH	Monument	*Lr34*; *Sr24*; *Yr17*
7	SSD; MH	Keldin	
8	TSD; NH	CM82036/Jerry/4/*Lr50*/Jerry//Falcon/3/Moats	*Fhb1*; *Qfhs.ifa-5A; Lr46*; *Yr17*

[1] T = tall, SD= semi-dwarf; TSD = tall semi-dwarf; SSD = short semi-dwarf; CH = cold-hardy, MH = moderately cold-hardy, NH = non-cold-hardy; W = white seed.
[2] Parents 3, 6, and 7 have inadequate bacterial leaf streak resistance.
[3] *Lr* = leaf rust resistance locus, *Sr* = stem rust resistance locus; *Yr* = stripe rust resistance locus; *Fhb* = FHB resistance QTL; *Qfhs.ifa-5A* = FHB resistance QTL; 1B.1R = wheat rye translocation; *tsn1* = tan spot insensitivity allele.

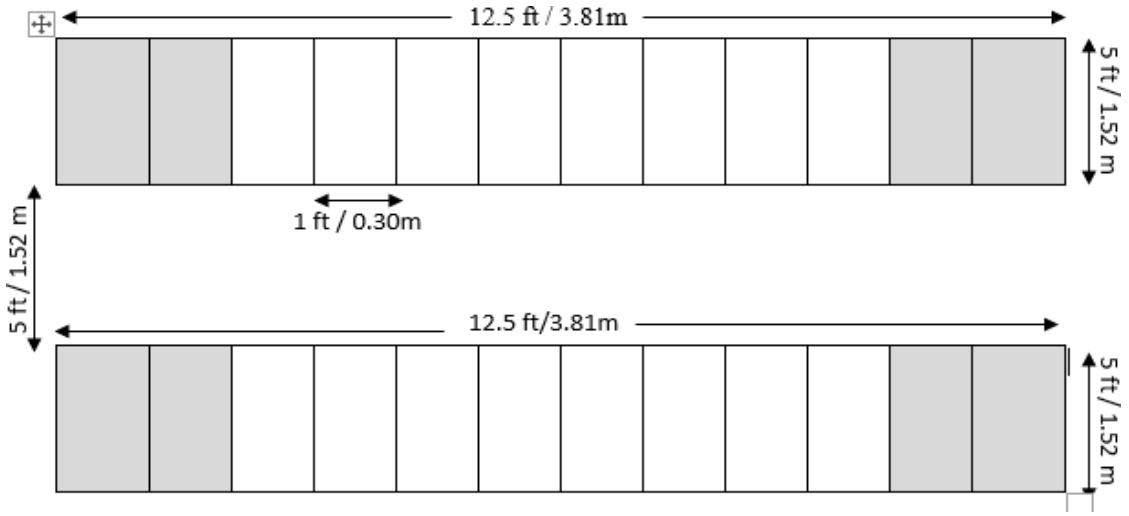


Figure 6. Layout of the check-plot field trial at Casselton, 2020. There were 25 blocks (with the two outmost blocks shown here). Each block consisted of twelve 5 ft rows where two adjacent rows constituted a plot. The grey shaded plots show the positions of the check plots. Check plots were planted to the variety Ideal.

Table 10. Crosses made among eight winter wheat parents

Cross	Parents	Pedigree
19K331	1 X 2	CM82036/Jerry//Jerry-*Lr56*/3/ Broadview/SD07W083-4
19K438	2 X 3	Broadview/SD07W083-4/3/Radiant/RCATL33//Ideal
19K89	4 X 2	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Broadview/SD07W083-4
19K365	5 X 6	Norstar-*Fhb1, Sr39*/Monument
19K94	4 X 6	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Monument
19K368	5 X 7	Norstar-*Fhb1, Sr39*/Keldin
19K97	4 X 7	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Keldin
19K132	4 X 8	Norstar*Fhb1*/Jerry//TX09D1119/Buteo/5/CM82036/Jerry/4/*Lr50*/Jerry//Falcon/3/Moats

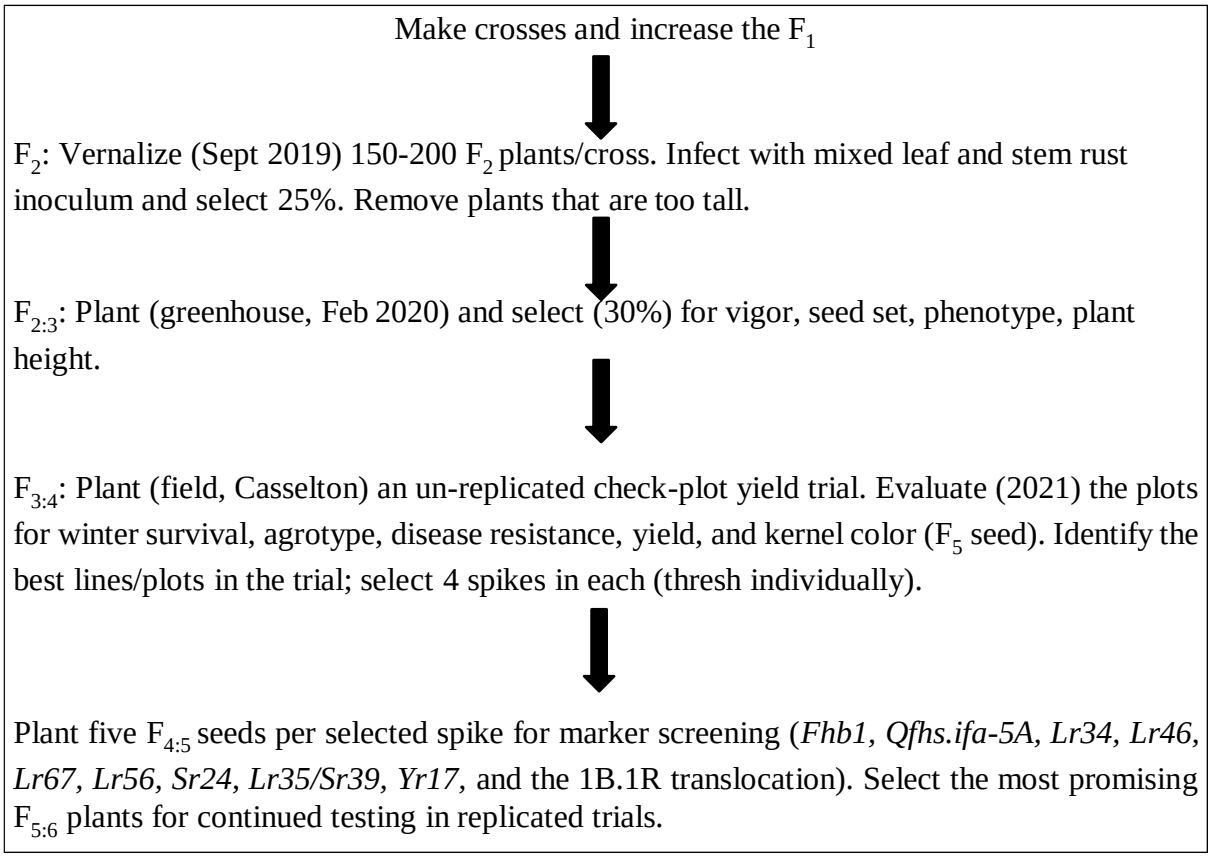


Figure 7. Outline of the inbreeding and selection scheme that was employed.

Phenotypic Measurements and Selection

In the early spring (2021), plots were evaluated for plant stand, both visually and by using drone pictures. Plant stand in the spring was influenced both by plant establishment in the fall and winter survival. A rating scale (1 to 10) was used for evaluating plant stand with 1 =

poorest (10%) stand and 10 = best (100%) stand. Generally, a survival score ≥ 9 is considered good. Due to the extremely dry summer of 2021, no significant natural disease infections occurred that could be scored in the field. During ripening, five single spikes (from different plants) were selected in each plot and kept for possible use during the final marker selection step. Spike selection was based on plant height (semi-dwarf plants were favored), health and phenotype as well as spike health, fertility, and productivity. All the plots were harvested to determine seed yield (grams/plot) and measure test weight (lbs/bu).

Data Analysis

The data of the replicated control plots for plant stand, grain yield, and test weight were used to assign a theoretical control value to each of the test plots. For this purpose, the data of the 4-6 closest check plots were weighted based on their respective distances from the test plot (these distances were measured from the midpoint of each plot). Control plots closest to the test plot were weighted higher. The actual (measured) performance of each test plot was then expressed as a percentage of the estimated performance of the control in that plot to also obtain an adjusted test plot performance. These calculations were done in MS Excel.

Marker Analyses

Following the data analyses and consideration of all the phenotypic data, the nine most promising families were identified. About five $F_{4:5}$ seeds per each of four selected spikes were planted in the greenhouse for marker screening (*Fhb1, Qfhs.ifa-5A, Lr34, Lr46, Lr67, Lr56, Sr24, Lr35/Sr39, Yr17,* and the 1B.1R translocation).

The SSR marker analyses were done by the USDA-ARS, North Central Small Grains Genotyping Laboratory at NDSU, Fargo following their in-house protocols. Seedling leaves from the selected plants were cut and DNA was extracted by using robotic equipment

(https://wheat.pw.usda.gov/GenotypingLabs/fargo). The *Fhb1*-TaHRC-KASP marker (Su. et al., 2018) was used for the detection of *Fhb1*. SSR marker *Xwmc44* was used for the detection of *Lr46* (https://maswheat.ucdavis.edu/protocols/*Lr46*). A *Lr34* marker (Krattinger et al., 2009) was used for the detection of *Lr34*. A CIMMYT designed CAPS marker (cs7BLNLRR) (https://maswheat.ucdavis.edu/protocols/Lr68) was used for the detection of *Lr68*. *Yr17* was detected using a marker described by Helguera et al. (2003). SSR marker *Xbarc71* was used for detecting *Sr24* (https://maswheat.ucdavis.edu/protocols/Sr24).

Results and Discussion

Unreplicated Check Plot Yield Trial

In the summer, data were recorded for agronomic (yield, test weight) and phenotypic measurements (plant height, plot stand) of the trial. Weighted control plot data (based on their respective distances from the test plots) were used for the adjustment of the test plot data. This adjustment was done in an attempt to equal out soil/environmental variability and allow for more accurate comparison among test plots. The complete trial data pertaining to all entries and measurements of the un-replicated yield trial are given in Addendum Table A7. The data ranges and averages recorded in this trial are summarized in Table 12. Nine entries were selected from the trial based on their yield, test weight and plot stand data and their data only are summarized in (Table 12). The average yield for all the entries except the checks ranged from 542.2 to 1080.2 g whereas the test weights ranged from 56.5 to 62.6 lbs/bu. The check variety Ideal performed very well in the trial. For Ideal, plot stand ranged from 7 to 9.5 with an average of 8.57. For all the other (test plot) entries plot stand was similar to Ideal (6-10; average = 8.38). The average yield, test weight, winter survival, and height of all the entries except checks were 840.87 gram,

60.55 lbs/bu, 8.35 and 27.5 cm and that of ideal were 1026.44 gm, 62.41 lbs/bu, 8.57, and 25.76 cm, respectively.

Table 11. Agronomic and phenotypic data pertaining to nine entries that were selected in an un-replicated yield trial (field).

Entry	Row numbers		Actual test plot yield (g)	Predicted check (Ideal) yield (g)	Relative test plot yield (%)	Test weight (lbs/bu)	Plot stand (0-10)	Plant height (inches)
	1st row	2nd row						
19K89-1	8003	8004	652.6	683.47	95	61	10	24
19K89-3	8007	8008	976	810.38	120	61.2	9	26
19K89-6	8017	8018	826	853.55	97	59.7	9	26
19K194-6	8055	8056	1022	1047.56	98	59.6	8	31
19K132-1	8149	8150	1080.2	1106.06	98	61.1	8	30
19K365-4	8231	8232	969.8	1005.48	96	61.3	9	30
19K368-8	8261	8262	1007.8	966.21	104	60.9	9.5	33
19K438-9	8291	8292	870.6	855.65	102	62.1	8	34
19K438-12	8297	8298	939.6	963.77	97	60.2	8	29

Table 12. Summary of the observed range and mean values of all the lines, selected lines, and checks for traits measured in the field trial at Casselton (2020).

	Plot stand (1 to 10)[1]	Plant height (inches)	Yield (g)	Test weight (lbs/bu)
Range for lines	6 to 10	17 to 34	542.2 to 1080.2	56.5 to 62.6
Average for lines	8.38	27.6	829.03	60.61
Range for selected lines	8 to 10	24 to 34	652.6 to 1080.2	59.7 to 62.1
Average for selected lines	8.81	29	915.32	60.93
Range for lines not selected	6 to 10	17 to 33	542.2 to 1062.8	56.5 to 62.6
Average for lines not selected	8.24	27.5	821.19	60.5
Range for check (Ideal)	7 to 9.5	22 to 30	583.6 to 1254.4	60.2 to 63.6
Average for check (Ideal)	8.57	25.8	1026.44	62.4

[1] Plot stand was evaluated on a 1 (worst) -10 (best) scale in the spring

Marker Screening

Nine entries were selected based on the agronomic and phenotypic trial data and approximately 20 seeds of each selection were tested for the presence of the markers. The

complete marker data of all plants are provided in (Addendum Table A8). Positive marker results were obtained regarding *Fhb1, Lr34, Lr46, Lr68, Yr17* and the 1BL.1RS translocation. The results showed that the *Lr34* marker detected heterozygotes in two families; whereas the *Lr46* and 1BL.1RS markers detected heterozygotes in three families each (Addendum Table A8). *Lr56* occurred in only one parent and was expected to segregate in cross 19K94; however, it was not detected in any of the four F_4-derived families of this cross and was likely lost by chance. The alleles of the *Qfhs.ifa-5A* markers *Xbarc180* and *Xbarc186* were not suitably polymorphic in the parental lines and were not useful for predicting the presence of *Qfhs.ifa-5A* in crosses 19K89, 19K132, 19K331 and 19K438 in which it was expected to segregate. Thus, while this locus was not detected due to inadequacy of the markers used, it may be present in the progeny of the latter four crosses. The *Sr24* and *Sr39* markers did not detect these two genes among the selected progenies.

The marker data were used to select a group of 143 promising F_5 plants with which to establish inbred lines for continued testing. Two of the nine originally selected (yield) lines completely lacked *Fhb1,* whereas the remaining seven lines showed regular presence of *Fhb1*. As a result, lines 19K89-6 and 19K438-12 were discarded. The data relevant to the selected F_5 plants in the remaining seven families (representing six of the original eight crosses) are summarized in Table 14. There also was residual segregation within some of the families: 19K89-3 (*Lr34*; 2 plants), 19K94-6 (*Lr34*, 1B.1R; 5 plants), 19K365-4 (1B.1R; 7 plants), 19K368-8 (*Lr34, Lr46*; 8 plants), and 19K438-9 (*Lr46;* 4 plants). The predicted frequencies of the rust resistance genes varied within families: *Lr34* (0.29-1.0), *Lr46* (0.04-1.0), 1B.1R translocation (0.2-0.6).

Table 13. Summary of lines selected for inbred line development

Entry	Pedigree	Number of plants	Markers detected
19K89-1	Norstar*Fhb1*/Jerry//TX09D1119/Buteo/3/Broadview/SD07W083-4	20	*Fhb1, Lr34, Lr46, Yr17*
19K89-3	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Broadview/SD07W083-4	9	*Fhb1, Lr46, Yr17*
19K89-3	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Broadview/SD07W083-4	11	*Fhb1, Lr34, Lr46, Yr17*
19K94-6	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Monument	8	*Fhb1, Yr17*
19K94-6	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Monument	3	*Fhb1, Lr46, 1B1R, Yr17*
19K94-6	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Monument	9	*Fhb1, 1B1R, Yr17*
19K132-1	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/6/CM82036/ Jerry/3/*Lr50*/Sup//Jerry/4/Falcon/5/Moats	16	*Fhb1, Lr46, Yr17*
19K132-1	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/6/CM82036/ Jerry/3/*Lr50*/Sup//Jerry/4/Falcon/5/Moats	4	*Fhb1, Lr46, 1B1R, Yr17*
19K365-4	Norstar-*Fhb1*, *Sr39*//Monument	13	*Fhb1, Lr34, Lr68*
19K365-4	Norstar-*Fhb1*, *Sr39*//Monument	5	*Fhb1, Lr34, Lr68, 1B1R*
19K368-8	Norstar-*Fhb1*, *Sr39*//Keldin	11	*Fhb1, Lr68*
19K368-8	Norstar-*Fhb1*, *Sr39*//Keldin	2	*Fhb1, Lr34, Lr46, Lr68*
19K368-8	Norstar-*Fhb1*, *Sr39*//Keldin	11	*Fhb1, Lr34, Lr68*
19K438-9	Broadview/SD07W083-4 /3/Radiant/RCATL33//Ideal	20	*Fhb1, Lr34, Yr17*
19K438-9	Broadview/SD07W083-4 /3/Radiant/RCATL33//Ideal	1	*Fhb1, Lr34, Lr46, Yr17*

Table 14. Summary of marker screening results for the selected F_5 lines

	Frequency of selected F_5 plants with critical markers[1]						
Entry	**Number**	***Fhb1***	***Lr34***	***Lr46***	***Yr17***	***Lr68***	**1B.1R**
19K89-1	20	1.0	1.0	1.0	1.0	0	0
19K89-3	20	1.0	0.55	1.0	1.0	0	0
19K94-6	20	1.0	0.0	0.15	1.0	0	0.6
19K132-1	20	1.0	0	1.0	1.0	0	0.2
19K365-4	18	1.0	1.0	0	0	1.0	0.27
19K368-8	24	1.0	0.29	0.58	0	1.0	0
19K438-9	21	1.0	1.0	0.04	1.0	0	0

Conclusion

Most of the winter wheat planted in North Dakota is grown in the southwestern and northwestern regions of the state (USDA-NASS, 2019). In 2018, the average yields were 2500 kg/ha (northwest) and 2720 kg/ha (southwest), respectively. Spring wheat typically has a higher protein content than winter wheat (Ransom, 2019a, 2019b). According to the NDSU extension department the average protein content of winter wheat varieties in North Dakota is around 13.5%. The low protein content of winter wheat contributes to its low market price making it a less attractive option to producers. Other problems of winter wheat cultivars in ND include lack of effective FHB resistance, susceptibility to diseases such as the cereal rusts and bacterial leaf streak and extreme winter conditions which can kill most of the plants if the cultivar lacks cold survival genes. Very limited genetic variation for FHB resistance occurs in winter wheat germplasm suitable for cultivation in ND. Significant levels of FHB resistance are available in spring wheat and can be transferred to winter wheat; however, this inevitably leads to decreased winter hardiness and low yield (Buerstmayr, 2009).

The main goal of this pre-breeding study was to develop semi-dwarf inbred lines that are high-yielding, winter-hardy and have FHB resistance and significant leaf, stem, and stripe rust resistance. Towards this end, eight parental winter wheat varieties and lines that each possess a

diverse set of useful genes for disease resistance and agrotype were used to produce eight crosses. Eight cross combinations were chosen that each involved at least one parent with *Fhb1,* and selection was aimed at establishing new inbred lines, each of which would have *Fhb1*. The crosses also permitted pyramiding of different subsets of useful genes in addition to *Fhb1*. Few reliable and specific disease resistance markers are available in wheat, and it is highly likely that additional rust, bacterial leaf streak and tan spot resistance genes were present among the parents. Continued testing of the lines should reveal the presence of such genes if they were retained by chance among the inbred lines. An important aspect of the study was to develop lines with comparable yield to the control, Ideal, yet having *Fhb1*. No selection was done for the presence of *Fhb1* prior to the point when the highest yielding derivatives from each cross had been selected, thus, it appeared likely that high yielding lines with this gene can be bred.

The selected lines will now continue to be evaluated in advanced yield trials to determine their suitability for varietal release. The material will also be valuable for the ongoing improvement of the winter wheat breeding population.

References

Anderson, J.A., R.W. Stack, S. Liu, B.L. Waldron, A.D. Fjeld, C. Coyne, B. Moreno-Sevilla, J.M. Fetch, Q.J. Song, P.B. Cregan, and R.C. Frohberg. 2001. DNA markers for Fusarium head blight resistance QTLs in two wheat populations. Theor. Appl. Genet. 102:1164-1168.

Bai, G., and G. Shaner. 2004. Management and resistance in wheat and barley to Fusarium head blight. Annu. Rev. Phytopathol. 42:135-161.

Bariana H.S., and R. A. McIntosh. 1993. Cytogenetic studies in wheat. XV. Location of rust resistance genes in VPM1 and their genetic linkage with other disease resistance genes in chromosome 2A. Genome. 36:476-82.

Bhardwaj S.C., S.K. Nyar, M. Prashar, J. Kumar, M. K. Menon, and S.B. Singh. 1990. A pathotype of *Puccinia graminis* f. sp. *tritici* on *Sr24* in India. Cereal Rusts and Powdery Mildews Bulletin. 18:35-38.

Buerstmayr, H., B. Steiner, L. Hartl, M. Griesser, N. Angerer, D. Lengauer, T. Miedaner, B. Schneider, and M. Lemmens. 2003. Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. II. Resistance to fungal penetration and spread. Theor. Appl. Genet. 107:503-508.

Buerstmayr, H., M. Lemmens, L. Hartl, L. Doldi, B. Steiner, M. Stierschneider, and P. Ruckenbauer. 2002. Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. I. Resistance to fungal spread (Type II resistance). Theor. Appl. Genet. 104:84-91.

Dyck, P.L., D.J. Samborsk, and R.G. Anderson. 1966. Inheritance of adult plant leaf rust resistance derived from common wheat varieties 'Exchange' and 'Frontana'. Can. J. Genet. Cytol. 8: 665–671.

Herrera-Foessel S.A., R.P. Singh, J. Huerta-Espino, G.M. Rosewarne, S.K. Periyannan, L. Viccar, V. Calvo-Salazar, C. Lan, and E.S. Lagudah. 2012. *Lr68***:** a new gene conferring slow rusting resistance to leaf rust in wheat. Theoretical and Applied Genetics, 124:1475-1486.

Kerber E.R., and P. L. Dyke. 1990. Transfer to hexaploid wheat of linked genes for adult-plant leaf rust and seedling stem rust resistance from amphiploid of *Aegilops speltoides* **x** *Triticum monococcum***.** Genome. 33:530-537.

Kolmer, J.A., R.P. Singh, D.F. Garvin, L. Viccars, H.M. William, J. Huerta-Espino, et al. 2008. Analysis of the rust resistance region in wheat germplasm. Crop Sci. 48: 1841-1852.

Krattinger, S.G., E.S. Lagudah, W. Spielmeyer, R.P. Singh, J. Huerta-Espino, H. McFadden, et al. 2009. A putative ABC transporter confers durable resistance to multiple fungal pathogens in wheat. Science 323:1360-1363.

Liu, S., M.O. Pumphrey, B.S. Gill, H.N. Trick, J.X. Zhang, J. Dolezel, B. Chalhoub, and J.A. Anderson. 2008. Toward positional cloning of *FHB1*, a major QTL four Fusarium head blight resistance in wheat. Cer. Res. Comm. 36:195-201.

Mago R., H.S. Bariana, I.S. Dundas, W. Spielmeyer, G.J. Lawrence, A.J. Pryor, and J.G. Ellis. 2005. Development of PCR markers for the selection of wheat stem rust resistance genes *Sr24* and *Sr26* in diverse wheat germplasm. Theoretical and Applied Genetics. 111:496-504.

Marais, G.F., B. McCallum, and A. Marais. 2006. Leaf rust and stripe rust resistance genes derived from *Aegilops sharonensis*. Euphytica 149: 373–380

Martínez F, R.E. Niks, R.P. Singh, and D. Rubiales. 2001. Characterization of *Lr46*, a gene conferring partial resistance to wheat leaf rust. Hereditas. 135:111-114.

Mesterházy, A. 1995. Types and components of resistance to Fusarium head blight of wheat. Plant Breeding. 114: 377-386.

Ransom, J. A. Green, S. Simsek, A. Friskop, M. Breiland, T. Friesen, M. Ostlie. 2019b. North Dakota hard red spring wheat-Variety trial results for 2019 and selection guide. NDSU Extension Publication. A574-19.

Ransom, J., F. Marais, S. Simsek, A. Friskop, E. Eriksmoen, J. Rickertsen, G. Pradhan. 2018. North Dakota hard winter wheat-Variety trial results for 2018 and selection guide. NDSU Extension Publication. A1196-18.

Ransom, J., F. Marias, S. Simsek, A. Friskop, E. Eriksmoen, B. Hanson, G. Pradhan. 2019a. North Dakota hard red winter wheat-Variety trial results for 2019 and selection guide. NDSU Extension Publication. A1196-19.

Risk, J.M., L.L. Selter, H. Chauhan, S.G. Krattinger, J. Kumlehn, et al. 2013. The wheat *Lr34* gene provides resistance against multiple fungal pathogens in barley. Plant Biotech. J. 11: 847–854.

Singh, R.P., A. Mujeeb-Kazi, and J. Huerta-Espino. 1998. *Lr46*: A Gene Conferring Slow-Rusting Resistance to Leaf Rust in Wheat. Phytopathology. 88:890-894.

Singh, R.P., D.P. Hodson, J. Huerta-Espino, Y. Jin, S. Bhavani, P. Njau, et al. 2011. The emergence of Ug99 races of the stem rust fungus is a threat to world. Annu. Rev. Phytopathol. 49:465-81.

Smith E.L., A.M. Schlehuber, H. C. Young Jr, and L.H. Edwards. 1968. Registration of agent wheat. Crop Sci. 8:511-512.

Su, Z., S. Jin, and D. Zhang, D. 2018. Development and validation of diagnostic markers for *Fhb1* region, a major QTL for Fusarium head blight resistance in wheat. 2018 *Theor Appl Genet* 131:2371–2380

Waldron, B.L., B. Moreno-Sevilla, J.A. Anderson, R.W. Stack, and R.C. Frohberg. 1999. RFLP Mapping of QTL for Fusarium Head Blight Resistance in Wheat. Crop Sci. 39:805-811.

APPENDIX

Table A1. Polymorphic chromosome 5A SNP loci that were used in the study and their map locations.

Cluster	Map distance(cM)	No. of markers	Markers
1	15.5 - 15.6	19	78172, 79362, 73529, 1332, 56717, 8259, 11086, 36601, 58164, 73530, 76484, 76692, 80018, 80166, 80633, 80732, 81284, 4738, 8225
2	15.6	3	8460, 8704, 8709
3	15.6	5	9264, 50844, 50979, 64755, 64757
4	15.6 - 15.8	3	66960, 9305, 9504
5	16.6	1	64756
6	19.9	1	8649
7	19.9	1	27297
8	19.9	3	33441, 52121, 66040
9	23.5	1	28466
10	25.2 - 25.6	3	7371, 7817, 80733
11	26.5	11	9821, 11400, 34969, 39861, 56402, 70992, 77596, 79257, 80757, 8504, 79912
12	35.3 - 35.9	4	78836, 71078, 76028, 78834
13	36.5- 36.8	5	12084, 48151, 48152, 76999, 79581
14	38.7	1	43006
15	38.7 - 38.9	4	6775, 46908, 14839, 32340
16	39.0 - 39.2	3	9955, 23381, 11167
17	39.6	2	77864, 80508
18	40.0	1	63884
19	42.0	1	81308
20	42.8	1	51581
21	42.9	2	77797, 78786
22	43.3	8	7316, 8266, 78149, 81037, 54468, 75108, 78583, 3903
23	43.3	2	4800, 4826
24	43.3	2	6696, 11025
25	43.3	1	12787
26	43.2 - 43.4	9	79332, 80855, 7718, 12010, 49259, 8437, 76153, 77525, 79428
27	45.0- 45.7	15	80907, 45248, 787, 9138, 9139,9723, 53640, 65855, 72022, 74436, 13508, 38790, 51874, 71584, 7967
28	46.0 - 46.07	6	66579, 2016, 61629, 34498, 65499, 80563
29	48.0	1	1040
30	49.02 - 49.2	4	35587, 45593, 71919, 44603
31	49.4	1	25919
32	49.4	1	79038
33	49.7 - 49.73	6	10665, 54141, 73508, 10054, 10858, 10938
34	50.3 - 50.5	2	43493, 10855
35	51.1	1	10795
36	51.4	1	77393
37	53.2 - 53.25	3	45170, 38892, 63860
38	53.5	20	4112, 44479, 66908, 79915, 218, 388, 3506, 3647, 6959, 7125, 7558, 7653, 7654, 10998, 11110, 12016, 23355, 35711, 35903, 36452
39	53.5	1	39228
40	53.5	7	43034, 43912, 45690, 46440, 54774, 59706, 71628

Table A1. Polymorphic chromosome 5A SNP loci that were used in the study and their map locations (continued).

Cluster	Map distance(cM)	No. of markers	Markers
41	53.5	1	75833
42	53.5	9	76850, 77387, 78594, 79283, 79916, 80120, 81283, 8651, 73141
43	56.5	1	12306
44	57.0- 57.9	3	39499, 4229, 78720
45	59.3	1	53912
46	62.7	3	80142, 37150, 52493
47	70.3	1	72977
48	76.8	1	27298
49	81.1 - 81.9	4	75341, 72387, 6184, 10965
50	84.1 - 84.5	5	64483, 73552, 62170, 31180, 79725
51	86.2	1	26844
52	86.3 - 86.3	1	73200
53	88.0	1	25393
54	89.9 - 89.9	2	78793, 62360
55	90.5	1	48364
56	91.3	3	61908, 47327, 79251
57	93.2	1	78872
58	94.5	1	78536
59	98.4	3	80494, 7241, 80493
60	104.9	1	76979
61	105.3 - 105.9	2	12226, 48788
62	109.4	1	61152
63	111.2	1	15035
64	113.1	1	58284
65	113.1	2	33039, 80888
66	114.5 - 114.9	3	7052, 11676, 66080
67	115.2 - 115.8	2	59300, 27941
68	116.1	1	7528
69	117.7	1	10029
70	117.7	1	11245
71	119.8	1	50494
72	119.9	1	55699
73	120.1- 120.4	3	9669, 41874, 77376
74	122.7	1	10313
75	124.6	1	7014
76	125.1 - 125.6	7	5368, 47624, 56756, 77333, 77334, 77335, 80828
77	127.6	1	5178
78	130.9	1	8070
79	130.9	5	13571, 38700, 77714, 75795, 7730
80	137.8- 137.9	2	12333, 9855
81	139.8	1	24813
82	139.8	3	26751, 59605, 80843
83	141.7	2	9800, 66038
84	141.7	1	73634
85	141.7	1	6768
86	147.3	1	5731
87	148.3	3	23682, 11328, 36199

Table A2. Summary of polymorphic chromosome 5A SNP data obtained with respect to the parents, F_1, and F_2 (CP46-C) populations that were studied. The PI 277012-derived chromosome regions are indicated in dark grey. The light gray map regions derive from non-PI 277012 sources.

Marker cluster[a]	Map Distance (cM)	GP80 map[b]	Novus-4 map[c]	GP80	Novus-4	19CP-29	18M6(F_1)	ND Noreen	F_2 CP46C-6	F_2 CP46C-9
1	15.5 - 15.6			BB	BB	AA	AB	AA	BB	BB
2	15.6			AA	BB	BB	AB	BB	BB	BB
3	15.6			AA	AA	BB	AB	BB	AA	AA
4	15.6 - 15.8			AA	BB	BB	AB	BB	BB	BB
5	16.6			AA	AA	BB	AB	BB	AA	AA
6	19.8			AA	AA	BB	AB	BB	AA	AA
7	19.89			BB	AA	AA	AA	BB	AA	AA
8	19.89			AA	AA	BB	AB	BB	AA	AA
9	23.4			BB	BB	AA	AB	AA	AB	BB
10	25.2 - 25.6			AA	AA	BB	AB	AA	AA	AA
11	26.5			BB	BB	AA	AB	AA	BB	BB
12	35.3 - 35.9			BB	BB	AA	AB	AA	AB	BB
13	36.5 - 36.8			AA	AA	BB	AB	BB	AB	AA
14	38.7			AA	AA	BB	AB	BB	AA	AA
15	38.7 - 38.9			AA	BB	BB	AB	BB	BB	BB
16	39.0 - 39.2			BB	AA	AA	AB	AA	AA	AA
17	39.6			BB	BB	AA	AB	BB	BB	BB
18	39.9			BB	AA	AA	AB	AA	AA	AA
19	42.0			AA	BB	BB	BB	BB	BB	BB
20	42.7			AA	AA	BB	AB	BB	AA	AA
21	42.8			BB	AA	BB	AB	BB	AA	AA
22	43.26			BB	BB	AA	AB	AA	BB	BB
23	43.26			BB	AA	BB	AB	AA	AA	AA
24	43.26			BB	BB	AA	AB	AA	BB	BB
25	43.26			BB	AA	AA	AA	AA	AA	AA
26	43.2 - 43.4			BB	BB	AA	AB	AA	BB	BB
27	45.08 - 45.7			BB	BB	AA	AB	BB	BB	BB
28	46.05- 46.07			AA	AA	BB	AB	BB	AA	AA
29	47.9			BB	AA	AA	AA	AA	AA	AA
30	49.02- 49.2			BB	AA	AA	AA	AA	AA	AA
31	49.38			BB	AA	AA	AB	BB	AA	AA
32	49.38			AA	BB	BB	BB	AA	BB	BB
33	49.7 - 49.73			BB	BB	AA	AB	BB	BB	BB
34	50.3 - 50.5			AA	AA	BB	AB	AA	AA	AA
35	51.1			BB	AA	BB	AB	AA	AA	AA
36	51.4			AA	AA	BB	AB	AA	AA	AA
37	53.2 - 53.25			BB	AA	BB	AB	BB	AA	AA
38	53.4			BB	BB	AA	AB	AA	BB	BB
39	53.4			BB	AA	BB	AB	BB	AA	AA
40	53.4			AA	AA	BB	AB	BB	AA	AA
41	53.4			BB	AA	BB	AB	BB	AA	AA
42	53.4			AA	AA	BB	AB	BB	AA	AA
43	56.4			AA	AA	BB	AB	BB	AA	AA
44	57.08 - 57.92			BB	AA	AA	AA	AA	AA	AA
45	59.2			AA	BB	AA	AB	BB	BB	BB
46	62.7			BB	AA	BB	AB	AA	AA	AA

Table A2. Summary of polymorphic chromosome 5A SNP data obtained with respect to the parents, F_1, and F_2 (CP46-C) populations that were studied (continued). The PI 277012-derived chromosome regions are indicated in dark grey. The light gray map regions derive from non-PI 277012 sources(continued).

Marker cluster[a]	Map Distance (cM)	GP80 map[b]	Novus-4 map[c]	GP80	Novus-4	19CP-29	18M6(F_1)	ND Noreen	F_2 CP46C-6	F_2 CP46C-9
47	70.3			AA	BB	AA	AB	AA	BB	BB
48	76.8			AA	BB	BB	BB	BB	BB	BB
49	81.1 - 81.9			AA	BB	AA	AB	AA	BB	BB
50	84.1 - 84.5			BB	AA	BB	AB	BB	AB	AA
51	86.2			BB	BB	AA	AB	AA	AB	BB
52	86.3 - 86.35			BB	AA	AA	AA	AA	AB	BB
53	88.02			BB	BB	AA	AB	AA	AB	BB
54	89.9 - 89.9			AA	BB	BB	BB	BB	AB	AA
55	90.5			AA	BB	BB	BB	BB	AB	AA
56	91.30			AA	BB	BB	BB	BB	AB	AA
57	93.2			BB	AA	AA	AA	AA	AB	BB
58	94.4			BB	AA	AA	AA	BB	BB	BB
59	98.4			BB	AA	AA	AA	BB	BB	BB
60	104.8			BB	AA	AA	AB	AA	BB	BB
61	105.3 - 105.9			AA	BB	BB	AB	BB	AA	AA
62	109.4			BB	BB	AA	AB	AA	BB	BB
63	111.2			AA	AA	BB	AB	BB	AA	AA
64	113.1			AA	AA	BB	AB	BB	AA	AA
65	113.14			AA	BB	BB	AB	BB	AA	AA
66	114.5 - 114.9			BB	AA	BB	BB	AA	BB	BB
67	115.2 - 115.8			BB	AA	BB	BB	BB	BB	BB
68	116.0			AA	BB	BB	AB	AA	AA	AA
69	117.6			BB	BB	AA	AB	AA	BB	BB
70	117.6			BB	AA	AA	AB	AA	BB	BB
71	119.8			BB	BB	AA	AB	AA	BB	BB
72	119.89			BB	AA	AA	AA	BB	AA	AA
73	120.1 - 120.4			BB	BB	AA	AB	AA	BB	BB
74	122.70			BB	BB	AA	AB	BB	BB	BB
75	124.5			BB	BB	AA	AB	AA	BB	AB
76	125.1 - 125.6			BB	AA	AA	AB	AA	AA	AA
77	127.6			AA	AA	BB	AB	BB	AA	AB
78	130.9			AA	AA	BB	AB	BB	AA	AB
79	130.90			BB	AA	AA	AB	AA	AA	AA
80	137.8 - 137.9			BB	AA	AA	AB	AA	AA	AA
81	139.7			BB	AA	AA	AB	AA	AA	AA
82	139.75			AA	AA	BB	AB	BB	AA	AB
83	141.74			AA	BB	BB	AB	BB	BB	BB
84	141.74			BB	BB	AA	AB	BB	BB	AB
85	141.74			BB	AA	AA	AB	AA	AA	AA
86	147.25			BB	AA	AA	AB	AA	AA	AA
87	148.3			BB	AA	AA	AB	AA	AA	AB

[a] SNP clusters defined in Table A1.

[b] A GP80 map was obtained from Ganaparthi (2020). Dark grey and light grey regions indicate PI 277012-derived and non-PI 277012-derived chromatin, respectively.

[c] A Novus-4 map was provided by Marais (2021 – Personal Communication). Dark grey and light grey regions indicate PI 277012-derived and non-PI 277012-derived chromatin, respectively.

Table A3. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen. Polymorphism for 21 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.1* region are shown. Alleles in orange color derive from PI 277012/GP80. Light grey regions do not derive from PI 277012 suggesting that CP46C-6 was a heterozygote having two different lengths of introgressed PI 277012 chromatin.

Markers	33441	52121	66040	8504	9821	34969	39861	56402	70992	79257	80757	71078	78836	76028	78834	12084	48151	48152	76999	79581	43006
Map Distance (cM)	19.9	19.9	19.9	26.5	26.5	26.5	26.5	26.5	26.5	26.5	26.5	35.4	35.4	35.9	35.9	36.6	36.6	36.6	36.6	36.9	38.7
GP80	AA	BB	AA	BB	BB	BB	AA	AA	AA	AA	AA	AA	BB	AA	BB	AA	AA	AA	AA	BB	AA
Novus-4	AA	BB	AA	BB	BB	BB	AA	AA	AA	AA	AA	AA	BB	AA	BB	AA	AA	AA	AA	BB	AA
19CP-29	BB	AA	BB	AA	AA	AA	BB	BB	BB	BB	BB	BB	AA	BB	AA	BB	BB	BB	BB	AA	BB
18M6(F_1)	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
ND Noreen	BB	AA	BB	AA	AA	AA	BB	BB	BB	BB	BB	BB	AA	BB	AA	BB	BB	BB	BB	AA	BB
CP46C-6(F_2)	AA	BB	AA	BB	BB	BB	AA	AA	AA	AA	AA	AB	AB	AB	AB	AB	AB	AB	AB	AB	AA
CP46C-9(F_2)	AA	BB	AA	BB	BB	BB	AA	AA	AA	AA	AA	AA	BB	AA	BB	AA	AA	AA	AA	BB	AA
ND Noreen	BB	AA	BB	AA	AA	AA	BB	BB	BB	BB	BB	BB	AA	BB	AA	BB	BB	BB	BB	AA	BB
CP46C-6(F_2)	AA	BB	AA	BB	BB	BB	AA	AA	AA	AA	AA	AB	AB	AB	AB	AB	AB	AB	AB	AB	AA
CP46C-6(F_2) Progeny																					
20M1-1	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-2	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-3	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-4	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-5	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-6	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-7	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-8	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-9	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-10	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB

Table A3. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen (continued). Polymorphism for 21 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.1* region are shown. Alleles in orange color derive from PI 277012/GP80. Light grey regions do not derive from PI 277012 suggesting that CP46C-6 was a heterozygote having two different lengths of introgressed PI 277012 chromatin (Continued).

Markers	33441	52121	66040	8504	9821	34969	39861	56402	70992	79257	80757	71078	78836	76028	78834	12084	48151	48152	76999	79581	43006
Map Distance (cM)	19.9	19.9	19.9	26.5	26.5	26.5	26.5	26.5	26.5	26.5	26.5	35.4	35.4	35.9	35.9	36.6	36.6	36.6	36.6	36.9	38.7
20M1-11	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-12	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-13	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-14	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-15	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-16	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-17	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-18	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-19	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-20	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-21	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-22	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-23	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-24	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-25	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-26	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-27	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-28	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-29	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-30	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-31	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-32	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB

Table A3. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen (continued). Polymorphism for 21 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.1* region are shown. Alleles in orange color derive from PI 277012/GP80. Light grey regions do not derive from PI 277012 suggesting that CP46C-6 was a heterozygote having two different lengths of introgressed PI 277012 chromatin (Continued).

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20M1-33	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-34	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AB	AB
20M1-35	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-36	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-37	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-38	(ii)	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-39	(ii)	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-40	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-41	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-42	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-43	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-44	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-45	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-46	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-47	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-48	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-49	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-50	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-51	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-52	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-53	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-54	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-55	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB

Table A3. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen (continued). Polymorphism for 21 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.1* region are shown. Alleles in orange color derive from PI 277012/GP80. Light grey regions do not derive from PI 277012 suggesting that CP46C-6 was a heterozygote having two different lengths of introgressed PI 277012 chromatin (Continued).

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20M1-56	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-57	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-58	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-59	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-60	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-61	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-62	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-63	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-64	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-65	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-66	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-67	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-68	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-69	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-70	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-71	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-72	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-73	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-74	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-75	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-76	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-77	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-78	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB

Table A3. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen (continued). Polymorphism for 21 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.1* region are shown. Alleles in orange color derive from PI 277012/GP80. Light grey regions do not derive from PI 277012 suggesting that CP46C-6 was a heterozygote having two different lengths of introgressed PI 277012 chromatin (Continued).

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20M1-79	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-80	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-81	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-82	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-83	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-84	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-85	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-86	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-87	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-88	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-89	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-90	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-91	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-92	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-93	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-94	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-95	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-96	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-97	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-98	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-99	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-100	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-101	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB

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Map Distance (cM)	**19.9**	**19.9**	**19.9**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**35.4**	**35.4**	**35.9**	**35.9**	**36.6**	**36.6**	**36.6**	**36.6**	**36.9**	**38.7**
20M1-102	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-103	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-104	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-105	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-106	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-107	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	NC	NC	AA	BB	BB	BB	BB	AA	AB
20M1-108	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	NC	BB	AA	AB
20M1-109	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-110	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AA	AB
20M1-111	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	AA	BB	BB	BB	BB	AA	AB
20M1-112	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
ND Noreen	BB	AA	BB	AA	AA	AA	BB	BB	BB	BB	BB	BB	AA	BB	AA	BB	BB	BB	BB	AA	BB
CP46C-9(F_2)	AA	BB	AA	BB	BB	BB	AA	AA	AA	AA	AA	AA	BB	AA	BB	AA	AA	AA	AA	BB	AA
CP46C-9(F_2) progeny																					
20M1-113	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-114	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-115	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-117	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-118	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-119	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB

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Map Distance (cM)	**19.9**	**19.9**	**19.9**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**26.5**	**35.4**	**35.4**	**35.9**	**35.9**	**36.6**	**36.6**	**36.6**	**36.6**	**36.9**	**38.7**
20M1-120	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-121	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-122	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-123	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-124	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-125	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-126	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-127	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-128	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-129	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-130	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-131	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB
20M1-132	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB

Table A4. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen. Polymorphism for 15 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.2* region are shown. Alleles in orange color derive from PI 277012/GP80. The non-polymorphic region (grey) suggests that loci 59300 and 27941 may be incorrectly mapped.

Markers	**76979**	**12226**	**48788**	**61152**	**15035**	**58284**	**33039**	**80888**	**7052**	**11676**	**66080**	**59300**	**27941**	**10029**	**11245**
Map distance (cM)	**104.9**	**105.4**	**106.0**	**109.4**	**111.2**	**113.1**	**113.1**	**113.1**	**114.5**	**115.0**	**115.0**	**115.3**	**115.8**	**117.7**	**117.7**
GP80	BB	AA	BB	BB	AA	AA	AA	AA	BB	AA	BB	BB	BB	BB	BB
Novus-4	AA	BB	AA	BB	AA	AA	BB	BB	AA	BB	AA	AA	AA	BB	AA
19CP29	AA	BB	AA	AA	BB	BB	BB	BB	BB	AA	BB	BB	BB	AA	AA
18M6(F_1)	AB	AB	AB	AB	AB	AB	AB	AB	BB	AA	BB	BB	BB	AB	AB
ND Noreen	AA	BB	AA	AA	BB	BB	BB	BB	AA	BB	AA	BB	BB	AA	AA
46C-6(F_2)	BB	AA	BB	BB	AA	AA	AA	AA	BB	AA	BB	BB	BB	BB	BB
46C-9(F_2)	BB	AA	BB	BB	AA	AA	AA	AA	BB	AA	BB	BB	BB	BB	BB
ND Noreen	AA	BB	AA	AA	BB	BB	BB	BB	AA	BB	AA	BB	BB	AA	AA
46C-6(F_2)	BB	AA	BB	BB	AA	AA	AA	AA	BB	AA	BB	BB	BB	BB	BB
46C-6(F_2) Progeny															
20M1-1	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-2	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-3	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-4	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-5	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-6	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-7	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-8	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-9	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-10	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-11	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-12	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-13	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-14	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-15	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-16	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-17	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-18	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-19	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-20	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-21	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-22	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-23	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-24	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-25	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-26	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-27	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-28	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-29	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB

Table A4. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen (continued). Polymorphism for 15 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.2* region are shown. Alleles in orange color derive from PI 277012/GP80. The non-polymorphic region (grey) suggests that loci 59300 and 27941 may be incorrectly mapped (Continued).

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Map distance (cM)	104.9	105.4	106.0	109.4	111.2	113.1	113.1	113.1	114.5	115.0	115.0	115.3	115.8	117.7	117.7
20M1-30	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-31	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-32	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-33	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-34	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-35	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-36	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-37	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-38	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-39	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-40	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-41	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-42	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-43	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-44	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-45	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-46	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-47	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-48	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-49	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-50	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-51	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-52	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-53	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-54	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-55	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-56	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-57	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-58	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-59	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-60	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-61	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-62	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-63	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-64	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-65	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-66	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-67	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-68	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-69	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB

Table A4. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen (continued). Polymorphism for 15 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.2* region are shown. Alleles in orange color derive from PI 277012/GP80. The non-polymorphic region (grey) suggests that loci 59300 and 27941 may be incorrectly mapped (Continued).

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20M1-70	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-71	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-72	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-73	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-74	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-75	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-76	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-77	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-78	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-79	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-80	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-81	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-82	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-83	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-84	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-85	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-86	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-87	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-88	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-89	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-90	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-91	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-92	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-93	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-94	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-95	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-96	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-97	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-98	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-99	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-100	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-101	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-102	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-103	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-104	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-105	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-106	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-107	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-108	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-109	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB

Table A4. F_1 progeny obtained when two F_2 homozygotes (CP46C-6 and CP46C-9) were backcrossed to ND Noreen (continued). Polymorphism for 15 markers believed to occur within or close to the QTL *Qfhb.rwg-5A.2* region are shown. Alleles in orange color derive from PI 277012/GP80. The non-polymorphic region (grey) suggests that loci 59300 and 27941 may be incorrectly mapped (Continued).

Markers	**76979**	**12226**	**48788**	**61152**	**15035**	**58284**	**33039**	**80888**	**7052**	**11676**	**66080**	**59300**	**27941**	**10029**	**11245**
Map distance (cM)	**104.9**	**105.4**	**106.0**	**109.4**	**111.2**	**113.1**	**113.1**	**113.1**	**114.5**	**115.0**	**115.0**	**115.3**	**115.8**	**117.7**	**117.7**
20M1-110	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-111	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-112	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
ND Noreen	AA	BB	AA	AA	BB	BB	BB	BB	AA	BB	AA	BB	BB	AA	AA
46C-9(F_2)	BB	AA	BB	BB	AA	AA	AA	AA	BB	AA	BB	BB	BB	BB	BB
46C-9(F_2) progeny															
20M1-113	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-114	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-115	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-117	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-118	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-119	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-120	AB	AB	AB	AB	AB	AB	AB	BB	AB	AB	AB	BB	BB	AB	AB
20M1-121	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-122	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-123	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-124	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-125	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-126	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-127	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-128	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-129	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-130	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-131	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB
20M1-132	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	AB	BB	BB	AB	AB

Table A5. Percentage of ND Noreen background recovered for each chromosome and the number of polymorphic SNPs that were involved.

	Background recovered (%)																					Total
Chr.	**1A**	**1B**	**1D**	**2A**	**2B**	**2D**	**3A**	**3B**	**3D**	**4A**	**4B**	**4D**	**5A**	**5B**	**5D**	**6A**	**6B**	**6D**	**7A**	**7B**	**7D**	
SNPs	**62**	**61**	**22**	**34**	**27**	**10**	**42**	**30**	**11**	**37**	**42**	**10**	**25**	**46**	**11**	**24**	**36**	**5**	**27**	**43**	**15**	
Plants																						
20M1-1	0.67	0.57	0.74	0.99	0.59	0.70	0.98	0.97	0.91	0.97	0.71	0.95	0.60	0.67	0.55	0.67	0.74	0.90	0.67	0.94	0.97	**0.78**
20M1-2	0.76	0.84	0.80	0.79	0.78	0.50	0.94	0.78	0.73	0.57	0.94	0.65	0.92	0.74	0.82	0.90	0.74	0.60	0.61	0.51	0.60	**0.74**
20M1-3	0.76	0.76	0.74	0.75	0.63	0.50	0.94	0.53	0.59	0.72	0.93	0.55	0.90	0.83	0.86	0.56	0.64	0.60	0.89	0.58	0.73	**0.71**
20M1-4	0.64	0.94	0.61	0.88	0.89	0.80	0.83	0.93	0.82	0.57	0.57	0.90	0.92	0.77	0.55	0.77	0.76	0.90	0.69	0.87	0.80	**0.78**
20M1-5	0.60	0.70	0.80	0.76	0.94	0.80	0.94	0.80	0.73	0.92	0.79	0.90	0.62	0.87	0.86	0.94	0.78	0.60	0.61	0.81	0.83	**0.79**
20M1-6	0.64	0.86	0.61	0.54	0.78	0.50	1.00	0.88	0.91	0.81	0.55	0.50	0.80	0.71	0.82	0.98	0.96	0.60	0.98	0.84	0.90	**0.77**
20M1-7	0.65	0.65	0.87	0.75	0.61	0.70	0.92	0.67	0.68	0.50	0.83	0.85	0.92	0.83	0.64	0.94	0.76	0.60	0.50	0.56	0.90	**0.73**
20M1-8	0.60	0.67	0.85	0.56	0.59	1.00	0.82	0.63	0.64	0.61	0.75	0.90	0.90	0.71	0.59	0.92	0.92	0.90	0.63	0.70	0.90	**0.75**
20M1-9	0.85	0.60	0.85	0.54	0.70	0.80	0.90	0.88	0.95	0.58	0.80	0.60	0.92	0.67	0.68	0.63	0.93	0.90	0.63	0.83	0.73	**0.76**
20M1-10	0.62	0.70	0.61	0.96	0.87	0.85	0.70	0.97	0.77	0.58	0.56	0.55	0.90	0.75	0.55	0.56	0.76	0.90	0.70	1.01	0.87	**0.75**
20M1-11	0.65	0.94	0.93	0.82	0.81	0.50	0.56	0.88	0.82	0.50	0.92	0.95	0.62	0.80	0.59	0.90	0.93	0.90	0.61	0.72	0.73	**0.77**
20M1-12	0.60	0.58	0.54	0.97	0.96	0.80	0.98	0.87	0.82	0.86	0.88	0.95	0.60	0.72	0.86	0.96	0.78	0.90	0.81	0.72	1.00	**0.82**
20M1-13	0.57	0.81	0.54	0.54	0.78	0.70	0.56	0.88	0.82	0.73	0.63	0.60	0.86	0.71	0.95	0.58	0.83	0.60	0.65	0.73	0.73	**0.71**
20M1-14	0.82	0.78	0.93	0.57	0.63	0.85	0.95	0.93	0.91	0.85	0.94	0.95	0.78	0.82	0.68	0.56	0.56	0.90	0.57	0.90	0.63	**0.79**
20M1-15	0.58	0.56	0.80	0.81	0.59	0.50	0.68	0.97	0.95	0.66	0.57	0.95	0.66	0.66	0.64	0.90	0.78	0.60	0.59	1.01	0.73	**0.72**
20M1-16	0.72	0.63	0.80	0.91	0.87	1.00	0.58	0.58	0.59	0.78	0.57	0.95	0.80	0.65	0.55	0.96	0.74	0.60	0.93	0.51	0.90	**0.74**
20M1-17	0.93	0.69	0.67	0.60	0.89	0.50	0.79	0.57	0.73	0.61	0.70	0.65	0.60	0.82	0.82	0.54	0.75	0.60	0.83	0.51	0.70	**0.69**
20M1-18	0.58	0.62	0.54	0.84	0.70	1.00	0.79	0.70	0.82	0.85	0.93	0.60	0.64	0.73	0.64	0.96	0.76	0.60	0.61	0.63	0.77	**0.73**
20M1-19	0.94	0.95	0.70	0.51	0.91	0.95	0.85	0.88	0.73	0.57	0.94	0.65	0.54	0.85	0.64	0.50	0.76	0.60	0.54	0.86	0.73	**0.74**
20M1-20	0.84	0.66	0.91	0.81	0.61	0.80	0.96	0.88	0.91	0.66	0.68	0.95	0.94	0.65	0.55	0.52	0.74	0.60	0.61	0.95	0.73	**0.76**
20M1-21	0.87	0.57	0.65	0.54	0.98	0.80	0.55	0.87	0.82	0.85	0.94	0.85	0.82	0.85	0.91	0.96	0.76	0.60	0.87	0.57	0.87	**0.79**
20M1-22	0.69	0.55	0.61	0.79	0.59	0.50	0.70	0.82	0.77	0.85	0.65	0.60	0.60	0.73	0.64	0.98	0.94	0.90	0.98	0.71	0.57	**0.72**
20M1-23	0.67	0.83	0.74	0.91	0.61	0.70	0.81	0.93	0.95	0.59	0.64	0.75	0.72	0.86	0.77	0.81	0.75	0.60	0.57	0.51	0.80	**0.74**
20M1-24	0.89	0.72	0.87	0.94	0.72	0.50	0.81	0.75	0.64	0.85	0.56	0.60	0.62	0.82	0.77	0.56	0.76	0.90	0.65	1.01	0.77	**0.75**
20M1-25	0.65	0.66	0.93	0.72	0.81	0.65	0.79	0.88	0.82	0.64	0.77	0.55	0.90	0.82	0.82	0.56	0.94	0.90	0.56	0.74	0.53	**0.74**
20M1-26	0.62	0.60	0.87	0.96	0.52	0.70	0.79	0.55	0.68	0.64	0.95	1.00	0.86	0.65	0.55	0.54	0.94	0.90	0.89	0.72	0.80	**0.75**
20M1-27	0.65	0.67	0.80	0.99	0.69	1.00	0.98	0.97	0.86	0.74	0.63	0.50	0.60	0.72	0.55	0.54	0.92	0.90	0.98	0.90	0.57	**0.77**
20M1-28	0.58	0.57	0.59	0.94	0.67	0.95	0.67	0.62	0.59	0.68	0.60	0.55	0.60	0.70	0.82	0.92	0.58	0.90	0.67	0.70	0.87	**0.70**
20M1-29	0.86	0.74	0.80	0.60	0.59	0.50	0.57	0.67	0.73	0.81	0.62	0.55	0.68	0.83	0.77	0.85	0.96	0.60	0.85	1.01	0.90	**0.74**
20M1-30	0.65	0.63	0.74	0.57	0.80	0.50	0.79	0.67	0.68	0.66	0.55	0.55	0.54	0.78	0.73	0.96	0.74	0.90	0.61	0.56	0.60	**0.68**
20M1-31	0.63	0.68	0.85	0.69	0.69	0.85	0.81	0.62	0.68	0.80	0.67	0.65	0.90	0.80	0.73	0.94	0.60	0.60	0.65	0.72	0.80	**0.73**
20M1-32	0.62	0.72	0.61	0.68	0.91	0.80	0.70	0.88	0.86	0.84	0.87	0.95	0.90	0.79	0.77	0.56	0.56	0.60	0.83	0.67	0.90	**0.76**
20M1-33	0.62	0.94	0.87	0.87	0.72	0.80	0.75	0.53	0.68	0.57	0.85	0.55	0.66	0.79	0.64	0.54	0.94	0.90	0.67	0.88	0.70	**0.74**
20M1-34	0.65	0.78	0.67	0.94	0.70	1.00	0.89	0.55	0.64	1.00	0.71	1.00	0.62	0.74	0.86	0.54	0.93	0.60	0.96	0.80	0.67	**0.77**
20M1-35	0.82	0.62	0.54	0.53	0.65	0.70	0.79	0.53	0.59	0.89	0.82	0.60	0.56	0.77	0.82	0.96	0.75	0.90	0.52	0.90	0.90	**0.72**

Table A5. Percentage of ND Noreen background recovered for each chromosome and the number of polymorphic SNPs that were involved (continued).

	Background recovered (%)																					Total
Chr.	**1A**	**1B**	**1D**	**2A**	**2B**	**2D**	**3A**	**3B**	**3D**	**4A**	**4B**	**4D**	**5A**	**5B**	**5D**	**6A**	**6B**	**6D**	**7A**	**7B**	**7D**	
SNPs	**62**	**61**	**22**	**34**	**27**	**10**	**42**	**30**	**11**	**37**	**42**	**10**	**25**	**46**	**11**	**24**	**36**	**5**	**27**	**43**	**15**	
Plants																						
20M1-36	0.85	0.70	0.54	0.59	0.54	0.70	0.95	0.55	0.64	0.80	0.55	0.55	0.62	0.83	0.68	0.73	0.93	0.60	0.94	0.63	0.57	**0.69**
20M1-37	0.79	0.77	0.83	0.65	0.81	1.00	0.52	0.53	0.68	0.65	0.56	0.60	0.90	0.78	0.64	0.60	0.54	0.90	0.85	0.51	0.93	**0.72**
20M1-38	0.89	0.67	0.93	0.93	0.52	0.70	0.64	0.80	0.82	0.80	0.62	0.55	0.60	0.71	0.59	0.98	0.94	0.60	0.89	0.63	0.77	**0.74**
20M1-39	0.85	0.93	0.87	0.96	0.76	0.70	0.76	0.85	0.91	0.95	0.88	0.95	0.84	0.77	0.82	0.56	0.94	0.90	0.85	0.56	0.77	**0.83**
20M1-40	0.93	0.77	0.87	0.84	0.94	1.00	0.85	0.88	0.91	0.66	0.94	0.90	0.66	0.73	0.91	0.71	0.94	0.60	0.56	1.01	0.83	**0.83**
20M1-41	0.60	0.83	0.61	0.69	0.69	0.80	0.99	0.97	0.82	0.92	0.60	0.55	0.60	0.66	0.73	0.98	0.94	0.90	0.85	0.63	0.97	**0.78**
20M1-42	0.57	0.72	0.54	0.81	0.87	0.70	0.57	0.88	0.82	0.65	0.73	0.90	0.90	0.75	0.82	0.90	0.74	0.90	0.63	0.70	0.60	**0.75**
20M1-43	0.62	0.62	0.87	0.59	0.57	0.70	0.82	0.97	0.86	0.66	0.68	1.00	0.88	0.70	0.82	0.83	0.57	0.90	0.93	1.01	0.87	**0.78**
20M1-44	0.81	0.57	0.63	0.81	0.59	0.70	0.70	0.88	0.95	0.99	0.71	0.60	0.56	0.79	0.64	0.94	0.56	0.60	0.56	0.51	0.77	**0.71**
20M1-45	0.57	0.56	0.67	0.81	0.78	0.70	0.68	0.53	0.59	0.72	0.60	0.60	0.82	0.72	0.77	0.96	0.75	0.60	0.81	0.85	0.87	**0.71**
20M1-46	0.85	0.75	0.74	0.53	0.52	0.70	0.74	0.87	0.82	0.86	0.60	0.55	0.64	0.77	0.86	0.56	0.76	0.60	0.94	0.51	0.57	**0.70**
20M1-47	0.67	0.56	0.74	0.56	0.89	1.00	0.65	0.75	0.73	0.66	0.93	0.65	0.60	0.80	0.64	0.92	0.57	0.90	0.76	0.90	0.87	**0.75**
20M1-48	0.87	0.55	0.80	0.99	0.70	1.00	0.81	0.53	0.73	0.92	0.89	0.65	0.94	0.71	0.73	0.96	0.78	0.90	0.70	1.01	0.93	**0.81**
20M1-49	0.92	0.79	0.80	0.90	0.74	0.50	0.51	0.53	0.59	0.85	0.67	1.00	0.78	0.74	0.73	0.52	0.58	0.90	0.94	0.90	0.67	**0.74**
20M1-50	0.64	0.94	0.67	0.76	0.52	0.50	0.86	0.55	0.55	0.66	0.71	0.85	0.92	0.78	0.59	0.79	0.76	0.60	0.76	0.80	0.60	**0.71**
20M1-51	0.57	0.56	0.61	0.71	0.89	0.80	0.70	0.88	0.77	0.53	0.82	0.60	0.62	0.70	0.55	0.58	0.93	0.90	0.85	0.51	0.87	**0.71**
20M1-52	0.93	0.95	0.80	0.53	0.54	0.70	1.00	0.88	0.91	0.66	0.94	0.90	0.70	0.86	0.91	0.54	0.75	0.60	0.83	0.69	0.60	**0.77**
20M1-53	0.60	0.55	0.63	0.94	0.80	0.50	0.61	0.62	0.59	0.80	0.69	0.55	0.90	0.80	0.64	0.73	0.72	0.60	0.96	0.51	0.70	**0.69**
20M1-54	0.88	0.58	0.87	0.63	0.87	0.50	0.67	0.53	0.68	0.91	0.57	0.90	0.92	0.75	0.86	0.94	0.57	0.60	0.93	0.91	0.83	**0.76**
20M1-55	0.66	0.57	0.63	0.97	0.69	0.80	0.70	0.85	0.86	0.84	0.55	0.95	0.60	0.82	0.68	0.63	0.74	0.90	0.70	0.73	0.83	**0.75**
20M1-56	0.81	0.57	0.70	0.57	0.70	1.00	0.54	0.90	0.77	0.84	0.92	0.95	0.88	0.72	0.64	0.85	0.92	0.90	0.61	0.52	0.77	**0.77**
20M1-57	0.88	0.65	0.61	0.78	0.87	0.70	0.93	0.88	0.82	0.91	0.57	0.85	0.96	0.76	0.86	0.58	0.93	0.60	0.89	0.80	0.80	**0.79**
20M1-58	0.65	0.70	0.87	0.51	0.57	0.50	0.56	0.88	0.82	0.54	0.75	0.95	0.92	0.80	0.77	0.94	0.96	0.90	0.81	0.88	0.53	**0.75**
20M1-59	0.84	0.95	0.89	0.72	0.52	0.50	0.73	0.90	0.77	0.54	0.80	0.90	0.98	0.83	0.77	0.63	0.94	0.60	0.67	0.98	0.80	**0.77**
20M1-60	0.56	0.59	0.87	0.96	0.59	0.85	0.68	0.53	0.68	0.84	0.56	0.60	0.60	0.74	0.82	0.85	0.90	0.90	0.85	0.63	0.90	**0.74**
20M1-61	0.75	0.84	0.59	0.97	0.59	0.50	0.69	0.63	0.68	0.92	0.65	0.95	0.92	0.79	0.77	0.94	0.75	0.90	0.59	0.69	0.60	**0.75**
20M1-62	0.75	0.59	0.67	0.65	0.87	0.70	0.65	0.65	0.77	0.68	0.67	1.00	0.56	0.74	0.73	0.77	0.74	0.60	0.63	0.51	0.60	**0.69**
20M1-63	0.87	0.76	0.87	0.91	0.78	0.50	0.57	0.93	0.82	0.96	0.56	0.95	0.80	0.64	0.77	0.88	0.56	0.90	1.00	1.01	0.77	**0.80**
20M1-64	0.64	0.95	0.78	0.74	0.70	0.55	0.64	0.95	0.86	0.84	0.56	0.55	0.88	0.72	0.82	0.92	0.96	0.90	0.52	0.51	0.63	**0.74**
20M1-65	0.88	0.58	0.61	0.93	0.59	0.50	0.65	0.62	0.50	0.93	0.94	0.90	0.90	0.70	0.73	0.90	0.93	0.90	0.63	0.51	0.93	**0.75**
20M1-66	0.81	0.58	0.59	0.94	0.50	0.50	0.79	0.68	0.64	0.72	0.55	0.60	0.82	0.86	0.64	0.85	0.94	0.90	0.76	0.70	0.93	**0.73**
20M1-67	0.67	0.58	0.54	0.96	0.50	0.70	0.93	0.57	0.68	0.57	0.73	0.90	0.60	0.64	0.68	0.96	0.94	0.60	0.87	0.51	0.73	**0.71**
20M1-68	0.83	0.67	0.74	0.96	0.61	1.00	0.79	0.88	0.77	1.00	0.74	1.00	0.82	0.82	1.00	0.52	0.56	0.90	0.89	0.92	0.80	**0.82**
20M1-69	0.89	0.70	0.80	0.91	0.98	0.80	0.56	0.88	0.86	0.54	0.79	1.00	0.68	0.85	0.59	0.98	0.94	0.90	0.81	0.78	0.70	**0.81**
20M1-70	0.89	0.55	0.65	0.94	0.61	0.70	0.99	0.88	0.86	0.65	0.94	0.75	0.88	0.75	0.91	0.77	0.57	0.60	0.70	0.80	0.87	**0.77**
20M1-71	0.89	0.70	0.87	0.59	0.61	0.70	0.54	0.97	0.95	0.86	0.71	0.95	0.88	0.85	0.73	0.96	0.76	0.90	0.59	0.51	0.80	**0.78**
20M1-72	0.90	0.84	0.93	0.60	0.91	0.70	0.71	0.88	0.91	0.65	0.94	0.65	0.62	0.78	0.64	0.52	0.93	0.90	0.65	0.66	0.77	**0.77**
20M1-73	0.92	0.89	0.67	0.62	0.81	0.70	0.79	0.62	0.55	0.92	0.86	0.95	0.62	0.78	0.91	0.94	0.96	0.90	0.94	0.90	0.80	**0.81**

Table A5. Percentage of ND Noreen background recovered for each chromosome and the number of polymorphic SNPs that were involved (continued).

	Background recovered (%)																				Total
Chr.	**1A**	**1B**	**1D**	**2A**	**2B**	**2D**	**3A**	**3B**	**3D**	**4A**	**4B**	**4D**	**5A**	**5B**	**5D**	**6A**	**6B**	**6D**	**7A**	**7B**	**7D**	
SNPs	**62**	**61**	**22**	**34**	**27**	**10**	**42**	**30**	**11**	**37**	**42**	**10**	**25**	**46**	**11**	**24**	**36**	**5**	**27**	**43**	**15**	
Plants																						
20M1-74	0.71	0.57	0.65	0.71	0.78	0.50	0.82	0.97	0.82	0.72	0.71	0.60	0.56	0.63	0.50	0.56	0.54	0.60	0.57	0.63	0.80	**0.66**
20M1-75	0.72	0.67	0.70	0.56	0.80	0.50	0.80	0.55	0.59	0.92	0.67	0.95	0.84	0.75	0.68	0.88	0.79	0.60	0.69	1.01	0.93	**0.74**
20M1-76	0.88	0.61	0.63	0.93	0.87	1.00	0.98	0.88	0.86	0.86	0.70	1.00	0.60	0.67	0.59	0.54	0.76	0.90	0.78	0.74	0.90	**0.80**
20M1-77	0.93	0.95	0.70	0.56	0.80	0.50	0.99	0.88	0.82	0.77	0.83	0.60	0.90	0.76	0.82	0.85	0.75	0.60	0.91	0.51	0.63	**0.76**
20M1-78	0.95	0.79	0.87	0.94	0.89	0.80	0.98	0.85	0.77	0.92	0.57	0.85	0.90	0.83	0.82	0.54	0.76	0.90	0.63	0.72	0.90	**0.82**
20M1-79	0.93	0.95	0.67	0.88	0.72	1.00	0.90	0.97	0.95	0.69	0.85	0.60	0.92	0.77	0.73	0.90	0.96	0.90	0.54	0.90	0.73	**0.83**
20M1-80	0.68	0.89	0.61	0.93	0.61	0.50	0.79	0.97	0.82	0.72	0.56	0.60	0.86	0.66	0.73	0.96	0.65	0.60	0.81	0.90	0.60	**0.74**
20M1-81	0.90	0.73	0.65	0.91	0.67	0.80	0.98	0.97	0.82	0.73	0.60	0.85	0.86	0.83	0.68	0.54	0.56	0.90	0.85	0.56	0.80	**0.77**
20M1-82	0.74	0.93	0.67	0.56	0.80	0.70	0.65	0.88	0.77	0.88	0.70	0.65	0.84	0.68	0.68	0.54	0.54	0.90	0.89	0.51	0.80	**0.73**
20M1-83	0.89	0.84	0.87	0.71	0.91	0.95	0.98	0.93	0.95	0.58	0.94	0.60	0.72	0.79	0.55	0.98	0.76	0.90	0.67	0.84	0.90	**0.82**
20M1-84	0.62	0.83	0.87	0.94	0.61	0.50	0.55	0.88	0.95	0.91	0.79	0.95	0.86	0.76	0.82	0.98	0.76	0.60	0.56	0.69	0.93	**0.78**
20M1-85	0.83	0.94	0.93	0.87	0.69	0.50	0.85	0.83	0.86	0.86	0.69	0.65	0.86	0.74	0.82	0.96	0.76	0.60	0.96	1.01	0.87	**0.81**
20M1-86	0.68	0.84	0.67	0.91	0.91	1.00	0.54	0.75	0.73	0.57	0.94	0.95	0.60	0.75	0.55	0.60	0.94	0.90	0.61	0.78	0.80	**0.76**
20M1-87	0.94	0.94	0.74	0.90	0.61	0.50	0.85	0.97	0.77	0.54	0.57	0.85	0.92	0.83	0.91	0.60	0.54	0.90	0.89	0.51	0.73	**0.76**
20M1-88	0.61	0.68	0.91	0.69	0.83	0.50	0.67	0.62	0.73	0.97	0.77	0.55	0.60	0.72	0.86	0.56	0.76	0.90	0.83	1.01	0.73	**0.74**
20M1-89	0.65	0.71	0.61	0.56	0.87	0.70	0.80	0.88	0.95	0.99	0.55	0.55	0.80	0.68	0.86	0.92	0.78	0.60	0.56	0.72	0.57	**0.73**
20M1-90	0.63	0.86	0.76	0.69	0.61	0.80	0.79	0.78	0.64	0.85	0.56	0.50	0.88	0.80	0.64	0.94	0.57	0.90	0.63	0.80	0.73	**0.73**
20M1-91	0.58	0.84	0.67	0.74	0.70	0.80	0.85	0.88	0.95	0.84	0.81	0.95	0.60	0.68	0.95	0.56	0.94	0.90	0.59	1.01	0.77	**0.79**
20M1-92	0.89	0.67	0.83	0.59	0.50	0.70	0.83	0.97	0.91	0.81	0.65	0.60	0.86	0.84	1.00	0.60	0.54	0.80	0.96	0.64	0.97	**0.77**
20M1-93	0.58	0.68	0.54	0.62	0.61	0.65	0.73	0.97	0.86	0.84	0.94	0.90	0.80	0.72	0.68	0.88	0.89	0.90	0.56	0.65	0.70	**0.75**
20M1-94	0.65	0.60	0.87	0.54	0.50	0.50	0.52	0.53	0.59	0.84	0.56	0.85	0.92	0.68	0.86	0.90	0.75	0.90	1.00	0.90	0.77	**0.73**
20M1-95	0.76	0.57	0.61	0.94	0.89	0.70	0.69	0.58	0.64	0.70	0.77	0.75	0.88	0.83	0.77	0.54	0.94	0.90	0.93	0.90	0.67	**0.76**
20M1-96	0.60	0.61	0.61	0.90	0.61	0.90	0.56	0.85	0.82	0.97	0.77	0.60	0.62	0.67	0.77	0.96	0.94	0.60	0.57	0.63	0.77	**0.73**
20M1-97	0.86	0.92	0.83	0.88	0.63	0.90	0.83	0.88	0.82	0.96	0.86	0.90	0.80	0.66	0.86	0.69	0.74	0.90	0.87	0.83	0.63	**0.82**
20M1-98	0.66	0.94	0.93	0.53	0.54	0.55	0.65	0.97	0.86	0.64	0.79	0.55	0.88	0.71	0.82	0.60	0.57	0.90	0.93	0.80	0.73	**0.74**
20M1-99	0.63	0.89	0.85	0.60	0.78	0.55	0.82	0.87	0.82	0.77	0.88	0.60	0.70	0.77	0.68	0.54	0.93	0.90	0.83	0.80	0.83	**0.76**
20M1-100	0.61	0.66	0.83	0.76	0.87	1.00	0.77	0.60	0.73	0.76	0.93	0.95	0.66	0.66	0.64	0.90	0.72	0.60	0.59	1.01	0.63	**0.76**
20M1-101	0.77	0.70	0.65	0.97	0.85	0.50	0.62	0.67	0.77	0.68	0.71	0.90	0.60	0.87	0.73	0.98	0.57	0.60	0.98	0.85	0.73	**0.75**
20M1-102	0.60	0.61	0.87	0.65	0.59	0.50	0.70	0.88	0.95	0.66	0.60	0.60	0.72	0.85	0.64	0.94	0.74	0.60	0.61	0.51	0.90	**0.70**
20M1-103	0.90	0.75	0.85	0.54	0.63	0.65	0.77	0.88	1.00	0.69	0.80	0.90	0.72	0.78	0.86	0.88	0.76	0.90	0.96	0.83	0.60	**0.79**
20M1-104	0.89	0.61	0.80	0.94	0.69	1.00	0.79	0.53	0.64	0.85	0.57	0.55	0.98	0.68	0.82	0.98	0.56	0.60	0.93	0.52	0.97	**0.76**
20M1-105	0.63	0.57	0.87	0.87	0.50	0.50	0.82	0.68	0.86	0.86	0.60	0.95	0.62	0.70	0.68	0.94	0.76	0.90	0.63	0.51	0.77	**0.73**
20M1-106	0.70	0.59	0.87	0.76	0.89	0.80	0.80	0.53	0.59	0.93	0.94	0.70	0.72	0.68	0.77	0.71	0.75	0.60	0.89	0.77	0.63	**0.74**
20M1-107	0.64	0.90	0.85	0.91	0.72	0.70	0.65	0.53	0.73	0.53	0.85	0.90	0.90	0.67	0.68	0.65	0.79	0.60	0.87	0.69	0.57	**0.73**
20M1-108	0.92	0.95	0.93	0.59	0.98	0.80	0.56	0.88	0.95	0.95	0.83	0.55	0.54	0.64	0.64	0.94	0.74	0.90	0.89	0.51	0.97	**0.79**
20M1-109	0.90	0.60	0.54	0.96	0.89	1.00	0.79	0.62	0.64	0.62	0.75	0.55	0.62	0.70	0.59	0.56	0.72	0.60	0.69	0.63	0.87	**0.71**
20M1-110	0.70	0.54	0.67	0.91	0.59	0.70	0.73	0.53	0.68	0.86	0.87	0.65	0.88	0.78	0.91	0.96	0.74	0.90	0.74	0.63	0.77	**0.75**
20M1-111	0.72	0.96	0.70	0.59	0.98	0.80	0.57	0.85	0.91	0.89	0.69	0.70	0.88	0.76	0.86	0.96	0.75	0.90	0.98	0.74	0.93	**0.82**

Table A5. Percentage of ND Noreen background recovered for each chromosome and the number of polymorphic SNPs that were involved (continued).

	Background recovered (%)																					Total
Chr.	**1A**	**1B**	**1D**	**2A**	**2B**	**2D**	**3A**	**3B**	**3D**	**4A**	**4B**	**4D**	**5A**	**5B**	**5D**	**6A**	**6B**	**6D**	**7A**	**7B**	**7D**	
SNPs	**62**	**61**	**22**	**34**	**27**	**10**	**42**	**30**	**11**	**37**	**42**	**10**	**25**	**46**	**11**	**24**	**36**	**5**	**27**	**43**	**15**	
Plants																						
20M1-112	0.81	0.95	0.67	0.97	0.89	0.50	0.56	0.88	0.86	0.86	0.74	0.95	0.62	0.79	0.64	0.77	0.75	0.60	0.56	0.88	0.70	**0.76**
20M1-113	0.70	0.91	0.76	0.90	0.80	0.50	0.57	0.53	0.55	0.64	0.56	0.55	0.62	0.78	0.68	0.52	0.56	0.60	0.70	1.01	0.80	**0.68**
20M1-114	0.83	0.70	0.96	0.88	0.89	1.00	0.76	0.53	0.59	0.50	0.83	0.85	0.60	0.78	0.68	0.90	0.57	0.60	0.61	0.90	0.83	**0.75**
20M1-115	0.67	0.60	0.83	0.93	0.87	0.80	0.77	0.62	0.59	0.53	0.55	0.65	0.60	0.84	0.73	0.54	0.74	0.60	0.61	1.01	0.73	**0.70**
20M1-117	0.78	0.60	0.70	0.91	0.78	0.70	0.80	0.60	0.59	0.54	0.63	0.50	0.60	0.83	0.73	0.96	0.79	0.60	0.70	1.01	0.93	**0.73**
20M1-118	0.68	0.62	0.89	0.88	0.78	0.70	0.77	0.53	0.55	0.51	0.55	0.50	0.58	0.73	0.68	0.54	0.57	0.60	0.63	1.01	0.73	**0.67**
20M1-119	0.69	0.95	0.80	0.88	0.63	0.70	0.69	0.62	0.55	0.53	0.58	0.85	0.60	0.80	0.68	0.65	0.74	0.60	0.70	0.90	0.53	**0.70**
20M1-120	0.78	0.85	0.89	0.88	0.65	0.70	0.77	0.53	0.55	0.62	0.87	0.55	0.60	0.78	0.68	0.54	0.74	0.60	0.67	1.01	0.60	**0.71**
20M1-121	0.82	0.93	0.76	0.93	0.67	0.50	0.55	0.62	0.64	0.93	0.87	0.85	0.60	0.78	0.77	0.63	0.58	0.60	0.63	1.01	0.60	**0.73**
20M1-122	0.69	0.96	0.96	0.91	0.80	0.50	0.56	0.53	0.59	0.61	0.86	0.90	0.58	0.74	0.68	0.73	0.57	0.60	0.59	0.90	0.80	**0.72**
20M1-123	0.78	0.83	0.87	0.96	0.72	0.85	0.70	0.62	0.55	0.86	0.64	0.85	0.60	0.78	0.73	0.54	0.78	0.60	0.65	0.90	0.80	**0.74**
20M1-124	0.70	0.96	0.80	0.96	0.80	0.70	0.56	0.53	0.55	0.85	0.87	0.60	0.60	0.82	0.77	0.54	0.57	0.70	0.59	0.90	0.87	**0.73**
20M1-125	0.66	0.59	0.70	0.93	0.74	1.00	0.80	0.53	0.59	0.93	0.86	0.60	0.60	0.76	0.77	0.54	0.74	0.60	0.72	1.01	0.57	**0.73**
20M1-126	0.75	0.60	0.89	0.90	0.78	0.50	0.56	0.62	0.55	0.78	0.55	0.55	0.58	0.76	0.73	0.85	0.76	0.60	0.63	1.01	0.80	**0.70**
20M1-127	0.78	0.62	0.76	0.96	0.89	1.00	0.77	0.60	0.59	0.85	0.82	0.85	0.60	0.77	0.77	0.96	0.75	0.60	0.61	1.01	0.53	**0.77**
20M1-128	0.71	0.95	0.87	0.90	0.74	0.80	0.76	0.60	0.64	0.53	0.86	0.90	0.62	0.80	0.73	0.96	0.65	0.60	0.61	0.90	0.53	**0.75**
20M1-129	0.73	0.94	0.93	0.96	0.67	0.55	0.77	0.53	0.55	0.78	0.86	0.55	0.58	0.78	0.77	0.90	0.57	0.60	0.69	0.90	0.53	**0.72**
20M1-130	0.77	0.61	0.89	0.87	0.78	0.50	0.54	0.62	0.59	0.92	0.56	0.80	0.60	0.73	0.73	0.92	0.75	0.60	0.67	1.01	0.70	**0.72**
20M1-131	0.72	0.80	0.93	0.96	0.89	1.00	0.68	0.53	0.59	0.92	0.86	0.85	0.58	0.78	0.73	0.96	0.58	0.60	0.69	0.90	0.77	**0.78**
20M1-132	0.68	0.59	0.67	0.88	0.65	0.55	0.76	0.62	0.59	0.92	0.83	0.85	0.60	0.77	0.77	0.90	0.58	0.60	0.59	0.90	0.83	**0.72**
Avg.	**0.74**	**0.73**	**0.76**	**0.79**	**0.73**	**0.72**	**0.75**	**0.75**	**0.75**	**0.76**	**0.74**	**0.75**	**0.74**	**0.76**	**0.73**	**0.77**	**0.76**	**0.74**	**0.74**	**0.77**	**0.76**	**0.75**

Table A6. Evaluation of parents and $B_1F_{1:2}$ families for FHB resistance in the first greenhouse trial.

Plants	No. of inoculated spikes	Infected spikelets	Total spikelets	Total infection (%)
20M1-1	3	27	39	0.62
20M1-2	3	8	45	0.11
20M1-3	3	4	28	0.14
20M1-4	3	8	43	0.12
20M1-5	4	9	54	0.09
20M1-6	3	15	43	0.28
20M1-7	4	9	60	0.07
20M1-8	3	6	41	0.07
20M1-9	3	7	46	0.09
20M1-10	2	2	27	0.07
20M1-11	3	15	46	0.26
20M1-12	3	8	42	0.12
20M1-13	3	10	38	0.18
20M1-14	3	7	35	0.11
20M1-15	3	7	37	0.11
20M1-16	3	11	39	0.21
20M1-17	3	15	41	0.29
20M1-18	3	9	39	0.15
20M1-19	4	15	50	0.22
20M1-20	3	7	47	0.09
20M1-21	3	7	50	0.08
20M1-22	3	8	40	0.13
20M1-23	3	10	38	0.18
20M1-24	2	4	24	0.17
20M1-25	2	8	34	0.24
20M1-26	3	8	46	0.11
20M1-27	3	9	45	0.13
20M1-28	3	8	34	0.15
20M1-29	3	16	40	0.33
20M1-30	3	7	39	0.10
20M1-31	3	6	42	0.07
20M1-32	3	19	43	0.37
20M1-33	3	6	36	0.08
20M1-34	3	11	43	0.19
20M1-35	2	8	24	0.33
20M1-36	3	15	41	0.29
20M1-37	4	12	56	0.14
20M1-38	3	23	45	0.44
20M1-39	3	7	38	0.11
20M1-40	3	11	39	0.21
20M1-41	3	17	35	0.40
20M1-42	3	12	46	0.20
20M1-43	3	13	42	0.24
20M1-44	3	29	42	0.62
20M1-45	2	4	25	0.16
20M1-46	3	15	41	0.29
20M1-47	2	15	57	0.23
20M1-48	3	6	39	0.08
20M1-49	3	17	39	0.36
20M1-50	3	15	47	0.26

Table A6. Evaluation of parents and $B_1F_{1:2}$ families for FHB resistance in the first greenhouse trial (continued).

Plants	No. of inoculated spikes	Infected spikelets	Total spikelets	Total infection (%)
20M1-51	3	28	42	0.60
20M1-52	4	9	59	0.08
20M1-53	3	27	49	0.49
20M1-54	3	16	38	0.34
20M1-55	3	17	41	0.34
20M1-56	3	21	43	0.42
20M1-57	3	6	40	0.08
20M1-58	4	10	62	0.10
20M1-59	3	9	51	0.12
20M1-60	4	15	56	0.20
20M1-61	3	8	38	0.13
20M1-62	3	12	39	0.23
20M1-63	3	14	40	0.28
20M1-64	3	10	45	0.16
20M1-65	3	13	54	0.19
20M1-66	3	14	50	0.22
20M1-67	3	7	41	0.10
20M1-68	4	23	59	0.32
20M1-69	3	13	40	0.25
20M1-70	3	6	44	0.07
20M1-71	4	12	56	0.14
20M1-72	4	18	56	0.25
20M1-73	3	6	37	0.08
20M1-74	3	21	43	0.42
20M1-75	3	29	38	0.68
20M1-76	3	17	38	0.37
20M1-77	3	8	40	0.13
20M1-78	3	6	36	0.08
20M1-79	3	14	49	0.22
20M1-80	2	11	31	0.35
20M1-81		Did not flower		
20M1-82	4	15	60	0.18
20M1-83	3	17	41	0.34
20M1-84	3	7	39	0.10
20M1-85	3	7	40	0.10
20M1-86	3	9	41	0.15
20M1-87	4	22	52	0.35
20M1-88	3	18	47	0.32
20M1-89	3	7	39	0.10
20M1-90	3	11	39	0.21
20M1-91	3	15	39	0.31
20M1-92	3	20	38	0.45
20M1-93	3	16	41	0.32
20M1-94	3	10	42	0.17
20M1-95	4	8	52	0.08
20M1-96	3	14	38	0.29
20M1-97	2	3	27	0.11
20M1-98	3	8	39	0.13
20M1-99	3	25	36	0.61

Table A6. Evaluation of parents and $B_1F_{1:2}$ families for FHB resistance in the first greenhouse trial (continued).

Plants	No. of inoculated spikes	Infected spikelets	Total spikelets	Total infection (%)
20M1-100	3	15	50	0.24
20M1-101	3	10	44	0.16
20M1-102	3	10	43	0.16
20M1-103	3	11	34	0.24
20M1-104	3	9	38	0.16
20M1-105	2	7	24	0.29
20M1-106	3	7	40	0.10
20M1-107	3	7	39	0.10
20M1-108	3	9	47	0.13
20M1-109	3	19	43	0.37
20M1-110	2	3	32	0.09
20M1-111	3	9	40	0.15
20M1-112	3	27	47	0.51
20M1-113	3	8	36	0.14
20M1-114	4	18	58	0.24
20M1-115	4	20	55	0.29
20M1-117	4	16	55	0.22
20M1-118	2	2	24	0.08
20M1-119	2	5	33	0.15
20M1-120	3	8	41	0.12
20M1-121	3	11	42	0.19
20M1-122	3	29	45	0.58
20M1-123	3	20	43	0.40
20M1-124	2	7	30	0.23
20M1-125	2	11	47	0.19
20M1-126	4	12	56	0.14
20M1-127	3	12	41	0.22
20M1-128	3	10	40	0.18
20M1-129	3	18	42	0.36
20M1-130	3	7	40	0.10
20M1-131	3	21	42	0.43
20M1-132	3	18	40	0.38
GP 80	3	3	28	0.11
Novus-4	2	5	26	0.19
19CP29	3	24	42	0.57
ND Noreen	3	13	36	0.36

Table A7. Agronomic traits and yield data obtained in an un-replicated yield trial that was planted at Casselton in 2020.

Entry	Row numbers		Actual plot yield (g)	Predicted check yield (g)[1]	Relative yield (%)[2]	Test weight (lbs/bu)	Winter survival (0-10)	Plant height (inches)
	1st row	2nd row						
Check	8001(Ideal)	8002(Ideal)	583.6	583.6	100	62.2	9.5	22
19K89-1	8003	8004	652.6	683.47	95	61	10	24
19K89-2	8005	8006	547	744.2	74	61.6	8.5	23
19K89-3	8007	8008	976	810.38	120	61.2	9	26
19K89-4	8009	8010	663.4	810.38	82	60.7	9	26
Check	8011(Ideal)	8012(Ideal)	837.6	837.6	100	63.1	8	24
Check	8013(Ideal)	8014(Ideal)	946	946	100	62.7	9	25
19K89-5	8015	8016	831	885.14	94	61	8.5	17
19K89-6	8017	8018	826	853.55	97	59.7	9	26
19K89-7	8019	8020	747.6	835.3	90	61	9	26
19K89-8	8021	8022	542.2	821.21	66	59.5	8.5	24
Check	8023(Ideal)	8024(Ideal)	815.8	815.8	100	63.2	8.5	23
Check	8025(Ideal)	8026(Ideal)	738	738	100	62.5	7.5	23
19K89-9	8027	8028	711.4	843.04	84	61	8	26
19K89-10	8029	8030	703.6	895.71	79	60.2	8	27
19K89-11	8031	8032	818.6	929.78	88	61	9	25
19K89-12	8033	8034	772	973.22	79	61.2	8.5	26
Check	8035(Ideal)	8036(Ideal)	1049	1049	100	63.3	9	27
Check	8037(Ideal)	8038(Ideal)	1052.4	1052.4	100	63.6	8.5	25
19K89-13	8039	8040	907.6	1013.13	90	60	8.5	26
19K94-1	8041	8042	680.4	989.39	69	59.9	8.5	25
19K94-2	8043	8044	782	972.32	80	60.3	7.5	26
19K94-3	8045	8046	628.4	952.64	66	61.6	8.5	25

Table A7. Agronomic traits and yield data obtained in an un-replicated yield trial that was planted at Casselton in 2020 (continued).

Entry	Row numbers		Actual plot yield (g)	Predicted check yield (g)[1]	Relative yield (%)[2]	Test weight (lbs/bu)	Winter survival (0-10)	Plant height (inches)
	1st row	2nd row						
Check	8047(Ideal)	8048(Ideal)	926.2	926.2	100	62.7	8.5	23
Check	8049(Ideal)	8050(Ideal)	966	966	100	63.2	8.5	25
19K94-4	8051	8052	858	1005.95	85	59.4	8	26
19K94-5	8053	8054	932.6	1029.2	91	61.1	8.5	27
19K94-6	8055	8056	1022	1047.56	98	59.6	8	31
19K94-7	8057	8058	835.2	1075.71	78	60	8	30
Check	8059(Ideal)	8060(Ideal)	1131.2	1131.2	100	61.9	8.5	25
Check	8061(Ideal)	8062(Ideal)	1083.6	1083.6	100	62	8.5	26
19K94-8	8063	8064	586.6	1068.83	55	56.5	7.5	24
19K94-9	8065	8066	976.2	1059.9	92	60	9.5	27
19K94-10	8067	8068	730.4	1053.86	69	60.6	8	27
19K94-11	8069	8070	940.8	1048.2	90	61.3	8.5	32
Check	8071(Ideal)	8072(Ideal)	1043.8	1043.8	100	63.5	9.5	24
Check	8073(Ideal)	8074(Ideal)	1019	1019	100	62.7	9.5	24
19K94-12	8075	8076	664.4	1046.04	64	60.5	8	26
19K94-13	8077	8078	676.6	1058.69	64	59.5	9	25
19K94-14	8079	8080	799.8	1065.63	75	61.1	9	26
19K94-15	8081	8082	824.8	1072.1	77	59.9	7.5	29
Check	8083(Ideal)	8084(Ideal)	1079.6	1079.6	100	62.8	8	26
Check	8085(Ideal)	8086(Ideal)	1112.4	1112.4	100	60.6	8.5	29
19K94-16	8087	8088	973.8	1103.6	88	60	8.5	28
19K94-17	8089	8090	898.8	1096.26	82	57.7	8.5	26
19K94-18	8091	8092	882.6	1089.74	81	60.5	9	31

Table A7. Agronomic traits and yield data obtained in an un-replicated yield trial that was planted at Casselton in 2020 (continued).

Entry	Row numbers		Actual plot yield (g)	Predicted check yield (g)[1]	Relative yield (%)[2]	Test weight (lbs/bu)	Winter survival (0-10)	Plant height (inches)
	1st row	2nd row						
19K97-15	8139	8140	966	1118.31	86	61.2	7.5	29
19K97-16	8141	8142	841.07	1094.17	77	59.6	7	32
Check	8143(Ideal)	8144(Ideal)	1054.4	1054.4	100	63	8	27
Check	8145(Ideal)	8146(Ideal)	1105	1105	100	61	8	24
19K97-17	8147	8148	834.1	1104.96	75	61.1	7	27
19K132-1	8149	8150	1080.2	1106.06	98	61.1	8	30
19K132-2	8151	8152	860.8	1106.75	78	60.5	7.5	31
19K132-3	8153	8154	829.6	1105.49	75	59.8	8.5	30
Check	8155(Ideal)	8156(Ideal)	1098	1098	100	62.2	8	27
Check	8157(Ideal)	8158(Ideal)	1051	1051	100	61.8	9	26
19K132-4	8159	8160	899.8	1084.75	83	61	9	27
19K132-5	8161	8162	1010.4	1096.68	92	60	9.5	27
19K132-6	8163	8164	956.2	1099.78	87	60.1	9	26
19K132-7	8165	8166	692	1098.06	63	61.9	7	25
Check	8167(Ideal)	8168(Ideal)	1088.6	1088.6	100	63.5	8.5	25
Check	8169(Ideal)	8170(Ideal)	1114.2	1114.2	100	62.1	8.5	25
19K132-8	8171	8172	773.53	1106.04	70	60.5	8.5	27
19K132-9	8173	8174	866.8	1110.06	78	60.4	8	27
19K132-10	8175	8176	694.6	1121	62	59.2	7	24
19K132-11	8177	8178	1062.8	1146.84	93	60.4	7	28
Check	8179(Ideal)	8180(Ideal)	1207.8	1207.8	100	61.9	9	28
Check	8181(Ideal)	8182(Ideal)	1152.4	1152.4	100	62.5	9.5	27
19K132-12	8183	8184	760	1114.5	68	61.5	7.5	28

Table A7. Agronomic traits and yield data obtained in an un-replicated yield trial that was planted at Casselton in 2020 (continued).

Entry	Row numbers		Actual plot yield (g)	Predicted check yield (g)[1]	Relative yield (%)[2]	Test weight (lbs/bu)	Winter survival (0-10)	Plant height (inches)
	1st row	2nd row						
19K132-13	8185	8186	841.4	1090.99	77	59.8	8.5	27
19K132-14	8187	8188	934.8	1070.35	87	60.7	9	28
19K132-15	8189	8190	823	1034.82	80	60.7	8	29
Check	8191(Ideal)	8192(Ideal)	958.8	958.8	100	62	8	27
Check	8193(Ideal)	8194(Ideal)	1058.6	1058.6	100	62.5	8.5	26
19K132-16	8195	8196	703.6	1050.05	67	60.4	9	27
19K132-17	8197	8198	915.2	1049.48	87	62.4	8.5	27
19K132-18	8199	8200	875.8	1051.48	83	61.2	8.5	28
19K132-19	8201	8202	910.2	1054.8	86	61.3	8.5	27
Check	8203(Ideal)	8204(Ideal)	1058.6	1058.6	100	62.5	9.5	26
Check	8205(Ideal)	8206(Ideal)	1045	1045	100	62.4	9	27
19K331-1	8207	8208	841.4	1035.57	81	60.5	9	28
19K331-2	8209	8210	790.6	1027.71	77	60.8	9.5	33
19K331-3	8211	8212	928.6	1021.26	91	61.4	9	28
19K331-4	8213	8214	904.4	1014.35	89	61.9	8.5	29
Check	8215(Ideal)	8216(Ideal)	1008	1008	100	63.1	8.5	25
Check	8217(Ideal)	8218(Ideal)	831.2	831.2	100	62.2	9	22
19K331-5	8219	8220	862	931.28	93	60.6	8.5	28
19K365-1	8221	8222	808.4	980	82	61.4	8	28
19K365-2	8223	8224	878.6	1011.63	87	61.4	8.5	27
19K365-3	8225	8226	883.4	1055.58	84	61.7	8	29
Check	8227(Ideal)	8228(Ideal)	1140.03	1140.03	100	62.8	8.5	27
Check	8229(Ideal)	8230(Ideal)	974.2	974.2	100	60.5	9.5	28
19K365-4	8231	8232	969.8	1005.48	96	61.3	9	30

Table A7. Agronomic traits and yield data obtained in an un-replicated yield trial that was planted at Casselton in 2020 (continued).

Entry	Row numbers		Actual plot yield (g)	Predicted check yield (g)[1]	Relative yield (%)[2]	Test weight (lbs/bu)	Winter survival (0-10)	Plant height (inches)
	1st row	2nd row						
19K365-5	8233	8234	650.6	1012.87	64	61.2	8.5	27
19K365-6	8235	8236	712	1012.12	70	61.2	7	27
19K365-7	8237	8238	756.6	1007.43	75	61	8	28
Check	8239(Ideal)	8240(Ideal)	1003.2	1003.2	100	62.7	8	24
Check	8241(Ideal)	8242(Ideal)	1122.8	1122.8	100	63.2	8.5	27
19K368-1	8243	8244	948.2	1077.07	88	59.4	8	30
19K368-2	8245/8246	8259/8260	1418.8	1098.56	129	60.4	9.5	32
19K368-3	8247/8248	8257/8258	1126.8	1110.81	101	60.1	9.5	33
19K368-4	8249	8250	873.8	1135.66	77	60.3	9	33
Check	8251(Ideal)	8252(Ideal)	1179	1179	100	60.2	8.5	27
Check	8253(Ideal)	8254(Ideal)	1065.6	1065.6	100	62.5	8.5	28
19K368-5	8255	8256	801.2	1053.2	76	59.6	8.5	30
19K368-8	8261	8262	1007.8	966.21	104	60.9	9.5	33
Check	8263(Ideal)	8264(Ideal)	928	928	100	62.5	8	24
Check	8265(Ideal)	8266(Ideal)	1011	1011	100	62.2	7.5	23
19K438-1	8267	8268	896.77	988.04	91	60.4	8	26
19K438-2	8269	8270	638.6	974.77	66	59.5	8.5	22
19K438-3	8271	8272	804.8	968.68	83	62	8.5	26
19K438-4	8273	8274	542.6	942.96	58	59.6	9	25
Check	8275(Ideal)	8276(ideal)	872.6	872.6	100	62.2	8	26
Check	8277(Ideal)	8278(Ideal)	1019.4	1019.4	100	61.9	7	25
19K438-5	8279	8280	832	989.37	84	60.9	6.5	30
19K438-6	8281	8282	636.8	980.34	65	61.4	6.5	30

Table A7. Agronomic traits and yield data obtained in an un-replicated yield trial that was planted at Casselton in 2020 (continued).

Entry	Row numbers		Actual plot yield (g)	Predicted check yield (g)[1]	Relative yield (%)[2]	Test weight (lbs/bu)	Winter survival (0-10)	Plant height (inches)
	1st row	2nd row						
19K438-7	8283	8284	643.8	980.34	66	61	6	31
19K438-8	8285	8286	869.6	979.53	89	62.6	8	29
Check	8287(Ideal)	8288(Ideal)	1001.2	1001.2	100	63.3	7.5	26
Check	8289(Ideal)	8290(Ideal)	828.4	828.4	100	62.9	8	24
19K438-9	8291	8292	870.6	855.65	102	62.1	8	34
19K438-10	8293	8294	849.2	970.8	87	61.6	8.5	30
19K438-11	8295	8296	905.22	967.21	94	60.9	8	30
19K438-12	8297	8298	939.6	963.77	97	60.2	8	29
Check	8299(Ideal)	8300(Ideal)	957.6	957.6	100	63.1	8.5	25

[1] Predicted yield of Ideal (check) at that test plot location.
[2] Actual test plot yield expressed as percentage of the predicted check yield at that test plot location

Table A8. Marker screening results for all the F_5 plants that were analyzed in the study.

S. No.	Entry #	Pedigree	Spike #	Seed #	Markers detected
1	19K89-1		1	1	*Fhb1, Lr34, Lr46, Yr17*
2		Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Broadview/SD07W083-4	1	2	*Fhb1, Lr34, Lr46, Yr17*
3			1	3	*Fhb1, Lr34, Lr46, Yr17*
4			1	4	*Fhb1, Lr34, Lr46, Yr17*
5			1	5	*Fhb1, Lr34, Lr46, Yr17*
6			2	1	*Fhb1, Lr34, Lr46, Yr17*
7			2	2	*Fhb1, Lr34, Lr46, Yr17*
8			2	3	*Fhb1, Lr34, Lr46, Yr17*
9			2	4	*Fhb1, Lr34, Lr46, Yr17*
10			3	1	*Fhb1, Lr34, Lr46, Yr17*
11			3	2	*Fhb1, Lr34, Lr46, Yr17*
12			3	3	*Fhb1, Lr34, Lr46, Yr17*
13			3	4	*Fhb1, Lr34, Lr46, Yr17*
14			4	1	*Fhb1, Lr34, Lr46, Yr17*
15			4	2	*Fhb1, Lr34, Lr46, Yr17*
16			4	3	*Fhb1, Lr34, Lr46, Yr17*
17			4	4	*Fhb1, Lr34, Lr46, Yr17*
18			4	5	*Fhb1, Lr34, Lr46, Yr17*
19			5	1	*Fhb1, Lr34, Lr46, Yr17*
20			5	2	*Fhb1, Lr34, Lr46, Yr17*
1	19K89-3	Norstar*Fhb1*/Jerry//TX09D1119/Buteo/3/Broadview/SD07W083-4	1	1	*Fhb1, Lr46, Yr17*
2			1	2	*Fhb1, Lr46, Yr17*
3			1	3	*Fhb1, Lr46, Yr17*
4			1	4	*Fhb1, Lr46, Yr17*
5			1	5	*Fhb1, Lr34, Lr46, Yr17*
6			2	1	*Fhb1, Lr34, Lr46, Yr17*
7			2	2	*Fhb1, Lr34, Lr46, Yr17*
8			2	3	*Fhb1, Lr34, Lr46, Yr17*
9			2	4	*Fhb1, Lr34, Lr46, Yr17*
10			2	5	*Fhb1, Lr34, Lr46, Yr17*
11			3	1	*Fhb1, Lr34, Lr46, Yr17*
12			3	2	*Fhb1, Lr34, Lr46, Yr17*
13			3	3	*Fhb1, Lr34, Lr46, Yr17*
14			3	4	*Fhb1, Lr34, Lr46, Yr17*
15			3	5	*Fhb1, Segr Lr34, Lr46, Yr17*
16			4	1	*Fhb1, Segr Lr34, Lr46, Yr17*

Table A8. Marker screening results for all the F_5 plants that were analyzed in the study (continued).

S. No.	Entry #	Pedigree	Spike #	Seed #	Markers detected
17			4	2	*Fhb1, Lr46, Yr17*
18			4	3	*Fhb1, Lr46, Yr17*
19			4	4	*Fhb1, Lr34, Lr46, Yr17*
20			4	5	*Fhb1, Lr46, Yr17*
1	19K89-6	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Broadview/SD07W083-4	1	1	*Lr46, Yr17*
2			1	2	*Lr46, Yr17*
3			1	3	*Lr46, Yr17*
4			1	4	*Lr46, Yr17*
5			1	5	*Lr46, Yr17*
6			2	1	*Lr46, Yr17*
7			2	2	*Lr46, Yr17*
8			2	3	*Lr46, Yr17*
9			2	4	*Lr46, Yr17*
10			2	5	*Lr46, Yr17*
11			3	1	*Lr46, Yr17*
12			3	2	*Lr46, Yr17*
13			3	3	*Lr46, Yr17*
14			3	4	*Lr46, Yr17*
15			3	5	*Lr46, Yr17*
16			4	1	*Lr46, Yr17*
17			4	2	*Lr46, Yr17*
18			4	3	*Lr46, Yr17*
19			4	4	*Lr46, Yr17*
20			4	5	*Lr46, Yr17*
1	19K94-6	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/3/Monument	1	1	*Fhb1, Segr 1B1R, Yr17*
2			1	2	*Fhb1 Yr17*
3			1	3	*Fhb1, Segr Lr46, Yr17*
4			1	4	*Fhb1, Segr Lr46, Segr 1B1R, Yr17*
5			1	5	*Fhb1, 1B1R, Yr17*
6			2	1	*Fhb1, 1B1R, Yr17*
7			2	2	*Fhb1, 1B1R, Yr17*
8			2	3	*Fhb1, 1B1R, Yr17*
9			2	4	*Fhb1, Segr 1B1R, Yr17*
10			2	5	*Fhb1, 1B1R, Yr17*
11			3	1	*Fhb1, Yr17*
12			3	2	*Fhb1, Segr 1B1R, Yr17*
13			3	3	*Fhb1, Segr Lr46, 1B1R, Yr17*

Table A8. Marker screening results for all the F_5 plants that were analyzed in the study (continued).

S. No.	Entry #	Pedigree	Spike #	Seed #	Markers detected
14			3	4	*Fhb1, 1B1R, Yr17*
15			3	5	*Fhb1, Segr Lr46, 1B1R, Yr17*
16			4	1	*Fhb1, Segr Lr46, 1B1R, Yr17*
17			4	2	*Fhb1, Lr46, 1B1R, Yr17*
18			4	3	*Fhb1, Segr Lr46, Segr 1B1R, Yr17*
19			4	4	*Fhb1, Lr46, 1B1R, Yr17*
20			4	5	*Fhb1, Lr46, 1B1R, Yr17*
1	19K132-1	Norstar-*Fhb1*/Jerry//TX09D1119/Buteo/6/	1	1	*Fhb1, Lr46, Yr17*
2		CM82036/Jerry/3/Lr50/Sup//Jerry/4/Falcon/5/Moats	1	2	*Fhb1, Lr46, Yr17*
3			1	3	*Fhb1, Lr46, Yr17*
4			1	4	*Fhb1, Lr46, 1B1R, Yr17*
5			1	5	*Fhb1, Lr46, Yr17*
6			2	1	*Fhb1, Lr46, Yr17*
7			2	2	*Fhb1, Lr46, Yr17*
8			2	3	*Fhb1, Lr46, Yr17*
9			2	4	*Fhb1, Lr46, Yr17*
10			2	5	*Fhb1, Lr46, Yr17*
11			3	1	*Fhb1, Lr46, Yr17*
12			3	2	*Fhb1, Lr46, Yr17*
13			3	3	*Fhb1, Lr46, 1B1R, Yr17*
14			3	4	*Fhb1, Lr46, 1B1R, Yr17*
15			3	5	*Fhb1, Lr46, 1B1R, Yr17*
16			4	1	*Fhb1, Lr46, Yr17*
17			4	2	*Fhb1, Lr46, Yr17*
18			4	3	*Fhb1, Lr46, Yr17*
19			4	4	*Fhb1, Lr46, Yr17*
20			4	5	*Fhb1, Lr46, Yr17*
1	19K365-4	Norstar-*Fhb1, Sr39*//Monument	1	1	*Fhb1, Lr34, Lr68, Segr 1B1R*
2			1	2	*Fhb1, Lr34, Lr68, Segr 1B1R*
3			1	3	*Fhb1, Lr34, Lr68, 1B1R*
4			1	4	*Fhb1, Lr34, Lr68*
5			1	5	*Fhb1, Lr34, Lr68, Segr 1B1R*
6			2	1	*Fhb1, Lr34, Lr68, 1B1R*
7			2	2	*Fhb1, Lr34, Lr68, 1B1R*
8			2	3	*Fhb1, Lr34, Lr68, 1B1R*
9			2	4	*Fhb1, Lr34, Lr68, Segr 1B1R*
10			2	5	*Fhb1, Lr34, Lr68, 1B1R*

Table A8. Marker screening results for all the F_5 plants that were analyzed in the study (continued).

S. No.	Entry #	Pedigree	Spike #	Seed #	Markers detected
11			3	1	*Fhb1, Lr34, Lr68, Segr 1B1R*
12			3	2	*Fhb1, Lr34, Lr68, Segr 1B1R*
13			3	3	*Fhb1, Lr34, Lr68, Segr 1B1R*
14			3	4	*Fhb1, Lr34, Lr68*
15			3	5	*Fhb1, Lr34, Lr68*
16			4	1	*Fhb1, Lr34, Lr68*
17			4	2	*Fhb1, Lr34, Lr68*
18			4	3	*Fhb1, Lr34, Lr68*
1	19K368-8	Norstar-*Fhb1, Sr39*//Keldin	1	1	*Fhb1, Segr Lr46, Lr68*
2			1	2	*Fhb1, Lr46, Lr68*
3			1	3	*Fhb1, Segr Lr46, Lr68*
4			1	4	*Fhb1, Segr Lr46, Lr68*
5			1	5	*Fhb1, Lr68*
6			2	1	*Fhb1, Lr34, Lr46, Lr68*
7			2	2	*Fhb1, Lr34, Lr68*
8			2	3	*Fhb1, Lr34, Lr68*
9			2	4	*Fhb1, Lr34, Segr Lr46, Lr68*
10			2	5	*Fhb1, Lr34, Lr46, Lr68*
11			3	1	*Fhb1, Segr Lr34, Lr68*
12			3	2	*Fhb1, Segr Lr34, Lr68*
13			3	3	*Fhb1, Segr Lr34, Lr68*
14			3	4	*Fhb1, Lr34, Lr68*
15			3	5	*Fhb1, Lr46, Lr68*
16			4	1	*Fhb1, Lr46, Lr68*
17			4	2	*Fhb1, Lr46, Lr68*
18			4	3	*Fhb1, Lr46, Lr68*
19			4	4	*Fhb1, Lr46, Lr68*
20			4	5	*Fhb1, Segr Lr34, Segr Lr46, Lr68*
21			5	1	*Fhb1, Segr Lr34, Lr68*
22			5	2	*Fhb1, Segr Lr34, Segr Lr46, Lr68*
23			5	3	*Fhb1, Segr Lr34, Segr Lr46, Lr68*
24			5	4	*Fhb1, Lr34, Segr Lr46, Lr68*
1	19K438-9	Broadview/SD07W083-4 /3/Radiant/RCATL33//Ideal	1	1	*Fhb1, Lr34, Yr17*
2			1	2	*Fhb1, Lr34, Yr17*
3			1	3	*Fhb1, Lr34, Yr17*
4			1	4	*Fhb1, Lr34, Yr17*
5			1	5	*Fhb1, Lr34, Yr17*

Table A8. Marker screening results for all the F_5 plants that were analyzed in the study (continued).

S. No.	Entry #	Pedigree	Spike #	Seed #	Markers detected
6			2	1	*Fhb1, Lr34, Yr17*
7			2	2	*Fhb1, Lr34, Yr17*
8			2	3	*Fhb1, Lr34, Yr17*
9			2	4	*Fhb1, Lr34, Yr17*
10			2	5	*Fhb1, Lr34, Yr17*
11			3	1	*Fhb1, Lr34, Yr17*
12			3	2	*Fhb1, Lr34, Yr17*
13			3	3	*Fhb1, Lr34, Yr17*
14			3	4	*Fhb1, Lr34, Yr17*
15			3	5	*Fhb1, Lr34, Yr17*
16			4	1	*Fhb1, Lr34, Yr17*
17			4	2	*Fhb1, Lr34, Segr Lr46, Yr17*
18			4	3	*Fhb1, Lr34, Lr46, Yr17*
19			4	4	*Fhb1, Lr34, Segr Lr46, Yr17*
20			4	5	*Fhb1, Lr34, Segr Lr46, Yr17*
21			4	6	*Fhb1, Lr34, Segr Lr46, Yr17*
1	19K438-12	Broadview/SD07W083-4/3/Radiant/RCATL33//Ideal	1	1	*Lr34, Lr46, Yr17*
2			1	2	*Lr34, Lr46, Yr17*
3			1	3	*Lr34, Lr46, Segr 1B1R, Yr17*
4			1	4	*Lr34, Lr46, Segr 1B1R, Yr17*
5			1	5	*Lr34, Lr46, Segr 1B1R, Yr17*
6			2	1	*Lr34, Lr46, Segr 1B1R, Yr17*
7			2	2	*Lr34, Lr46, Yr17*
8			2	3	*Lr34, Lr46, Segr 1B1R, Yr17*
9			2	4	*Lr34, Lr46, Segr 1B1R, Yr17*
10			2	5	*Lr34, Lr46, Yr17*
11			3	1	*Lr34, Lr46, Yr17*
12			3	2	*Lr34, Lr46, Yr17*
13			3	3	*Lr34, Lr46, Yr17*
14			3	4	*Lr34, Lr46, Segr Yr17*
15			3	5	*Lr34, Lr46, Yr17*
16			3	6	*Lr34, Lr46, Yr17*
17			3	7	*Lr34, Lr46, Segr Yr17*

Table A9. Disease severity of parents and selected 20M1 progenies across replications at green house.

Entry	Rep.	Disease Severity
GP80	1	0.12
	2	0.14
	3	0.13
	4	0.11
	5	0.14
	6	0.12
Avg.		**0.13**
19CP29	1	0.87
	2	0.89
	3	0.91
	4	0.89
	5	0.82
	6	0.8
Avg.		**0.86**
ND Noreen	1	0.09
	2	0.08
	3	0.2
	4	0.17
	5	0.23
	6	0.14
Avg.		**0.15**
20M1-28-10	1	0.11
	2	0.2
	3	0.17
	4	0.16
	5	0.19
	6	0.13
Avg.		**0.16**
20M1-58-8	1	0.08
	2	0.12
	3	0.14
	4	0.11
	5	0.15
	6	0.14
Avg.		**0.12**
20M1-58-10	1	0.11
	2	0.12
	3	0.11
	4	0.1
	5	0.15
	6	0.14
Avg.		**0.14**

Table A9. Disease severity of parents and selected 20M1 progenies across replications at green house (continued).

Entry	Rep.	Disease Severity
Avg.		**0.12**
20M1-12-3	1	0.09
	2	0.1
	3	0.11
	4	0.12
	5	0.1
	6	0.08
Avg.		**0.1**
20M1-12-12	1	0.08
	2	0.13
	3	0.11
	4	0.08
	5	0.13
	6	0.08
Avg.		**0.10**
20M1-23-6	1	0.14
	2	0.07
	3	0.12
	4	0.09
	5	0.08
	6	0.12
Avg.		**0.10**
20M1-58-18	1	0.11
	2	0.1
	3	0.15
	4	0.09
	5	0.17
	6	0.12
Avg.		**0.12**
20M1-58-27	1	0.1
	2	0.09
	3	0.12
	4	0.18
	5	0.1
	6	0.16
Avg.		**0.13**
20M1-58-32	1	0.1
	2	0.1
	3	0.11
	4	0.08
	5	0.09
	6	0.09
Avg.		**0.10**