DEVELOPMENT OF BARLEY INTROGRESSION LINES CARRYING
THE LEAF RUST RESISTANCE GENES *Rph1* TO *Rph15*

A THESIS
SUBMITTED TO THE FACULTY OF THE GRADUATE SCHOOL
OF THE UNIVERSITY OF MINNESOTA
BY

MATTHEW JAMES MARTIN

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
MASTER OF SCIENCE

Adviser: Brian J. Steffenson

July, 2018

Matthew Martin copyright 2018

Acknowledgements

I would like to extend thanks to Dr. Brian Steffenson and Dr. Jerry Franckowiak for providing me the opportunity to bring to fruition this long-term project. Their insight and motivation improved the quality of the research. I would also like to express gratitude to my other committee members, Dr. Kevin Smith and Dr. Melania Figueroa for their critical reviews of the thesis and general support during my program. I would like to acknowledge work done by Dr. Jerry Franckowiak, Dr. Brian Steffenson, Dr. Oswaldo Chicaiza, and Juan Caffarel in the years before my involvement with this research. Their extensive technical work spent collecting, backcrossing, and screening sources of leaf rust resistance in barley made it possible for me to carry this project to a logical end. Additionally, I would like to thank Dr. Arnis Druka and Dr. Dragan Perovic (Julius Kuehn Institute in Gatersleben, Germany) for providing the genotyping data on the barley germplasm used in this research. During my time in Dr. Steffenson's lab, I have worked with many incredible people who I owe thanks to for challenging, assisting, teaching, and letting loose with me. My utmost thanks goes out to Tamas Szinyei for being a master technician and amazing friend, Oadi Matny for being a great co-worker and father figure around the lab, Ryan Johnson for being a constant beacon of hope and energy, Austin Case for his friendship, knowledge, and comedic relief, and Matthew Haas for his comradery and witty banter. I also offer thanks to the other graduate and undergraduate students who have come through our project as I have had the pleasure to befriend, teach, and learn from each of them. I have also had the opportunity to learn from many of the faculty in the Department of Plant Pathology and would like to thank them for their dedication to the student body and the mission of the department. I give a shoutout to the department administrators for being always willing to help in any way they can. Thanks are also in order to the Minnesota Agricultural Experiment Station (MAES) and the College of Food Agriculture and Natural Resource Sciences (CFANS) for providing excellent workspaces.

Dedication

This thesis is dedicated to my wife, Annie Martin, for always being there and believing in me. A very special sincere thanks to all of my family and friends outside the lab for their relentless encouragement and support.

Table of Contents

Acknowledgements	i
Dedication	ii
List of Tables	v
List of Figures	vi
Abstract	vii
Chapter One: General Introduction and Literature Review	1
Introduction	2
Literature Review	5
Genetics of Barley	5
Biology of <i>Puccinia hordei</i>	6
Impact of Barley Leaf Rust	9
Resistance of Barley to Leaf Rust	13
Genetics of Leaf Rust Resistance in Barley	16
Bowman Introgression Lines for Leaf Rust Resistance.....	26
Chapter Two: Phenotypic and Genotypic Characterization of Barley Introgression Lines Carrying the Leaf Rust Resistance Genes <i>Rph1</i> to <i>Rph15</i>	28
Introduction	29
Materials and Methods	32
Plant Materials and Development of Introgression Lines	32
Pathogen Isolates and Race Nomenclature	33
Experimental Design	35
Inoculation and Disease Phenotyping Protocols	35
Genotyping the Bowman Introgression Lines and Resistance Donors	38
Selection of Bowman Introgression Lines for the Leaf Rust Differential Set	40
Results and Discussion	40
Disease Phenotyping	40
Genotyping Resistant Donor Sources and their Derived Introgression Lines	42
Phenotypic and Genotypic Characterization of Introgression Lines Representing <i>Rph1</i> to <i>Rph15</i>	43
<i>Rph1.a</i>	43
<i>Rph2.t</i>	43
<i>Rph3.c</i>	45
<i>Rph4.d</i>	47
<i>Rph5.e</i> and <i>Rph5.f</i> (<i>Rph6</i>).....	48
<i>Rph7.g</i>	50
<i>Rph8.h</i>	51
<i>Rph9.i</i> and <i>Rph9.z</i> (<i>Rph12</i>)	51

<i>Rph10.o and Rph11.p</i>	53
<i>Rph13.x</i>	55
<i>Rph14.ab</i>	56
<i>Rph15.ad</i>	56
Other Leaf Rust Resistance Genes	58
<i>Rph</i> Gene Deployment and Effectiveness	62
Candidate Leaf Rust Resistance Genes	66
Summary	67
Literature Cited	88

List of Tables

Table 1: Accession details for Bowman and the 15 selected parental donors of leaf rust resistance	70
Table 2: Pedigree details for the <i>Rph1-15</i> introgression lines selected for the barley leaf rust differential set	71
Table 3: Race nomenclature of <i>Puccinia hordei</i> isolates used to select for leaf rust resistance genes <i>Rph1-15</i> in Bowman introgression lines.....	72
Table 4: Median infection types and general reactions of barley differential lines carrying leaf rust resistance genes <i>Rph1-24</i> to 12 global isolates of <i>P. hordei</i> ..	73
Table 5: Median infection types and general reactions of the resistant donor parents for <i>Rph1-15</i> and their derived Bowman introgression lines selected for the barley leaf rust differential set.....	74
Table 6: Infection type mode and range for the selected Bowman introgression lines carrying the leaf rust resistance genes <i>Rph1-15</i> against 12 isolates of <i>Puccinia hordei</i>	76
Table 7: Annotated genes involved in disease resistance and residing in the same contigs as BOPA SNP markers within Bowman introgression lines carrying nine of the 15 leaf rust resistance alleles used in this study.....	77
Appendix Table 1: Accession details for the 24 barley leaf rust differential lines used in this study.....	100
Appendix Table 2: Accession details for the barley cultivar Bowman and the 95 donors of leaf rust resistance used in this study	101
Appendix Table 3: Pedigrees and recognized <i>Rph</i> genes in the 95 Bowman introgression lines used in this study	106
Appendix Table 4: Details for the 12 <i>Puccinia hordei</i> isolates used to characterize 95 leaf rust resistant introgression lines in Bowman	111
Appendix Table 5: Raw infection types of Bowman introgression lines postulated to carry <i>Rph2</i> , <i>Rph3</i> , <i>Rph4</i> , <i>Rph5</i> , <i>Rph9</i> , or unknown resistance gene(s)	112
Appendix Table 6: Raw infection types of 50 Bowman introgression lines postulated to carry <i>Rph15</i>	116
Appendix Table 7: Raw infection types for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.....	120
Appendix Table 8: Raw infection types of barley differential lines carrying the leaf rust resistance genes <i>Rph1-24</i> in this and a previous study	128
Appendix Table 9: Annotated genes in the same contig as SNP markers residing in <i>Rph</i> -carrying introgressions.....	135

List of Figures

Figure 1: Examples of leaf rust infection types (ITs) observed on Bowman introgression lines.....	79
Figure 2: Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes <i>Rph1-15</i> selected for the barley leaf rust differential set	80

Abstract

Barley (*Hordeum vulgare*) is the fourth most planted and harvested grain crop in the world. In many production areas, barley is attacked by the leaf rust pathogen (*Puccinia hordei*), a basidiomycetous fungus that reduces both its yield and quality. Many leaf rust resistance genes, known as reaction to *P. hordei* (*Rph*) genes, have been described in barley. Resistance deployed in cultivars has often been transient due to the pertinacious nature of single gene virulence mutations in *P. hordei* populations. To differentiate genetic variants for virulence in *P. hordei* populations, plant pathologists use barley accessions with different resistance genes, referred to as a differential set. The barley leaf rust differential set evolved through time as more *Rph* genes were described. The sources of *Rph1-15* were derived from barleys with diverse geographic origins and agro-morphological traits. Ideal differential sets include lines where single resistance genes are backcrossed into the genetic background of an adapted susceptible cultivar. Sources for *Rph1-15*, along with many other *Rph* gene donors were backcrossed to the susceptible cultivar Bowman. Overall, 95 Bowman introgression lines for leaf rust resistance were developed, and a single line was recommended for use as the new backcrossed differential host line for each individual *Rph1-15* gene. Genomic positions and gene candidates for *Rph1-15* were characterized and the existence of possible new resistance genes was postulated. The new Bowman *Rph1-15* differential lines will facilitate the efficient virulence phenotyping of *P. hordei* and serve as valuable genetic stocks for *Rph* gene stacking and cloning in barley.

CHAPTER 1

General Introduction and Literature Review

INTRODUCTION

Barley (*Hordeum vulgare* L.) is the fourth most planted and harvested cereal grain crop in terms of global production behind wheat, corn, and rice (USDA, 2017). It is an important feedstuff for animals and essential raw ingredient for the brewing industry. Barley is desirable as a cultivated crop due to its shorter maturation period and ability to yield well under stressful environments such as extreme elevation, cold, and saline soils where other cereal grains cannot produce (Ullrich, 2011). Despite these advantages, barley is subject to the biotic stresses from both herbivores and pathogens capable of targeting all types of tissue. Leaf rust, a disease caused by the fungal plant pathogen *Puccinia hordei* Otth, is very common throughout the barley production regions of the world. Although leaf rust rarely destroys entire crops, it does cause enough damage to regularly challenge the economic sustainability of barley production and therefore merits the need for control (Newton et al., 1945; King & Polley, 1976; Johnson & Wilcoxson, 1979; Whelan et al., 1997; González et al., 2013). Deploying cultivars with genetic resistance to leaf rust is the most economically and environmentally sound means of managing the disease.

Over the past century, barley accessions from around the world have been reported to carry resistance to *P. hordei*. Currently, 26 major-effect leaf rust resistance genes (referred to as reaction to *P. hordei* (*Rph*) genes), have been described in *Hordeum* (Franckowiak et al., 1997; Park et al., 2015; Kavanagh et al., 2017; Ziems et al., 2017, Yu et al., In Press). Among these described genes, *Rph3*, *Rph7*, *Rph9*, and *Rph12* have been commonly used in breeding programs because they confer a high level of resistance, are broadly effective against different pathotypes of *P. hordei*, and exist in agronomically suitable genetic backgrounds. The use of these resistance genes in widely grown barley cultivars provided decades of effective leaf rust control in North America, Europe, and

Australia until virulence finally evolved in populations of *P. hordei* (Cotterill et al., 1992b; Steffenson et al., 1993; Dreiseitl and Steffenson, 2000; Park, 2003; Sandhu et al., 2016). The repeated episodes of resistance genes being overcome have increased the economic pressure of the disease on barley, warranting investment of more resources for control. By understanding the virulence diversity of regional *P. hordei* populations, pathologists can make informed decisions as to which combinations of *Rph* genes might provide more long-lasting leaf rust resistance. To this end, encouraging resistance gene stewardship could improve the economic and environmental sustainability of barley cropping systems.

Puccinia hordei is classified into different pathotypes (a.k.a. races) based on its infection type reactions or (ITs) across a panel of diverse barley lines that carry different resistance genes. Of the 26 described *Rph* genes, 17 were identified in barley cultivars or landraces of diverse origins, five from the barley progenitor *H. vulgare* subsp. *spontaneum*, and four from the secondary gene pool species *Hordeum bulbosum* L. Characterization of the original genetic stocks carrying *Rph1-25* embodied an immense amount of research, and this germplasm remains a useful resource for studying pathogen virulence. To characterize virulence diversity in pathogen populations, many pathologists have established sets of differential host lines, each presumed to carry a different resistance gene (Stakman et al., 1962; Person, 1976; Clifford, 1977). While such selected host lines have adequately served the purpose of typing virulence in the pathogen, they do have some limitations. First, because the lines have not always been genetically characterized for their resistance, some may actually carry more than one resistance gene. In such cases, avirulence genes in the pathogen will remain cryptic if their corresponding resistance gene is not present alone in a differential line. Moreover, epistasis within the host lines may confound the phenotypic expression of some resistance genes. The development of single

gene introgression lines in a uniform genetic background was a major advance for the efficient typing of pathogen virulence. With such a set of host lines, avirulence genes in the pathogen can be efficiently characterized without the problem of cryptic genes or genetic background effects. The use of an agronomically advanced recurrent parent for the differential set will facilitate experiments in the field because the lines will grow well and have uniform traits such as height and heading date. Introgression lines each possessing a single resistance gene in a uniform genetic background have been developed in a number of pathosystems including wheat leaf rust, wheat stripe rust, barley powdery mildew, and barley loose smut (Tapke, 1945; Clifford, 1977; Roelfs and Martens, 1988; Afanasenko et al., 2009).

Many researchers have investigated the genetics of leaf rust resistance in barley (Roane & Starling, 1967; Tan, 1978; Jin et al., 1993; Park & Karakousis, 2002). Mapping resistance gene loci can clarify intergenic relationships and enables the identification of morphological traits and/or molecular markers that are linked to the *Rph* genes. The advent of molecular marker technologies (Amplified Fragment Length Polymorphisms [AFLP], Restriction Fragment Length Polymorphisms [RFLP], isozyme, Single Nucleotide Polymorphisms [SNP], and Diversity Array Technologies [DArT], Weerasena, 2003; Ivandic et al., 1998; Druka et al., 2011; Golegaonkar et al., 2009) greatly facilitated the efficient mapping of *Rph* genes in barley. Moreover, the identification of mapping positions and molecular markers for *Rph* genes can facilitate the selection and transfer of resistance genes more efficiently than standard rust phenotyping in breeding programs. To develop a set of differential host lines for typing virulence in *P. hordei*, Chicaiza (1996) and Caffarel (2005) backcrossed the donor sources of *Rph1-15* into the susceptible two-rowed feed barley cultivar Bowman (PI 483237) (Franckowiak et al., 1985). The Bowman introgression lines were created to provide barley pathologists with a refined set of single gene host lines

for efficiently characterizing the virulence diversity of *P. hordei*. The introgression lines can also serve barley breeders as advanced genetic stocks for transferring the *Rph* genes into breeding germplasm with minimal linkage drag. (Franckowiak et al., 1985).

The objectives of this study were: 1) Use multi-pathotype testing to postulate the resistance gene identities of leaf rust resistance donors and introgression lines, 2) Use genotypic data to position the *Rph* gene-containing introgressions in the genome, and 3) Define a final differential set comprised of Bowman introgression lines for the barley leaf rust resistance genes *Rph1-15*.

LITERATURE REVIEW

Genetics of Barley

Barley is closely related to bread wheat (*Triticum aestivum*), rye (*Secale cereale*), oats (*Avena sativa*), and stiff brome (*Brachypodium distachyon*), all of which are members of the *Triticeae* tribe of the subfamily *Pooidae* in the grass family *Poaceae*. Other common cereals like rice (*Oryza sativa*) and corn (*Zea mays*) belong to other distantly related subfamilies. The global importance of these crops is driving research into their genetic sequences and genomic structures. This information allows for the interpretation of biological gene functions, interspecies relatedness, and evolutionary history.

The barley genome sequence is now assembled and physically sorted (Mayer et al., 2012; Mascher et al., 2017). It is comprised of 5,100 million base pairs (Mb) spread across seven haploid chromosomes ($2n=2x=14$). Like other plants, more than 80% of the barley genome consists of repetitive non-coding retrotransposon sequences (Ullrich, 2011). Less than 20% of the remaining

nucleotides hold an estimated 26,159 gene sequences that are unevenly distributed among the seven barley chromosomes, designated 1H to 7H (Mayer et al., 2012). Barley's diploid genome is larger and more diverse than rice (430 Mb) or corn (2,400 Mb), but smaller and simpler than bread wheat, which is an allohexaploid (17,000 Mb) (Gale and Devos, 1998). Genetic sequences from simpler model species can be applicable to more complex species. For example, an annotated homologous sequence in rice was used to hypothesize the function of a gene from a leaf rust resistant barley (Chen et al., 2010). Understanding homologous sequences between different species better informs how each species-specific gene has evolved and functions. In this regard, the discovery of disease resistance genes is of particular interest to pathologists and crop producers because of their use in agriculture.

Biology of *Puccinia hordei*

Puccinia hordei is the causal organism of barley leaf rust. It is one of 7,000 species described in the genus *Puccinia*. This genus is one of 100 genera in the basidiomycete family *Pucciniaceae*, the third largest family of fungi (Maier 2003). Other Latin binomials such as, *Uromyces hordei* P. Nielsen (1875), *Puccinia anomala* Rostr. (1878), and *Puccinia simplex* Erikss. & E. Henn. (1894), have been used in the past for this organism, but *Puccinia hordei* Otth (1871) has been accepted as the proper binomial for this organism (Stevenson and Johnson, 1946). Other common disease names such as brown rust or dwarf rust were frequently used in the older research works. According to a survey of the journal community for *Molecular Plant Pathology*, the *Pucciniaceae* are collectively considered the third most important fungal plant pathogens behind *Magnaporthe oryzae* (rice blast) and *Botrytis cinerea* (gray mold) (Dean et al., 2012).

Like many other *Puccinia* species attacking cereal crops, *P. hordei* is a heteroecious and macrocyclic rust pathogen. It is a highly specialized biotrophic pathogen that infects less than 20 plant species within a few genera. This host range is extremely small compared to a generalist plant pathogen like *B. cinerea* that can infect more than 200 plant species (Mains & Jackson 1924; D'Oliveira, 1949; Williamson et al., 2007). *Puccinia hordei* is heterothallic and therefore requires individuals of different mating types to fuse before undergoing sexual recombination. Due to its heteroecious nature, *P. hordei* requires two different genera of hosts (*Hordeum* and *Ornithogalum* species) in order to complete its life cycle. Urediniospores, teliospores, and basidiospores form on the *Hordeum* hosts, whereas pycniospores and aeciospores form on the *Ornithogalum* hosts. The optimal temperature for *P. hordei* growth ranges from 15 to 25°C. Like other cereal rust pathogens, *P. hordei* requires free moisture or high humidity to induce urediniospores germination and subsequent infection through leaf stomata (Clifford, 1985).

The life cycle of this pathogen begins with the gametophytic phase as teliospores germinate to produce spore-bearing basidia. The emergent haploid basidiospores are disseminated by wind, water, or insects to cause primary infection on the common Star-of-Bethlehem lily, (*O. umbellatum* L.) and other species of *Ornithogalum* such as *O. pyrenaicum* L., *O. narbonense* L., or *O. brachystachys* C. Koch (Mains and Jackson, 1924; D'Oliveira, 1961; Anikster & Wahl, 1979). The resulting spermagonia produce haploid pycniospores in pustules on the adaxial leaf surface. This spore is usually spread by insects and can only cause secondary re-infection on *Ornithogalum*. When pycnia of one mating type within a pustule are paired with pycnia of a different mating type from another pustule, their hyphae may fuse and produce the dikaryotic phase (N + N) of the fungus. Though this is considered a fertilization event, the two haploid nuclei do not recombine inside the hyphae, but rather remain as two distinct

haploid nuclei in a dikaryotic state. The newly formed dikaryotic hyphae proceed to grow downward through the mesophyll cells of the leaf and sporulate on the abaxial leaf margin to produce aeciospores. These aeciospores are wind-borne and do not re-infect *Ornithogalum*, but instead cause primary infections on cultivated (*H. vulgare*) and wild barley, *H. vulgare* subsp. *spontaneum* (Mains & Jackson, 1924; D'Oliveira, 1949). A few other species (*H. murinum* L., *H. chilense* Roem. & Schult., *H. bulbosum* L., and *Triticum aestivum* L.) can be infected by *P. hordei*, but are generally considered as non-hosts or near non-hosts (Mains, 1930; Straib, 1937; Rubiales & Niks, 1992). Infection of the grass hosts by aeciospores is the beginning of the sporophytic phase and results in the eruption of pustules bearing dikaryotic urediniospores on both adaxial and abaxial surfaces of the leaf. This phase of the life cycle is most prolific in terms of spore production because multiple generations of urediniospores can re-infect the barley host causing economic damage.

A urediniospore in contact with a susceptible host can germinate within ten hours when the environment is conducive (15 to 25°C and 80% or more humidity). A germ tube emerges from the urediniospore and grows across the leaf surface in search of stomata. If a stomate is found, the fungus forms an appressorium above the pore. The fungus enters the plant via a penetration peg and the resulting hypha forms a substomatal vesicle. Within 24 hours, most substomatal vesicles produce at least one haustorium, a specialized cell that penetrates the plant cell wall and interfaces with the plant cell membrane. Successful infection sites are often comprised of at least eight haustoria that secrete effector molecules into plant cells to facilitate parasitization. Using these acquired nutrients, infective hyphae grow and ramify to neighboring plant cells and begin the formation of uredinia bearing urediniospores (Niks, 1983; Chen et al., 2010). As mentioned above, urediniospores can re-infect barley multiple times during the growing season. However, as the crop approaches maturity, the

fungus begins to produce teliospores within the uredinia. This marks the full life cycle rotation for the fungus.

Impact of Barley Leaf Rust

Barley leaf rust rarely results in total crop loss, but infections can regularly reduce yields by 30% in moderate epidemics and by more than 60% in severe epidemics (Newton et al., 1945; Melville & Griffin, 1976; Johnson & Wilcoxson, 1979; Cotterill et al., 1992a; Griffey et al., 1994; Whelan et al., 1997; Woldeab et al., 2007). Besides yield loss, other negative impacts of the disease include reduced tillering, green leaf area, root volume, straw integrity, and most importantly reductions in grain quality (Newton et al., 1945; Whelan et al., 1997; González et al., 2013). Even in cases where yield loss is minimal, leaf rust can reduce grain quality from malting to feed grade, nullifying the premium paid to producers by the malting industry. Frequent dockage of barley from malting to feed grade may discourage future plantings of the crop by producers. Although fungicide use can protect the crop from leaf rust infection, the number of applications needed is generally not economical in many production systems. Leaf rust continues to challenge the sustainability of barley farming and merits research to quantify its impact and develop effective control measures.

The cosmopolitan nature of leaf rust has become more apparent as barley plantings have increased worldwide through the twentieth century. In the Western Hemisphere from Canada to Argentina, barley is subject to leaf rust infection. In Canada, Newton et al. (1944) found that barley not protected by sulfur dust suffered 24% yield loss from leaf rust infection, and the main component for loss was reduced kernel weight. Buchannon and Wallace (1962) recorded leaf rust severities as high as 32-53% on the barley cultivar Parkland in Canada. They considered leaf rust to be economically important in their experiments even

though other foliar diseases were more common at the time. In field experiments in the United States, Johnson and Wilcoxson (1979) reported back-to-back yield losses of 35% in 1976 and 42% in 1977. Soon after, Andres et al. (1983) discovered that the severe leaf rust epidemics of 1979 and 1981 in the Upper Midwest US were coincident with an evolutionary shift for greater virulence in the pathogen population. Griffey et al. (1994) investigated the effects of leaf rust on winter barleys in Virginia after another virulence shift in the *P. hordei* population. They recorded a maximum yield loss of 32% and estimated that for every incremental percent increase in leaf rust severity, yield was reduced by 0.42%. Griffey et al. (2004) also commented that the price paid for feed barley made applying fungicides for leaf rust control uneconomical. In another study in Virginia, Das et al. (2007) reported yield losses up to 33% due to leaf rust. They also found that in most cases, a single application of a triazol fungicide (Triadimefon) could adequately protect the crop from the disease. In Mexico, leaf rust has become an increasingly important factor limiting barley production. For example, Gonzalez et al. (2013) reported losses up to 30% in the cultivar Esmeralda, and the harvested grain could not be marketed as premium malting barley. They also noted that a single application of a triazole-based fungicide can control the disease, but this management strategy became uneconomical if more than one application was ever required. Pereyra and Ackerman (2007) observed leaf rust yield losses up to 25% in Uruguay. Although the fungicides used in this study were effective in controlling the disease, even a single treatment was not economical. As summarized above, yield losses due to leaf rust in North and South America were comparable ranging from 24-33% and in only a few cases was fungicide use considered economical.

Barley leaf rust is just as pervasive and damaging throughout the Eastern Hemisphere as it is in the Western Hemisphere. According to King (1972), England's barley plantings increased from 0.86 to 2.14 million hectares from 1957 to 1967, a time concurrent with widespread and high leaf rust infection, reaching an average severity of 12% on the flag leaf. From this foliar disease survey, King (1972) calculated that for every 1% severity of flag leaf infection, the yield was reduced by 0.66% and for every 1% severity on the penultimate leaves, the yield was reduced by 0.5%. These estimates are higher than the 0.42% incremental loss of yield for each 1% severity on the upper two leaves calculated by Griffey et al. (1994) in the US. In England, King and Polley (1976) corroborated these values and found that, for every 1% rust severity on the flag leaf, yield was lowered by 0.6%, equal to 27.6 kilograms/hectare. Melville and Griffin (1976) reported that leaf rust infections on barley in England reached epidemic levels in 1971, and that for every 1% increase in rust severity on the penultimate leaves caused a yield reduction of 0.77%. In their study, an excessive fungicide regimen of eight applications increased yield 17 to 31% compared to untreated plots with the increase being mostly due to higher grain weight.

Leaf rust also impacts barley producers in Eurasia and Africa. Although reports of outbreaks are common, specific estimates regarding yield losses are rare (Wilten, 1953; Martínez et al., 2001; van Niekerk et al., 2001; Woldeab et al., 2006; Sadravi et al., 2007). Rubiales et al. (1992) noted a leaf rust epidemic in Spain in 1989; Sadravi et al. (2007) reported a 1 million metric ton (MMT) yield loss in Iran in 1993; van Niekerk et al. (2001) noted an epidemic in South Africa in 1999; and Woldeab et al. (2006) estimated a 28% yield loss in Ethiopia in 2003. Although no yield loss studies were conducted recently in Germany or the former USSR, literature on this pathosystem is voluminous, indicating the importance of the disease (Straib, 1937; D'Oliveira, 1961; Grunewaldt-Stocker,

1983; Khokhlova, 1982; Nover & Lehmann, 1974; Shelko, 1974; Tan, 1977). In contrast to the above reports, Wilten (1953) stated that, at that time, leaf rust was not of economic concern in the Netherlands, which could perhaps be attributed to the effective disease resistance of their local barley cultivars at that time.

Compared to other regions in the world, it appears that barley in New Zealand and Australia is most impacted by leaf rust (Arnst et al., 1979; Lim & Gaunt, 1986; Cotterill et al., 1992a; Whelan et al., 1997; Murray & Brennan, 2010). In New Zealand, Arnst et al. (1979) observed widespread incidence of the disease and reported that frequent epidemics raised concerns about its economic impact in this region. After studying the pathogen population and its effect on the crop, he estimated that up to 21% yield loss was incurred, mainly due to lower grain weight. Also in New Zealand, Lim and Gaunt (1986) evaluated the effects of leaf rust and powdery mildew co-infections on the popular susceptible cultivar Zephyr. They found that late infection of leaf rust reduced yield by 10%, early infection of leaf rust reduced yield by 32%, and a late co-infection of both leaf rust and powdery mildew reduced yield as much as 52%. Whelan et al. (1997) also investigated yield loss in barley due to leaf rust in New Zealand and found the highest percent loss (63%) ever recorded in 1987 with a lower but still significant loss of 38% during the following year. Early leaf rust infections reduce yield more and do so by reducing tiller numbers, while later infections reduce yield less markedly by lowering the total number of grains and grain weight (Whelan et al., 1997). Similar to the findings in New Zealand, Cotterill et al. (1992a) in Australia recorded yield losses as high as 62% in the barley cultivar Gus during a moderate to severe leaf rust epidemic in 1990. Murray and Brennan (2010) organized coordinated disease surveys across the four major barley-producing regions in Australia to determine the value of disease control. From this work, leaf rust was ranked as the fourth most economically important disease of barley, causing an average yield loss worth 21 million Australian dollars. This average

dollar loss estimate was based on just a 2% yield reduction nationwide. Deployment of resistant cultivars saved an average of 47 million Australian dollars per annum for producers, providing a solid case for the importance of resistance breeding over the use of fungicides.

The economic importance of barley leaf rust worldwide is evident. Reductions in grain quality are often more devastating to producers than losses in grain yield. If long-lasting (durable) resistant cultivars can be developed, the need for expensive fungicides could be decreased, and producers could reap higher returns on their barley crops. While there are some cases of cultivars remaining resistant to leaf rust for decades, many varieties carrying single resistance genes have been rendered susceptible in just a few short years by genetic changes in *P. hordei* populations.

Resistance of Barley to Leaf Rust

Barley, wheat, and oat all diverged from a common ancestor 7 – 9 million years ago (mya) (Middleton et al., 2014). *Puccinia* species were probably among the first biotrophic fungal species to infect them as fossil evidence dates basidiomycete evolution to around 300 mya (Tiffney and Barghoorn, 1974, Krings et al., 2011). This lineage of fungal pathogens appears far more ancient than the cereal hosts and many other plants they infect today. Different types of rust can infect plants from relatively ancient to recently derived lineages. For each of the many thousand species of *Puccinia* to successfully co-evolve with all of these plants, a co-evolution model predicts that as more plant species emerged, the first rusts able to infect them were plurivorous, able to infect all individuals of that species. As the barley lineage flourished, diversifying selection for leaf rust resistance would encourage the evolution of many plants with different resistance factors. This has been predicted in the co-evolution model and also observed in

barley germplasm, as the catalog of known leaf rust resistance genes is much larger than genes for stem or stripe rust resistance (Anikster, 1989; Heath, 1991; Tosa, 1992). The host-parasite gene-for-gene concept first described in the flax rust pathosystem by H. H. Flor (1942) explains the genetic basis for plant-pathogen interactions, declaring that a gene product in the host plant can detect a gene product in the pathogen. Jones and Dangl (2006) later reviewed and summarized gene-for-gene concepts into a more advanced model of multifaceted plant immunity mechanisms. A plant's ability to mitigate microbial invasion encourages microbes to evolve ways to avoid detection by the plant. A theory about general plant-pathogen coevolution exists and is called the "arm's race theory". As plants evolve ways to recognize and respond to pathogens, in turn, pathogens evolve ways to down regulate plant defense responses and avoid recognition.

Against biotrophic pathogens like the rusts, there are two general classifications of host resistance: race-specific and non-race-specific, each thought to be controlled by different groups of genes. Both types of resistance can be expressed at either the seedling stage, the adult plant stage, or both stages (Berloo et al., 2001; Qi et al., 1998). Non-race-specific disease resistance is also referred to as partial resistance or quantitative minor effect resistance and often comprised of many coordinated genes each with their own small effect on general disease level. This type of resistance, conferred by quantitative trait loci (QTL), often restricts many pathogen races by reducing infection frequency, slowing the growth rate of infection, and decreasing spore productivity (Neervoort and Parlevliet, 1978). QTL for partial leaf rust resistance in barley have been thought to co-localize with genes encoding receptor-like kinases, wheat-induced-resistance (WIR1) homologues, and other genes like peroxidases, superoxide dismutase, and thaumatin, all of which have antimicrobial properties (Chen et al., 2010; Marcel et al., 2007). Some minor effect QTL have even been precisely

mapped close to annotated genes for defense response. Chen et al. (2010) hypothesized that a minor effect QTL for leaf rust resistance was modulating the expression of a defense response gene. Subedi (2017) recently discovered that this same minor effect QTL could be a kinase gene potentially involved in disease response signaling. Results from Chapter 2 also revealed the potential role of similarly annotated genes in leaf rust resistance.

Race-specific disease resistance genes exhibit a qualitative type of resistance against only specific races of the pathogen. These genes allow the plant to genetically recognize protein products of an avirulence gene secreted by the pathogen's haustoria (Ellis et al., 2014). Oftentimes, resistance is achieved after the plant molecularly senses the pathogen and signals a defense response. The role of a pathogen's effector molecule is to silence a plant's ability to signal the defense response (Dodds and Rathjen, 2010). If the plant's defense is mounted successfully, disease severity is reduced most commonly by the hypersensitive (HR) cell death response, which engulfs biotrophic pathogens like the rusts, choking their access to living host tissue. To achieve the HR response, different race-specific resistance factors can collaborate to detect microbial activity, bind pathogen avirulence molecules, and/or initiate signal transduction pathways that result in localized cell death (Jones and Dangl, 2006; Dodds and Rathjen, 2010; Ellis et al., 2014). A study by Bernardo et al. (2012) compared the disease resistance proteomes between Bowman and the *Rph15* Bowman introgression line (BW719) used in this study (Chapter 2). They discovered a group of 18 genes that were significantly differentially expressed upon infection by *P. hordei*, and the coordinated functions of these genes upregulated a defense response and diverted energy metabolism towards mounting this defense response.

In total, 26 barley leaf rust resistance loci have been identified across the barley genome (Park et al., 2015; Kavanagh et al., 2017; Ziemis et al., 2017, Yu et al., In Press). Initially referred to as *Puccinia anomola* (*Pa*) genes, these leaf rust resistance genes are now designated as reaction to *Puccinia hordei* (*Rph*) genes, reflecting the change in the accepted nomenclature for the pathogen (Franckowiak et al., 1997). Each unique *Rph* gene in the plant may interact with a unique avirulence gene in the pathogen. In any barley-producing region, the genetic diversity of the local pathogen dictates the effectiveness of an *Rph* gene. A group of barley accessions each carrying a different *Rph* gene is referred to as a differential set and is used to study virulence diversity in *P. hordei* populations.

Genetics of Leaf Rust Resistance in Barley

Inspired by previous genetic work in other cereal rust pathosystems, Waterhouse (1927) in Australia initiated the first study on the genetics of leaf rust resistance in barley. Through his investigations with several barley crosses, Waterhouse observed Mendelian inheritance of single completely dominant resistance genes. In the United States, Mains (1930) screened barley cultivars and various species of *Hordeum* to two different isolates of *P. hordei* and postulated that different resistance genes may exist in the germplasm and that diversity in the pathogen may be useful in detecting these genes. Researchers in other countries such as Canada, Germany, Argentina, and England investigated the resistance of hundreds of different barley accessions to local isolates of *P. hordei* and reported similar results with respect to diversity for resistance in the host and for virulence in the pathogen (Brown, 1931; Hey, 1931; Mains and Martini, 1932; Hirschhorn, 1933; Ronsdorf, 1935; D'Oliveira, 1939).

Continuing the research of Waterhouse (1927), Watson and Butler (1947) described the first two leaf rust resistance genes (i.e. *Pa* genes for *Puccinia anomala*, the old taxonomic epithet for *P. hordei*) from barley: *Pa1* from Minn. II 21.15 (Smooth Awn x Manchuria) and *Pa2* from No. 22 (Weider, CIho 1021). Henderson (1945) also studied the genetics of leaf rust resistance in Weider and reported a single incompletely dominant gene, which he assigned as *Pa*, inconsistent with Watson and Butler's previous designation for the gene in Weider. Henderson also found a different gene present in the cultivar Estate (CIho 3410; PI 57700) and designated it as *Pa₁*. In Canada, Zloten (1952) developed genetic populations with *Rph2*-carrying lines (Kwan, Ricardo, and CI 4219) reported to be resistant to Minnesotan *P. hordei* isolates in Henderson's work. He discovered evidence for single gene factors in Kwan and Ricardo, plus evidence for multiple resistance factors being present in CI 4219.

Stakman and his co-workers commented that the different genotypes used in various countries to study *P. hordei* diversity made it practically impossible to compare results (Stakman et al., 1933). This led to a study by Levine and Cherewick (1952) to determine which barley accessions, out of all previously reported ones used to study pathogen diversity around the world, were essential for separating North American isolates of *P. hordei* into different races. They found that nine selected barley lines differentiated the maximum number of *P. hordei* races. Those lines were Speciale (CIho 7536), Sudan (CIho 6489), Oderbrucker (CIho 1272), Reka I (CIho 5051), Gold (CIho 1145), Lechtaler (CIho 6488), Quinn (CIho 1024, PI 39401), Bolivia (CIho 1257, PI 36360), and Egypt 4 (CIho 6481). The efficiency of these nine genotypes in differentiating North American isolates of the pathogen led Roane (1962) to presume the existence of additional leaf rust resistance loci in barley than those few already described. Moreover, inconsistencies in the naming of resistance loci among several research groups (Waterhouse, 1927, Watson and Butler, 1947, and Henderson,

1945) also prompted Roane (1962) to study the genetics of the nine differential barley accessions selected by Levine and Cherewick (1952). Roane (1962) investigated the genetics of leaf rust resistance in the nine differential barleys by crossing them all to the susceptible cultivar Moore (CIho 7251). He also intercrossed all of the differential lines for tests of allelism. From this work, Roane (1962) detected six leaf rust resistance loci. Subsequently, Roane and Starling (1967) conducted genetic studies on other leaf rust resistant lines described by Henderson (1945) and Watson and Butler (1947) to provide comprehensive information on the identity of the six *Pa* genes they suspected to reside in the germplasm. According to their results, *Pa*₁ was present in Oderbrucker, an accession carrying the same allele as Minn. II 21.15, Especiale, and Sudan; *Pa*₂ was present in Weider, Reka I, Quinn, and Bolivia; *Pa*₃ was present in Estate; *Pa*₄ was present in Gold and Lechtaler; *Pa*₅ was present in Quinn; and another gene, tentatively designated *Pa*_x, was present in Bolivia, but had not yet been distinguished from *Pa*₅, the gene in Quinn. These accessions became the first genetic standards for future research on leaf rust resistance genes in barley.

As the discovery of new leaf rust resistance genes continued, distinguishing among them became more challenging. After the characterization of the first six genes by Roane and Starling (1967), a new gene, *Pa*_γ was postulated to be present in Cebada Capa (CIho 6193, PI 539113) and other accessions based on their much broader resistance spectrum against various races of *P. hordei* (Johnson, 1968; Dillard and Brown, 1969). Roane and Starling (1970) performed allelism tests between Cebada Capa and the previously described sources of *Pa* genes. After observing a lack of susceptible progeny in the F₂ generation of a Cebada Capa x Quinn cross, they concluded that *Pa*_γ was not a unique locus, but rather an allele at the *Pa*₅ locus. They also mentioned the possibility that *Pa*_γ and *Pa*₅ could actually comprise two very tightly linked loci, but the number of progeny needed to resolve this in an allelism test would be

much higher. Furthermore, after observing segregation for leaf rust resistance in a cross between Cebada Capa and Bolivia, they designated Pa_X as a new gene, Pa_6 . If Pa_Y had initially been described as a unique gene, it would have been Pa_6 , and then the Pa_6 gene named by Roane and Starling (1970) would likely have been considered an allele of Pa_5 . Besides the linkage between Pa_5 , Pa_X , and Pa_Y , Roane and Starling (1970) also reported a possible linkage between Pa_3 and Pa_5 . Although they clarified the genetic identities of Pa_{1-5} , their conclusions about Pa_X and Pa_Y were soon challenged.

More robust genetic approaches became necessary to resolve, with more confidence, the newly described leaf rust resistance genes. Around the same time as the genetic investigations by Roane and Starling (1970) in the United States, Frecha (1970) in Argentina published the discovery of a new gene he designated as Pa_Y , but it was from a different barley line called Forrajera Klein. In contrast to the results of Roane and Starling (1970), the cross between Forrajera Klein and Quinn yielded segregation in the F_2 generation, indicative of tight linkage between Pa_Y and Pa_5 (Frecha, 1970). Based on these results, Nover and Lehman (1974) first designated the gene in Forrajera Klein as Pa_7 . Parlevliet (1976) then determined that Pa_7 was also present in Cebada Capa after performing an allelism test between these two lines. Tan (1978) used crosses between trisomic lines and both Cebada Capa and Estate in order to position Pa_7 to chromosome 3H and determine that Pa_3 from Estate was not linked on this same chromosome. After the identity of Pa_7 was clarified, Zhong et al. (2003) developed backcross lines derived from Bolivia (Pa_{2+6}) that were postulated to carry singly either Pa_2 or Pa_6 . The derived line carrying Pa_6 only was used in an allelism test with Magnif 102, which carries Pa_5 . Strong evidence that Pa_5 and Pa_6 are actually alleles of the same gene was provided when no susceptible progeny were found from more than 2,000 F_2 plants evaluated. The additional genetic analysis involving trisomic lines and monogenic backcross lines provided

important data to resolve the relationships among the genes *Pa*₃, *Pa*₅, *Pa*₆, and *Pa*₇.

The genetics of the next provisionally accepted leaf rust resistance gene remains poorly understood. Clifford (1977) cited a personal communication with B. H. Tan that a single gene reported in Egypt 4 should be named *Pa*₈. Unlike the other described lines carrying different *Pa* genes, this accession has a narrow spectrum of resistance to *P. hordei* (Clifford, 1977). The resistance gene in Egypt 4 went undetected in studies by Roane (1962) and Roane and Starling (1967), presumably because no *P. hordei* isolate with avirulence for Egypt 4 could be found. Although *Pa*₈ is accepted as a unique gene, its chromosomal position and allelic relationship with other genes have not been reported. The genomic locus associated with this gene is described in Chapter 2.

Another case of unresolved allelism for leaf rust resistance genes arose between the Ethiopian landrace Abyssinian (Clho1243, HOR2596) and the German cultivar Trumpf (PI 548762, HOR 4967). Both accessions were thought to carry the same gene, one that displayed a wider spectrum of resistance than previously seen in other barley accessions (Clifford and Udeogalanya, 1976). Crosses between Abyssinian and the other widely resistant sources of Estate (*Pa*₃) and Cebada Capa (*Pa*₇) yielded evidence that a new gene, *Pa*₉, must be present in Abyssinian (Tan, 1977). In Germany, the leaf rust resistance in cultivar Trumpf was overcome by virulent *P. hordei* isolates, spurring investigations into its genetic ancestry. The resistance in Trumpf may have been derived from the Ethiopian line HOR 2596 (Abyssinian or CI 1243), which was previously described as carrying *Pa*₉. Data from F₂ populations of reciprocal allelism crosses failed to reject a model predicting multiple incompletely dominant genes, indicating that HOR 2596 and Trumpf carry the same resistance gene (Walther, 1987). Other pathotype screening data indicated that Trumpf reacted differently

than Abyssinian (Jones and Clifford, 1980). Corroborating allelism and pathotype testing by Jin et al. (1993) led to the designation of a new leaf rust resistance gene (*Rph12*) in Trumpf, using the modern gene epithet, which will be used throughout this chapter. In a subsequent study, Borovkova et al. (1998) evaluated a large F_2 population of nearly 4,000 individuals from a cross between Abyssinian and Triumph (Clho 12307, PI 290195), the American imported source of Trumpf grown in the United States, and found no susceptible segregants. They concluded that *Rph9* and *Rph12* must be alleles of each other at the same locus. They also found that both genes were linked to the sequence-tagged site (STS) molecular marker *ABC155* on chromosome 5HL. The two genes were within 3 cM of each other. A third possible allele at this locus was described in the cultivar Cantala. This contention was based on the fact that no segregating F_3 families were observed from a cross between Cantala and Triumph (Dracatos et al., 2014). It remains unclear whether these genes are truly alleles at the same locus or are distinct but closely linked loci.

The gene in Trumpf was named *Rph12* because two new genes from wild barley had previously been described by Feuerstein et al. (1990). In the work by Feuerstein et al. (1990), 56 different *Hordeum vulgare* ssp. *spontaneum* accessions from Israel were backcrossed to the susceptible cultivar Clipper and two broadly resistant BC_3F_2 families were chosen for genetic analysis. A partially dominant gene was identified in the backcross line BC-8 (GSHO 1588) and was linked with the isozyme locus *Est2* on chromosome 3H. This gene was designated as *Rph10* without performing allelism tests with other genes positioned on the same chromosome. A different partially dominant resistance gene in another backcross line BC-67 (GSHO 1589) was linked with the isozyme loci *Acp3* and *Dip2* on chromosome 6H and designated as *Rph11* since no other leaf rust resistance genes had been described on this chromosome. This

pioneering work with wild barley was followed by other investigations mining disease resistance genes in other wild *Hordeum* germplasm.

The next several leaf rust resistance genes were discovered after evaluation of a diverse collection of wild and cultivated barley accessions (Jin and Steffenson, 1994). Using a panel of *P. hordei* isolates with virulence for genes *Rph1-Rph12*, these workers reported that wild barleys are typically more resistant than cultivated barleys and often carry multiple genes with dominant or recessive gene action. A later publication based on this initial work led to the discovery of barley accessions with resistance to widely virulent *P. hordei* isolates and the designation of two new leaf rust resistance genes (Jin et al., 1996). The wild barley-derived accession PI 531849 (BS 99-2-1) segregated for a single dominant gene when crossed with the susceptible cultivar Bowman, and its uniqueness was revealed in allelism tests with *Rph1-Rph12*. The gene in PI 531849 was designated as *Rph13* and was reported as possibly linked with *Rph9* on chromosome 5HL after a deficiency of susceptible F₂ progeny was observed in the cross PI 531849 x HOR 2596. PI 584760 (PI 531901-1, NDBLR01) segregated for a single incompletely dominant gene when crossed with Bowman. Higher infection types in some F₂ progeny classified as resistant from this cross suggested the incompletely dominant nature of this gene. Allelism tests with the donors of *Rph1-Rph13* confirmed a novel gene in PI 584760, which was designated as *Rph14*. The incompletely dominant nature of this gene was confirmed and complete co-segregation between this gene and the Diversity Arrays Technology (DART) marker *bPb-1664* on chromosome 2HS was observed (Golegaonkar et al., 2009). In addition to *Rph13* and *Rph14*, another resistance gene from wild barley was reported in accession PI 355447 (Chicaiza, 1996). This accession, along with several others, exhibited a high level of resistance to a wide array of *P. hordei* isolates. After backcrossing the resistance gene into Bowman and also performing allelism tests with the sources of *Rph1-14*, this new

single dominant resistance gene was named *Rph15*. Crossing PI 355447 with morphological marker stocks provided the initial genetic evidence that *Rph15* lies on chromosome 2HL between the six-rowed spike 1 (*vrs1*) locus and the ribbon grass (white streak 7, *wst7*) locus (Chicaiza, 1996). However, this placement of *Rph15* on the long arm of chromosome 2H would later be corrected.

The discovery of two other widely resistant *H. vulgare* ssp. *spontaneum* accessions led to the identification of another leaf rust resistance allele of *Rph15* by Ivandic et al. (1998). They found two wild barley accessions (HS078 and HS084) resistant to *P. hordei* isolates with virulence for *Rph7* and developed a doubled haploid population for each with the susceptible line, L94. After construction of Restriction Fragment Length Polymorphism (RFLP) marker maps, Ivandic et al. (1998) positioned the resistance loci of both HS078 and HS084 in the short arm of chromosome 2H via linkage with RFLP markers *MWG784* and *MWG2133*. They concluded the gene was novel and named it as *Rph16* (Ivandic et al., 1998). A later investigation by Weerasena et al. (2004) concluded that *Rph15* and *Rph16* are likely alleles of each other. Additionally, Amplified Fragment Length Polymorphism (AFLP) markers on the short arm of chromosome 2H, not the long arm, were found linked to *Rph15*. Moreover, there was no evidence of segregation observed in 1,027 F₂ individuals from the cross Bowman-*Rph15* (the backcross line from Chicaiza) x HS084 (one of Ivandic's parental wild barleys). One AFLP marker was long enough to be converted to a Cleaved Amplified Polymorphic Sequences (CAPS) marker, *P13M40*, making it amenable for marker-assisted selection in breeding (Weerasena et al., 2004). Another allele at the *Rph15/16* locus was described in a different wild barley accession, *Hordeum spontaneum* 677 (Kopahnke et al., 2004). Without the appropriate allelism tests or fine-mapping data, it is uncertain whether these genes lie at separate linked loci or are alleles at the same locus.

Other barley leaf rust resistance genes were identified in the secondary gene pool species, *Hordeum bulbosum*. In New Zealand, Pickering (1995, 1997, and 2000) successfully introgressed large segments of chromatin from different *H. bulbosum* accessions into two *H. vulgare* cultivars bred for high levels of horizontal leaf rust resistance. The first introgression line was produced as an F₃ recombinant inbred line between *H. bulbosum* accession 81882 x *H. vulgare* cultivar Vada (PI 243182). The introgression was mapped to the short arm of chromosome 2H and the resistance gene was designated as *Rph17*. The next introgression line was produced as a BC₂F₂ line derived from *H. bulbosum* accession 2032 x *H. vulgare* cultivar Emir (CIho 11790). This introgression was mapped to the long arm of chromosome 2H and the resistance gene was designated as *Rph18*. Within these large segments of introgressed chromatin, it is possible that more than one resistance gene might be present. Johnston et al. (2013) created another introgression line 182Q20 between *H. bulbosum* accession A17-1 x *H. vulgare* cultivar Golden Promise (PI 342079). Many recombination events were assayed in a large population of F₂ progeny to map this resistance gene (*Rph22*) to the long arm of chromosome 2H (Johnston et al., 2013). Of the two *H. bulbosum*-derived genes of *Rph18* and *Rph22* positioned in chromosome 2HL, flanking polymerase chain reaction (PCR) markers of *WEB114/WEB113* and *WEB115/WEB114* were identified, respectively. These genes are considered to have a qualitative effect on resistance even though their observed modes of action and potential gene transcripts are generally considered horizontal quantitative resistance mechanisms (Pickering et al., 2000; Pickering et al., 1995; Pickering et al., 1998).

Two recently characterized seedling resistance genes were identified from two older cultivars Reka I and Ricardo (PI 328933, HOR 2471). Originally used as a differential host line by Levine and Cherewick (1952), Reka I was likely derived from the Australian cultivar Prior released in 1905. Reka I is also known

to carry *Rph2* and an uncharacterized gene, named *RphP* (Cotterill et al., 1995). Park and Karakousis (2002) mapped *RphP* to the long arm of chromosome 7H in a doubled haploid population between Chebec x Harrington. They then performed an allelism test between Estate and Prior that provided evidence of repulsion-phase linkage between *Rph3* and *RphP* around 28 cM on 7HL, allowing them to designate *RphP* as *Rph19*. Since 1945, the cultivar Ricardo has also been known to carry two resistance genes, *Rph2* and one additional gene. Using bulked segregant analysis and single sequence repeat (SSR) genotyping on an F₃ population, the uncharacterized gene from Ricardo was mapped on chromosome 4H (Sandhu et al., 2012). Since this was the only leaf rust resistance gene mapped to this chromosome, it was declared unique and given the gene designation of *Rph21*.

The remaining barley leaf rust resistance genes were first described as quantitative trait loci (QTL). *Rph20* is an adult plant resistance (APR) gene that was identified from the Australian cultivar Flagship (Hickey et al., 2011). In a doubled haploid population between Flagship x ND24260, the QTL *qRphFlag*, contributed by Flagship, accounted for 64-85% of the phenotypic variation in the field and was associated with the DArT marker *bPb-0837* in the short arm of chromosome 5H. Also from this population, ND24260 donated a weaker effect resistance QTL, designated as *qRphND*, that was detected in chromosome 6H and only accounted for 2-5% of disease variation. The origin of *Rph20* traces back to *Hordeum laevigatum*, which contributed the major effect non-hypersensitive APR in other cultivars such as Vada and Emir. Another APR gene, *Rph23*, was described in a different Australian cultivar Yerong (Derevnina et al., 2013; Singh et al., 2015). The largest effect resistance QTL from Yerong (*qRphYer-7H*) accounted for 18-20% of the phenotypic variation and was very closely linked to the co-dominant SSR marker *Ebmac0603* in the short arm of chromosome 7H. The QTL from ND24260 reported by Hickey et al. (2011) was

later characterized as *Rph24* (Ziems et al., 2017). Barleys carrying a combination of *Rph20* with *Rph23*, *Rph24*, or other small effect resistance QTL displayed enhanced levels of APR compared to barleys carrying a single QTL. *Rph25* was first identified as the QTL, *RphFT*, in the Chinese cultivar Fong Tien during an investigation of diverse barley germplasm for seedling resistance (Kavanagh et al., 2017). Using DArT marker analysis in a doubled haploid population, *Rph25* was positioned to chromosome 5HL near the *Rph9/Rph12* locus. Although *Rph25* only provides resistance to a single Australian *P. hordei* isolate and was not considered to be of much value for breeding programs, it could still be useful in combination with other resistance genes. The most recently described leaf rust resistance gene is *Rph26*, derived from a cross between the *Hordeum bulbosum* accession A17 and the cultivar Emir (Yu et al., in press). The resultant line 200A12 contains this novel *H. bulbosum* resistance introgression in chromosome 1HL. A group of five separate QTLs within this introgression was collectively designated as *Rph26*. They were inherited in an incompletely dominant fashion and were reported to be less effective in heterozygous individuals. Leaf rust resistance genes with quantitative resistance mechanisms can have qualitative effects on disease severity.

Bowman Introgression Lines for Leaf Rust Resistance

The current collection of barley accessions used as leaf rust differentials by various research groups is genetically diverse. Agronomic and genetic differences among these lines can make them difficult to study and use in breeding programs. Also, some of these lines even carry combinations of resistance genes that effectively encrypt the actions of single genes. Isolating individual *Rph* genes into a common susceptible cultivar is ideal both for normalizing the agronomic characteristics of the differential set and for revealing

the true resistance spectrum of single resistance genes (Person, 1959; Clifford, 1977).

In recognition of these research limitations, the barley leaf rust resistance genes *Rph1-15* were backcrossed to the susceptible cultivar Bowman to develop a set of near-isogenic differential host lines (Chicaiza, 1996; Caffarel, 2005). Between 1-10 backcrosses were used to develop 95 Bowman introgression lines representing these leaf rust resistance genes, their potential allelic variants, and perhaps yet undescribed genes for resistance. In this study, 12 diverse pathotypes of *P. hordei* were used to compare the phenotypic reaction patterns between the introgression lines and their respective donor lines. Altogether this effort was intended to develop a new barley leaf rust differential set consisting of single-gene introgression lines in a uniform genetic background.

As part of a large, comprehensive genetic study of barley, Druka et al. (2011) genotyped barley mutants backcrossed to Bowman to investigate morphological gene mutants as single gene lines. The germplasm included the 95 leaf rust introgression lines, but these genotype data have not been published. More recently, Genotyping-by-Sequencing (GBS) Single Nucleotide Polymorphism (SNP) data were generated for these 95 lines to better define the introgressions carrying the resistance genes. In Chapter 2 of this thesis, a comprehensive phenotypic and genotypic analysis of these 95 introgression lines was completed with the goal of selecting a refined set of single gene host lines for characterizing the virulence diversity of *P. hordei*.

CHAPTER 2

PHENOTYPIC AND GENOTYPIC CHARACTERIZATION
OF BARLEY INTROGRESSION LINES CARRYING THE
LEAF RUST RESISTANCE GENES *Rph1* TO *Rph15*

Introduction

Barley (*Hordeum vulgare* L.) ranks fourth among cereal crops in the world with an average annual production from 2014-2017 of 145 million metric tons (MMT) from ~50 million planted hectares (MHa) (USDA, 2017). *Puccinia hordei* Otth, the causal agent of barley leaf rust, is one of the most ubiquitous pathogens challenging the sustainability of the crop across the world. Under moderate epidemics, yield losses around 30% are common in barley, but can reach up to 63% during severe epidemics (Levine and Cherewick, 1952; King and Polley, 1976; Cotterill et al., 1992a; Griffey et al., 1994). Breeding cultivars with resistance is the preferred method of controlling barley leaf rust because fungicide applications, while effective, are rarely economical and can contaminate the environment. To date, 26 major effect resistance genes, referred to as Reaction to Puccinia hordei (*Rph*) genes, have been identified and mapped to unique positions across the barley genome (Park et al., 2015; Kavanagh et al., 2017; Ziemis et al., 2017; Yu et al., in press). While many of these resistance genes have been rendered ineffective in deployed cultivars due to the appearance of new virulent pathotypes of *P. hordei*, they still provide a means to characterize virulence phenotypes in pathogen populations.

To differentiate virulence phenotypes within pathogen populations, researchers compile sets of host genotypes that are postulated to carry unique resistance genes. These collections of resistant host lines are referred to as differential sets. Stakman and Levine (1922) developed one of the first differential sets in their pioneering research to characterize the virulence diversity of the wheat stem rust pathogen, *Puccinia graminis* f. sp. *tritici*. From the evaluation of many wheat lines, they selected 12 that were thought to carry distinct resistance genes. This wheat stem rust differential set became an international standard for characterizing races of *P. graminis* f. sp. *tritici*. After the initial research by

Stakman and Levine (1922), differential host lines were established for many other pathosystems such as flax rust, wheat stripe rust, wheat stem rust, and oat crown rust (Flor, 1942; Zimmer et al., 1963; Roelfs and Martens, 1988; and Chen et al., 2014). With barley, a number of differential sets have been developed to type virulence to such diverse pathogens as *Cochliobolus sativus* (spot blotch pathogen), *Pyrenophora teres* f. sp. *teres* (net form net blotch pathogen), *Blumeria graminis* f. sp. *hordei* (powdery mildew pathogen), *Puccinia striiformis* f. sp. *hordei* (stripe rust pathogen), and *Puccinia hordei* (leaf rust pathogen) (Levine and Cherewick, 1952; Kolster, 1986; Valjavec-Gratian and Steffenson, 1997; Afanasenko et al., 2009; Wan and Chen, 2014; Park et al., 2015).

Levine and Cherewick (1952) proposed the first barley leaf rust differential set, highlighting the difficulties of comparing virulence data derived from different sets of host lines used by workers around the world. This original set of nine lines was used internationally for 25 years (Moseman and Roane, 1959; Nover and Lehmann, 1974; Shelko, 1974), until genetic studies by Roane and Starling (1970) identified redundancies within this first set. Soon afterwards, a proposal for an international barley leaf rust differential set was forwarded by Clifford (1977) and comprised of eight lines, each with a different recognized resistance gene. As new resistance genes were characterized, this set was expanded and has been regularly used by various international research groups (Yahyaoui et al., 1988; Steffenson and Jin 1992; Brodny and Rivadeneira, 1996; van Niekirk et al., 2001; Park, 2003; Germán et al., 2005; Jones, 2005; Woldeab et al., 2006; Czembor et al., 2007). The current barley leaf rust differential set is comprised of cultivars, landraces, and wild barley introgression lines originating from around the world. Although this differential set is comprised of accessions that, in most cases, are known to carry only a single *Rph* gene, their diverse genetic backgrounds can sometimes affect the expression of resistance. For example, *Rph3* confers a strong hypersensitive reaction to many *P. hordei* isolates when

present in the barley cultivar Ribari, but gives incomplete resistance when transferred into the susceptible line L94 (Niks et al., 2011). Also, epistasis can occur in differential lines carrying more than one *Rph* gene, thereby confounding the true reaction types of the genes present. Finally, due to the diverse agronomic phenotypes of these lines, comparisons for leaf rust resistance can be difficult in the field due to differences in heading date, plant height, and leaf morphology. The current barley leaf rust differential set has been successfully used to characterize the virulence of *P. hordei* isolates in many studies; however, its utility can be improved.

An ideal and efficient differential set for characterizing pathogen virulence is one comprised of lines that each carry a single resistance gene backcrossed into the uniform genetic background of a single susceptible genotype (Person, 1959). With successive backcrossing, differential sets comprised of near-isogenic lines have been developed for the wheat leaf rust, wheat stripe rust, barley leaf scald, and barley powdery mildew pathogens (Kolster et al., 1987; Patil et al., 2002; Dyck, 2011; Chen et al., 2014). A future intent to develop backcrossed differential lines for the wheat stem rust, barley net form net blotch, and the barley leaf rust pathosystems have been communicated in the literature (Clifford, 1977; Roelfs and Martens, 1988; Steffenson and Jin, 1992; Afanasenko et al., 2009). With respect to this effort in the barley leaf rust pathosystem, the donor lines for the resistance genes *Rph1-15* were first crossed to the susceptible feed barley cultivar Bowman (PI 483237) as part of a large effort to develop backcross lines for major morphological markers in barley (Chicaiza, 1996; Caffarel, 2005; Druka et al., 2011). Multiple sources for many of these *Rph* genes were backcrossed to Bowman to compare the reactions between alleles of the same gene. The cultivar Bowman was selected as the recurrent parent based on its superior agronomic traits, adaptation across diverse environments, and broad susceptibility to isolates of the leaf rust pathogen (Franckowiak et al., 1985).

Chicaiza (1996) initiated development of 95 Bowman introgression lines carrying different *Rph* alleles. Fifteen of these lines were initially recommended to represent *Rph1-15* by Caffarel (2005). As part of a large, comprehensive genetic study of barley, Druka et al. (2011) utilized Barley Oligonucleotide Pooled Assay (BOPA) SNP markers to genotype hundreds of Bowman introgression lines with mutations for altered morphological traits. This germplasm also included Bowman lines with introgressions from 95 leaf rust resistant donors, but these genotyping data were not previously published. Genotyping-by-sequencing (GBS) SNP data were recently collected for these 95 Bowman introgression lines, plus their respective original donors. The main objective of this investigation was to characterize the phenotypic and genotypic polymorphisms of barley introgression lines carrying leaf rust resistance genes. This was done by evaluating the 95 introgression lines for their infection types (ITs) to 12 global isolates of the leaf rust pathogen and genotyping with SNP markers. These data were used to describe the phenotypic differences among alleles at the same loci, the precise physical positions for the leaf rust resistance gene introgressions across the barley genome, and the existence of putative new resistance genes in the germplasm. In turn, these data were used to select an improved set of differential host lines each carrying a single *Rph* gene (from *Rph1* to *Rph15*) for typing virulence in *P. hordei*.

Materials and Methods

Plant Materials and Development of Introgression Lines

The donors for *Rph1-24* were obtained from the original investigators or the United States Department of Agriculture-Agricultural Research Service (USDA-ARS) National Small Grains Collection (NSGC) in Aberdeen, Idaho. Data on the line names, accession numbers, recognized *Rph* genes, country of origin,

and donors for these resistance sources are given in Appendix Table 1. When the program to introgress leaf rust resistance genes into Bowman (PI 483237) was first initiated, only *Rph1* to *Rph15* were described (Chicaiza, 1996) (Table 1). Thus, only these 15 leaf rust resistance genes are included in the final differential set of Bowman introgression lines (Table 2). The main focus of this investigation was on the 15 introgression lines (*Rph1-15*) selected for use as differential host lines. However, we also characterized the other 80 leaf rust resistant donors used as parents in the larger resistance introgression effort. To develop the introgression lines, a single F₁ progeny from each cross between Bowman and the *Rph* donor parent was backcrossed to Bowman. From each resulting BC₁F₂ population, a single progeny that closely resembled the respective donor parent's ITs to a selected pathotype of *P. hordei* was transplanted and used in backcrossing. Details regarding the accession number, origin, improvement status, pedigrees, and derived BW lines for Bowman and the 95 donor parents given in Appendix Table 2. Appendix Table 3 details the respective donor parents, numbers of backcross generations, pedigrees, and postulated gene identities for each of the 95 introgression lines.

Pathogen Isolates and Race Nomenclature

From a global collection of more than 350 isolates of *P. hordei*, Chicaiza (1996) and Caffarel (2005) selected 12 diverse ones for use in the introgression project. These isolates had unique virulence combinations capable of differentiating each leaf rust resistance gene under study. Together, the panel of isolates provided redundant avirulence and virulence phenotypes for all *Rph* genes, except *Rph15* for which no virulent isolate was available. The panel was assembled to provide avirulence to each gene in at least two of the 12 isolates. Redundancies for virulence and avirulence in an isolate panel help validate that each resistance gene is functioning independently.

In this study, four of the same isolates from Chicaiza (1996) and Caffarel (2005) were used, but eight alternative isolates were selected from the global collection (Fetch et al., 1998). Together, this modified panel of 12 *P. hordei* isolates improved the balance between avirulence and virulence redundancies for *Rph1* to *Rph14* and also added virulence for *Rph15*, the widely effective resistance gene first described by Chicaiza (1996). Complete details for the avirulence/virulence (*Avr/Vir*) formulae (Clifford, 1985), coded triplet octal notations (Gilmour, 1972), and North American rust race codes (Roelfs and Martens, 1988) for the 12 *P. hordei* isolates are listed in Table 3. Donors of these *P. hordei* isolates and their origins of collection are detailed in Appendix Table 4.

These different race nomenclatural systems have been used to represent the virulence phenotypes of *P. hordei* in order to simplify comparisons among isolates. In the *Avr/Vir* system, the *Rph* genes for which the isolate is avirulent are listed first followed by a slash and then the *Rph* genes for which the isolate is virulent. In the coded triplet octal notation system, each resistance gene of the differential set is aligned chronologically from right to left. Across all of the *Avr/Vir* reactions, reaction types of three resistance genes (genes 1-3, or 4-6, or 7-9) are used together as a triple-digit value to encode a single numeral from 1 to 7 that abbreviates the virulence combinations (Gilmour, 1973; Limpert and Müller, 1994). Isolates with different virulence combinations will have different numerical codes. In this scheme, higher numbers indicate wider virulence. Instead of using numerals to name pathotypes of the pathogen, under the North American system, rust isolates are named according to an alphabetical letter code (Roelfs and Martens, 1988; Wan and Chen, 2014). The *Avr/Vir* reactions of the differential lines are grouped sequentially in groups of four and the different possible four-digit reaction patterns are converted into alphabetical letters from “B” to “S”. In this race code system, letters furthest from the beginning of the alphabet imply wider virulence.

Experimental Design

Leaf rust evaluations of resistance donors and derived introgression lines were performed in a greenhouse (GH) on the Saint Paul campus of the University of Minnesota. These evaluations were conducted in three separate experiments, each with two replicates, in the fall/spring GH season of 2016-17 and the spring GH season of 2017-18. Also included in the experiment were several replications of the recurrent parent Bowman and the differential host lines for *Rph1-24*. The medium used for growing the plants was a 50:50 mixture of steam-sterilized soil and a commercial potting mix (SS #8 F2 mix; Sun Gro Horticulture brand, Agawam, MA). Plastic cells measuring 3.8 cm wide and 21 cm deep (RL98 tray with SC10 Super cells; Ray Leach Cone-tainerTM brand, Stuewe and Sons Inc, Tangen, OR) were filled with the growth medium into which three seeds of each line were sown. After sowing, a slow release fertilizer was applied according to the manufacturer's instructions (Osmocote 14-14-14; Scotts brand, Marysville, OH).

Inoculation and Disease Phenotyping Protocols

Inoculum for each isolate was produced in an isolated greenhouse on seedlings of the susceptible barley cultivar Moore (CIho 7251). Prior to inoculation, plants were grown in a rust-free GH with a day/night temperature of 24/20°C and 16-hour photoperiod supplemented by 1000-watt high-pressure sodium (HPS) bulbs emitting $350\text{-}400\ \mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$. Seedlings were grown in peat pots (Dioni trays containing 7x9 cm peat pots; FertilUSA, Slatington, PA) filled with the same growing mix detailed above. For ease of handling, the peat pots were set into 16-count plastic trays. When coleoptiles first emerged from the soil, the plants were treated with a maleic acid hydrazide solution ($380\cdot\text{mg}\cdot\text{L}^{-1}$) to retard their growth, making the collection of urediniospores more convenient. The

12 stock isolates of *P. hordei* were stored as urediniospores in a -80°C freezer. To prepare the inoculum for inoculation, each sample was individually heat shocked for 15 minutes in a 45°C water bath. Then, the urediniospores were rehydrated in a chamber at 80% relative humidity for a minimum of 3 hours. Rehydrated spores were suspended in a light mineral oil (Soltrol 170, Chevron Phillips Chemical Company LP, The Woodlands, TX) at a concentration of $10 \cdot \text{mg} \cdot \text{ml}^{-1}$. Suspended urediniospores were aerosolized using a custom spore inoculator (GRA-301, Tallgrass Solutions, Manhattan, KS) pressured by a pump (DOA-P704-AA, GAST, Benton Harbor, MI) set to 34.5 kPa. Fully emerged primary leaves of cultivar Moore seedlings were inoculated at a rate of approximately 24 μg per plant. Urediniospores of individual isolates were collected on separate days 12-14 days post inoculation using a large cyclone spore collector (GRA-101; Tallgrass Solutions) attached to a shop vacuum (WS0250VA; Workshop Wet/Dry Vacs, Saint Louis, MI). Urediniospores were desiccated over silica crystals for 3-7 days and then placed inside size “00” gelatin capsules (Fagron, Saint Paul, MN). These capsules were then placed inside of cryovials (T311-2; Simport, Beloeil QC, Canada) for long term storage at -80°C.

For the phenotyping experiments, the fully expanded primary leaves (8-9 days old) of accessions were inoculated with the same inoculum concentration as detailed above. To reduce the risk of phytotoxicity, the Soltrol 170 oil carrier was volatilized from the leaf surfaces by 2 hours of gentle fanning from an oscillating fan under 400-watt HPS lamps. For the infection period, inoculated seedlings were placed in mist chambers at 22°C and ~100% relative humidity in the dark. The mist was generated by household ultrasonic humidifiers (V5100NS; Vicks, Cincinnati, OH) run for ten minutes at the outset and thereafter for two continuous minutes at 15-minute intervals. After the infection period, plants were allowed to dry off slowly before being incubated in a GH with a day/night temperature of

20/15°C and 16-hour photoperiod supplemented by 1000-watt HPS bulbs emitting 350-400 $\mu\text{mol}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$. Once returned to the GH, each full cone-rack (N=98) was fertilized with 10•g•L⁻¹ of a water-soluble fertilizer (20-10-20; N-P-K; J.R. Peter's, Inc.; Allentown, PA). Then, the cones were spaced out at half density to allow for full light penetration on the leaves.

Leaf rust infection types (ITs) were assessed on the barley germplasm at 10 and 12 days post inoculation (dpi). ITs were scored using the original 0-4 rating scale developed by Mains (1930) with the additional notations of "+" and "-" to indicate larger or smaller size variations of classically described uredinia (Levine and Cherewick, 1952) and "c" and "n" to indicate the presence of chlorotic or necrotic reactions (Moseman and Roane, 1959), respectively. Complex ITs were recorded when more than one IT was observed on a single leaf. For example, an IT of 213- indicates that all three pustule types were present on the same leaf, and that the "2" type pustules were the most common, followed by the "1" type, with "3-" type pustules being the least common. ITs from "0" to "23-c" were considered resistant (R), and ITs from "23-" to "4" were considered susceptible (S) (Steffenson et al. 1993). The presence of chlorosis in a "23-c" reaction indicates some level of incompatibility and is therefore not considered susceptible like the "23-" reaction lacking a chlorotic response. Among the ITs classified into the general category of R, two phenotypic classes were either highly resistant (ITs "0" to "0;12") or moderate (M) reactions (ITs "12" to "23-c"). Figure 1 displays the range of infection types grouped within each of the three reaction classifications.

Genotyping the Bowman Introgression Lines and Resistance Donors

As part of a large comprehensive study to genetically characterize the introgression of various morphological traits into Bowman barley, Druka et al. (2011) utilized the BOPA_1 and BOPA_2 SNP marker sets to genotype more than 800 Bowman introgression lines for 3,072 SNP loci. As part of this study, Druka et al. (2011) also SNP genotyped the 95 leaf rust introgression lines, but these data were not included in the publication. Full details on the SNP genotyping methods used are described in Druka et al. (2011). SNP sequences were obtained from The Triticeae Toolbox (<https://triticeaetoolbox.org/barley/>) and queried through the BARLeY genome EXplorer (Barlex; <https://apex.ipk-gatersleben.de/apex/f?p=284:10>) in order to obtain updated POPulation SEQuencing (POPSEQ) positions (in CentiMorgans [cM]) according to Mascher et al. (2013). An unpublished consensus map combining eight barley linkage and consensus maps was designed as part of a meta-analysis study by Ahmad Sallam (personal communication) and used to update the cM positions for any SNP lacking information in Barlex. Estimated physical positions for the BOPA SNPs were obtained from an unpublished dataset provided by Shiaoman Chao (USDA-ARS, Fargo, ND).

In addition to the BOPA SNP genotyping, cooperators at the Julius-Kühn Institute (Quedlinburg, Germany), under the direction of Dragan Perovic, performed Genotyping-by-Sequencing (GBS) for Bowman, the 95 resistance donors, and the 95 introgression lines. DNA was extracted from seedlings at the two-leaf stage according to the methodology of Stein et al. (2001). Then, the genomic DNA was cleaved, repaired, and quantified according to Wendler et al. (2014). With 96-well plates, three separate genotyping runs were made on a MiSeq instrument (Illumina, Inc., San Diego, CA) using the Illumina Kit V3 and 150 total paired-end cycles. Starting libraries consisted of genomic fragments

with an average length of 453 base pairs (bp) at an average concentration of 39.5 nanomolar (nM). Working libraries were diluted to an average DNA concentration of $0.64 \text{ ng} \cdot \mu\text{l}^{-1}$ according to Qubit quantification. The average library concentration used for the MiSeq runs was 8.7 picomols (pM), and the average 96-well cluster density was $1201.7 \text{ K} \cdot \text{mm}^2$. Across the three MiSeq runs, 87.5% of the clusters passed quality filtration, representing a matrix of multi-sequence alignment across 3,952.7 megabases (Mb) of the 5,100 Mb genome. Trim Galore software from Babraham Bioinformatics was utilized to trim the first 13 bp of the adaptor sequence from the amplified genomic sequences. SNP reads were aligned against the reference barley genome sequence “150831 barley pseudomolecules.fasta” (Mascher et al., 2017) using the BWA-MEM algorithm (Li and Durbin, 2009). Missing marker data was imputed using Beagle (Browning and Browning, 2016). SAMtools Mpileup software was used to call SNPs (Li, 2011). Execution of the GBS pipeline was done through the open online Galaxy platform (<https://galaxyproject.org/>). A total of 41,460 SNP markers were included in a vcf data format before performing quality assessment. Unmapped markers and those that were either monomorphic or had more than 20% heterozygosity among the parental lines were removed. In total, 19,593 SNP markers were retained and used in the final analyses. The GBS marker profiles for all of the lines were converted into a Microsoft Excel spreadsheet in order to compare the genotypes between the donors and their respective introgression lines. Non-Bowman type markers within the introgression lines represent regions where donor sequences were retained. Graphical genotypes for the introgression lines selected to represent *Rph1-15* are displayed in Figure 2 and were drawn using MapChart software (Voorrips 2002).

Selection of Bowman Introgression Lines for the Leaf Rust Differential Set

Within a group of introgression lines presumed to carry the same *Rph* gene, only one was selected to represent that gene in the final barley leaf rust differential set. The selection of introgression lines to best represent the *Rph1-15* alleles in a Bowman background was based on several criteria. The first criterion was based on parentage. Preference was placed on introgression lines derived from the most commonly used historical differentials. The second criterion was whether the introgression line exhibited the same or similar reaction pattern to its original donor parent in response to the 12 *P. hordei* isolates. The third criterion was whether the introgression line retained donor chromatin at a position matching the previously described genomic location of the respective *Rph* gene, if known. The final criterion was based on size of the transferred chromosomal segment(s) in the introgression lines according to both the BOPA and GBS SNP datasets. Introgression lines with the greatest number of backcrosses and the smallest introgression(s) were given the priority in the final selection process.

Results and Discussion

Disease Phenotyping

The median ITs of the 24 *Rph* donors to the 12 isolates of *P. hordei* are presented in Table 4. These median ITs were based on three independent experimental replications and represent the central IT tendencies for each interaction. In each experiment, the 12 *P. hordei* isolates produced clear differential reaction patterns for each *Rph* gene, except for the reaction patterns between *Rph5* versus *Rph6*, *Rph9* versus *Rph12*, and *Rph10* versus *Rph11*. Two of these three gene pairs are likely alleles of the same gene (Borovkova et al., 1998; Zhong et al., 2003), but *Rph10* and *Rph11* have always been

considered unique genes and not alleles. Multiple different reaction patterns among the other *Rph* genes were observed in both the donor parents and their respective introgression lines. Out of the 95 introgression lines, only a single line carries *Rph1*, *Rph7*, *Rph8*, *Rph10*, *Rph11*, *Rph13*, or *Rph14*. Three introgression lines carry *Rph4*, six introgression lines carry *Rph2*, *Rph3*, *Rph5/6*, or *Rph9/12*, and 50 introgression lines carry *Rph15*. The remaining 11 introgression lines potentially carry other genes for leaf rust resistance. Phenotypic comparisons among groups of introgression lines considered to carry the same genes are detailed in Appendix Table 5 (for *Rph2*, 3, 4, 5, and 9) and Appendix Table 6 (for *Rph15* and *Rph15+*). The phenotypes for all of the parental lines (i.e. Bowman and the *Rph* donors) are detailed in Appendix Table 7. Additional IT data for the 24 leaf rust resistance sources from earlier seed increase generations and from an earlier investigation (Fetch et al. 1998) are given in Appendix Table 8.

The median ITs and general reactions (GRs) of *Rph1-15* introgression lines selected for the differential set and their respective donor parents to the 12 *P. hordei* isolates are given in Table 5. The GRs display whether the IT is classified as resistant (R), moderate (M), or susceptible (S). The mode and range of ITs for the selected introgression lines are presented in Table 6. The mode displays the most commonly observed IT across the three experiments, while the range conveys an inference of the variation observed (i.e. lowest and highest ITs). No GR differences were observed between the donor lines and respective introgression lines for *Rph1*, *Rph7*, and *Rph15* (Table 5). The introgression lines selected for *Rph3*, *Rph5*, *Rph10*, *Rph11*, *Rph12*, and *Rph14* had higher ITs (i.e. more compatible GRs) than their respective donor parents for one to three of the isolates, and those selected for *Rph2*, *Rph4*, *Rph6*, *Rph9*, and *Rph13* had higher ITs (i.e. more compatible GRs) than their respective donor parents for four or more of the isolates. In other cases, introgression lines selected for *Rph5*, *Rph6*,

Rph8, *Rph10*, *Rph11*, and *Rph14* had lower ITs (i.e. less compatible GRs) than their respective donor parents to one or a few isolates.

Genotyping Resistant Donor Sources and their Derived Introgression Lines

Initially, a total of 981 polymorphic BOPA SNP markers were used to analyze the genomes of Bowman and the 95 introgression lines (Druka et al., 2011). Increasing the marker density by 20-fold, the GBS dataset comprised 19,593 polymorphic markers that were used to genotype not just Bowman and the 95 introgression lines, but also their respective *Rph* donor parents. In both SNP datasets, chromosomal regions containing blocks of “non-Bowman” type markers were considered introgressions from the *Rph* donor parents. Introgressions for *Rph1-15* were detected with both the BOPA and GBS SNP datasets, the exceptions being with *Rph4* and *Rph14*, which were only observed with the GBS dataset. For each *Rph*-associated introgression, the physical positions from the GBS dataset coincided with the physical distances of the SNP markers (data not shown). Graphical genotypes of the 15 *Rph* introgression lines selected for the leaf rust differential set were constructed from the GBS SNP dataset (Figure 2). Annotated defense genes on the same contigs as BOPA SNP markers within the *Rph* gene-containing donor chromatin of these selected 15 introgression lines are listed in Table 7. Every annotated gene found on the same contig as a BOPA SNP marker within *Rph* gene-containing introgressions are listed in Appendix Table 9.

Phenotypic and Genotypic Characterization of Introgression Lines representing Rph1 to Rph15

Rph1.a

Rph1 was originally described in the experimental line Minn. II 21.15, which was derived from the cultivar Manchuria (CIho 2330). The gene was subsequently reported in the Portuguese cultivar Sudan and several other accessions (Watson and Butler, 1947; Roane and Starling, 1967). Based on trisomic analysis with another *Rph1* donor source (Speciale, CIho 7536), Tuleen and McDaniel (1971) positioned the gene in chromosome 2H. Sudan was the only source of *Rph1* used in this study. Its derived introgression line, BW682, exhibited an identical general reaction pattern (Table 2). In BW682, BOPA SNP markers were detected between 2.2 and 7.4 cM in chromosome 2HS, confirming the original chromosome assignment of Tuleen and McDaniel (1971). GBS SNP markers placed this ~16.3 megabase (Mb) introgression from 6.8 to 23.1 Mb in chromosome 2HS (Figure 2). No other introgressions were detected in this introgression line. BW682 was selected as the *Rph1* source for the differential host set. Although it was the only introgression line carrying this gene, it has the desirable attributes of being phenotypically similar to its *Rph1* donor source against *P. hordei* and carries just one small introgression.

Rph2.t

Many lines of different origin are presumed to carry *Rph2* (Franckowiak et al., 1997). Although the current barley leaf rust differential set employs the South American landrace Peruvian to represent *Rph2*, the gene was originally described in the Australian line Weider and subsequently identified in the Australian cultivar Reka I, in combination with *Rph19* (Watson and Butler, 1947;

Roane and Starling, 1967; Park and Karakousis, 2002). In another study, crosses between the barley line Q21861 (PI 584766) and different sources of *Rph2* (Peruvian, PI 531840, PI 531841, and Bolivia) indicated that this gene is linked with various markers in chromosome 5HS near the centromere (Borovkova et al., 1997). In this study, six different donors of *Rph2* (Peruvian, Bolivia, Reka I, BS-78-3-4, Juliaca, and MSI3351) were used to develop six introgression lines (BW737, BW742, BW743, BW744, BW739, and BW732, respectively) in Bowman. The general reactions to *P. hordei* among these six *Rph2*-introgression lines were mostly similar (Appendix Table 5). Only minor general reaction differences from highly resistant to moderate or from moderate to susceptible were detected to several *P. hordei* isolates. Line BW739, with the gene derived from Juliaca (PI 39151), was highly resistant to isolate AUS 220, while most of the other introgression lines carrying *Rph2* were susceptible. This suggests that BW739 may carry an allele of *Rph2* that is different from the other sources or that it possibly carries an additional resistance gene within its chromosome 1HS or 3HL introgressions. Considering the moderately sized 5HS introgressions and slight phenotypic differences observed among these six lines, it was not possible to determine whether or not they carry identical alleles at the *Rph2* locus or perhaps other linked genes that together confer a similar reaction pattern. As discussed by Jin and Steffenson (1996), allelism tests should be made among these six *Rph2*-introgression lines to resolve this genetic question.

All of the six introgression lines that putatively carry *Rph2* have a common donor segment (>300 Mb) in chromosome 5HS, thus corroborating the initial mapping position of the gene reported by Borovkova et al. (1997). Although other introgressions were detected in these six lines, they occurred in positions that were unique to each member of the group. The best representative among them is BW743. This introgression line exhibits a phenotype very similar to its donor parent, Reka I, was developed with six backcrosses, and also matches the

reaction pattern of the other *Rph2* donors. BOPA SNP markers detected three introgressions (1H, 5H, and 6H) within BW743, and the introgression carrying *Rph2* was initially positioned from 60 to 121 cM in chromosome 5HS. The GBS SNP markers confirmed that BW743 carries the smallest chromosome 5HS introgression (38.6 to 349.8 Mb; Figure 2) within the *Rph2* group (Appendix Table 5). Despite having two other small introgressions in chromosomes 1H and 6H, BW743 was the best candidate for the *Rph2* source in the leaf rust differential set.

Rph3.c

Originally discovered in the Egyptian cultivar Aim (PI 57650), *Rph3* is also present in Estate (PI 57700), a landrace also from Egypt (Henderson, 1945; Roane and Starling, 1967). These two accessions have been widely used as the sources for *Rph3* in differential sets (Levine and Cherewick, 1952; Moseman and Greeley, 1965; Nover and Lehman, 1974; Khokhlova, 1982). Tan (1978) crossed Estate to the susceptible cultivar Moore and first hypothesized the location of *Rph3* in either chromosome 7H or 2H. Jin et al. (1993) confirmed the chromosome 7HL position of *Rph3* in Estate through its linkage to the morphological marker *Xnt1* (Xantha seedling 1). In this study, infection types between Estate and its derived introgression line, BW746, were nearly identical in response to the 12 *P. hordei* isolates (Table 5). Definitive evidence for the chromosome 7HL position of *Rph3* was revealed through molecular marker data. In BW746, BOPA SNP markers were detected between 119.8 and 126.1 cM in chromosome 7HL. GBS SNP markers placed this ~8.5 Mb introgression at 628.4 to 636.9 Mb in 7HL (Figure 2). Five other alleles of *Rph3* were hypothesized to exist in the introgression lines BW675, BW687, BW740, BW745, and BW747 derived from Gija/Tanekase, PI 531901-2, Q21861, PC249, and PI 466324, respectively (Appendix Table 5). BW746 was selected as the *Rph3* source for

the final differential set because it exhibits a phenotype to *P. hordei* that is very similar to its donor source, carries only a single introgression, was developed using more backcrosses than other *Rph3*-introgression lines, and was derived from the *Rph3* donor that has been most widely used in the past.

In this study, both phenotype and genotype data strongly suggested that *Rph3* was present in Q21861. The reaction patterns of Q21861 and BW740 were more similar to donor and introgression lines containing *Rph3* than to those carrying *Rph2*. If Q21861 and BW740 did not carry *Rph3*, they would be susceptible to isolates Ger 7, 98-20, and 94-8 (Appendix Table 5). At the BC₁ generation, there are at least 13 introgressions in BW740. There is an introgression in chromosome 7HL near the *Rph3* locus and no introgression in chromosome 5H near the *Rph2* locus. Previously, using bulked segregant analysis, Poulsen et al. (1995) revealed that Q21861 carries an allele of *Rph2*, but the presence of *Rph3* was not detected. In the investigation of Borovkova et al. (1997), a *P. hordei* isolate avirulent for both *Rph2* and *Rph3* was used during their genetic investigation of Q21861 and *Rph3* was not detected in their study either, adding further evidence that these two genes do not co-exist in this line. Hypothetically, if Q21861 carries both *Rph2* and *Rph3*, then *Rph2* could have been separated from *Rph3* in the segregating populations studied by Borovkova et al. (1997), explaining why their observed resistance segregation fit a single-gene model. Either Q21861 carries multiple genes for leaf rust resistance and its copy of *Rph2* was not captured in the introgression line BW740, or the source of Q21861 used in this or the previous studies was incorrect.

Rph4.d

First described in the cultivar Gold (CIho 1145), *Rph4* was also found to exist in Lechtaler (CIho 6488), Franger (PI 180669), and in this study, the cultivar Batna (CIho 3391), also reported to carry *Rph2* (Roane and Starling, 1967 & 1970; Jin and Steffenson, 1996). Previously, the resistance genes in Gold and Lechtaler were designated as the same allele (*Rph4.d*) at the *Rph4* locus. Using trisomic analysis, Tan (1978) mapped *Rph4* in chromosome 1H. In this study, the three *Rph4* sources of Gold, Lechtaler, and Batna were used to develop the introgression lines of BW752, BW753, and BW738, respectively. The general reactions between Gold and BW752 were more similar than those between Lechtaler and BW753 (Appendix Tables 5 and 7). Both BW752 and BW753 contained a BOPA SNP marker at the telomere of chromosome 1HS. GBS analysis detected overlapping introgressions (from ~52 to 290 kilobases [Kb]) near the telomere of chromosome 1HS in both lines, confirming the original 1HS chromosomal assignment for *Rph4* by Tan (1978). BW752 was selected as the *Rph4* source for the final differential set because, despite having three other introgressions in addition to chromosome 1HS, it has a phenotype very similar to its donor source (Figure 2). Moreover, this introgression line was derived from Gold, the *Rph4* donor most widely used as a differential line in the past.

In two previous studies, Batna was reported to carry *Rph2* based on its reaction to a suite of different *P. hordei* isolates (Chicaiza 1996; Jin and Steffenson 1996). In this study, phenotypic and genotypic analysis of Batna and its derived introgression line BW738 revealed the possible presence of *Rph4*. Batna and BW738 exhibited general reactions that were very similar to each other and also to the other *Rph4*-carrying donor and introgression lines (Gold, Lechtaler, BW752, and BW753) (Appendix Table 5). In contrast, some of their general reactions were distinct from the *Rph2* donors and their derived

introgression lines. GBS SNP markers revealed a single donor segment in BW738 that overlapped with the 1HS telomeric introgression of the *Rph4*-carrying lines of BW752 and BW753. Again, if the seed source used in this study is correct, then based on these data, it appears that Batna and BW738 likely carry an allele of *Rph4* and not *Rph2* as previously hypothesized. Another new leaf rust resistance gene that is more broadly effective than *Rph4* was discovered in chromosome 1HS near the *Rph4* locus (Konig et al., 2012). This gene was derived from the Yugoslavian landrace MBR1012 and was given the temporary designation of *RphMBR1012*. This new gene segregated from *Rph4* in an allelism test and will likely be designated as a new *Rph* gene (D. Perovic, M. Martin, and B. Steffenson, unpublished).

Rph5.e and *Rph5.f* (*Rph6*)

Rph5 was originally described as one of two *Rph* genes in the Australian landrace Quinn (PI 39401) and was bred into the Argentinian cultivar Magnif 102 (PI 337140) (Roane and Starling, 1967). In a trisomic study, Tan (1978) positioned *Rph5* from Quinn in chromosome 3H and described its tight linkage with *Rph7* from Cebada Capa. Roane and Starling (1970) first described *Rph6* as one of two *Rph* genes (with *Rph2*) in the Bolivian landrace eponymously named Bolivia (PI 36360). Using progeny testing and *P. hordei* isolates with differential virulence patterns, Zhong et al. (2003) developed a line (Bolivia-*Rph6*) carrying only the putative *Rph6* gene. Molecular mapping and bulked segregant analysis positioned the putative *Rph6* gene in chromosome 3H, near *Rph5* and also *Rph7* (Zhong et al., 2003), corroborating the previous results of Tan (1978). Allelism tests between Bolivia-*Rph6* and Magnif 102 (*Rph5*) and Cebada Capa (*Rph7*) revealed that *Rph6* was allelic with *Rph5* and closely linked with *Rph7*. Although isolates of *P. hordei* (e.g. Ger 11, Mex 930001, 99-26) are capable of differentiating reaction types between the original donor lines of Magnif 102 and

Bolivia-*Rph6* (Fetch et al., 1998), it is still unknown whether these two genes are really alleles of the same gene or are different tightly linked genes at the 3HS locus.

None of the *P. hordei* isolates used in this study were capable of differentiating *Rph5* from *Rph6* in either the donor or introgression lines. Both BW755 and BW756 had overlapping introgressions in chromosome 3HS. For *Rph5* in BW755, only a single BOPA SNP marker from the donor was detected and that was near the 3HS telomere at 1 cM. GBS SNP markers revealed a single ~6.6 Mb introgression from 586 Kb to 7.2 Mb in chromosome 3HS of BW755 (Figure 2). For *Rph6* in BW756, BOPA SNP markers from the donor were detected between 0 and 8.4 cM, while GBS SNP markers placed this ~10.4 Mb introgression from 1.1 to 11.5 Mb also in chromosome 3HS (Figure 2). A second 5.4 cM introgression in chromosome 2HS of BW756 was detected by BOPA SNP markers, but not by GBS SNP markers. Since *Rph5* and *Rph6* are presumed to be allelic, we concur with the *Rph6.f* to *Rph5.f* epithet change as suggested by Franckowiak (1997) in the Barley Genetics Stock (BGS) 575 description. Based on both phenotypic and genotypic results, other alleles of *Rph5* were postulated in the introgression lines derived from PI 531990 (BW676) (Jin and Steffenson, 1994) and the three landraces from Tunisia (Yahyaoui 1986): namely Tunisia 17 (BW757), Tunisia 25 (BW748), and Tunisia 29 (BW750). BW755 and BW756 were selected as the introgression lines representing *Rph5.e* and *Rph5.f* for the final differential set because their phenotypes were very similar to their donor sources, they had fewer introgressions, and their respective donors (Magnif 102 and Bolivia-*Rph6*) were most widely used in the differential set in the past.

Rph7.g

Rph7 was first described in the Argentinian cultivar Cebada Capa and subsequently in Forrajera Klein and other cultivars (Frecha, 1970, Nover and Lehman, 1974). This gene was first positioned in chromosome 3HS by Tan (1978) through trisomic analysis and later corroborated with molecular mapping by Brunner et al. (2000) and Graner et al. (2000). In this study, the general reaction patterns of Cebada Capa and BW758 were identical. Genotype analysis of BW758 identified BOPA SNP markers from the donor between 0 cM and 8.5 cM in the telomeric region of chromosome 3HS, while GBS SNP markers placed this ~12.4 Mb introgression between 438 Kb and 12.8 Mb (Figure 2). This introgression overlaps with those found for *Rph5.e* and *Rph5.f* in BW755 and BW756, respectively, corroborating previously reported evidence of close linkage between the *Rph5/6* and *Rph7* loci (Johnson, 1968; Roane and Starling, 1970; Frecha, 1970; Parlevliet, 1975; and Tan, 1978). Since the telomeric introgression for *Rph7* is longer than that for *Rph5/6* (Zhong et al., 2003), the *Rph7* locus likely exists proximal from the *Rph5/6* locus between 7.2 and 12.8 Mb in chromosome 3HS (Figure 2). Only a single introgression was detected in BW758, and no other introgression lines were hypothesized to carry *Rph7*. BW758 was selected as the *Rph7* source for the final differential set because it carries a single introgression and is the only source of this gene introgressed into Bowman.

Rph8.h

In some earlier investigations, Egypt 4 was used as a universal susceptible in the differential set (Levine and Cherewick 1952; Roane and Starling 1967). Tan (1977a) first described *Rph8* in Egypt 4 after observing its leaf rust resistance during a seedling screening experiment. It is the only reported source of *Rph8* and was used to develop the introgression line BW759 in this study. No studies have been done to assign *Rph8* to a chromosome. The general reactions of BW759 were similar to Egypt 4 in most cases; however, BW759 exhibited moderate reactions to a few *P. hordei* isolates (ARG231, 98-20, and 94-8) for which Egypt 4 was susceptible. This result was likely due to transferring *Rph8* into the Bowman genetic background. Genotypic analysis of BW759 detected BOPA SNP markers from the donor between 24.7 and 38.2 cM in chromosome 2HS. Additionally, GBS SNP markers placed this ~25.8 Mb introgression between 32.7 and 58.5 Mb in the same chromosome (Figure 2). BW759 was selected as the source of *Rph8* for the differential set because no other introgressions exist in BW759, and no other introgression lines were postulated to carry this gene.

Rph9.i and *Rph9.z* (*Rph12*)

Based on their broad resistance to a panel of *P. hordei* isolates virulent for *Rph1-8*, the Ethiopian landrace Hor 2596 and German cultivar Trumpf were both thought to carry a new resistance gene (Clifford and Udeogalanya 1976). Tan (1977a) named the gene *Rph9* after observing segregation in F₂ populations made between landraces considered to carry *Rph9* and the donors of *Rph1-8*. The leaf rust resistance gene carried by Trumpf was investigated after it was rendered susceptible in the field by a virulence shift in the leaf rust pathogen. Walther (1987) noted Hor 2596 may be in the pedigree for Trumpf; however, data

from an allelism test did not unequivocally demonstrate that both lines share the same gene. In a later study, the gene in Trumpf was designated as *Rph12* after Jin et al. (1993) observed not only different reaction types between Hor 2596 and Trumpf to races of *P. hordei*, but also segregation in an allelism test between the two accessions. They also reported the first chromosomal assignment of this gene after linkage between *Rph12* and the semi-smooth awn (*r* locus) in chromosome 5HL was observed. Borovkova et al. (1998) evaluated a much larger population (3,800 F₂ progeny) between Hor 2596 and Trumpf and found no susceptible segregants. Based on this result, they concluded that the gene in Trumpf is an allele of *Rph9*, and designated it as *Rph9.z*. Another allele at this locus was recently described in the Australian cultivar Cantala and mapped to 5HL (Dracatos et al., 2014). Introgression lines BW760 and BW761 were developed from the donor sources of Hor 2596 and Trumpf, respectively. The general reactions between Hor 2596 and Triumph were nearly identical with only isolate Neth 28 differentiating the two (Table 5). This result, plus the definitive allelism test conducted by Borovkova et al. (1998), supports the contention that the originally assigned genes of *Rph9* and *Rph12* are indeed different alleles at the same locus. The chromosome 5H location for *Rph9.z* (and therefore *Rph9.i* by lieu of linkage), originally postulated by Jin et al. (1993), was confirmed by genotyping in this study as both BW760 and BW761 had overlapping introgressions (from 605 to 614 Mb) in chromosome 5HL. In BW760, BOPA SNP markers from the donors were detected between 114 to 135.1 cM and in BW761, between 119 to 155.8 cM. GBS SNP markers revealed ~36.4 and ~77.7 Mb introgressions from 578.3 to 614.7 Mb and from 577.5 to 655.3 Mb in BW760 and BW761, respectively (Figure 2). No other introgressions were detected in either of these introgression lines. Since the current genetic evidence indicates that *Rph9* and *Rph12* are alleles, we concur with the recommended allele designation of *Rph9.i* for the gene in Hor 2596 and *Rph9.z* for the gene in Trumpf as suggested by Franckowiak (1997) in the BGS 333 description. Although four

other introgression lines were hypothesized to carry the same gene, BW760 and BW761 were selected as the sources for *Rph9.i* and *Rph9.z* because they were derived from the original differential sources of these genes, developed with more generations of backcrossing, and their general reactions were the most similar to their donor parents (Appendix Table 5).

Rph10.o and *Rph11.p*

Rph10 and *Rph11* were originally derived from two wild barley (*Hordeum vulgare* subsp. *spontaneum*) accessions, Bar Giyyora 30 and Maalot 17, respectively (Feuerstein et al., 1990). Backcross selections made from crosses between the Australian cultivar Clipper and Bar Giyyora 30 (Clipper BC8) and Maalot 17 (Clipper BC67) became the donor sources of *Rph10* and *Rph11*, respectively, in the barley leaf rust differential set. Currently, these selections are the only known sources for these genes. *Rph10* and *Rph11* were positioned in 3HL and 6HL, respectively, using isozyme markers (Feuerstein et al., 1990). Bowman introgression line BW683 was derived from Clipper BC8, whereas BW684 was derived from Clipper BC67. Although reported as being broadly resistant to leaf rust in Australia, neither Clipper BC8 nor Clipper BC67 exhibited highly resistant reactions to the panel of 12 *P. hordei* isolates used in this study. Both the donor lines and their respective introgression lines produced a similar pattern of general reactions to the leaf rust pathogen, with moderate ITs predominating (Table 5). In BW683, BOPA SNP markers identified an introgression from 52.8 to 68.2 cM in chromosome 3HL, supporting the original chromosome assignment for *Rph10* established by Feuerstein et al. (1990). GBS SNP markers placed this ~79.7 Mb introgression from 490.3 to 570 Mb in 3HL (Figure 2). In BW684, one BOPA SNP marker from the donor was identified at 117.6 cM in chromosome 6HL. The GBS SNP markers placed this ~10.9 Mb introgression from 561.9 to 572.8 Mb in the same chromosome. No other

introgressions were detected in BW684 (Figure 2). These data confirm the original chromosome 6HL assignment for *Rph11* by Feuerstein et al. (1990).

In BW683, although the BOPA SNP marker data highlighted two additional introgressions in chromosome 3HS and 7HL, the GBS SNP data indicated a third introgression of ~5.9 Mb within the same region as the 6HL introgression containing *Rph11* in BW684. The 6HL introgression in BW683 is not entirely comprised of non-Bowman type alleles and is perhaps an example of a data quality issue with the detection of this introgression. In previous studies by Feuerstein et al. (1990) and Druka et al. (2011), it is possible that the 6HL introgression in BW683 was not detected due to a paucity of markers. Few scattered markers (mostly isozymes and morphological) were available in the study by Feuerstein et al. (1990). Although a larger number of BOPA SNP markers (3,072) were available in the study by Druka et al. (2011), it is still possible that their distribution failed to detect the smaller 6HL introgression in BW683. The potential presence of overlapping 6HL introgressions in both BW683 and BW684 raises a question about whether *Rph10* exists as a unique gene in chromosome 3HL or is an allele of the resistance gene at the *Rph11* locus in chromosome 6HL. *Rph10* and *Rph11* exhibited very similar general reactions to *P. hordei* in this study, a common hallmark of resistance alleles (Table 5). Additional research should be done to resolve the mapping position of *Rph10* and whether it could be allelic to *Rph11*, whose mapping position was well established. Without stronger evidence, *Rph10* is still assumed to exist in 3HL and *Rph11* in 6HL. BW683 and BW684 were selected as the *Rph10* and *Rph11* sources, respectively, for the final differential set because no other donor sources of these genes are known.

Rph13.x

PI 531849 is an experimental barley line from the United Kingdom and was originally derived from a wild barley accession. It exhibited a high level of resistance to a panel of isolates virulent for *Rph1-12* and segregated for a single resistance gene when crossed to Bowman (Jin and Steffenson, 1994). Subsequent allelism tests with the *Rph1-12* donors produced two-gene and three-gene (i.e. the cross with Bolivia having both *Rph2* and *Rph6*) segregation ratios, indicating that the leaf rust resistance gene in PI 531849 was unique. Jin et al. (1996) designated the gene as *Rph13*. BW685, the introgression line derived from PI 531849, had a general reaction pattern similar to its donor, but showed a lower level of resistance to four of the *P. hordei* isolates (ARG231, Race 4, 94-8, and 90-3) tested in this study (Table 5). Jin et al. (1996) observed a deficiency of susceptible F₂ progeny in a cross between PI 531849 and Hor 2596 suggesting that this locus was possibly linked with the *Rph9* locus in chromosome 5HL. However, BOPA SNP data for BW685 revealed only one introgression between 103.7 and 154.9 cM in chromosome 3HL. The GBS SNP markers identified two introgressions in 3HL, one of ~30.6 Mb from 640.3 to 670.9 Mb and the second nearly 14 Mb from 684.6 to 698.5 Mb (Figure 2). BW685 was selected as the *Rph13* source for the differential set because PI 531849 is the only known source of this gene, its general reaction pattern matched its donor parent, and no other introgressions were detected in BW685 by either BOPA or GBS SNP data.

Rph14.ab

Rph14 was identified in the Egyptian line PI 584760, which was originally selected from PI 531901-1 (NDBLR01) (Jin et al., 1996). Crosses between PI 584760 and Bowman revealed a single incompletely dominant gene. Allelism tests between this line and the sources of *Rph1-13* confirmed this gene as novel and hence it was designated *Rph14*. Using Diversity Arrays Technology (DART) markers, Golegaonkar et al. (2009) mapped *Rph14* to chromosome 2HS. The general reactions between PI 584760 and its introgression line BW686 were similar (Table 5). The *P. hordei* isolates Race 4, WGCC, and Neth 28 produced more compatible infections on BW686, while one isolate, ARG231, was more compatible on the parent (Table 5). In BW686, BOPA SNP markers revealed an introgression around 42 cM in 2HS, confirming the initial gene mapping results of Golegaonkar et al. (2009). GBS SNP markers placed this ~11.6 Mb introgression from 45.7 to 57.3 Mb in chromosome 2HS (Figure 2). Two other introgressions were detected in BW686, one in chromosome 1HL and another in chromosome 6HL. BW686 was selected as the *Rph14* source for the differential set because no other donor sources of this gene are known, its general reaction pattern was similar to its parent, and no other introgressions were detected.

Rph15.ad

Rph15 was identified in a wild barley accession (PI 355447) from Israel by Chicaiza (1996). It has a wider resistance spectrum than any of the *Rph1-14* genes to diverse *P. hordei* isolates (Table 4). Crosses made between PI 355447 and morphological marker stocks suggested a possible chromosome 2HL position for the gene based on linkage to the six-rowed spike 1 (*vsr1*) locus and the ribbon grass (white streak 7, *wst7*) locus. Soon after *Rph15* was described, Ivandic et al. (1998) identified another new leaf rust resistance gene (designated

Rph16) in a different accession of wild barley. This gene was mapped in chromosome 2HS using restriction fragment length polymorphism (RFLP) markers. Weeraseena et al. (2004) conducted an allelism test between *Rph15* and *Rph16* and concluded that these genes were likely alleles at the same locus. They also mapped *Rph15* to the same locus as *Rph16* using amplified fragment length polymorphism (AFLP) markers. Kopahnke et al. (2004) investigated another widely effective leaf rust resistance gene from a different wild barley accession (*H. spontaneum* 677). Based on a doubled haploid population genotyped with AFLP markers, they positioned this gene at or very near the *Rph15/Rph16* locus.

In this study, 50 different wild barley accessions with a wide resistance spectrum similar to the *Rph15* donor (PI 355447) were used to develop introgression lines in cultivar Bowman (Appendix Table 6). Subsequent phenotypic and genotypic analysis revealed that all of these introgression lines had broad spectrum resistance and carried an introgression at the same location. Of these 50 introgression lines, just five carry a single introgression at the *Rph15/Rph16* locus. In BW719, BOPA SNP markers from the donor were detected between 39.7 and 44 cM in chromosome 2HS, and GBS SNP markers placed this ~12.4 Mb introgression from 44.8 to 57.3 Mb (Figure 2). BW719 was selected to represent *Rph15* in the differential set because it was derived from the wild barley accession PI 355447, has a single introgression at the *Rph15/Rph16* locus, and retained the least amount of donor chromatin.

The introgression for *Rph15* overlaps with the introgressions for *Rph14* and *Rph8*. The introgression for *Rph8* begins more than 10 Mb distal from the *Rph14* and *Rph15* introgressions and ends about 1.2 Mb proximal from their ending positions. This indicates that *Rph8* may lie in 2HS somewhere either in the distal region between 32.7 and 44.8 Mb or in the proximal region between

57.3 and 58.5 Mb. The introgression for *Rph14* begins about 900 Kb proximal from the starting position for *Rph15*, and both of these introgressions end at the same position. This suggests that either *Rph15* lies somewhere in the 900 Kb region between 44.8 and 45.7 Mb or both of these genes are tightly linked within the 11.6 Mb region from 45.7 to 57.3 Mb. Additionally, the chromosome 2HS introgression carrying *Rph15* in three introgression lines (BW723, BW724, and BW725) was translocated into chromosome 4HS (Franckowiak, personal communication).

Other Leaf Rust Resistance Genes

BW731, BW677, BW741, and BW754 were all derived from leaf rust resistance donors not previously thought to carry *Rph9* (PI 466247, PI 531853, and Quinn) and had introgressions spanning the same physical positions as the *Rph9*-carrying introgressions from BW760 and BW761. BW731, derived from wild barley accession PI 466247, exhibited enhanced levels of resistance to several *P. hordei* isolates compared to other introgression lines carrying *Rph9* alleles (Appendix Table 5). This result could be due to a single *Rph9* allele providing a wider resistance spectrum or possibly an additional seedling resistance QTL in BW731. In comparison to other *Rph9*-carrying introgression lines, the 5HL *Rph9*-introgression in BW731 begins at a position more proximal, overlapping with the reported positions of two seedling QTLs (*Rphq7* and *Rphq17*) at ~155.6 cM (Schnaithmann et al., 2014). Additionally, it also carries introgressions in chromosomes 3H and 7H. The introgression in 3HS overlaps with the *Rph5* locus, and the one in chromosome 7H coincides with two other previously reported seedling resistance QTL, *Rphq1* and *Rphq8* (Marcel et al. 2007, Schnaithmann et al. 2014). It is unlikely that BW731 carries an allele of *Rph5* based on the differential reactions it exhibits to specific *P. hordei* isolates. A more likely possibility is that the enhanced resistance is due to one or more

seedling QTL present in the line. Derived from another wild barley accession (PI 531853), BW677 is a different introgression line that expressed enhanced resistance to some *P. hordei* isolates compared to other *Rph9*-carrying lines (Appendix Table 5). BW677 has an additional introgression in 2HL that coincides with the previously reported location of another seedling resistance QTL, *Rphq11* (Marcel et al. 2007). The last two introgression lines with phenotypes similar to *Rph9* lines were BW741 and BW754, both derived from Quinn and sharing identical general reactions with each other. Both BW741 and BW754 have an introgression in 5HL coinciding with the *Rph9* locus and their general reaction patterns are most similar to other *Rph9*-carrying introgression lines. The donor parent Quinn is known to carry both *Rph2* and *Rph5*, and was not previously postulated to carry an allele at the *Rph9* locus. An allelism test between BW741 or BW754 and either Hor 2596 or Triumph is necessary in order to resolve the identity of the other *Rph* gene in Quinn.

Of the 50 introgression lines postulated to carry *Rph15* from wild barley, three (BW697, BW691, and BW709) displayed enhanced levels of resistance to one or two of the *P. hordei* isolates (Appendix Table 6). BW697, derived from PI 391121, showed a highly resistant reaction to isolate Neth 28, unseen in other lines thought to solely carry *Rph15*. The BOPA and GBS SNP marker data revealed a ~13.1 Mb introgression in chromosome 3HS that overlaps with both the *Rph5/6* and *Rph7* loci. Neither *Rph5/6* nor *Rph7* confers resistance to isolate Neth 28, indicating that either this line has an *Rph15* translocation at this locus or a potentially new gene exists in this region with a resistance spectrum similar to *Rph15* alleles. BW691, derived from PI 391045, was moderate in its reaction to isolate 90-3 and highly resistant to Neth 28. It has introgressions in every chromosome except for chromosome 1H, some of which co-locate to previously reported QTL for leaf rust resistance at the seedling stage. While the presence of any of these QTL has not been confirmed in BW691, it should be noted that

introgressions coincident with seedling resistance QTL exist, such as one unnamed QTL in chromosome 3HL reported by Jafary (2006), *Rphq10* in 4HS, *Rphq20* in 4HL, *Rphq4* in 5HS, and *Rphq7* or *Rphq16* in 5HL (Marcel et al., 2007; Marcel et al., 2008; Schnaithmann et al., 2014). BW709, derived from PI 405399, exhibited a moderate level of resistance to isolates 90-3 and Neth 28 (Appendix Table 6). While BOPA SNP markers revealed introgressions only in chromosomes 2HS and 4HL, GBS SNP markers indicated that additional introgressions exist in chromosomes 1HL and 2HL. In addition to likely carrying *Rph15*, BW709 could also contain the chromosome 2H QTL of *Rphq6* and/or *Rphq18* or the chromosome 4H QTL of *Rphq5* and/or *Rphq20* (Marcel et al., 2007; Schnaithmann et al., 2014).

Eleven introgression lines had a reaction pattern unlike that of *Rph1-15*. Some of these lines contain other major resistance genes beyond these 15 genes and others contain introgressions that coincide with several previously reported seedling leaf rust resistance QTL. Two members of this group, BW735 and BW736, were respectively derived from the *Hordeum bulbosum* introgression lines 81882 (Vada/*H. bulbosum*), which carries *Rph17*, and 38P18/9/4 (Emir/*H. bulbosum*), which carries *Rph18*. BW735 has an introgression in chromosome 2H that overlaps with the *Rph17* region previously reported by Pickering (1997), but also several other introgressions as well. BW736 has an introgression in 2H that overlaps with the *Rph18* region previously reported by Pickering (1999), but again carries several other introgressions. Both BW735 and BW736 exhibited reaction patterns very similar to their respective parents (Appendix Tables 5 and 7, respectively), indicating the successful transfer and expression of the respective resistance genes in a Bowman background. BW726, an F₃ inbred line derived from the wild barley accession, HS584, exhibited highly resistant reactions to all 12 *P. hordei* isolates used in this study and carries a total of 16 distinct introgressions that span all arms of the seven chromosomes except 6HL.

Unless a new gene for resistance exists in one of the many introgressions of BW726, the only known *Rph* genes that could result in this phenotype would be a combination of *Rph13* and *Rph15*. BW672, derived from another wild barley accession (PI 466373), has an introgression overlapping the position of *Rph8* in chromosome 2H, but its reaction pattern is different to some *P. hordei* isolates in comparison to the reactions for *Rph8.h* in BW759 (Table 5). Other introgressions that may be affecting the reaction spectrum of BW672 overlap with the reported positions of the seedling resistance QTL, *Rphq10* in chromosome 4HS and *Rph11* in chromosome 6HL (Marcel et al., 2007).

The seven remaining introgression lines exhibited partially or fully incompatible reactions to a few of the *P. hordei* isolates, indicating that they carry at least one effective resistance gene. The three most resistant members of this group (BW727, BW728, and BW749) each have an introgression that overlaps with the reported region of *Rphq21* in chromosome 1HS near the centromere (Marcel et al., 2008). While BW728 and BW749 carry only one introgression that possibly contains *Rphq21*, BW727 carries several other introgressions that overlap with the positions of other reported QTL for leaf rust resistance. This may explain why, out of these remaining seven introgression lines, BW727 is the least compatible with isolates of *P. hordei*. It is also possible that BW749 carries an allele of *Rph19* in chromosome 7HL because it has a coincident introgression in this region and also a reaction pattern similar to this gene derived from the cultivar Prior.

The remaining four introgression lines were generally susceptible, but exhibited an incompatible reaction to at least one isolate of *P. hordei* (Appendix Table 5). Based on their introgression positions, each of these four lines may contain different combinations of *Rph* genes and/or previously reported seedling resistance QTL. BW668 expressed a moderate reaction to four of the *P. hordei*

isolates and may carry *Rphq9* in chromosome 7H (Qi et al., 1998, Marcel et al., 2007). BW698 expressed a moderate reaction to three *P. hordei* isolates and may contain *Rphq6* in chromosome 2H, *Rphq8* in chromosome 7H, and/or *Rphq19* in chromosome 4H. BW701 only occasionally exhibited a moderate response against a few *P. hordei* isolates in just one of three experiments. Although it contains an introgression in chromosome 6H coincident with the purported location of *Rphq3*, without a more consistent resistant reaction across replications, it is unlikely BW701 contains this QTL (Marcel et al., 2007, Schnaithmann et al., 2014). Finally, BW751 does not appear to carry any introgressions. It gave a moderate reaction in only one experiment against a single *P. hordei* isolate, which is likely due to environmental differences between the experimental replications.

Rph Gene Deployment and Effectiveness

Many *Rph* genes have been deployed in barley cultivars grown across diverse production regions of the world. Despite the long track record of failure, most of these deployed cultivars were bred with only a single leaf rust resistance gene. Except for *Rph2* and *Rph7*, nearly all other *Rph* genes deployed in cultivars have failed to provide lasting resistance in any of the production regions where they have been grown. *Rph1* was first released in the Minnesota cultivar Manchuria in 1918 and was found to be susceptible to an isolate in 1925 (Mains, 1930). Unlike *Rph1*, *Rph2* was first deployed in the South American landrace Peruvian in the early 20th century and did not succumb to a virulent *P. hordei* race until 1960 in Argentina (D'Oliveira, 1961). Henderson (1945) first described *Rph3* in the Egyptian line Aim, but the donor source more widely used in breeding is the Egyptian landrace Estate. *Rph3* has been deployed in Europe, Oceania, and North America. In the Czech Republic, the cultivar Karat with *Rph3* was released in 1981 and by 1988 a virulent race had rendered this gene

completely ineffective (Dreiseitl and Steffenson, 2000). Before the release of *Rph3*-containing barley varieties, fifty years of virulence surveys did not detect virulence for *Rph3* in the nation's *P. hordei* population. Several Australian barley varieties (Fitzroy, Yarra, Starmalt, and others) with *Rph3* were released in the early 2000's and the resistance conferred by this gene was rendered ineffective as early as 2008 (Park, 2013). In the southeastern United States, *Rph3* was deployed in the cultivar Doyce in 2003 and was rendered susceptible by mutations in the pathogen in 2010 (Rouse et al., 2013). Virulence for *Rph4* is very common around the world. Cultivars carrying *Rph4* often possess other genes for leaf rust resistance in production regions where this virulence abounds, such as with the Australian cultivar Tallon, known to carry both *Rph4* and *Rph12* (Christopher et al., 2007). *Rph5* and *Rph6* have been deployed in Australia and the Americas. *Rph5* is present in the Australian cultivar Quinn and was incorporated into the Argentinian cultivar Magnif 102. *Rph6* is carried by the South American landrace Bolivia. Reports of these cultivars succumbing to virulent *P. hordei* isolates are not well documented, but virulence for both of these genes is common around the world (Rintelen, 1974; Khokhlova, 1982; Andres et al., 1983; Yahyaoui et al., 1988; Manisterski, 1989; Cotterill et al., 1995). One of the most durable leaf rust resistance genes used in agriculture is *Rph7*, a gene widely used in the United States and derived from the cultivar Cebada Capa. Varieties carrying *Rph7* were released in the Mid-Atlantic region of the United States starting in 1968 with the cultivar Hanover. A virulent isolate was not identified until 22 years later in 1990 (Steffenson et al., 1993). *Rph8* has apparently never been deployed in a widely grown cultivar to our knowledge. *Rph9* has been largely utilized in Australia and was first deployed in 1989. By 1991, a virulent isolate was collected on the cultivar Tallon in Tasmania and hypothesized to have been introduced to Australia from New Zealand (Cotterill et al., 1992b). *Rph12* was incorporated into European and Australian cultivars and rendered ineffective by shifts in pathogen virulence within a very short time. In

Germany, the cultivar Trumpf with *Rph12* was released in 1973, and virulent pathotypes of *P. hordei* were observed as early as 1977. In Australia, cultivar Franklin with *Rph12* was released in 1989 and rendered susceptible by *Rph12*-virulent isolates in 1993 (Dreiseitl and Steffenson, 2000; Park, 2003). *Rph10* and *Rph11* were both backcrossed into the cultivar Clipper, but have not been deployed in agriculture. Neither *Rph13*, *Rph14*, nor *Rph15* have been deployed in cultivars at this time.

In a study by Fetch et al. (1998), more than 350 isolates of *P. hordei* from around the world were examined on a differential set consisting of the original donors of genes *Rph1-15*. The frequencies of virulence to each of these genes were reported by region. On a global scale, *Rph14* and *Rph15* were the most widely effective genes being resistant to 98% and >99% of the isolates, respectively. The next most widely effective genes were *Rph3*, *Rph7*, *Rph9*, and *Rph12*, which were all resistant to 95% of the isolates. *Rph2*, *Rph10*, *Rph11*, and *Rph13* were resistant against 30 to 50% of the evaluated isolates, while *Rph5* and *Rph6* were resistant to about 20% of the isolates. *Rph1*, *Rph4*, and *Rph8* were the least effective genes, conferring resistance to <10% of the isolates.

Strong regional differences for *P. hordei* virulence have been reported. In North America, no virulence has been detected for *Rph12*, *Rph14*, or *Rph15* and less than 1% of the isolates were virulent on *Rph3* and *Rph7* (Fetch et al., 1998). In the last four years (2014-2017) of United States *P. hordei* surveys, virulence for genes *Rph1-14* has been detected while *Rph15* resistance has remained intact (M. Rouse, personal communication). The genes *Rph7*, *Rph9*, *Rph12*, *Rph13*, and *Rph15* were all effective against a South American collection of the pathogen. However, an isolate with virulence for *Rph9* and *Rph12* was recently detected in Uruguay (Hickey et al., 2011). *Rph15* was effective against all *P. hordei* isolates from Africa, while *Rph3*, *Rph13*, and *Rph14* were effective against

89%. *Rph3* and *Rph7* were reported to be effective against all isolates in Morocco, Tunisia, Egypt, and Ethiopia (Yahyaoui et al., 1988; Woldeab et al., 2006). In Israel, Jordan, and Syria, only *Rph3* and *Rph7* have remained effective against the pathogen, while a few reports have listed *Rph7* and *Rph9* virulence as very rare (Yahyaoui et al., 1988; Brodny and Rivadeneira, 1996). In Europe, no virulence has been detected for *Rph15*, while *Rph14* virulence remains rare. *Rph7* has only been overcome by 8% of the European *P. hordei* isolates. It appears that *Rph7* is still effective in the United Kingdom and Spain as no virulence for the gene has been reported. No recent reports of *P. hordei* virulence from Spain have been published since the work of Shtaya et al. (2006). The same is true for the United Kingdom since the elimination of barley leaf rust from the UK Cereal Pathogen Surveys (Jones, 2005; Shtaya et al., 2006). In Australia, only *Rph7* remains effective as a singly deployed resistance gene. No virulence has been detected for the non-deployed genes of *Rph11*, *Rph14*, or *Rph15* in Australia (Park, 2003; Park et al., 2015).

According to Fetch et al. (1998), it is advisable to combine multiple major effect resistance genes with minor effect resistance genes in order to provide durable leaf rust resistance. In barley production regions today, the most effective genes for combining are *Rph7*, *Rph9*, and *Rph12-15*. In Africa, *Rph5* and *Rph6* may also be worth including because they have not previously been deployed in cultivars. Although virulence for these genes in North and East African *P. hordei* populations has been detected, those isolates with virulence are not the predominantly isolated pathotypes (Yahyaoui and Sharp, 1987; Woldeab et al., 2006). Aside from *Rph1-15*, *Rph20* has shown broad effectiveness at the seedling and adult plant stage against the 12 isolates tested in this study. Other resistance genes such as *Rph21*, and *Rph23-25* may also be useful for gene pyramiding. Until the large linkage blocks containing the *H. bulbosum*-derived genes of *Rph17*, *Rph18*, and *Rph22* can be reduced to minimize linkage drag,

these genes may be difficult to utilize in elite breeding programs. Towards this effort, Pickering et al. (2000) dissected the genomic region where *Rph18* was introgressed into cultivated barley from *H. bulbosum*. Their work will help elucidate the causal gene for *Rph18* and make available a better genetic resource for this gene. In addition to the major effect *Rph* genes, minor effect QTLs should also be targeted for gene stacking. Derived from the broadly resistant cultivar Vada, *Rphq2* and *Rphq11* both significantly reduce disease severity at the seedling stage, while *Rphq3* and *Rphq4* are both effective at the adult plant stage (Qi et al., 1998; Spaner et al., 1998; Marcel et al., 2007).

Candidate Leaf Rust Resistance Genes

Within the 15 introgression lines selected for the final barley leaf rust differential set, we explored whether annotated genes on the same contigs as the BOPA SNPs might be present by performing BLAST searches against the barley reference sequence of the cultivar Morex (Mascher et al., 2017). The entire list for the annotated gene constructs are compiled in Appendix Table 9. From this list, only genes implicated in disease resistance functions were collated into Table 7. In total, 41 genes with some putative disease resistance function were found on the same contigs as BOPA SNP markers from within these *Rph*-gene containing introgressions. Six of these genes have leucine rich repeat receptor-like kinase domains that are most commonly associated with disease resistance against biotrophic pathogens (Ellis et al., 2014). These were found in the introgressions for *Rph2.t*, *Rph9.i*, *Rph10.o*, *Rph9.z*, and *Rph13.x*. Several other genes implicated in disease resistance were also identified. Peroxidases within a plant's apoplast are involved in signaling the presence of pathogens by creating bursts of reactive oxygen species (Chen et al., 2010). Ubiquitinases catalyze the attachment of ubiquitin that signals potentially invasive proteins for degradation (Zhou et al., 2017). Cysteine proteases have recently been found to signal

downstream defense responses such as hypersensitive cell death (Misas-Villamil et al., 2017). Pectin lyases may be involved in transducing signals downstream of the initial recognition event (Cao, 2012). Finally, reticulon-like proteins are involved in trafficking and activating certain immune receptors (Lee et al., 2011). These types of genes, along with various kinds of transcription factors were found within the introgressions for *Rph1.a*, *Rph2.t*, *Rph3.c*, *Rph7.g*, *Rph9.i*, *Rph10.o*, *Rph9.z*, *Rph13.x*, and *Rph15.ad* (Table 7).

Summary

The development of introgression lines each possessing a single *Rph* gene in the genetic background of the susceptible cultivar Bowman is an ideal genetic resource for characterizing the virulence diversity of *P. hordei*. By developing these introgression lines through repeated backcrossing, the effect of individual *Rph* genes can be assessed without influence from other possible epistatic resistance or various agro-morphological traits. Moreover, since Bowman is an agronomically advanced and widely adapted cultivar, the differential set developed and characterized in this study can be grown in many different environments to assess the virulence of naturally occurring *P. hordei* isolates and also the effect of individual *Rph* genes in conferring resistance. In this study, we characterized the resistance spectra and chromosomal positions of 95 *Rph* introgression lines in a Bowman background using 12 global isolates of *P. hordei* and SNP genotyping. The results validated the successful transfer of genes *Rph1-15* and provided important new data on their chromosomal positions based on the physical map of barley.

Fifteen introgression lines were selected to represent *Rph1-15* in a proposed new barley leaf rust differential set. Eleven of these 15 lines carry a single introgression that agrees with previously described mapping positions for

the respective *Rph* genes. The other four differential lines carried multiple introgressions, with one at the putative location of the gene. Highly similar phenotypes and overlapping introgressions were found between *Rph5* and *Rph6*, *Rph9* and *Rph12*, and *Rph10* and *Rph11*; thus, it was not possible to determine whether these pairs represent different tightly linked genes or alleles of the same gene. In the future, an allelism test between BW683 and BW684 should be performed to determine whether *Rph10* and *Rph11* are unique genes or alleles of the same gene. Allelism tests have already been used to study the relationships of *Rph5/Rph6* and *Rph9/Rph12*, and these two pairs of genes appear to be alleles of each other. In these cases, seed stocks for the respective introgression lines could be mutated to knockout the causal gene(s). Tight genetic linkage was observed among *Rph5*, *Rph6*, and *Rph7* in chromosome 3H and also among *Rph8*, *Rph14*, and *Rph15* in chromosome 2H. For introgressions of different genes that overlap in the same chromosome segment, identifying the physical positions of the causal genes would clarify their exact physical positions.

Eleven of the 95 introgression lines exhibited reaction patterns distinct from those exhibited by *Rph1-15*. Many of these lines contained introgressions that coincided with several previously reported seedling leaf rust resistance QTL. Whether or not QTLs exist in any of these lines was not confirmed. One highly resistant backcross line (BW697) may contain either an *Rph15* translocation, a novel allele at the *Rph5* or *Rph7* locus, or a closely linked gene that has not yet been described. Allelism tests should be performed between lines containing *Rph5*, *Rph6*, *Rph7*, and BW697.

Currently, the major effect leaf rust resistance genes of *Rph3*, *Rph7*, *Rph9*, *Rph12*, *Rph14* and *Rph15* are the most broadly effective in barley. The respective introgression lines for these genes, BW746, BW758, BW760, BW761, BW686, and BW719, could be the preferred resource for the stacking or cloning

of these resistance genes. For the 15 introgression lines selected for the final leaf rust differential set, annotated gene constructs within their *Rph* gene-containing introgressions were recorded. Many genes with disease resistance functions were found and six genes with leucine rich repeat receptor-like kinase descriptions may represent the candidate genes for *Rph2.t*, *Rph9.i*, *Rph9.z*, and *Rph13.x*. A mutant gene knockout approach could be applied to seed stocks of these introgression lines to determine which particular candidate gene(s) may be responsible for the disease resistance effect.

Table 1. Accession details for Bowman and the 15 selected parental donors of leaf rust resistance.

Accession Name	Accession Number ¹	Origin	Improvement Status ²	Gene(s)	Allele(s) ³
Bowman	PI 483237	North Dakota, USA	Cultivar	NA	NA
Sudan	CIho 6489	Lisbon, Portugal	Unknown	<i>Rph1</i>	<i>Rph1.a</i>
Reka I	CIho 5051	New South Wales, Australia	Cultivar	<i>Rph2+19</i>	<i>Rph2.t+19.ah</i>
Estate	PI 57700	Sharqiya, Egypt	Landrace	<i>Rph3</i>	<i>Rph3.c</i>
Gold	CIho 1145	Malmöhus, Sweden	Cultivar	<i>Rph4</i>	<i>Rph4.d</i>
Magnif 102	PI 337140	Buenos Aires, Argentina	Cultivar	<i>Rph5</i>	<i>Rph5.e</i>
Bolivia	PI 36360	Oruro, Bolivia	Landrace	<i>Rph6</i>	<i>Rph2.r+Rph5.f</i>
Cebada Capa	PI 539113	Argentina	Cultivar	<i>Rph7</i>	<i>Rph7.g</i>
Egypt 4	CIho 6481	Germany	Unknown	<i>Rph8</i>	<i>Rph8.h</i>
Hor2596	CIho 1243	Ethiopia	Landrace	<i>Rph9</i>	<i>Rph9.i</i>
Clipper BC8	GSHO 1588	Australia	<i>H.v.s.</i> backcross line	<i>Rph10</i>	<i>Rph10.o</i>
Clipper BC67	GSHO 1589	Australia	<i>H.v.s.</i> backcross line	<i>Rph11</i>	<i>Rph11.p</i>
Triumph	PI 290195	German	Cultivar	<i>Rph12</i>	<i>Rph9.z</i>
BS 99-2-1	PI 531849	England	Breeding line	<i>Rph13</i>	<i>Rph13.x</i>
NDBLR01	PI 584760	Egypt	Unknown	<i>Rph14</i>	<i>Rph14.ab</i>
703	PI 355447	Israel	<i>H.v.s.</i> accession	<i>Rph15</i>	<i>Rph15.ad</i>

¹. Accession numbers are a combination of an abbreviation and a numerical identifier. PI stands for Plant Introduction, CIho stands for Cereal Intestigation in Hordeum, and GSHO stands for Genetic Stock of Hordeum.

². Improvement status indicates the level of domestication for each of the differential lines. *Hordeum vulgare* subsp. *spontaneum* and *Hordeum bulbosum* are respectively abbreviated as *H.v.s.* and *H.b.*

³. *Rph* allele symbols were assigned according to Franckowiak et al. (1997).

Table 2. Pedigree details for the *Rph1-15* introgression lines selected for the barley leaf rust differential set.

BW Line¹	Donor Parent	Generation	<i>Rph</i> Allele²	Pedigree³
BW682	Sudan	BC ₅ F ₃	<i>Rph1.a</i>	Bowman*6/Sudan
BW743	Reka I	BC ₇ F ₃	<i>Rph2.t</i>	Bowman*8/Reka I
BW746	Estate	BC ₁₀ F ₃	<i>Rph3.c</i>	Bowman*11//Estate/3.2 uz als MM
BW752	Gold	BC ₅ F ₃	<i>Rph4.d</i>	Gold/6*Bowman
BW755	Magnif 102	BC ₇ F ₃	<i>Rph5.e</i>	Magnif 102/8*Bowman
BW756	Bolivia	BC ₇ F ₃	<i>Rph5.f</i>	Bowman*8/Bolivia
BW758	Cebada Capa	BC ₇ F ₃	<i>Rph7.g</i>	Bowman*8/4/ND7771//Cebada Capa/MT81195
BW759	Egypt 4	BC ₅ F ₃	<i>Rph8.h</i>	Bowman*6/Egypt 4
BW760	Hor2596	BC ₇ F ₃	<i>Rph9.i</i>	Bowman*8/Hor 2596
BW683	Clipper BC8	BC ₅ F ₃	<i>Rph10.o</i>	Clipper BC8/6*Bowman
BW684	Clipper BC67	BC ₅ F ₃	<i>Rph11.p</i>	Bowman*6/Clipper BC67
BW761	Triumph	BC ₈ F ₃	<i>Rph9.z</i>	Bowman*9/Triumph
BW685	PI 531841	BC ₆ F ₃	<i>Rph13.x</i>	Bowman*7/PI 531849
BW686	PI 584760	BC ₄ F ₃	<i>Rph14.ab</i>	Bowman*5/PI 584760
BW719	PI 355447	BC ₇ F ₃	<i>Rph15.ad</i>	PI 355447/8*Bowman

^{1.} Bowman leaf rust resistance introgression lines are abbreviated as "BW" followed by a number.

^{2.} *Rph* allele symbols were assigned according to Franckowiak et al. (1997).

^{3.} The pedigree used to develop each Bowman introgression line.

Table 3. Race nomenclature¹ of *Puccinia hordei* isolates² used to select for leaf rust resistance genes *Rph1-15* in Bowman introgression lines.

Isolate	Octal Notation	Race Code	Avr/Vir	Rph Vir
BR (Octal 11)	00011	MBBC	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23/24	1
ARG231	00041	LGBC	1, 2, 3, 4, 5, 6, 7, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 23/8, 22, 24	3
Aus220	00060	BQBC	1, 2, 3, 4, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 20, 21, 22, 23/5, 6, 19, 24	4
Race 4	21200	BCGH	1, 2, 3, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22/4, 10, 23, 24	4
Ger 7	00012	HBBC	1, 2, 3, 4, 5, 6, 7, 9, 11, 12, 13, 15, 16, 17, 18, 19, 20, 21, 22, 23/8, 10, 14, 24	4
WGCC	00060	BQBC	1, 2, 3, 4, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 20, 21, 23/5, 6, 19, 22, 24	5
98-20	11113	RDGM	3, 5, 6, 9, 10, 11, 12, 14, 15, 17, 18, 19, 20, 21, 22/1, 2, 4, 7, 8, 13, 16, 23, 24	9
94-8	14413	RBMM	2, 3, 5, 6, 7, 10, 11, 13, 14, 15, 17, 18, 20, 21/1, 4, 8, 9, 12, 16, 19, 22, 23, 24	10
BRS 76-12	00275	PRBC	2, 5, 6, 7, 9, 11, 12, 13, 14, 15, 17, 18, 20, 21/1, 3, 4, 8, 10, 16, 19, 22, 23, 24	10
90-3	67673	RRTK	3, 4, 9, 13, 14, 15, 17, 18, 20/1, 2, 5, 6, 7, 8, 10, 11, 12, 16, 19, 21, 22, 23, 24	15
Neth 28	24767	STMH	3, 5, 7, 10, 11, 1, 17, 18/1, 2, 4, 6, 8, 9, 12, 14, 15, 16, 19, 20, 21, 22, 23, 24	16
92-7	24777	TTMH	11, 13, 14, 15, 17, 18, 20/1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12, 16, 19, 21, 22, 23, 24	17

¹. Octal notation, North American rust code, and *avirulence/virulence* formulae race nomenclature systems were as described by Gilmour (1973; Limpert and Müller, 1994), Roelfs and Martens (1988), and Clifford (1985), respectively.

². Isolate names were given according to the original epithet provided by the collector.

³. *Rph Vir* indicates the number of *Rph* genes for which an isolate has virulence.

Table 4. Median infection types¹ and general reactions² of barley differential lines carrying leaf rust resistance genes *Rph1*-24 to 12 isolates of *Puccinia hordei*.

Isolate ⁴	Genes	Differential ³																							
		<i>Rph1</i>	<i>Rph2</i>	<i>Rph3</i>	<i>Rph4</i>	<i>Rph5</i>	<i>Rph6</i>	<i>Rph7</i>	<i>Rph8</i>	<i>Rph9</i>	<i>Rph10</i>	<i>Rph11</i>	<i>Rph12</i>	<i>Rph13</i>	<i>Rph14</i>	<i>Rph15</i>	<i>Rph16</i>	<i>Rph17</i>	<i>Rph18</i>	<i>Rph19</i>	<i>Rph20</i>	<i>Rph21</i>	<i>Rph22</i>	<i>Rph23</i>	<i>Rph24</i>
		<i>Sudan</i>	<i>Peruvian</i>	<i>Estate</i>	<i>Gold</i>	<i>Magnif 102</i>	<i>Bolivia New</i>	<i>Cebada Capa</i>	<i>Egypt 4</i>	<i>Hor 2596</i>	<i>Clipper BC 8</i>	<i>Clipper BC 67</i>	<i>Triumph</i>	<i>PI 531849</i>	<i>PI 584760</i>	<i>I95-282-2</i>	<i>HOR15560</i>	<i>81882/BS1</i>	<i>38P18/8/1/10</i>	<i>Prior</i>	<i>Flagship</i>	<i>Ricardo</i>	<i>182Q20/1/M1</i>	<i>Yerong</i>	<i>ND24260</i>
BR (Octal 11)		21	0;1c	0;1c	23-c	0;1N	0;1cn	0;cN	0;2C	10;cn	120;	120;	0;1-cn	10;cN	0;1n	0;1-cn	2	0;12c	00;	0;1-	0;1c	0;1	12	21	33-
ARG231		213c	0;1	00;1-	21	21c	12C	0;n	3-2	0;N	21c	21c	1-0;cN	0;1c	12	0;n	210;	0;1-	00;n	10;	0;1c	0;1-	23-1	0;12c	3-2
WGCC		1-0;n	10;	0;1-	0;12	3-2	23	00;1-	23c	0;1N	210;c	23-c	0;1c	0;1N	21c	0;N	0;1	0;1c	00;	33-	10;c	0;12c	0;3-	21	33-
Ger 7		0;1	213-c	00;1	3-2	10;	120;c	0;1-c	0;1-cn	10;cN	23-	21c	0;1-	0;1-	0;1-n	0;1-cn	0;1-	0;1-c	00;	12c	0;1c	213	21	3-2	3
Race 4		0;1c	0;12c	00;1-	10;c	213c	213-	0;n	3	0;1c	23-	21c	10;c	0;1	23-1	0;1-cn	0;1-	1-0;c	00;	120;	10;c	0;1c	2	0;12	33+
Aus 220		0;1	10;c	0;1-c	0;1c	3	3-2	00;	23-c	0;1-c	2C	2c	0;1-cn	0;1-N	10;c	0;cn	0;1-	1-0;	00;	3-2	0;1c	10;	23-1	21c	3-2
98-20		33-	23-1	00;1	3-2	1	120;	33-	33-c	120;c	12c	23-c	10;c	23-1	12c	1-0;cn	32	11-c	00;	23-c	0;12c	213-c	213-	33-	3
94-8		3-	21c	00;1	23	0;1	10;c	0;	3-2	3-2	12c	23-c	3-	23-c	12c	0;1-cn	23-	0;1	00;	3-2c	0;12c	21	33-	33-	3-2
BRS 76-12		3-2	213-c	3-2	3-2	23-c	213-c	0;n	3	10;c	23-1	21c	0;1-c	0;1	210;c	0;N	3-2	10;c	00;	23-1	10;c	210;c	3-20;	33-	33-
Neth 28		3-	3-3	213c	0;12	33-	3-2	33-	3	1c	3	3-2c	33-	0;1c	21c	12cn	3-2	23-1c	00;	33-	23-c	33-	3-0;	33-	3
90-3		3	32	0;12	3-	23-1c	33-0;	12c	33+	33-	23-c	23-c	3	0;1c	3-2c	33-	3-	23-c	00;	3-2c	3-2c	3-0;	3-21	3	33+
92-7		33-	3-	3-2c	3-	33-	33-	33-	3	3-2	33-	23-c	3-	10;c	21	0;1-cn	3-	23-c	00;	33-	23-c	3-	3	3	33+

¹ The infection types displayed in the matrix represent the median score recorded across three experiments. Infection types were scored using the original 0-4 rating scale developed by Mains (1930) with the additional notations of "+" and "-" to indicate larger or smaller size variations of classically described uredinia (Levine and Cherevick, 1952) and "c" and "n" to indicate the presence of chlorotic or necrotic reactions (Moseman and Roane, 1959), respectively. Complex ITs were recorded when more than one IT was observed on a single leaf. For example, an IT of 213- indicates that all three pustule types exist on the same leaf and that the "2" type pustules were the most common, followed by the "1" type, with "3-" type pustules being the least common.

² General reactions are different classes of infection types. Infection types from "0;" to "0;12" are classified as highly resistant ("R", green cells), infections types from "12" to "23-c" are classified as moderately resistant ("M", yellow cells), and infection types from "23-" to "33+" are classified as susceptible ("S", red cells) (Steffenson et al., 1993).

³ The differential lines representing *Rph1*-24 were selected from the most commonly used sources of these genes (Roane, 1962; Steffenson and Jin, 1992; Park et al., 2015; Ziems et al., 2017). Seed stocks of *Rph1*-15 were provided by Brian J. Steffenson; Robert Park provided stocks of *Rph16*-21, and *Rph23*; *Rph22* was obtained from the Norgen genebank; and *Rph24* was provided by Jerome D. Franckowiak.

⁴ Isolates used in this study were selected from a global collection of over 350 isolates of *P. hordei* (Fetch et al., 1998). Four of the isolates in this panel were also used to develop the Bowman leaf rust introgression lines (Chicaiza, 1996; Caffarel, 2005). The remaining isolates were chosen to balance redundancies for virulence and avirulence across *Rph1*-15.

Table 5. Median infection types¹ and general reactions² of the resistant donor parents³ for *Rph1-15* and their derived Bowman introgression lines⁴ selected for the barley leaf rust differential set.

		<i>P. hordei</i> Isolates																							
		BR (Octal 11)		ARG231		Aus 220		Race 4		Ger 7		WGCC		98-20		94-8		BRS 76-12		90-3		Neth 28		92-7	
Host line	Allele	IT	GR	IT	GR	IT	GR	IT	GR	IT	GR	IT	GR	IT	GR	IT	GR	IT	GR	IT	GR	IT	GR	IT	GR
Sudan	<i>Rph1.a</i>	3-2c	S	3-0;	S	0;1	R	0;1-	R	0;1-	R	0;12	R	33-	S	33-	S	33-	S	3	S	33-	S	3-	S
BW682	<i>Rph1.a</i>	3-2c	S	3-0;c	S	0;1-	R	0;1-	R	0;1-	R	0;12	R	3	S	33-	S	33+	S	33+	S	3	S	3	S
Reka 1	<i>Rph2.t+Rph19.ah</i>	0;n	R	120;	M	0;1-	R	0;1	R	0;1-	R	0;12	R	3-2	S	23-1	S	3-2	S	33-	S	33-	S	3	S
BW743	<i>Rph2.t</i>	120;c	M	120;	M	23-c	M	213-c	M	3-2	S	120;	M	3	S	3-2	S	213-c	M	33-	S	3-	S	3-	S
Estate	<i>Rph3.c</i>	0;1-c	R	00;1	R	0;1-c	R	00;1-	R	00;2	R	00;2	R	00;1c	R	00;1	R	3-	S	0;12c	R	213-c	M	3-2c	S
BW746	<i>Rph3.c</i>	0;1c	R	0;1-c	R	0;1-c	R	0;1c	R	00;1	R	0;1c	R	0;cn	R	0;	R	3-2c	S	21c	M	23-	S	3-	S
Gold	<i>Rph4.d</i>	23-c	M	23-c	M	0;1	R	10;	R	3-2	S	0;12	R	3-2	S	3-2c	S	3-2	S	33-	S	0;12	R	33-	S
BW752	<i>Rph4.d</i>	3-2	S	21	M	120;	M	120;c	M	3	S	120;c	M	3	S	3-2	S	3-2	S	33-	S	120;c	M	3	S
Magnif 102	<i>Rph5.e</i>	0;1-n	R	11-cn	R	3-2c	S	213-	M	120;c	M	3-2	S	120;	M	0;1	R	23-c	M	3-2c	S	33-	S	33-	S
BW755	<i>Rph5.e</i>	0;1	R	23-c	M	3	S	213-c	M	120;c	M	3	S	10;c	R	10;c	R	3-2c	S	33-	S	3-	S	33-	S
Bolivia	<i>Rph2.r+Rph5.f</i>	0;1-	R	0;1-n	R	0;1	R	0;	R	0;1	R	0;1	R	23-0;	S	23-0;	S	210;	M	3	S	33-	S	3	S
BW756	<i>Rph5.f</i>	0;1	R	3-2	S	3	S	213-c	M	21c	M	3	S	120;c	M	10;c	R	3-2c	S	3-3	S	3-	S	3-	S
Cebada Capa	<i>Rph7.g</i>	00;n	R	0;1-cn	R	00;	R	0;1-c	R	0;1-c	R	00;	R	3-20;	S	0;1-cn	R	0;n	R	213-c	M	33-c	S	33-	S
BW758	<i>Rph7.g</i>	0;1-cn	R	0;1-cn	R	00;n	R	0;1-cn	R	0;1-c	R	0;n	R	3-2	S	0;1-cn	R	0;1-cn	R	21c	M	33-	S	33-	S
Egypt 4	<i>Rph8.h</i>	0;1n	R	3-2	S	23-c	M	3	S	0;1-c	R	23-c	M	3-c	S	3-2c	S	3-2	S	3	S	33-	S	3	S
BW759	<i>Rph8.h</i>	0;1-cn	R	23-c	M	21c	M	3	S	0;1-c	R	23-c	M	213-	M	23-c	M	3-2c	S	3	S	3-	S	3-	S
Hor 2596	<i>Rph9.i</i>	0;1N	R	0;n	R	0;1-cn	R	0;1-c	R	1-0;cn	R	0;1-c	R	10;c	R	3-2c	S	10;c	R	3	S	120;c	M	23-	S
BW760	<i>Rph9.i</i>	11-c	R	21c	M	213-c	M	213-	M	0;1c	R	213-c	M	23-c	M	3-2	S	12c	M	3	S	3-2	S	3	S
Clipper BC8	<i>Rph10.o</i>	120;	M	12c	M	10;c	R	23-	S	23-	S	12c	M	120;c	M	10;	R	23-1c	M	23-c	M	3	S	33-	S
BW683	<i>Rph10.o</i>	21c	M	12	M	12c	M	3-2c	S	23-c	M	21c	M	3-2c	S	23-c	M	23-c	M	33-	S	23-c	M	23-c	M
Clipper BC67	<i>Rph11.p</i>	21	M	21c	M	23-c	M	21c	M	21c	M	23-c	M	23-c	M	23-c	M	21c	M	23-c	M	3-2c	S	23-c	M
BW684	<i>Rph11.p</i>	2c	M	213-c	M	21c	M	213-c	M	23-c	M	21c	M	23-c	M	23-c	M	23-1c	M	33-	S	23-1c	M	23-c	M
Triumph	<i>Rph9.z</i>	0;1-	R	0;1	R	0;1-c	R	10;c	R	0;1-c	R	0;1-c	R	10;c	R	3-	S	0;1-c	R	3	S	3	S	3	S
BW761	<i>Rph9.z</i>	0;1	R	0;12c	R	120;	M	0;12c	R	0;1c	R	10;c	R	10;c	R	3-3	S	10;c	R	3	S	3-2	S	33-	S
PI 531849	<i>Rph13.x</i>	0;1-	R	0;1c	R	00;1-	R	0;1	R	0;1-	R	0;1-	R	23-1	S	23-c	M	0;1	R	0;1c	R	0;1c	R	0;1	R
BW685	<i>Rph13.x</i>	0;1	R	210;	M	0;1-c	R	21	M	10;c	R	0;1	R	3-2	S	33-	S	10;c	R	12c	M	10;c	R	10;	R
PI 531901-1	<i>Rph14.ab</i>	0;1n	R	23-	S	21c	M	213-	M	0;cn	R	10;c	R	210;c	M	210;c	M	21c	M	3-	S	23-c	M	3-2	S
BW686	<i>Rph14.ab</i>	00;1c	R	21	M	12c	M	33-	S	0;1-cn	R	21c	M	210;c	M	21c	M	213-c	M	33-	S	3-2	S	3-	S
PI 355447	<i>Rph15.ad</i>	0;1-cn	R	0;1-	R	0;cn	R	0;1-cn	R	0;cn	R	0;cn	R	0;1-cn	R	0;1-cn	R	0;cn	R	3-	S	23-cn	M	0;1cn	R
BW719	<i>Rph15.ad</i>	0;1-cn	R	0;cn	R	0;cn	R	1-0;cn	R	0;1-cn	R	0;cn	R	10;cn	R	1-0;cn	R	0;cn	R	3-3	S	21cn	M	0;1-cn	R

Table 5 Footnotes.

- ¹ The infection types displayed in the matrix represent the median score recorded across three experiments. Infection types were scored using the original 0-4 rating scale developed by Mains (1930) with the additional notations of “+” and “-” to indicate larger or smaller size variations of classically described uredinia (Levine and Cherewick, 1952) and “c” and “n” to indicate the presence of chlorotic or necrotic reactions (Moseman and Roane, 1959), respectively. Complex ITs were recorded when more than one IT was observed on a single leaf. For example, an IT of 213- indicates that all three pustule types exist on the same leaf and that the “2” type pustules were most common, followed by the “1” type, with “3-” type pustules being the least common.
- ² General reactions are different classes of infection types. Infection types from "0;" to "0;12" are classified as highly resistant ("R", green cells), infections types from "12" to "23-c" are classified as moderately resistant ("M", yellow cells), and infection types from "23-" to "33+" are classified as susceptible ("S", red cells) (Steffenson et al., 1993). GRs in italics denote cases where different reactions occurred between the donor parent and its introgression line.
- ³ The host lines are listed sequentially from *Rph1-15* with the original donor parent above each respective introgression line.
- ⁴ The Bowman introgression lines are abbreviated using the letters BW and a three digit number identifier.

Table 6. Infection type mode¹ and range² for the selected Bowman introgression lines carrying the leaf rust resistance genes *Rph1-15* against 12 isolates of *Puccinia hordei*.

		Isolates of <i>P. hordei</i>																							
		BR (Octal 11)		ARG231		Aus 220		Race 4		Ger 7		WGCC		98-20		94-8		BRS 76-12		90-3		Neth 28		92-7	
Line	Allele ³	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R
BW682	<i>Rph1.a</i>	3-	2 to 3-	3-	0; to 3-	0;	0; to 2	0;	0; to 1	0;	0; to 1-	0;	0; to 2	3	3- to 3+	3	3- to 3+	3	1 to 3+	3	3 to 3+	3	3- to 3	3	2 to 3+
BW743	<i>Rph2.t</i>	1	0; to 2	1	0; to 3-	2	1 to 3-	2	0; to 3-	3-	2 to 3-	2	0; to 3-	3	2 to 3+	3-	0; to 3	3-	1 to 3-	3-	3- to 3	3-	2 to 2-	3-	2 to 3-
BW746	<i>Rph3.c</i>	0;	0; to 1	0;	0; to 1-	0;	0; to 1	0;	0; to 2	0	0 to 2	0;	0; to 1	0;	0; to 1	0;	0; to 1-	3-	2 to 3	2	0; to 3-	3-	2 to 3-	3-	2 to 3-
BW752	<i>Rph4.d</i>	3-	1 to 3-	2	0; to 3-	1	0; to 2	1	0; to 2	3	2 to 3	1	0; to 3-	3	1 to 3	3	1 to 3+	3-	2 to 3	3	2 to 3	1	0; to 2	3	3
BW755	<i>Rph5.e</i>	1	0; to 1	2	1 to 3-	3	2 to 3	2	0; to 3-	1	0; to 2	3	2 to 3	1	0; to 2	1	0; to 1	3-	0; to 3	3-	3- to 3	3-	2 to 3-	3-	3- to 3
BW756	<i>Rph5.f</i>	1	0; to 1	3-	2 to 3-	3	3- to 3	2	1 to 3-	2	1 to 3-	3	2 to 3	1	0; to 2	1	0; to 1	3-	0; to 3	3-	2 to 3	3-	2 to 3	3-	2 to 3
BW758	<i>Rph7.g</i>	0;	0; to 1-	0;	0; to 1-	0	0 to 0;	0;	0; to 1-	0;	0; to 1	0;	0; to 1-	3-	2 to 3+	0;	0; to 1	0;	0; to 1	2	1 to 3-	3-	2 to 3	3-	3- to 3
BW759	<i>Rph8.h</i>	0;	0; to 1-	2	1 to 3-	2	1 to 3-	3	3 to 3+	0;	0; to 1	2	1 to 3-	2	1 to 3-	2	1 to 3-	3-	2 to 3-	3	2 to 3	3-	2 to 3	3-	3- to 3
BW760	<i>Rph9.i</i>	1	0; to 2	2	0; to 2	2	1 to 3-	1	0; to 3-	1	0; to 2	2	0; to 3-	2	1 to 3-	3-	1 to 3-	1	0; to 2	3	3- to 3	3-	2 to 3	3	3- to 3
BW683	<i>Rph10.o</i>	2	1 to 3-	1	0; to 2	1	0; to 2	2	0; to 3-	2	1 to 3-	2	0; to 3-	3-	1 to 3-	2	0; to 3-	2	1 to 3-	3	3- to 3	2	1 to 3-	2	1 to 3-
BW684	<i>Rph11.p</i>	2	1 to 3-	2	1 to 3-	2	1 to 3-	2	1 to 3-	2	1 to 3-	2	0; to 3-	2	2 to 3-	2	1 to 3-	2	0; to 3-	3	3- to 3+	2	1 to 3-	2	1 to 3-
BW761	<i>Rph9.z</i>	1	0; to 1	1	0; to 2	1	0; to 2	1	0; to 2	0;	0; to 1	1	0; to 1	1	0; to 2	3	1 to 3+	1	0; to 2	3	3- to 3	3-	2 to 3	3-	2 to 3
BW685	<i>Rph13.x</i>	0;	0; to 2	1	0; to 2	0;	0; to 1	2	1 to 3-	0;	0; to 2	0;	0; to 1	3-	2 to 3-	3	3- to 3+	1	0; to 2	1	0; to 3-	1	0; to 2	1	0; to 2
BW686	<i>Rph14.ab</i>	0;	0 to 1	2	0; to 3-	1	0; to 2	3	2 to 3	0;	0; to 1-	2	1 to 3-	2	0; to 2	2	0; to 2	2	1 to 3-	3-	2 to 3	3-	2 to 3-	3-	2 to 3
BW719	<i>Rph15.ad</i>	1-	0; to 1-	0;	0; to 1-	0;	0;	1-	0; to 1-	0;	0; to 1-	0;	0; to 1-	1	0; to 1	1-	0; to 1	0;	0;	3-	3- to 3	2	1 to 3-	0;	0; to 1

¹ M = Infection type mode: the most common single infection type (IT) observed across three experiments.

² R = Range of observed ITs: the lowest and highest ITs observed across three experiments.

³ *Rph* allele symbols were designated according to Franckowiak et al. (1997).

Table 7. Annotated genes involved in disease resistance and residing in the same contigs as BOPA SNP markers within Bowman introgression lines carrying nine¹ of the 15 leaf rust resistance alleles used in this study.

BW Line²	<i>Rph</i> introgression³	BOPA_SNP⁴	Genes on contig⁵	Annotation⁶
BW682	<i>Rph1.a</i>	2_1377	AK361947	Zinc finger (Ran-binding) family protein
BW743	<i>Rph2.t</i>	2_1253	MLOC_54218.3	Protein kinase superfamily protein LENGTH=390
BW743	<i>Rph2.t</i>	1_1432	MLOC_73036.2	Leucine Rich Repeat family protein
BW743	<i>Rph2.t</i>	2_0697	MLOC_64255.1	Transmembrane protein
BW743	<i>Rph2.t</i>	1_0240	MLOC_54831.1	Thioredoxin m
BW743	<i>Rph2.t</i>	2_0332	AK248962.1	Ubiquitin activating enzyme 2
BW743	<i>Rph2.t</i>	2_0461	AK251412.1	Peroxidase superfamily protein
BW743	<i>Rph2.t</i>	2_0372	MLOC_6055.1	Ubiquitin-conjugating enzyme variant
BW743	<i>Rph2.t</i>	2_1200	MLOC_52504.2	WRKY transcription factor
BW743	<i>Rph2.t</i>	2_0265	AK365065	Trehalose-6-phosphate phosphatase
BW743	<i>Rph2.t</i>	1_1281	MLOC_11773.1	Trehalose-6-phosphate synthase
BW743	<i>Rph2.t</i>	2_1121	MLOC_34809.1	Cathepsin L-like cysteine proteinase
BW746	<i>Rph3.c</i>	1_0797	MLOC_50091.1	Senescence-associated protein DH
BW746	<i>Rph3.c</i>	2_1160	MLOC_60881.1	WD-repeat protein 57
BW758	<i>Rph7.g</i>	1_0112	AK249901.1	Bowman-Birk type trypsin inhibitor
BW758	<i>Rph7.g</i>	2_0595	MLOC_36532.1	Ubiquitin-conjugating enzyme 22
BW760	<i>Rph9.i</i>	2_0653	MLOC_53949.2	Leucine-rich repeat receptor-like protein kinase
BW760	<i>Rph9.i</i>	2_1247	MLOC_4611.1	Protein kinase
BW760	<i>Rph9.i</i>	1_1090	AK373628	Transcription factor
BW760	<i>Rph9.i</i>	1_1080	AK364080	Cysteine proteinase
BW760	<i>Rph9.i</i>	1_0292	AK371585	Succinate dehydrogenase subunit A
BW760	<i>Rph9.i</i>	2_0731	MLOC_6875.2	Reticulon-like protein B8
BW683	<i>Rph10.o</i>	1_0728	AK358661	Chitinase
BW683	<i>Rph10.o</i>	1_0335	MLOC_11772.1	RING finger and CHY zinc finger domain-containing protein 1
BW683	<i>Rph10.o</i>	2_1305	MLOC_50950.1	NBS-LRR-like protein
BW683	<i>Rph10.o</i>	2_0931	MLOC_63920.1	WD-40 repeat family protein

Table 7. Annotated genes involved in disease resistance and residing in the same contigs as BOPA SNP markers within Bowman introgression lines carrying nine¹ of the 15 leaf rust resistance alleles used in this study.

BW Line²	<i>Rph</i> introgression³	BOPA_SNP⁴	Genes on contig⁵	Annotation⁶
BW761	<i>Rph9.z</i>	2_0653	MLOC_53949.2	Leucine-rich repeat receptor-like protein kinase
BW761	<i>Rph9.z</i>	2_1247	MLOC_4611.1	Protein kinase
BW761	<i>Rph9.z</i>	1_1090	AK373628	Transcription factor
BW761	<i>Rph9.z</i>	2_1241	MLOC_57253.1	Receptor-like protein kinase 2
BW761	<i>Rph9.z</i>	1_1080	AK364080	Cysteine proteinase
BW761	<i>Rph9.z</i>	1_0292	AK371585	Succinate dehydrogenase subunit A
BW761	<i>Rph9.z</i>	2_0731	MLOC_6875.2	Reticulon-like protein B8
BW761	<i>Rph9.z</i>	2_0876	AK367513	Remorin
BW761	<i>Rph9.z</i>	2_0686	AK364134	Kinase family protein
BW761	<i>Rph9.z</i>	2_0536	MLOC_13213.1	Pectin lyase-like superfamily protein
BW685	<i>Rph13.x</i>	2_0612	MLOC_37645.1	Zinc finger CCCH domain-containing protein
BW685	<i>Rph13.x</i>	1_0842	MLOC_17018.1	Peptidyl-prolyl cis-trans isomerase
BW685	<i>Rph13.x</i>	2_1427	AK369539	NBS-LRR disease resistance protein homologue
BW719	<i>Rph15.ad</i>	2_1153	MLOC_44144.1	CW-type Zinc Finger family protein

¹ No genes for defense were found in the *Rph*-allele containing introgressions for *Rph4.d*, *Rph5.e*, *Rph5.f*, *Rph8.h*, *Rph11.p*, or *Rph14.ab*.

² BW lines are the Bowman leaf rust resistance introgression lines whose introgressions carry annotated genes for defense.

³ *Rph* gene residing in an introgression of the corresponding BW line.

⁴ BOPA SNP markers residing on the same genetic contig as annotated defense genes.

⁵ Annotated genes for defense residing on the same contig as Barley Oligonucleotide Polymorphic Assay (BOPA) SNP markers.

⁶ The listed gene annotation from the barley genome explorer provided by the Leibniz-Institut für Pflanzengenetik und Kulturpflanzenforschung (IPK, Gatersleben, Germany).

Figure 1. Examples of leaf rust infection types (ITs) observed on Bowman introgression lines. Infection types were scored using the original 0-4 rating scale developed by Mains (1930) with the additional notations of "+" and "-" to indicate larger or smaller size variations of classically described uredinia (Levine and Cherewick, 1952) and "c" and "n" to indicate the presence of chlorotic or necrotic reactions (Moseman and Roane, 1959), respectively. Complex ITs were recorded when more than one IT was observed on a single leaf. For example, an IT of 213- indicates that all three pustule types exist on the same leaf and that the "2" type pustules were the most common, followed by the "1" type, with "3-" type pustules being the least common. General reactions are different classes of infection types. Infection types from "0;" to "0;12" are classified as highly resistant ("R", green cells), infections types from "12" to "23-c" are classified as moderately resistant ("M", yellow cells), and infection types from "23-" to "33+" are classified as susceptible ("S", red cells) (Steffenson et al., 1993).

Figure 2. Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes *Rph1-15* selected for the barley leaf rust differential set. Physical distances along the genome are represented in centimorgans (cM). Bold sections within a chromosome are the regions of retained donor chromatin.

Figure 2 continued. Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes *Rph1-15* selected for the barley leaf rust differential set.

Figure 2 continued. Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes *Rph1-15* selected for the barley leaf rust differential set.

Figure 2 continued. Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes *Rph1-15* selected for the barley leaf rust differential set.

Figure 2 continued. Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes *Rph1-15* selected for the barley leaf rust differential set.

Figure 2 continued. Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes *Rph1-15* selected for the barley leaf rust differential set.

Figure 2 continued. Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes *Rph1-15* selected for the barley leaf rust differential set.

Figure 2 continued. Graphical genotypes for Bowman introgression lines carrying leaf rust resistance genes *Rph1-15* selected for the barley leaf rust differential set.

Literature Cited

- Afanasenkov, O.S., M. Jalli, H.O. Pinnschmidt, O. Filatova, and G.J. Platz. 2009. Development of an international standard set of barley differential genotypes for *Pyrenophora teres* f. sp. *teres*. Plant Pathol. 58:665–676.
- Andres, M.W., R.D. Wilcoxson, A.P. Roelfs, and Z.A. Pretorius. 1983. Races of *Puccinia hordei* in the United States from 1979-1982. Plant Dis. 67:1336–1337.
- Anikster, Y. 1989. Host specificity versus plurivory in barley leaf rusts and their microcyclic relatives. Mycol. Res. 93:175–181.
- Anikster, Y., and I. Wahl. 1979. Coevolution of the rust fungi on *Gramineae* and *Liliaceae* and their hosts. Annu. Rev. Phytopathol. 17:367–403.
- Arnst, B.J., J.W. Martens, G.M. Wright, P.A. Burnett, and F.R. Sanderson. 1979. Incidence, importance and virulence of *Puccinia hordei* on barley in New Zealand. Annu. Appl. Biol. 92:185–190.
- Babraham Bioinformatics. 2012. Trim Galore script. Cambridge, England. "http://www.bioinformatics.babraham.ac.uk/projects/trim_galore/".
- Berloo, R. v, H. Aalbers, A. Werkman, and R.E. Niks. 2001. Resistance QTL confirmed through development of QTL-NILs for barley leaf rust resistance. Mol. Breed. 8:187–195.
- Bernardo, L., B. Prinsi, A.S. Negri, L. Cattivelli, L. Espen, and G. Valè. 2012. Proteomic characterization of the *Rph15* barley resistance gene-mediated defence responses to leaf rust. BMC Genomics 13(1).
- Borovkova, I.G., B.J. Steffenson, Y. Jin, A. Kilian, A. Kleinhofs, and T.K. Blake. 1997. Identification and mapping of a leaf rust resistance gene in barley line Q21861. Genome 40:236–241.
- Borovkova, I.G., Y. Jin, and B.J. Steffenson. 1998. Chromosomal location and genetic-relationship of leaf rust resistance genes *Rph9* and *Rph12* in barley. Phytopathol. 88:76–80.
- Brodny, U., and M. Rivadeneira. 1996. Physiologic specialization of *Puccinia hordei* in Israel and Ecuador. Can. J. Plant Pathol. 18:375–378.
- Brown, A.M. 1931. Physiologic specialization in the dwarf leaf rust of barley. Rep. Dom. Bot. 1929:58–60.
- Browning, B.L., and S.R. Browning. 2016. Genotype imputation with millions of reference samples. Am. J. Hum. Genet. 98:116–126.
- Brunner, S., B. Keller, and C. Feuillet. 2000. Molecular mapping of the *Rph7.g* leaf rust resistance gene in barley (*Hordeum vulgare* L.). Theor. Appl. Genet. 101:783–788.
- Buchannon, K.W., and H.A.H. Wallace. 1962. Note on the effect of leaf diseases on yield bushel weight and thousand-kernal weight of Parkland barley. Can. J. Plant Sci. 42:534–536.
- Caffarel, J.C. 2005. Leaf rust studies in barley using Bowman backcross-derived lines. M.S. thesis. North Dakota State University, Fargo.

- Cao, J. 2012. The pectin lyases in *Arabidopsis thaliana*: Evolution, selection and expression profiles. PLoS One 7(10).
- Chen, X., C.A. Hackett, R.E. Niks, P.E. Hedley, C. Booth, A. Druka, T.C. Marcel, A. Vels, M. Bayer, I. Milne, J. Morris, L. Ramsay, D. Marshall, L. Cardle, and R. Waugh. 2010. An eQTL analysis of partial resistance to *Puccinia hordei* in barley. PLoS One 5(1): e8598.
- Chicaiza, O. 1996. Genetic control of leaf rust. Ph.D. thesis. North Dakota State University, Fargo.
- Christopher, M., E. Mace, D. Jordan, D. Rodgers, P. McGowan, I. Delacy, P. Banks, J. Sheppard, D. Butler, and D. Poulsen. 2007. Applications of pedigree-based genome mapping in wheat and barley breeding programs. Euphytica 154:307–316.
- Clifford, B.C. 1977. Monitoring virulence in *Puccinia hordei*: a proposal for the choice of host genotypes and survey procedures. Cereal Rusts and Powdery Mildews Bull. 5:34–38.
- Clifford, B.C. 1985. Chapter 6: Barley leaf rust. In: Roelfs, A.P. and W.R. Bushnell, The Cereal Rusts. Academic Press, Inc. Orlando, FL. p. 180-210.
- Clifford, B.C., and A.C.C. Udeogalanya. 1976. Hypersensitive resistance of barley to brown rust (*Puccinia hordei* Otth.). 4th Eur. Mediterr. Cereal Rusts Conf. Interlaken:27–29.
- Cotterill, P.J., R.F. Park, and R.G. Rees. 1995. Pathogenic Specialization of *Puccinia hordei* Otth. in Australia, 1966-1990. Aust. J. Agric. Res. 46:127–134.
- Cotterill, P.J., R.G. Rees, G.J. Platz, and R. Dill-Macky. 1992a. Effects of leaf rust on selected Australian barleys. Aust. J. Exp. Agric. 32:747–751.
- Cotterill, P.J., R.G. Rees, and W.A. Vertigan. 1992b. Detection of *Puccinia hordei* virulent on the *Pa9* and Triumph resistance genes in barley in Australia. Australas. Plant Pathol. 21:32–34.
- Czembor, J.H., H.J. Czembor, G. Attene, R. Papa, V. Agraria, V.D. Nicola, and M. Dago. 2007. Leaf rust resistance in selections from barley landraces collected in Sardinia. Plant Breed. Seed Sci. 56:73–84.
- D'Oliveira, B. 1939. Studies on *Puccinia anomala* Rost. I. physiologic races on cultivated barleys. Ann. Appl. Biol. 26:56–82.
- D'Oliveira, B. 1949. Aecidia of barley dwarf rust. Nature 164:880–881.
- D'Oliveira, B. 1961. Ideas concerning the evolution and distribution of cereal rusts. Recent Adv. Bot. 1:564–567.
- Das, M.K., C.A. Griffey, R.E. Baldwin, C.M. Waldenmaier, M.E. Vaughn, A.M. Price, and W.S. Brooks. 2007. Host resistance and fungicide control of leaf rust (*Puccinia hordei*) in barley (*Hordeum vulgare*) and effects on grain yield and yield components. Crop Prot. 26:1422–1430.

- Dean, R., J.A.L. Van Kan, Z.A. Pretorius, K.E. Hammond-Kosack, A.D. Pietro, P.D. Spanu, J.J. Rudd, M. Dickman, R. Kahmann, J. Ellis, and G.D. Foster. 2012. The Top 10 fungal pathogens in molecular plant pathology. *Mol. Plant Pathol.* 13:414–430.
- Derevnina, L., D. Singh, and R.F. Park. 2013. Identification and characterization of seedling and adult plant resistance to *Puccinia hordei* in Chinese barley germplasm. *Plant Breed.* 132:571–579.
- Dillard, M.W., and A.R. Brown. 1969. Inheritance of reaction to race 8 of *Puccinia hordei* Otth. in two barley crosses. *Crop Sci.* 9:677–678.
- Dmochowska-Boguta, M., S. Alaba, Y. Yanushevska, U. Piechota, E. Lasota, A. Nadolska-Orczyk, W.M. Karlowski, and W. Orczyk. 2015. Pathogen-regulated genes in wheat isogenic lines differing in resistance to brown rust *Puccinia triticina*. *BMC Genomics* 16:1–17.
- Dodds, P. and J. Rathjen. 2010. Plant immunity: Towards an integrated view of plant-pathogen interactions. *Nat. Rev. Genet.* 1:539–548.
- Dracatos, P.M., M.S. Khatkar, D. Singh, and R.F. Park. 2014. Genetic mapping of a new race specific resistance allele effective to *Puccinia hordei* at the *Rph9/Rph12* locus on chromosome 5HL in barley. *BMC Plant Biol.* 14:382.
- Dreiseitl, A., and B.J. Steffenson. 2000. Postulation of leaf-rust resistance genes in Czech and Slovak barley cultivars and breeding lines. *Plant Breed.* 119:211–214.
- Druka, A., J.D. Franckowiak, U. Lundqvist, N. Bonar, J. Alexander, K. Houston, S. Radovic, F. Shahinnia, V. Vendramin, M. Morgante, N. Stein, and R. Waugh. 2011. Genetic dissection of barley morphology and development. *Plant Physiol.* 155:617–627.
- Dyck, P. 1977. Genetics of leaf rust reaction in three introductions of common wheat. *Can. J. Genet. Cytol.* 19:711–716.
- Ellis, J., E. Lagudah, W. Spielmeyer, and P. Dodds. 2014. The past, present and future of breeding rust resistant wheat. *Front. Plant Sci.* 5:641.
- Fetch, T., B.J. Steffenson, and Y. Jin. 1998. Worldwide virulence of *Puccinia hordei* on barley. *Phytopathology* 88:S28.
- Feuerstein, U., A.H.D. Brown, and J.J. Burdon. 1990. Linkage of rust resistance genes from wild barley (*Hordeum spontaneum*) with isozyme markers. *Plant Breed.* 104:318–324.
- Flor, H.H. 1942. Inheritance of pathogenicity in *Melampsora lini*. *Phytopathology* 32:653–669.
- Franckowiak, J.D., A.E. Foster, B.D. Pederson, and R.E. Pyler. 1985. Registration of 'Bowman' barley. *Crop Sci.* 25:883.
- Franckowiak, J.D., Y. Jin, and B.J. Steffenson. 1997. Recommended allele symbols for leaf rust resistance genes in barley. *Barley Genet. Newsl.* 27:36–44.
- Frecha, J.H. 1970. Inheritance of the resistance to *Puccinia hordei* Otth. in barley. *Bol. Genet.* 7:1–8.

- Gale, M.D., and K.M. Devos. 1998. Comparative genetics in the grasses. *Proc. Natl. Acad. Sci.* 95:1971–1974.
- Germán, S., and S. Pereyra. 2007. Winter Crops Day. INIA LA Estanzuela:8–24. (In Spanish)
- Gilmour, J. 1973. Octal notation for designating physiologic races of plant pathogens. *Nature* 242:620.
- Golan, T., Y. Anikster, J.G. Moseman, and I. Wahl. 1978. A new virulent strain of *Puccinia hordei*. *Euphytica* 27:185–189.
- Golegaonkar, P.G., H. Karaoglu, and R.F. Park. 2009. Molecular mapping of leaf rust resistance gene *Rph14* in *Hordeum vulgare*. *Theor. Appl. Genet.* 119:1281–1288.
- González, M.G., M.Z. Díaz, R. Huerta, and S. Solano. 2013. Efficiency of three fungicides to control the leaf rust in malting barley. *J. Mexican Agric. Sci.* 4:1237–1250. (In Spanish)
- Graner, A., S. Streng, and A. Drescher. 2000. Molecular mapping of the leaf rust resistance gene *Rph7* in barley. *Plant Breeding* 119:389–392.
- Griffey, C.A., M.K. Das, R.E. Baldwin, and C.M. Waldenmaier. 1994. Yield losses in winter barley resulting from a new race of *Puccinia hordei* in North America. *Plant Dis.* 78:256–260.
- Grunewaldt-Stöcker, G. 1983. Development of a differential set for the race analysis of *Puccinia hordei* Otth. *J. Phytopathology* 107: 309–317.
- Heath, M.C. 1991. The role of gene-for-gene interactions in the determination of host species specificity. *Phytopathology* 81: 127–130.
- Henderson, M.T. 1945. Studies of sources of resistance and inheritance of reaction to leaf rust, *Puccinia anomala* Rostr., in barley. Ph.D. Thesis. University of Minnesota, Saint Paul.
- Hey, A. 1931. Contribution to the specialization of the barley rust, *Puccinia simplex* Erikss. and Henn. Work from the Biological Imperial Institute for land and forestry 19(3): 227–261. (In German)
- Hickey, L.T., W. Lawson, G.J. Platz, M. Dieters, V.N. Arief, S. Germán, S. Fletcher, R.F. Park, D. Singh, S. Pereyra, and J.D. Franckowiak. 2011. Mapping *Rph20*: A gene conferring adult plant resistance to *Puccinia hordei* in barley. *Theor. Appl. Genet.* 123:55–68.
- Hirschhorn, J. 1933. Two rusts of barley, new for Argentina. *J. Fac. Agron.* 19:390–397. (In Spanish)
- Hubbard, A., S. Wilderspin, S. Holdgate, and H. Road. October 2017. United Kingdom Cereal Pathogen Virulence Survey 2017 Annual Report.
- Ivancic, V., U. Walther, and A. Graner. 1998. Molecular mapping of a new gene in wild barley conferring complete resistance to leaf rust (*Puccinia hordei* Otth). *Theor. Appl. Genet.* 97:1235–1239.

- Jafary, H., L.J. Szabo, and R.E. Niks. 2006. Innate nonhost immunity in barley to different heterologous rust fungi is controlled by sets of resistance genes with different and overlapping specificities. *Mol. Plant. Microbe. Interact.* 19:1270–9.
- Jin, Y., G.H. Cui, B.J. Steffenson, and J.D. Franckowiak. 1996. New leaf rust resistance genes in barley and their allelic and linkage relationships with other *Rph* genes. *Phytopathology* 86:887–890.
- Jin, Y., G.D. Statler, J.D. Franckowiak, and B.J. Steffenson. 1993. Linkage between leaf rust resistance genes and morphological markers in barley. *Phytopathology* 83:230–233.
- Jin, Y., and B.J. Steffenson. 1994. Inheritance of resistance to *Puccinia hordei* in cultivated and wild barley. *J. Hered.* 85:451–454.
- Jin, Y., and B.J. Steffenson. 1996. A multi-allelic series at the *Rph2* locus for leaf rust resistance in barley. *Cereal Rusts Powdery Mildews Bull.* 24:74–75.
- Johnson, R. 1968. The genetics of resistance of some barley varieties to *Puccinia hordei*. *Eur. Mediterr. Cereal Rust Conf. Oeiras, Portugal*:160–162.
- Johnson, D.A., and R.D. Wilcoxson. 1979. Yield losses of fast and slow rusting barleys infected with *Puccinia hordei*. *Plant Dis. Report.* 63:764–768.
- Johnston, P.A., R.E. Niks, V. Meiyalaghan, E. Blanchet, and R.A. Pickering. 2013. *Rph22*: mapping of a novel leaf rust resistance gene introgressed from the non-host *Hordeum bulbosum* L. into cultivated barley (*Hordeum vulgare* L.). *Theor. Appl. Genet.* 126:1613–25.
- Jones, E.R.L. 2005. Brown rust of barley. UK Cereal Pathogen Virulence Survey.
- Jones, E.R.L., and B.C. Clifford. 1980. Brown rust of barley. UK Cereal Pathogen Virulence Survey:61–65.
- Jones, J.D.G., and J.L. Dangl. 2006. The plant immune system. *Nature* 444:323–329.
- Kavanagh, P.J., D. Singh, U.K. Bansal, and R.F. Park. 2017. Inheritance and characterization of the new and rare gene *Rph25* conferring seedling resistance in *Hordeum vulgare* against *Puccinia hordei*. *Plant Breed.* 136:908–912.
- Khokhlova, A.P. 1982. Genes for resistance against leaf rust of barley. Works on applied botany genetics and selection. 71:63–67. (In Russian)
- King, J.E. 1972. Surveys of foliar diseases of spring barley in England and Wales 1967-70. *Plant Pathol.* 21:23–35.
- King, J.E., and R.W. Polley. 1976. Observations on the epidemiology and effect on grain yield of brown rust in spring barley. *Plant Pathol.* 25:63–73.
- Kølster, P., L. Munk, O. Stølen, and J. Løhde. 1986. Near-isogenic barley lines with genes for resistance to powdery mildew. *Crop Sci.* 26:903–907.
- Kopahnke, D., M. Nachtigall, F. Ordon, and B.J. Steffenson. 2004. Evaluation and mapping of a leaf rust resistance gene derived from *Hordeum vulgare* subsp. *spontaneum*. *Czech J. Genet. Plant Breed.* 40:86–90.

- Krings, M., N. Dotzler, J. Galtier, and T.N. Taylor. 2011. Oldest fossil basidiomycete clamp connections. *Mycoscience* 52:18–23.
- Lee, H.Y., C.H. Bowen, G. V. Popescu, H.-G. Kang, N. Kato, S. Ma, S. Dinesh-Kumar, M. Snyder, and S.C. Popescu. 2011. Arabidopsis RTNLB1 and RTNLB2 reticulon-like proteins regulate intracellular trafficking and activity of the FLS2 immune receptor. *Plant Cell Online* 23:3374–3391.
- Levine, M.N., and W.J. Cherewick. 1952. Studies on dwarf leaf rust of barley. USDA Tech. Bull. 1046:1–17.
- Li, H. 2011. A statistical framework for SNP calling, mutation discovery, association mapping and population genetical parameter estimation from sequencing data. *Bioinformatics* 27 (21):2987-93.
"http://samtools.sourceforge.net/mpileup.shtml".
- Li, H. and R. Durbin. 2009. Fast and accurate short read alignment with Burrows-Wheeler Transform. *Bioinformatics* 25:1754-60. "http://bio-bwa.sourceforge.net/".
- Lim, L.G., and R.E. Gaunt. 1986. The effect of powdery mildew (*Erysiphe graminis* f. sp. *hordei*) and leaf rust (*Puccinia hordei*) on spring barley in New Zealand. I. Epidemic development, green leaf area and yield. *Plant Pathol.* 35:44–53.
- Limpert, E., and K. Müller. 1994. Designation of pathotypes of plant pathogens. *J. Phytopathol.* 140:346-358.
- Mains, E.B. 1930. Host specialization of barley leaf rust, *Puccinia anomala*. *Phytopathology* 20:873–882.
- Mains, E.B., and H.S. Jackson. 1924. Aecial stages of the leaf rusts of rye, *Puccinia dispersa* Erikss. and Henn., and of barley, *P. anomala* Rostr., in the United States. *J. Agric. Res.* 28:1119–1126.
- Mains, E.B., and M.L. Martini. 1932. Susceptibility of barley to leaf rust (*Puccinia anomala*) and to powdery mildew (*Erysiphe graminis hordei*). USDA Tech. Bull. 295:1–33.
- Marcel, T.C., R. Aghnoum, J. Durand, R.K. Varshney, and R.E. Niks. 2007. Dissection of the barley 2L1.0 region carrying the “*Laevigatum*” quantitative resistance gene to leaf rust using near-isogenic lines (NIL) and subNIL. *Mol. Plant. Microbe. Interact.* 20:1604–15.
- Marcel, T.C., B. Gorguet, M.T. Ta, Z. Kohutova, A. Vels, and R.E. Niks. 2008. Isolate specificity of quantitative trait loci for partial resistance of barley to *Puccinia hordei* confirmed in mapping populations and near-isogenic lines. *New Phytol.* 177:743–755.
- Martínez, F., R.E. Niks, and D. Rubiales. 2001. Partial Resistance to leaf rust in a collection of ancient Spanish barleys. *Hereditas* 135:199–203.
- Mascher, M., H. Gundlach, A. Himmelbach, S. Beier, S.O. Twardziok, T. Wicker, V. Radchuk, C. Dockter, P.E. Hedley, J. Russell, M. Bayer, L. Ramsay, H. Liu, G. Haberer, X.Q. Zhang, Q. Zhang, R.A. Barrero, L. Li, S. Taudien, M. Groth, M. Felder, A. Hastie, H. Šimková, H. Stanková, J. Vrána, S. Chan, M.

- Munõz-Amatriaín, R. Ounit, S. Wanamaker, D. Bolser, C. Colmsee, T. Schmutzer, L. Aliyeva-Schnorr, S. Grasso, J. Tanskanen, A. Chailyan, D. Sampath, D. Heavens, L. Clissold, S. Cao, B. Chapman, F. Dai, Y. Han, H. Li, X. Li, C. Lin, J.K. McCooke, C. Tan, P. Wang, S. Wang, S. Yin, G. Zhou, J.A. Poland, M.I. Bellgard, L. Borisjuk, A. Houben, J. Doleael, S. Ayling, S. Lonardi, P. Kersey, P. Langridge, G.J. Muehlbauer, M.D. Clark, M. Caccamo, A.H. Schulman, K.F.X. Mayer, M. Platzer, T.J. Close, U. Scholz, M. Hansson, G. Zhang, I. Braumann, M. Spannagl, C. Li, R. Waugh, and N. Stein. 2017. A chromosome conformation capture ordered sequence of the barley genome. *Nature* 544:427–433.
- Mayer, K.F.X., R. Waugh, P. Langridge, T.J. Close, R.P. Wise, A. Graner, T. Matsumoto, K. Sato, A. Schulman, R. Ariyadasa, D. Schulte, N. Poursarebani, R. Zhou, B. Steuernagel, M. Mascher, U. Scholz, B. Shi, K. Madishetty, J.T. Svensson, P. Bhat, M. Moscou, J. Resnik, G.J. Muehlbauer, P. Hedley, H. Liu, J. Morris, Z. Frenkel, A. Korol, H. Bergès, S. Taudien, M. Felder, M. Groth, M. Platzer, A. Himmelbach, S. Lonardi, D. Duma, M. Alpert, F. Cordero, M. Beccuti, G. Ciardo, Y. Ma, S. Wanamaker, F. Cattonaro, V. Vendramin, S. Scalabrin, S. Radovic, R. Wing, M. Morgante, T. Nussbaumer, H. Gundlach, M. Martis, J. Poland, M. Pfeifer, C. Moisy, J. Tanskanen, A. Zuccolo, M. Spannagl, J. Russell, A. Druka, D. Marshall, M. Bayer, D. Swarbreck, D. Sampath, S. Ayling, M. Febrer, M. Caccamo, T. Tanaka, S. Wannamaker, T. Schmutzer, J.W.S. Brown, G.B. Fincher, and N. Stein. 2012. A physical, genetic and functional sequence assembly of the barley genome. *Nature* 491:711–716.
- Melville, S.C., and G.W. Griffin. 1976. Effects of fungicide spraying on brown rust and yield in spring barley. *Plant Pathol.* 25:99–107.
- Middleton, C.P., N. Senerchia, N. Stein, E.D. Akhunov, B. Keller, T. Wicker, and B. Kilian. 2014. Sequencing of chloroplast genomes from wheat, barley, rye and their relatives provides a detailed insight into the evolution of the Triticeae tribe. *PLoS One* 9:1–12.
- Misas-Villamil, J.C., R.A.L. van der Hoorn, and G. Doehlemann. 2016. Papain-like cysteine proteases as hubs in plant immunity. *New Phytol.* 212:902–907.
- Murray, G.M., and J.P. Brennan. 2010. Estimating disease losses to the Australian barley industry. *Australas. Plant Pathol.* 39:85–96.
- Neervoort, W.J., and J.E. Parlevliet. 1978. Partial resistance of barley to leaf rust *Puccinia hordei*. Analysis of the components of partial resistance in eight barley cultivars. *Euphytica* 27:33–39.
- Newton, M., B. Peterson, and W.O.S. Meredith. 1945. The effect of leaf rust of barley on the yield and quality of barley varieties. *Can. J. Res.* 23:212–218.
- van Niekerk, B.D., Z.A. Pretorius, and W.H.P. Boshoff. 2001. Occurrence and pathogenicity of *Puccinia hordei* on barley in South Africa. *Plant Dis.* 85:713–717.

- Niks, R.E. 1983. Comparative histology of partial resistance and the nonhost reaction to leaf rust pathogens in barley and wheat seedlings. *Phytopathology* :60–64.
- Niks, R.E., J.E. Parlevliet, P. Lindhout, and Y. Bai. 2011. Breeding crops with resistance to diseases and pests. Wageningen Academic, Wageningen.
- Nover, V.I., and C.O. Lehmann. 1974. Resistance in the barley and wheat collection Gatersleben. 18. Screening spring barleys for their reaction to leaf rust (*Puccinia hordei* Otth.). *Crop* 22: 25–43. (In German)
- Patil, V., A. Bjornstad, H. Magnus, and J.M. Key. 2002. Resistance to scald (*Rhynchosporium secalis*) in barley (*Hordeum vulgare*) studied by near-isogenic lines: II. Diallel analysis of near-isogenic lines. *Hereditas* 137:186–197.
- Park, R.F. 2003. Pathogenic specialization and pathotype distribution of *Puccinia hordei* in Australia, 1992 to 2001. *Plant Dis.* 87:1311–1316.
- Park, R.F. 2013. Barley leaf rust pathotype compromises WA. *Farming Ahead* 262:56–58.
- Park, R.F., P.G. Golegaonkar, L. Derevnina, K.S. Sandhu, H. Karaoglu, H.M. Elmansour, P.M. Dracatos, and D. Singh. 2015. Leaf rust of cultivated barley: Pathology and control. *Annu. Rev. Phytopathol.* 53:565–589.
- Park, R.F., and A. Karakousis. 2002. Characterization and mapping of gene *Rph19* conferring resistance to *Puccinia hordei* in the cultivar 'Reka 1' and several Australian barleys. *Plant Breed.* 121:232–236.
- Parlevliet, J.E. 1976. The genetics of seedling resistance to leaf rust, *Puccinia hordei* Otth. in some spring barley cultivars. *Euphytica* 25:249–254.
- Person, C. 1959. Gene-for-gene relationships in host : parasite systems. *Can. J. Bot.* 37:1101–1130.
- Pereyra, S., and M.D. d Ackermann. 2007. Use of fungicides to control leaf rust of barley and wheat. INIA LA Estanzuela. (In Spanish)
- Pickering, R.A., A.M. Hill, M. Michel, and G.M. Timmerman-Vaughan. 1995. The transfer of a powdery mildew resistance gene from *Hordeum bulbosum* L. to barley (*H. vulgare* L.) chromosome 2 (21). *Theor. Appl. Genet.* 91:1288–1292.
- Pickering, R.A., S. Malyshev, A. Johnston, V. Korzun, M. Menke, and I. Schubert. 2000. Locating introgressions of *Hordeum bulbosum* chromatin within the *H. vulgare* genome. *Theor. Appl. Genet.* 100:27–31.
- Pickering, R.A., B.J. Steffenson, A.M. Hill, and I.G. Borovkova. 1998. Association of leaf rust and powdery mildew resistance in a recombinant derived from a *Hordeum vulgare* x *Hordeum bulbosum* hybrid. *Plant Breed.* 117:83–84.
- Poulsen, D.M.E., R.J. Henry, R.P. Johnston, J.A.G. Irwin, and R.G. Rees. 1995. The use of bulk segregant analysis to identify a RAPD marker linked to leaf rust resistance in barley. *Theor. Appl. Genet.* 91:270–273.

- Qi, X., R.E. Niks, P. Stam, and P. Lindhout. 1998. Identification of QTLs for partial resistance to leaf rust (*Puccinia hordei*) in barley. *Theor. Appl. Genet.* 96:1205–1215.
- Rintelen, J. 1976. Physiological specialization of *Puccinia hordei* Otth. and reactions of barley varieties and cultivars to physiological races. *Proc. 3rd Int. Barley Genet. Symp.* p. 464–467.
- Roane, C.W. 1962. Inheritance of reaction to *Puccinia hordei* in barley. 1. Genes for resistance among North American race differentiating varieties. *Phytopathology* 52:1288–1295.
- Roane, C.W., and T.M. Starling. 1967. Inheritance of reaction to *Puccinia hordei* in barley. II. Gene symbols for loci in differential cultivars. *Phytopathology* 57:66–68.
- Roelfs, A.P., and J.W. Martens. 1988. An international system of nomenclature for *Puccinia graminis* f. sp. *tritici*. *Phytopathology* 78:526–533.
- Ronsdorf, L. 1935. Further studies on the detection of biological breeds of the barley rust, *Puccinia simplex* Erikss. and Henn. *Phytopathology Magazine* 8:237–243. (In German)
- Rouse, M., C.A. Griffey, and W.S. Brooks. 2013. First detection of *Puccinia hordei* virulence to barley leaf rust resistance gene *Rph3* and combination with virulence to *Rph7* in North America. *Plant Dis.* 97:838.
- Rubiales, D., and R.E. Niks. 1992. Histological responses in *Hordeum chilense* to brown and yellow rust fungi. *Plant Pathol.* 41:611–617.
- Sadravi, M., Y. Ono, M. Pei, and K. Rahnama. 2007. Fourteen rusts from northeast Iran. *J. Plant Pathol.* 89:191–202.
- Sandhu, K.S., K.L. Forrest, S. Kong, U.K. Bansal, D. Singh, M.J. Hayden, and R.F. Park. 2012. Inheritance and molecular mapping of a gene conferring seedling resistance against *Puccinia hordei* in the barley cultivar Ricardo. *Theor. Appl. Genet.* 125:1403–1411.
- Sandhu, K.S., D. Singh, and R.F. Park. 2016. Characterization of leaf rust resistance in international barley nurseries. *J. Plant Breed. Crop Sci.* 8:117–125.
- Schnaithmann, F., D. Kopahnke, and K. Pillen. 2014. A first step toward the development of a barley NAM population and its utilization to detect QTLs conferring leaf rust seedling resistance. *Theor. Appl. Genet.* 127:1513–25.
- Schweizer, P., and N. Stein. 2011. Large-scale data integration reveals colocalization of gene functional groups with meta-QTL for multiple disease resistance in barley. *Mol. Plant-Microbe Interact.* 24:1492–1501.
- Shelko, L.G. 1974. Race differentiation of the causative agent and immunity sources of barley to dwarf rust. *Bull. of Appl. Bot. Genet. Plant Breed.* 53:105–112. (In Russian)
- Shtaya, M.J.Y., J.C. Sillero, and D. Rubiales. 2006. Screening for Resistance to Leaf Rust (*Puccinia hordei*) in a Collection of Spanish Barleys. *Breed. Sci.* 56:173–177.

- Singh, D., P.M. Dracatos, L. Derevnina, M. Zhou, and R.F. Park. 2015. *Rph23*: A new designated additive adult plant resistance gene to leaf rust in barley on chromosome 7H. *Plant Breed.* 134:62–69.
- Spaner, D., L.P. Shugar, T.M. Choo, I. Falak, K.G. Briggs, W.G. Legge, D.E. Falk, S.E. Ullrich, N.A. Tinker, B.J. Steffenson, and D.E. Mather. 1998. Mapping of disease resistance loci in barley on the basis of visual assessment of naturally occurring symptoms. *Crop Sci.* 38:843–850.
- Stakman, E.C., D.M. Stewart, and W.Q. Loegering. 1962. Identification of physiologic races of *Puccinia graminis* var. *tritici*. *USDA Agric. Res. Serv.* E617:5–53.
- Stakman, E.C., M.N. Levine, C.J. T, and K. Isenbeck. 1933. Determination of physiological breeds of phytopathogenic fungi. November 8 Leopoldina treatises of Kais. Lopoldinisch-Carolinisch Duetschland Academy. *The Naturalist* 3:281–336. (In German)
- Stein, N., G. Herren, and B. Keller. 2001. A new DNA extraction method for high-throughput marker analysis in a large-genome species such as *Triticum aestivum*. *Plant Breed.* 120:354–356.
- Steffenson, B.J., Y. Jin, and C.A. Griffey. 1993. Pathotypes of *Puccinia hordei* with virulence for the barley Leaf rust resistance gene *Rph7* in the United States. *Plant Dis.* 77:867–869.
- Stevenson, J.A., and A.G. Johnson. 1946. The nomenclature of the barley leaf rust. *Plant Dis. Report.* 30:372.
- Straib, W. 1937. On the question of the rust species occurring on *Hordeum murinum* L. and the independence of *Puccinia hordei*. *Rep. German Bot. Soc.* 55:120–126. (In German)
- Subedi, S. 2017. Identification of the gene underlying the *Rphq2* resistance of barley (*Hordeum vulgare*) to barley leaf rust (*Puccinia hordei*). M.S. thesis. Wageningen University and Research, Wageningen.
- Tan, B.H. 1977a. A new gene for resistance to *Puccinia hordei* in certain Ethiopian barleys. *Cereal Rusts Bull.* 5:39–43.
- Tan, B.H. 1977b. Evaluating host differentials of *Puccinia hordei*. *Cereal Rusts Bull.* 5:17–23.
- Tan, B.H. 1978. Verifying the genetic relationships between three leaf rust resistance genes in barley. *Euphytica* 27:317–323.
- Tiffney, B.H., and E.S. Barghoorn. 1974. The fossil record of the fungi. *Occasional Pap. Farlow Herb. Cryptogam. Bot.* 7:1–42.
- Tosa, Y. 1992. A model for the evolution of formae speciales and races. *Phytopathology*:728–730.
- Tuleen, N.A., and M.E. McDaniel. 1971. Location of genes *Pa* and *Pa5*. *Barley Newsl.* 15:106–107.
- Ullrich, S.E. 2011. *Barley: Production, Improvement, and Uses*. Wiley-Blackwell, Ames, IA, USA.

- USDA. 2017. World Agricultural Production. Circ. Ser. August 2016 (WAP 8-16):1–29.
- Valjavec-Gratian, M., and B.J. Steffenson. 1997. Pathotypes of *Cochliobolus sativus* on Barley in North Dakota. Plant Dis. 81:1275–1278.
- Voorrips, R.E. 2002. MapChart: Software for the graphical presentation of linkage maps and QTLs. J. Hered. 93:77–78.
- Walther, U. 1987. Inheritance of resistance to *Puccinia hordei* Otth in the spring barley variety Trumpf. Cereal Rusts Bull. 15:20–26.
- Wan, A., and X. Chen. 2014. Virulence characterization of *Puccinia striiformis* f. sp. *tritici* using a new set of Yr single-gene line differentials in the United States in 2010. Phytopathology 98:1534–1542.
- Waterhouse, W.L. 1927. Studies in the inheritance of resistance to leaf rust, *Puccinia anomala* Rostr., in crosses of barley. I. J. Proc. R. Soc. New South Wales 61:218–247.
- Watson, I.A., and F.C. Butler. 1947. Resistance to barley leaf rust (*Puccinia anomala* Rost.). Proc. Linn. Soc. New South Wales 72:379–386.
- Weerasena, J.S., B.J. Steffenson, and A.B. Falk. 2004. Conversion of an amplified fragment length polymorphism marker into a co-dominant marker in the mapping of the *Rph15* gene conferring resistance to barley leaf rust, *Puccinia hordei* Otth. Theor. Appl. Genet. 108:712–9.
- Wendler, N., M. Mascher, C. Nöh, A. Himmelbach, U. Scholz, B. Ruge-Wehling, and N. Stein. 2014. Unlocking the secondary gene-pool of barley with next-generation sequencing. Plant Biotechnol. J. 12:1122–1131.
- Whelan, H.G., R.E. Gaunt, and W.R. Scott. 1997. The effect of leaf rust (*Puccinia hordei*) on yield response in barley (*Hordeum vulgare* L.) crops with different yield potentials. Plant Pathol. 46:397–406.
- Williamson, B., B. Tudzynski, P. Tudzynski, and J.A.L. Van Kan. 2007. *Botrytis cinerea*: The cause of grey mould disease. Mol. Plant Pathol. 8:561–580.
- Wiltén, W. 1953. Fighting dwarf rust (*Puccinia simplex*) in summer barley. Notebook of the Natl. Committee for Brewers 17:72–79. (In Dutch)
- Woldeab, G., C. Fininsa, H. Singh, and J. Yuen. 2006. Virulence spectrum of *Puccinia hordei* in barley production systems in Ethiopia. Plant Pathol. 55:351–357.
- Woldeab, G., J. Yuen, C. Fininsa, and H. Singh. 2007. Barley leaf rust (*Puccinia hordei* Otth) in three production systems and practices in Ethiopia. Crop Prot. 26:1193–1202.
- Yahyaoui, A.H. 1986. Epidemiology of *Puccinia hordei* and new sources of resistance in barley. Ph.D. Thesis. Montana State University, Bozeman.
- Yahyaoui, A.H., E.L. Sharp, and M. Reinhold. 1988. New sources of resistance to *Puccinia hordei* in barley land race cultivars. Phytopathology 78:905–908.

- Yu, X., H.Y. Kong, V. Meiyalaghan, S. Casonato, S. Chng, E.E. Jones, R.C. Butler, R. Pickering, and P.A. Johnston. 2018. Genetic mapping of a barley leaf rust resistance gene *Rph26* introgressed from *Hordeum bulbosum*. Theor. Appl. Genet. In press.
- Zhong, S., R.J. Effertz, Y. Jin, J.D. Franckowiak, and B.J. Steffenson. 2003. Molecular mapping of the leaf rust resistance gene *Rph6* in barley and its linkage relationships with *Rph5* and *Rph7*. Phytopathology 93:604–609.
- Zhou, B., R. V. Mural, X. Chen, M.E. Oates, R.A. Connor, G.B. Martin, J. Gough, and L. Zeng. 2017. A subset of ubiquitin-conjugating enzymes is essential for plant immunity. Plant Physiol. 173:1371–1390.
- Ziems, L.A., L.T. Hickey, G.J. Platz, J.D. Franckowiak, P.M. Dracatos, D. Singh, and R.F. Park. 2017. Characterisation of *Rph24*: A gene conferring adult plant resistance to *Puccinia hordei* in barley. Phytopathology 107: 834-841.
- Zimmer, D.E., J.F. Schafer, and F.L. Patterson. 1963. Mutations for virulence in *Puccinia coronata*. Phytopathology 53:171-176.
- Zloten, R.R. 1952. The inheritance of reaction to leaf rust in barley. M.S. thesis. University of Manitoba, Winnipeg.

Appendix Table 1. Accession details for the 24 barley leaf rust differential lines used in this study.

Line Name ¹	Accession Number ²	Gene	Country	Improvement Status ³	Donor ⁴
Sudan	CIho 6489	<i>Rph1</i>	Portugal	Cultivar	Brian Steffenson
Peruvian	CIho 2441	<i>Rph2</i>	Peru	Landrace	Brian Steffenson
Estate	CI 3410	<i>Rph3</i>	Egypt	Landrace	Brian Steffenson
Gold	CIho 1145	<i>Rph4</i>	Sweden	Cultivar	Brian Steffenson
Magnif 102	PI 337140	<i>Rph5</i>	Argentina	Cultivar	Brian Steffenson
Bolivia New	CI 1257	<i>Rph6</i>	Bolivia	Landrace	Brian Steffenson
Cebada Capa	CI 6193	<i>Rph7</i>	Argentina	Cultivar	Brian Steffenson
Egypt 4	CI 6481	<i>Rph8</i>	Egypt	Unknown	Brian Steffenson
Hor 2596	CI 1243	<i>Rph9</i>	Ethiopia	Landrace	Brian Steffenson
Clipper BC 8	GSHO 1588	<i>Rph10</i>	Israel	<i>H.v.s.</i> introgression	Brian Steffenson
Clipper BC 67	GSHO 1589	<i>Rph11</i>	Israel	<i>H.v.s.</i> introgression	Brian Steffenson
Triumph	PI 290195	<i>Rph12</i>	Germany	Cultivar	Brian Steffenson
PI 531849	PI 531849	<i>Rph13</i>	Unknown	<i>H.v.s.</i> introgression	Brian Steffenson
PI 584760	PI 584760	<i>Rph14</i>	Egypt	Breeding line	Brian Steffenson
I95-282-2	PI355447/5* Bowman	<i>Rph15</i>	Israel	<i>H.v.s.</i> introgression	Brian Steffenson
HOR15560	HS078 + HS084	<i>Rph16</i>	Israel	<i>H.v.s.</i> introgression	Robert Park
81882/BS1	-	<i>Rph17</i>	Unknown	<i>H.b.</i> introgression	Robert Park
38P18/8/1/10	-	<i>Rph18</i>	Unknown	<i>H.b.</i> introgression	Robert Park
Prior	CI 5051	<i>Rph2+19</i>	Australia	Cultivar	Robert Park
Flagship	-	<i>Rph20</i>	Australia	Cultivar	Robert Park
Ricardo	PI 328933	<i>Rph2+21</i>	Denmark	Cultivar	Robert Park
182Q20/1/M1	-	<i>Rph22</i>	Unknown	<i>H.b.</i> introgression	NorGen
Yerong	PI 573730	<i>Rph23</i>	Australia	Cultivar	Robert Park
ND24260	-	<i>Rph24</i>	United States	Breeding line	Jerry Franckowiak

¹. The original names given for each of the differential lines for *Rph1-24*.

². The accession numbers for each differential line used to search in genebank databases.

³. Improvement status indicates the level of domestication for each of the differential lines. *Hordeum vulgare* subsp. *spontaneum* and *Hordeum bulbosum* are respectively abbreviated as *H.v.s.* and *H.b.*

⁴. The research scientist who provided stocks for each of the differential lines.

Appendix Table 2. Accession details for the barley cultivar Bowman and the 95 donors of leaf rust resistance used in this study.

BW Line¹	Parental Accession²	Other ID	Origin	Status³	Pedigree⁴
-	Bowman	PI 483237	North Dakota, USA	Cultivar	Klages//Fergus/Nordic/3/ND115 6/4/Hector
BW667	PI 405338	HS 733	Israel	Wild	-
BW668	PI 405169	HS 534	Israel	Wild	-
BW669	PI 405298	HS 689	Israel	Wild	-
BW670	PI 391007	HS 520	Israel	Wild	-
BW671	PI 466470	6	Northern Israel	Wild	-
BW672	PI 466373	40	Southern Israel	Wild	-
BW673	PI 405305	HS 697	Israel	Wild	-
BW674	PI 466245	3157	Israel	Wild	-
BW675	Gija/Tanekase	-	-	-	-
BW676	PI 531990	2712	Egypt	Wild	-
BW677	PI 531853	2280	Northern Israel	Wild	-
BW678	PI 354926	HS 527	Israel	Wild	-
BW679	PI 282610	11001	Israel	Wild	-
BW680	PI 391049	HS 705	Israel	Wild	-
BW681	PI 391044	HS 665	Israel	Wild	-
BW682	Clho 6489	Sudan	Lisbon, Portugal	Unknown	-
BW683	Clipper BC8	GSHO 1588	Australia	Introgression line	-
BW684	Clipper BC67	GSHO 1589	Australia	Introgression line	-
BW685	PI 531849	BS 99-2-1	England	Introgression line	Berac*3/HS 2986
BW686	PI 531901-1	PI 584760	Egypt	Wild	Selection from PI 531901
BW687	PI 531901-2	PI 584761	Egypt	Wild	Selection from PI 531901

Appendix Table 2 continued. Accession details for the barley cultivar Bowman and the 95 donors of leaf rust resistance used in this study.

BW Line¹	Parental Accession²	Other ID	Origin	Status³	Pedigree⁴
BW688	PI 354940	HS 708	Israel	Wild	-
BW689	PI 405277	HS 661	Israel	Wild	-
BW690	PI 354923	HS 498	Israel	Wild	-
BW691	PI 391045	HS 697	Israel	Wild	-
BW692	PI 296841	Dinoor No. 445	Southern Israel	Wild	-
BW693	PI 354932	HS 610	Israel	Wild	-
BW694	PI 391002	HS 506	Israel	Wild	-
BW695	PI 391072	-	Northern Israel	Wild	-
BW696	PI 391087	-	Northern Israel	Wild	-
BW697	PI 391121	-	Jerusalem, Israel	Wild	-
BW698	PI 405154	HS 515	Israel	Wild	-
BW699	PI 405194	HS 564	Israel	Wild	-
BW700	PI 405201	HS 572	Israel	Wild	-
BW701	PI 405215	HS 587	Israel	Wild	-
BW702	PI 405219	HS 591	Israel	Wild	-
BW703	PI 405233	HS 607	Israel	Wild	-
BW704	PI 405289	HS 676	Israel	Wild	-
BW705	PI 405308	HS 701	Israel	Wild	-
BW706	PI 405332	HS 727	Israel	Wild	-
BW707	PI 405335	HS 730	Israel	Wild	-
BW708	PI 405364	HS 774	Israel	Wild	-
BW709	PI 405399	HS 819	Israel	Wild	-
BW710	PI 405235	HS 609	Israel	Wild	-

Appendix Table 2 continued. Accession details for the barley cultivar Bowman and the 95 donors of leaf rust resistance used in this study.

BW Line¹	Parental Accession²	Other ID	Origin	Status³	Pedigree⁴
BW711	PI 405311	HS 704	Israel	Wild	-
BW712	PI 354928	HS 595	Israel	Wild	-
BW713	PI 391000	HS 503	Israel	Wild	-
BW714	PI 391024	HS 585	Israel	Wild	-
BW715	PI 355444	665	Israel	Wild	-
BW716	PI 405227	HS 599	Israel	Wild	-
BW717	PI 405236	HS 610	Israel	Wild	-
BW718	PI 405354	HS 760	Israel	Wild	-
BW719	PI 355447	703	Israel	Wild	-
BW720	PI 391089	-	Northern Israel	Wild	-
BW721	PI 354937	HS 687	Israel	Wild	-
BW722	PI 391069	-	Northern Israel	Wild	-
BW723	PI 405179	HS 544	Israel	Wild	-
BW724	PI 405341	HS 740	Israel	Wild	-
BW725	PI 391004	HS 511	Israel	Wild	-
BW726	PI 405212	HS 584	Israel	Wild	-
BW727	PI 405209	HS 580	Israel	Wild	-
BW728	PI 355445	697	Israel	Wild	-
BW729	PI 355434	504	Israel	Wild	-
BW730	PI 405210	HS 581	Israel	Wild	-
BW731	PI 466247	3161-2	Israel	Wild	-
BW732	MSI 3351	-	-	-	Selection from Aim/MSI3351
BW733	PI 405220	HS 592	Israel	Wild	-
BW734	PI 466483	19	Northern Israel	Wild	-

Appendix Table 2 continued. Accession details for the barley cultivar Bowman and the 95 donors of leaf rust resistance used in this study.

BW Line¹	Parental Accession²	Other ID	Origin	Status³	Pedigree⁴
BW735	Vada/H. bulbosum	81882	New Zealand	Introgression Line	<i>H. bulbosum</i> introgression line
BW736	Emir/H. bulbosum	38P18/9/4	New Zealand	Introgression Line	<i>H. bulbosum</i> introgression line
BW737	Peruvian	CIho 2441	Cuzco, Peru	Landrace	Selection from PI 39150
BW738	Batna	CIho 3391	Algeria	Unknown	-
BW739	Juliaca	PI 39151	Puno, Peru	Landrace	-
BW740	Q21861	PI 584766	Western Australia	Unknown	CIMMYT line
BW741	Quinn	PI 39401	New South Wales, Australia	Landrace	-
BW742	Bolivia	PI 36360	Oruro, Bolivia	Landrace	-
BW743	Reka I	CIho 5051	New South Wales, Australia	Cultivar	Selection from Cape or Californian brewing type
BW744	PI 531841	BS 78-3-4	England	Introgression line	HJ 198*3/HS 2310
BW745	PC 249	PI 584765	Federal District Mexico	Unknown	CI12823/CI585//CI9805.16D/3/Cita 'S'/4/Gal//K1/2*CI2376/3/Gloria 'S'
BW746	Estate	PI 57700	Sharqiya, Egypt	Landrace	-
BW747	PI 466324	10	Haifa, Israel	Wild	-
BW748	Tunisia 25	-	-	-	-
BW749	Tunisia 33	-	-	-	-
BW750	Tunisia 29	-	-	-	-
BW751	Columbia	PI 494520	Montana, USA	Cultivar	Gus/Kombar

Appendix Table 2 continued. Accession details for the barley cultivar Bowman and the 95 donors of leaf rust resistance used in this study.

BW Line¹	Parental Accession²	Other ID	Origin	Status³	Pedigree⁴
BW752	Gold	CIho 1145	Malmöhus, Sweden	Cultivar	Selection from landrace in Gotlan region
BW753	Lehtaler	CIho 6488	Portugal	Unknown	-
BW754	Quinn	PI 39401	New South Wales, Australia	Landrace	-
BW755	Magnif 102	PI 337140	Buenos Aires, Argentina	Cultivar	Malteria Heda*4/Quinn
BW756	Bolivia	PI 36360	Oruro, Bolivia	Landrace	-
BW757	Tunisia 17	-	-	-	-
BW758	Cebada Capa	PI 539113	Argentina	Cultivar	-
BW759	Egypt 4	CIho 6481	Germany	Unknown	-
BW760	Hor 2596	CIho 1243	Ethiopia	Landrace	-
BW761	Triumph	PI 290195	Germany	Cultivar	Selection from Scotch Common

¹. Bowman leaf rust resistance introgression lines are abbreviated as "BW" followed by a numeric value.

². Accession numbers are a combination of an abbreviation and a numerical identifier. PI stands for Plant Introduction, CIho stands for Cereal Introduction in Hordeum, GSHO stands for Genetic Stock of Hordeum, and HS stands for Hordeum vulgare subsp. spontaneum.

³. Status indicates the level of domestication for each of the differential lines. *Hordeum vulgare* subsp. *spontaneum* and *Hordeum bulbosum* are respectively abbreviated as *H.v.s.* and *H.b.*

⁴. The known pedigrees used to develop each of the leaf rust resistant donor parents are listed.

Appendix Table 3. Pedigrees and recognized *Rph* genes in the 95 Bowman introgression lines used in this study.

BW Line¹	Donor Parent²	Generation³	Pedigree⁴	Gene Identity⁵
BW667	PI 405338	BC ₂ F ₃	PI405338/3*Bowman	<i>Rph15</i>
BW668	PI 405169	BC ₃ F ₃	Bowman*4/PI405169A	?
BW669	PI 405298	BC ₃ F ₃	PI405298/4*Bowman	<i>Rph15</i>
BW670	PI 391007	BC ₃ F ₃	PI391007/4*Bowman	<i>Rph15</i>
BW671	PI 466470	BC ₄ F ₃	PI466470/5*Bowman	<i>Rph15</i>
BW672	PI 466373	BC ₄ F ₃	Bowman*5//Moore/PI466373	?
BW673	PI 405303	BC ₅ F ₃	Bowman*6//Aim/PI405303	<i>Rph15</i>
BW674	PI 466245	BC ₆ F ₃	Bowman*7/PI466245	<i>Rph15</i>
BW675	Gija/Tanekase	BC ₆ F ₃	Bowman*7//Gija 119/Tanekase	<i>Rph3</i>
BW676	PI 531990	BC ₇ F ₃	Bowman*8/PI531990	<i>Rph5</i>
BW677	PI 531853	BC ₅ F ₃	Bowman*6/PI531853	<i>Rph9</i>
BW678	PI 354926	BC ₃ F ₃	Bowman*4/PI354926	<i>Rph15</i>
BW679	PI 282610	BC ₅ F ₃	Bowman*6/PI282610	<i>Rph15</i>
BW680	PI 391049	BC ₅ F ₃	PI391049/4*Bowman	<i>Rph15</i>
BW681	PI 391044	BC ₄ F ₃	PI391044/ND13944//4*Bowman	<i>Rph15</i>
BW682	Sudan	BC ₅ F ₃	Bowman*6/Sudan	<i>Rph1.a</i>
BW683	Clipper BC8	BC ₅ F ₃	Clipper BC8/6*Bowman	<i>Rph10.o</i>
BW684	Clipper BC67	BC ₅ F ₃	Bowman*6/Clipper BC67	<i>Rph11.p</i>
BW685	PI 531849	BC ₆ F ₃	Bowman*7/PI 531849	<i>Rph13.x</i>
BW686	PI 584760	BC ₄ F ₃	Bowman*5/PI 584760	<i>Rph14.ab</i>
BW687	PI 584761	BC ₄ F ₃	PI584761/5*Bowman	<i>Rph3</i>
BW688	PI 354940	BC ₄ F ₃	PI354940/5*Bowman	<i>Rph15</i>

Appendix Table 3 continued. Pedigrees and recognized *Rph* genes in the 95 Bowman introgression lines used in this study.

BW Line¹	Donor Parent²	Generation³	Pedigree⁴	Gene Identity⁵
BW689	PI 405277	BC ₄ F ₃	Bowman*5/PI405277	<i>Rph15</i>
BW690	PI 354923	BC ₄ F ₃	PI354923/5*Bowman	<i>Rph15</i>
BW691	PI 391045	BC ₄ F ₃	Bowman*5/PI391045A het Eam1	<i>Rph15</i> +
BW692	PI 296841	BC ₅ F ₃	PI296841/6*Bowman	<i>Rph15</i>
BW693	PI 354932	BC ₅ F ₃	PI354932/6*Bowman	<i>Rph15</i>
BW694	Pi 391002	BC ₅ F ₃	PI391002/6*Bowman	<i>Rph15</i>
BW695	PI 391072	BC ₅ F ₃	PI391072/6*Bowman	<i>Rph15</i>
BW696	PI 391087	BC ₅ F ₃	PI391087/6*Bowman	<i>Rph15</i>
BW697	PI 391121	BC ₅ F ₃	Bowman*6/PI391121	<i>Rph15</i> +
BW698	PI 405154	BC ₅ F ₃	Bowman*6/PI405154	?
BW699	PI 405194	BC ₅ F ₃	PI405194/6*Bowman	<i>Rph15</i>
BW700	PI 405201	BC ₅ F ₃	Bowman*6/PI405201	<i>Rph15</i>
BW701	PI 405215	BC ₅ F ₃	PI405215/ND13944//5*Bowman	?
BW702	PI 405219	BC ₅ F ₃	Bowman*6/PI405219	<i>Rph15</i>
BW703	PI 405233	BC ₅ F ₃	PI405233/6*Bowman	<i>Rph15</i>
BW704	PI 405289	BC ₅ F ₃	PI405289/6*Bowman	<i>Rph15</i>
BW705	PI 405308	BC ₅ F ₃	PI405308/6*Bowman	<i>Rph15</i>
BW706	PI 405332	BC ₅ F ₃	Bowman*6/PI405332	<i>Rph15</i>
BW707	PI 405335	BC ₅ F ₃	Bowman*6/PI405335	<i>Rph15</i>
BW708	PI 405364	BC ₅ F ₃	Bowman*6/PI405364	<i>Rph15</i>
BW709	PI 405399	BC ₅ F ₃	Bowman*6/PI405399	<i>Rph15</i> +
BW710	PI 405235	BC ₆ F ₃	Bowman*7/PI405235	<i>Rph15</i>

Appendix Table 3 continued. Pedigrees and recognized *Rph* genes in the 95 Bowman introgression lines used in this study.

BW Line¹	Donor Parent²	Generation³	Pedigree⁴	Gene Identity⁵
BW711	PI 405311	BC ₆ F ₃	Bowman*7/PI405311	<i>Rph15</i>
BW712	PI 354928	BC ₃ F ₃	PI354928/4*Bowman	<i>Rph15</i>
BW713	PI 391000	BC ₅ F ₃	PI391000/6*Bowman	<i>Rph15</i>
BW714	PI 391024	BC ₅ F ₃	PI391024/6*Bowman	<i>Rph15</i>
BW715	PI 355444	BC ₅ F ₃	Bowman*6/PI355444	<i>Rph15</i>
BW716	PI 405227	BC ₅ F ₃	Bowman*6/PI405227	<i>Rph15</i>
BW717	PI 405236	BC ₅ F ₃	PI405236/6*Bowman	<i>Rph15</i>
BW718	PI 405354	BC ₅ F ₃	Bowman*6/PI405354A	<i>Rph15</i>
BW719	PI 355447	BC ₇ F ₃	PI 355447/8*Bowman	<i>Rph15</i>
BW720	PI 391089	BC ₇ F ₃	PI391089/8*Bowman	<i>Rph15</i>
BW721	PI 354937	BC ₇ F ₃	Bowman*8/PI354937	<i>Rph15</i>
BW722	PI 391069	BC ₇ F ₃	Bowman*8/PI391069	<i>Rph15</i>
BW723	PI 405179	BC ₃ F ₃	PI405179/4*Bowman 0:cn	<i>Rph15</i>
BW724	PI 405341	BC ₅ F ₃	Bowman*6/PI405341	<i>Rph15</i>
BW725	PI 391004	BC ₆ F ₃	PI391004/7*Bowman Translocation	<i>Rph15</i>
BW726	PI 405212	F ₃	HS 584/Bowman	?
BW727	PI 405209	F ₄	HS 580/Bowman	?
BW728	PI 355445	BC ₃ F ₃	Bowman*4/PI355445	?
BW729	PI 355434	BC ₄ F ₃	Bowman*5/PI355434	<i>Rph15</i>
BW730	PI 405210	BC ₄ F ₃	PI405210/5*Bowman	<i>Rph15</i>
BW731	PI 466247	BC ₄ F ₃	PI466247/5*Bowman	<i>Rph9</i>
BW732	MSI 3351	BC ₅ F ₃	Bowman*6//AIM/MSI 3351	<i>Rph2</i>

Appendix Table 3 continued. Pedigrees and recognized *Rph* genes in the 95 Bowman introgression lines used in this study.

BW Line ¹	Donor Parent ²	Generation ³	Pedigree ⁴	Gene Identity ⁵
BW733	PI 405220	BC ₅ F ₃	PI405220/6*Bowman	<i>Rph15</i>
BW734	PI 466483	BC ₅ F ₃	PI466483/6*Bowman	<i>Rph15</i>
BW735	81882 (Vada/ <i>H. bulbosum</i>)	F ₄	81882 Rph17.af/Bowman	<i>Rph17</i>
BW736	38P18/9/4 (Emir/ <i>H. bulbosum</i>)	BC ₁ F ₃	Bowman*2/38P18-9-4	<i>Rph18</i>
BW737	Peruvian	BC ₂ F ₃	Bowman*3/PERUVIAN	<i>Rph2.b</i>
BW738	Batna	BC ₅ F ₃	BATNA/6*Bowman	<i>Rph4</i>
BW739	Juliaca	BC ₃ F ₃	Bowman*4/JULIACA 0;1	<i>Rph2.l</i>
BW740	Q21861	BC ₁ F ₃	Bowman*2/Q21861 Rph2.Q	<i>Rph3</i>
BW741	Quinn	BC ₄ F ₃	QUINN/5*Bowman	<i>Rph9</i>
BW742	Bolivia	BC ₄ F ₃	Bowman*5/BOLIVIA	<i>Rph2.r</i>
BW743	Reka I	BC ₇ F ₃	B0WMAN*8/REKA 1	<i>Rph2.t</i>
BW744	PI 531841	BC ₄ F ₃	PI 531841A/5*Bowman	<i>Rph2.y</i>
BW745	PC249	BC ₅ F ₃	PC249A/6*Bowman PI584765	<i>Rph3.aa</i>
BW746	Estate	BC ₁₀ F ₃	Bowman*11//CI3410/3.2 uz als MM	<i>Rph3.c</i>
BW747	PI 466324	BC ₆ F ₃	Bowman*7/PI466324	<i>Rph3.w</i>
BW748	Tunisia 25	BC ₄ F ₃	Bowman*5/TUNISIA 25	<i>Rph5</i>
BW749	Tunisia 33	BC ₅ F ₃	Bowman*6/TUNISIA 33	<i>Rph19?</i>
BW750	Tunisia 29	BC ₅ F ₃	Bowman*6/TUNISIA 29	<i>Rph5</i>
BW751	Columbia	BC ₅ F ₃	12022/3/CMB822/BOW//Columbia/4/ 6*Bowman	?
BW752	Gold	BC ₅ F ₃	GULL/6*Bowman	<i>Rph4.d</i>
BW753	Lechtaler	BC ₆ F ₃	LECHTALER/7*Bowman	<i>Rph4.d</i>

Appendix Table 3 continued. Pedigrees and recognized *Rph* genes in the 95 Bowman introgression lines used in this study.

BW Line¹	Donor Parent²	Generation³	Pedigree⁴	Gene Identity⁵
BW754	Quinn	BC7F3	Bowman*8/QUINN	<i>Rph9</i>
BW755	Magnif 102	BC7F3	MAGNIF/8*Bowman	<i>Rph5.e</i>
BW756	Bolivia	BC7F3	Bowman*8/BOLIVIA	<i>Rph5.f</i>
BW757	Tunisia 17	BC7F3	Bowman*8/TUNISIA 17	<i>Rph5</i>
BW758	Cebada Capa	BC7F3	Bowman*8/4/ND7771//CI6193/MT81 195	<i>Rph7.g</i>
BW759	Egypt 4	BC5F3	Bowman*6/EGYPT 4	<i>Rph8.h</i>
BW760	Hor2596	BC7F3	Bowman*8/HOR 2596	<i>Rph9.i</i>
BW761	Triumph	BC8F3	Bowman*9/TRIUMPH	<i>Rph9.z</i>

¹. Bowman leaf rust resistance introgression lines are abbreviated as "BW" followed by a numeric value.

². The leaf rust resistance donor parent for the corresponding Bowman introgression line.

³. The backcross generation achieved for each Bowman introgression line.

⁴. The pedigree used to develop each Bowman introgression line.

⁵. The *Rph* allele derived from each leaf rust resistance donor.

Appendix Table 4. Details for the 12 *Puccinia hordei* isolates used to characterize 95 leaf rust resistant introgression lines in Bowman.

Isolate ¹	Collector ²	City	State	Country	<i>Rph</i> Vir ⁴
BR (Octal 11)	B. Clifford / E. Jones	Aberystwyth	Wales	United Kingdom	1
Arg 231*	E. Antonelli	Castelar	-	Argentina	3
WGCC	C. Roane	-	Virginia	USA	4
Ger 7	U. Walther	Hadmersleben	Sachsen-Anhalt	Germany	4
Race 4*	C. Roane	-	Virginia	USA	4
Aus 220*	R. Rees	Toowoomba	Queensland	Australia	5
98-20	B. Steffenson	Blacksburg	Virginia	USA	9
94-8*	J. Franckowiak	Aleppo (Tel Hadya)	-	Syria	10
BRS 76-12	B. Clifford / E. Jones	Aberystwyth	Wales	United Kingdom	10
Neth 28	J. Parlievliet	Wageningen	Gelderland	Netherlands	15
90-3	A. Roelfs	Sha`ar HaGai	Jerusalem	Israel	16
92-7	A. Roelfs	-	-	Morocco	17

¹. The original isolate name designated by the original collector.

². The name of the research scientist who first collected the *P. hordei* isolate.

³. Avr/Vir displays the combinations of avirulence and virulence genes of each isolate, respectively.

⁴. *Rph* Vir indicates the number of *Rph* genes for which an isolate is virulent.

Asterisks indicate the four isolates also used by Chicaiza (1996).

Appendix Table 5. Raw infection types¹ of Bowman introgression lines postulated to carry *Rph2*, *Rph3*, *Rph4*, *Rph5*, *Rph9*, or unknown resistance gene(s).

BW Line ²	Gene ³	Isolates of <i>Puccinia hordei</i>																	
		BR (Octal 11)			ARG231			Aus 220			Race 4			Ger 7			WGCC		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
BW743*	<i>Rph2.t</i>	12-c	0;12c	120;c	213-	0;1c	120;	23-c	213-c	23-1	213-c	213-c	0;12c	32	33-	3-2	120;	23-1c	120;c
BW732	<i>Rph2</i>	21	0;12c	0;1	3-2	0;1-c	0;1	3-2	0;1c	0;1-cn	33+	0;12c	0;1cn	3+3	33-	3	0;1-c	3	0;12c
BW737	<i>Rph2.b</i>	21c	23-c	21c	21	0;1c	10;	23-1c	3-2c	3-2	120;	213-c	0;12c	3	3-	3-2c	21c	213-c	0;12
BW739	<i>Rph2.l</i>	21c	10;c	120;c	21c	0;1-c	0;12	21c	0;12c	0;12	3-21	213-	120;c	33+	33-	3	210;	0;12c	0;12c
BW742	<i>Rph2.r</i>	21	0;12c	10;c	12	0;1c	120;	3-2	213-c	23-1	120;	23-1c	120;c	23-	3	213-c	120;	3-2c	120;c
BW744	<i>Rph2.y</i>	21	0;12c	120;c	213-	0;12c	120;	23-1c	213-c	23-1	210;c	0;12c	0;12c	33+	33-	3	120;	213-c	0;12c
BW746*	<i>Rph3.c</i>	0;1-	0;1c	0;1c	0;1-	00;c	0;1-c	0;1-c	0;1-c	0;1	0;1-	0;1c	0;12c	00;1	00;cn	00;2	0;1c	0;1c	0;1c
BW675	<i>Rph3</i>	0;1-c	00;	0;1	0;1-	00;1	0;1-	0;1c	00;1c	0;1-	00;1-	0;1-	0;n	00;1-	00;n	00;2	0;1-c	0;1c	0;12c
BW687	<i>Rph3</i>	0;1-	00;1-cn	00;1	0;1-	0;c	00;	0;1-c	0;1-c	0;1-	0;1-	0;1-c	00;1	00;n	00;1-	00;	0;1-	0;12c	0;12
BW740	<i>Rph3</i>	0;1-	00;2	00;	0;1-	00;1-c	00;	0;1-c	0;1-c	00;	00;	0;1c	00;n	00;	00;2	00;1	00;1-	0;12c	00;n
BW745	<i>Rph3.aa</i>	10;c	0;1-c	0;1-	0;1-	0;1c	00;1	12c	0;12c	00;1	0;1-	0;12	00;1	00;1-	00;2	00;1	0;1c	0;12c	0;12
BW747	<i>Rph3.w</i>	0;1-	00;1-c	00;1	0;1-c	00;1-c	00;	0;1-c	0;1-c	0;1-	0;1-	0;1c	00;n	00;1	00;2	00;2	00;1-n	0;12c	0;12c
BW752*	<i>Rph4.d</i>	213-	3-2	3-c	21	23-c	120;c	210;c	0;1-c	120;	210;	120;c	0;1c	3-2	3	3	23-1	120;c	0;1
BW738	<i>Rph4</i>	21c	23-c	3-2	21c	23-c	120;c	0;1-c	0;12c	0;1	120;	213-c	0;12c	33-	33-	3	213-c	0;12c	0;12c
BW753	<i>Rph4.d</i>	23-1	3-2	3-2	12c	23-1	213-	10;c	0;12c	120;	12c	3	3-	3	3	3	33-	3	0;1c

Appendix Table 5 continued. Raw infection types¹ of Bowman introgression lines postulated to carry *Rph2*, *Rph3*, *Rph4*, *Rph5*, *Rph9*, or unknown resistance gene(s).

BW Line ²	Gene ³	Isolates of <i>Puccinia hordei</i>																	
		BR (Octal 11)			ARG231			Aus 220			Race 4			Ger 7			WGCC		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
BW755*	<i>Rph5.e</i>	1-1	0;1-c	0;1	12	3-2	23-c	3-2	3	3	213-c	3-2	120;c	21c	120;c	120;c	3-2	3	3
BW756*	<i>Rph5.f</i>	1-0;	0;1c	0;1	3-2	3-2	3-	33-	3	3	213-c	3-2	12	21c	213-c	21c	3-2	3	3
BW676	<i>Rph5</i>	1-1	0;1c	10;c	23-	3-2c	3-2	3-	3	3	33+	3-	23-c	213-	23-c	213-c	213-	3	3-
BW748	<i>Rph5</i>	11-	0;1c	10;	213-	3-2	213-	3-2	33-	3	33-	23-c	213-	23-1c	23-c	120;c	33-	3-	3-2
BW750	<i>Rph5</i>	10;c	0;1c	10;c	21c	23-c	3-2	3-3	33-	3	23-	33-	3-2	1c	33-	0;12c	21c	3	3-2
BW757	<i>Rph5</i>	1-0;	10;c	10;	21c	120;c	3-	3-2	3-2	3	23-	23-c	3-2	11-c	23-c	210;c	213-	33+	3
BW760*	<i>Rph9.i</i>	11-c	0;1c	120;	21c	120;c	21c	21c	213-c	3-2	213-	21c	0;1c	0;1-c	12c	0;1c	213-c	23-c	0;12c
BW761*	<i>Rph9.z</i>	11-c	0;1c	0;1	11-c	0;1-c	0;12c	21c	0;12c	120;	10;c	0;12c	0;1c	0;1-c	10;c	0;1c	11-c	10;cn	10;c
BW677	<i>Rph9</i>	21c	0;12c	12c	21c	0;1c	21c	213-c	0;1c	12	0;1c	23-c	0;12c	10;c	10;c	0;12c	21c	0;1c	0;1c
BW731	<i>Rph9</i>	1-0;c	0;1c	12c	0;1c	0;1c	0;1c	0;1-c	0;1c	0;1c	0;1c	10;c	0;1c	0;1c	0;1c	0;1c	10;c	0;1c	0;1c
BW741	<i>Rph9</i>	1-0;c	0;1-c	0;1c	10;c	0;1-c	120;c	21c	0;12c	12c	0;12c	0;12c	120;c	0;1c	10;cn	0;12c	11-c	12cn	10;c
BW754	<i>Rph9</i>	1-0;c	0;1c	0;1c	12c	0;cn	0;1c	12c	0;12c	12c	10;c	10;c	0;12c	0;1c	10;c	0;1c	10;c	120;c	10;c
BW735	?	00;n	00;	00;	00;n	00;n	00;	00;n	00;n	00;	00;	00;	00;	00;n	00;	00;	00;	00;	00;
BW736	?	00;n	00;	00;	00;n	00;n	00;	00;n	00;n	00;	00;	00;	00;	00;1-	0;	00;	00;	00;	00;
BW726	?	0;1-cn	0;cn	0;	0;1-cn		120;	00;1-n	00;n	0;n	0;1-cn	0;1c	0;1-c	0;1-cn		0;	0;1-cn	0;1c	0;
BW672	?	213-	23-c	0;1-cn	21	0;1-cn	0;1-cn	21c	0;1c	0;1-cn	0;1-c	10;c	0;1-c	33-	21c	0;1c	0;1-c	23-c	0;1-c
BW727	?	21	3-2c	23-1	12	3-21	3	3-2	33-	3	3	3-	213-	33+	33-	3	33-	33+	3-2
BW728	?	21	3-2c	23-c	213-	120;c	3-2	21c	120;c	120;c	3+3	3+	3	3+3	3	3	213-c	21c	10;c
BW749	?	213-c	120;c	210;c	21c	0;1c	120;	3-2c	213-c	3-2	3-3	21	213-	33-	3	3	23-1	213-c	21c
BW668	?	33-	3-2c	3-	23-	33-	23-c	21-c	120;c	3-2	33-	3	23-	3-2	3	3-2	213-c	23-c	21c
BW698	?	12-	213-c	120;c	3-2	33-	3-		23-c	21c	3-3	33-	3-	21	3	3-2	21	3-	21c
BW701	?	213-	33-	3-2	3-3	33-	3-	3-3	3	3	3+3	33-	3	3	3	3	23-1	3	3
BW751	?	23-	33-	3-	33-	3	3	3-2	33-	3	33-	3	3	23-	3	3	3	3	3

Appendix Table 5 continued. Raw infection types¹ of Bowman introgression lines postulated to carry *Rph2*, *Rph3*, *Rph4*, *Rph5*, *Rph9*, or unknown resistance gene(s).

BW Line ²	Gene ³	Isolates of <i>Puccinia hordei</i>																	
		98-20			94-8			BRS 76-12			90-3			Neth 28			92-7		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep ₂	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
BW743*	<i>Rph2.t</i>	23-	3+	3	3-2	33-	210;c	213-c	33-	213-c	33-	33-	3-	3-2c	3-	3-	3-2c	33-	3-
BW732	<i>Rph2</i>	3-2	33-	3-2	3-2c	33-	213-	213-c	3-2	213-c	33-	33-	3-2	3-2	3-2	3-	3	33-	3-
BW737	<i>Rph2.b</i>	3-2	3	3	3-	33-	23-	3-2	33-	213-	3	3-	33-	3-2	3-2	3-	3+3	3-	3
BW739	<i>Rph2.l</i>	23-1	3-	3	33-	3	210;	3-2c	23-c	3-2c	33-	33-	33-	33-	23-	3-2	33+c	33-	3-
BW742	<i>Rph2.r</i>	23-	3+	3	33-	3	210;c	3-2	23-c	23-1c	33-	3	3-	3-2	3-	3-	3-2c	33-	3-
BW744	<i>Rph2.y</i>	23-	33-	3-2	33-	3	23-	23-1c	3-2	23-1c	3-2	3-	33-	3-2	2	3-	33-	3-	3-
BW746*	<i>Rph3.c</i>	0;cn	0;1c	00;	0;1-c	00;1-c	0;	3-2c	33-c	23-c	120;c	21c	213-c	23-	23-	3-	3	3-	3-2
BW675	<i>Rph3</i>	0;1-cn	1-0;cn	00;1	0;1-c	00;1	00;	3-2	33-	210;c	21c	21c	213-c	33-	23-	3-2	33-	3	3
BW687	<i>Rph3</i>	0;	10;c	00;	0;1c	00;1-c	00;	23-1c	23-	213-	210;c	21c	213-c	213-c	213-	3-	23-	3-	
BW740	<i>Rph3</i>	0;1-c	00;1n	00;1	0;1-c	00;2c	00;	3-2	33-	210;c	210;c	12c	0;12c	33-	33-	3-21	33+	33-	3
BW745	<i>Rph3.aa</i>	0;1-c	00;1	00;	0;1-c	00;1c	00;	213-c	23-c	3-2	10;c	3-	213-c	23-	23-	3-2	33-	33-	
BW747	<i>Rph3.w</i>	00;2c	00;	00;	0;1-c	00;1-c	00;	3-2c	3-2c	3-2c	120;c	21c	210;c	3-	3-2	3	3-	3-	3
BW752*	<i>Rph4.d</i>	3	3	23-1	3-2	33+	21c	3-2	33-	23-c	33-	3	3-2	210;c	120;c	0;12c	3	3	3
BW738	<i>Rph4</i>	23-1	3	3	23-	33-	210;c	3-2c	23-c	3-2	3-2	3	33-	210;c	21	120;c	33-	33-	3
BW753	<i>Rph4.d</i>	33-	3	23-	3-2	33+	23-	3-2c	23-	23-1c	3-2	33-	3	213-	10;c	120;c	23-c	33-	3-

Appendix Table 5 continued. Raw infection types¹ of Bowman introgression lines postulated to carry *Rph2*, *Rph3*, *Rph4*, *Rph5*, *Rph9*, or unknown resistance gene(s).

BW Line ²	Gene ³	Isolates of <i>Puccinia hordei</i>																	
		98-20			94-8			BRS 76-12			90-3			Neth 28			92-7		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
BW755*	<i>Rph5.e</i>	120;c	10;c	0;12	10;c	10;c	0;1-	3-2c	33-	210;c	3-	33-	3	3-	3-	3-2	33-	33-	3-
BW756*	<i>Rph5.f</i>	120;c	10;c	120;	10;c	10;c	0;1-c	3-2c	33-c	210;	3-2	3	3-3	3-2	3	3-	3-2	33-	3-
BW676	<i>Rph5</i>	21c	00;1	120;	10;c	0;1c	0;12c	3-2c	33-	23-1c	33-c	33+	33-	3-2	3-	3-2	33-	3	3-
BW748	<i>Rph5</i>	12c	10;c	0;12	10;c	0;1c	0;1-	3-2c	3-c	213-c	3	3-2	3-2	3-2c	33-	3-	3	33-	3-
BW750	<i>Rph5</i>	10;c	10;	120;	10;c	0;1c	0;1-	33-	33-	210;c	3+	33-	3	3	3	3-2	3+3	33-	3-
BW757	<i>Rph5</i>	120;c	120;c	120;	120;c	10;c	0;1	23-c	21c	23-1	33-	33-	3-2	3-2	3	3-2	3-2	33-	3-
BW760*	<i>Rph9.i</i>	23-c	23-c	213-	3-2	33-	213-c	12c	10;c	21c	3	3-	3	23-c	33-	3-2	3-3		3
BW761*	<i>Rph9.z</i>	120;c	10;c	0;12c	3-3	33+	213-	10;c	10;c	120;c	3	3	33-	3-2c	33-	3-2	3-2c	33-	
BW677	<i>Rph9</i>	21c	3-2c	3-2	23-1c	23-c	3-2c	0;1-cn	10;c	0;1c	33-c	3	3-	3-2c	23-c	23-c	21c	2c	3-
BW731	<i>Rph9</i>	12c	10;c	213-	23-1c	213-c	21c	10;cn	10;c	0;12c	3-2	33-	3-	213-c	21c	213-c	21c	10;c	210;
BW741	<i>Rph9</i>	12c	12c	21c	3-2	3	3-c	10;cn	10;c	0;12c	3	33-	3	23-c	3-	3-2	3-2c	3-	3-
BW754	<i>Rph9</i>	12c	12c	21c	33-	33-	213-c	10;c	10;c	10;	3-	33-	3-	3-2	23-	3-	3	33-	3-
BW735	?	00;	00;	00;	00;	00;	00;	00;	00;	00;	00;	00;	00;	00;	00;	0;	00;	00;	00;
BW736	?	00;	0;	00;	00;	0;	00;	00;	0;	00;	00;	0;	00;n	00;	00;	00;	00;n	00;	00;
BW726	?	1-0;cn	10;c	0;1c	1-0;cn	1-0;cn	0;1-cn	0;cn	00;n	0;	210;	1c	0;12c	120;c	0;1c	0;1-c	0;1c	0;1c	0;1c
BW672	?	23-c	12cn	12c	213-	33-	10;c	21c	23-1	0;1	3	3	3	23-1	21c	213-c	3-2c	21c	3-2
BW727	?	3-2c	12c	210;	213-c	10;	120;	3-2	213-c	0;12	33-	3	3-2	3-	23-	3-2	3	3-	3-2
BW728	?	3-2c	21c	21	213-c	12c	213-	3-2	23-c	3-2c	3	33-	3-	3-	3-2	210;c	3	3	3
BW749	?	3	3	3-2	3-2	3+	23-1	3-c	3	3-2c	33-	33-	3-	3-2	33-	33-	3+3	33-	3-
BW668	?	3-2c	12c	23-1	213-c	21c	210;c	3-2c	33-	23-1c	33-	3-	3	23-1	21c	23-c	3-2c	3-	3-2
BW698	?	23-1		21	33-	3-	21c	33-	2c	210;	3-2	33-	33-	3-	3-	3-	33+	3	33-
BW701	?	33-	33+	3-2	3-2	3	23-c	33-	33-	210;c	3-2	3	3-	3-2	23-	3-2	3+3	3	3-2
BW751	?	3	33+	23-	33-	33+	23-c	3-2	33-	210;c	33+	33-	3	3	3	3-2	3+3	3	3-

¹ Infection types were scored using the original 0-4 rating scale developed by Mains (1930) with the additional notations of "+" and "-" to indicate larger or smaller size variations of classically described uredinia (Levine and Cherewick, 1952) and "c" and "n" to indicate the presence of chlorotic or necrotic reactions (Moseman and Roane, 1959), respectively. Complex ITs were recorded when more than one IT was observed on the surface of a single leaf. For example, an IT of 213- indicates that all three pustule types exist on the same leaf and that the "2" type pustules were the most common, followed by the "1" type, with "3-" type pustules being the least common. Infection types from "0;" to "0;12" are classified as highly resistant ("R", green cells), infections types from "12" to "23-c" are classified as moderately resistant ("M", yellow cells), and infection types from "23-" to "33+" are classified as susceptible ("S", red cells) (Steffenson et al., 1993).

² BW lines are the 95 Bowman leaf rust resistance introgression lines.

³ The *Rph* gene retained in each leaf rust resistant introgression line.

Asterisks indicate the introgression line selected to represent that *Rph* gene in the differential host set.

Appendix Table 6. Raw infection types¹ of 50 Bowman introgression lines postulated to carry *Rph15*.

BW Line ²	Gene ³	Isolates of <i>Puccinia hordei</i>																	
		BR (Octal 11)			ARG231			Aus 220			Race 4			Ger 7			WGCC		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
BW719*	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	1-cn	1-0;cn	1-0;cn	0;1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW667	<i>Rph15</i>	0;1-cn	1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	1-0;cn	1-0;cn	120;c	1-0;cn	1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW669	<i>Rph15</i>	0;1-cn	1-cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	1-0;cn	0;1-cn	1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW670	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	1cn	12cn	0;1-cn	0;cn	0;cn	1-0;c	0;cn	0;cn
BW671	<i>Rph15</i>	0;cn	0;cn	0;cn	1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	1-cn	1cn	21cn	0;cn	0;cn	0;cn	1cn	1cn	0;cn
BW673	<i>Rph15</i>	1-cn	0;cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	0;cn	1-0;cn	0;1-cn	0;1-cn	0;1-c	10;cn	0;1-cn	0;1-cn	0;cn	0;cn
BW674	<i>Rph15</i>	1-cn	0;cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;1-cn	0;cn	0;cn	1-0;cn	0;1-cn	0;1-cn	1-0;cn	10;cn	0;1-cn	0;1-cn	0;cn	1-0;cn
BW678	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	1-0;cn	1-0;cn	0;1-cn	0;1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW679	<i>Rph15</i>	0;1-cn	0;cn	0;1-cn	11-c	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;1-c	1cn	23-cn	1-0;cn	0;cn	0;cn	0;1-cn	0;1cn	1-0;cn
BW680	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	0;1cn	12cn	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;cn
BW681	<i>Rph15</i>	11-c	0;12cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	0;12c	1-0;cn	1cn	1-cn	0;1-c	0;cn	0;cn
BW688	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	1-0;cn	1cn	0;1-cn	0;cn	0;cn	0;cn
BW689	<i>Rph15</i>	1-cn	1-0;cn	0;1-cn	1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	0;1-cn	1-0;cn	1cn	0;1-cn	1-0;cn	0;cn	0;cn
BW690	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	1-cn	1-0;cn	10;cn	0;1-cn	1-0;cn	0;cn	0;cn
BW692	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;12c	0;1-cn	10;cn	0;1-cn	1-0;cn	0;cn	0;cn
BW693	<i>Rph15</i>	1-cn	1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;1-cn	0;cn	1-0;cn	0;1cn	12cn	1-0;cn	10;cn	1-cn	0;1-cn	0;cn	0;cn
BW694	<i>Rph15</i>	1-cn	1-cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	1-cn	0;1-	1-0;cn	10;cn	1-0;cn	1-0;cn	0;cn	0;cn
BW695	<i>Rph15</i>	1-cn	1-cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-cn	0;1cn	0;1cn	1-0;cn	1cn	1-0;cn	1-0;cn	0;cn	0;cn
BW696	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;cn	1cn	1-0;cn	1cn	0;1-cn	1-0;cn	0;cn	0;cn
BW699	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	1cn	0;1-cn	1cn	10;cn	1-0;cn	0;1-cn	0;cn
BW700	<i>Rph15</i>	21	1-0;cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	210;	0;cn	0;1-cn	1-0;cn	3-	0;1-cn	0;cn	1cn	0;cn
BW702	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;1-c	0;1-cn	0;cn	0;cn	0;1-cn	1-0;cn	0;1-cn	0;1cn	0;cn	0;cn	0;cn	1-cn	0;1-cn	0;1-cn
BW703	<i>Rph15</i>	1-cn	1-0;cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	0;1cn	0;1-cn	1cn	0;1-cn	1-0;cn	0;cn	0;cn
BW704	<i>Rph15</i>	1-cn	1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;1cn	1-0;cn	1cn	0;1-cn	1-0;cn	0;cn	0;cn
BW705	<i>Rph15</i>	1-cn	1-0;cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;1-cn	10;cn	0;cn	1-cn	1-0;cn	0;1-cn	0;1-cn	1-0;cn	0;cn	0;cn

Appendix Table 6 continued. Raw infection types¹ of 50 Bowman introgression lines postulated to carry *Rph15*.

BW Line ²	Gene ³	Isolates of <i>Puccinia hordei</i>																	
		BR (Octal 11)			ARG231			Aus 220			Race 4			Ger 7			WGCC		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
BW706	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;1cn	1-0;cn	1cn	0;1-cn	0;1-cn	0;cn	0;cn
BW707	<i>Rph15</i>	1-cn	0;cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	1-0;cn	1-0;cn	0;1-cn	1cn	0;1-cn	0;cn	0;cn	0;cn
BW708	<i>Rph15</i>	1-cn	1-0;cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	1cn	12cn	1-0;cn	1cn	0;1-cn	1-0;cn	0;cn	0;cn
BW710	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1cn	0;12cn	1-0;cn	1-cn	0;1-cn	0;1-cn	0;cn	0;cn
BW711	<i>Rph15</i>	1-cn	0;cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;1-cn	1-0;cn	1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW712	<i>Rph15</i>			0;1-cn			0;1-cn			0;cn			0;1-cn			0;1-cn			0;cn
BW713	<i>Rph15</i>	1-cn	0;cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1cn	0;1-cn	1-0;cn	0;1cn	0;1-cn	1-0;cn	0;cn	0;cn
BW714	<i>Rph15</i>	0;1-cn	0;cn	0;cn	1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;1-cn	1-0;cn	1cn	0;1-cn	0;1-cn	0;cn	0;cn	1-0;cn	0;cn	0;1-cn
BW715	<i>Rph15</i>	1-cn	0;cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	1cn	0;1-cn	0;cn	0;1-cn	0;cn	1-0;cn	0;cn	0;cn
BW716	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	1-0;cn	0;cn	0;1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW717	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	1-0;cn	0;1-cn	10;cn	0;1-cn	1-0;cn	0;cn	0;cn
BW718	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	1-0;cn	0;1-cn	10;cn	0;1-cn	1-0;cn	0;cn	0;cn
BW720	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	1-0;cn	0;1cn	10;cn	0;1-cn	0;1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW721	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	10;cn	1-0;cn	0;1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW722	<i>Rph15</i>	1-cn	0;cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	1-0;cn	0;1-cn	1-0;cn	0;1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW723	<i>Rph15</i>	1-cn	0;cn	0;cn	0;1-cn	0;1-cn	10;c	0;1-cn	0;cn	0;1-cn	10;cn	21cn	12	0;1-cn	0;cn	0;cn	11-c	0;1-cn	0;1-
BW724	<i>Rph15</i>	1-cn	0;cn	0;1-cn	1-cn	0;1-cn	10;c	0;1-cn	0;cn	0;1-cn	1-0;cn	12cn	0;l	0;1-cn	0;cn	0;1-cn	11-c	0;1cn	0;1-
BW725	<i>Rph15</i>	0;1-cn	0;1-cn	0;1-cn	1-cn	0;1-cn	10;	0;1-cn	0;cn	0;1-cn	11-c	21cn	10;	1-0;cn	0;cn	0;cn	11-	10;cn	0;1-
BW729	<i>Rph15</i>	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	0;1-cn	0;1-cn	0;1-cn	1-0;cn	0;1-cn	0;1-cn	1-0;cn	0;cn	0;cn
BW730	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;1-cn	1-0;c	1cn	10;cn	0;1-cn	0;cn	0;cn	1-0;cn	0;1-cn	0;cn
BW733	<i>Rph15</i>	1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;1cn	0;cn	0;cn	0;cn	1-0;cn	0;1c	0;1-cn	1-0;cn	1-cn	0;1-cn	1-cn	0;cn	0;cn
BW734	<i>Rph15</i>	0;cn	0;cn	0;cn	0;1-cn	0;1-cn	1	0;1-cn	0;cn	0;cn	1-0;cn	1cn	0;1-cn	1-0;cn	0;cn	0;cn	1-0;cn	0;cn	0;cn
BW697	<i>Rph15+?</i>	1-0;c	10;c	0;1c	1c	0;1c	10;c	0;1-c	0;1-cn	0;l	10;c	12c	1-0;cn	21c	21c	10;cn	10;c	0;1c	0;1-cn
BW691	<i>Rph15+?</i>	0;1-	0;1c	0;1-	10;	10;c	10;	12c	120;c	120;c	10;c	0;12c	0;1-	213-	23-c	0;12	0;1	3-2	0;12
BW709	<i>Rph15+?</i>	1-cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	1cn	12cn	1-0;cn	1cn	0;1-cn	0;1-cn	0;cn	0;cn

Appendix Table 6 continued. Raw infection types¹ of 50 Bowman introgression lines postulated to carry *Rph15*.

BW Line ²	Gene ³	Isolates of <i>Puccinia hordei</i>																	
		98-20			94-8			BRS 76-12			90-3			Neth 28			92-7		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
BW719*	<i>Rph15</i>	0;1-cn	10;cn	10;cn	1-0;cn	1-0;cn	1cn	0;cn	0;cn	0;cn	3-3	33-	3-	12-cn	21cn	213-	0;1-cn	0;1-cn	10;cn
BW667	<i>Rph15</i>	1-0;cn	1-0;cn	213-c	1-0;cn	0;1-cn	120;cn	0;1-cn	0;cn	0;cn	3-c	33-	3	21c	21cn	3-2	1-0;cn	21cn	10;cn
BW669	<i>Rph15</i>	0;1-cn	0;1-cn	12cn	1-0;cn	0;1-cn	10;cn	0;1-cn	0;cn	0;cn	33-	33-	3	23-1	12cn	3-2c	1-0;cn	1-0;cn	10;cn
BW670	<i>Rph15</i>	11-cn	1-0;cn	12cn	11-cn	12cn	12cn	0;1-cn	0;cn	0;cn	33-	33-	3	3-2	21c	3-2	1-0;cn	1-0;cn	10;cn
BW671	<i>Rph15</i>	21cn	11-cn	12cn	21cn	12cn	21cn	0;cn	0;cn	0;cn	3	33+	3-2	33-	22+	3-2	1-cn	1cn	12cn
BW673	<i>Rph15</i>	1-cn	1-0;cn	10;cn	1-0;cn	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	33-c	3	33-	3-2cn	12cn	21cn	1-0;cn	0;1-cn	10;cn
BW674	<i>Rph15</i>	1-cn	1cn	12cn	10;cn	1-0;cn	1cn	0;cn	0;cn	0;cn	33-c	33+	3-2	3-2cn	213-c	23-cn	0;1-cn	0;1-cn	10;cn
BW678	<i>Rph15</i>	1-0;cn	10;cn	12	1-0;cn	1-0;cn	21cn	0;cn	0;cn	0;cn	33-c	33-	3	3-2cn	21c	3-2	1-0;cn	0;1-cn	10;cn
BW679	<i>Rph15</i>	1-cn	12cn	21	1-0;cn	10;cn	10;cn	0;1-cn	0;cn	0;cn	33-c	33-	3	3-2	213-	3-2	1-0;cn	0;1-cn	10;cn
BW680	<i>Rph15</i>	11-cn	1cn	12cn	120;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	33-c	3	3	3-2	33+	3-2	12-cn	1-0;cn	10;cn
BW681	<i>Rph15</i>	11-c	10;cn	10;cn	1-0;cn	0;1-cn	10;cn	0;cn	0;cn	0;cn	33-	33+	33-	3-2	21cn	3-2	1-0;cn	1-0;cn	10;cn
BW688	<i>Rph15</i>	1-0;cn	00;	21cn	1-0;cn	0;1-cn	1cn	0;cn	0;cn	0;cn	33-c	33-	33-	23-cn	21cn	213-	1-0;cn	0;1-cn	0;1cn
BW689	<i>Rph15</i>	1-0;cn	0;1-cn	10;cn	1-0;cn	1-0;cn	1cn	0;cn	0;cn	0;cn	3c	33+	33-	23-c	21cn	213-	0;1-cn	1-0;cn	10;cn
BW690	<i>Rph15</i>	1-0;cn	10;cn	12cn	1-0;cn	1-0;cn	1cn	0;cn	0;cn	0;cn	3-2	3-	33-	23-	21cn	23-1	1-0;cn	0;1-cn	10;cn
BW692	<i>Rph15</i>	1-0;cn	10;c	10;cn	1-0;cn	0;1-cn	10;cn	0;cn	0;cn	0;cn	3-2c	33-	3-	213-c	213-c	23-cn	1-0;cn	1-0;cn	10;cn
BW693	<i>Rph15</i>	1-0;cn	10;cn	0;1-cn	1-0;cn	0;1-cn	10;cn	0;cn	0;cn	0;cn	3-c	3-	33-	23-	21cn	23-cn	1-0;cn	1-0;cn	1cn
BW694	<i>Rph15</i>	1-cn	10;cn	10;cn	1-cn	0;1-cn	1cn	0;1-cn	0;cn	0;cn	33-c	33-	3-	23-c	21cn	21cn	1-0;cn	1-0;cn	10;cn
BW695	<i>Rph15</i>	1-cn	1cn	10;cn	10;cn	10;cn	1cn	0;1-cn	0;cn	0;cn	33+c	33+	3-2	33-	21c	21cn	1-0;cn	1-0;cn	12cn
BW696	<i>Rph15</i>	1-0;cn	1-0;cn	1cn	1-0;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	33-c	3	33-	23-c	21cn	3-2	1-0;cn	0;1-cn	12cn
BW699	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	1-0;cn	0;1cn	0;cn	0;cn	0;cn	33-c	3-	3	23-c	21cn	3-2	1-0;cn	1-0;	1-cn
BW700	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	23-1	10;cn	1-0;cn	0;cn	0;cn	3-2c	3	3	213-cn	21cn	213-	12-cn	0;1-cn	10;cn
BW702	<i>Rph15</i>	1-0;cn	10;cn	12cn	1-0;cn	10;cn	1cn	0;cn	0;cn	0;cn	3-2	33-	3	213-	21cn	21cn	12-cn	1-0;cn	10;cn
BW703	<i>Rph15</i>	1-0;cn	10;cn	1cn	1-0;cn	1-0;cn	1cn	0;cn	0;cn	0;cn	33-c	33-	33-	213-c	21cn	21cn	1-0;cn	1-0;cn	10;cn
BW704	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	1-0;cn	1cn	0;cn	0;cn	0;cn	3c	33-c	3-	213-c	21cn	213-	1-0;cn	10;cn	0;1cn
BW705	<i>Rph15</i>	1-0;cn	1-0;cn	10;cn	1-0;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	33-	33-	3	23-	21cn	21cn	1-0;cn	1-0;cn	0;1-cn

Appendix Table 6. Raw infection types¹ of 50 Bowman introgression lines postulated to carry *Rph15*.

BW Line ²	Gene ³	Isolates of <i>Puccinia hordei</i>																	
		98-20			94-8			BRS 76-12			90-3			Neth 28			92-7		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
BW706	<i>Rph15</i>	1-0;cn	0;1-cn	1-0;cn	1-cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;cn	33-	3-	33-	12-cn	21cn	23-cn	1-0;cn	0;1-cn	10;cn
BW707	<i>Rph15</i>	1-0;cn	0;1-cn	10;cn	1-cn	0;1-cn	1cn	0;cn	0;cn	0;cn	3-3	23-	3-2	12-cn	21cn	21cn	0;1-cn	0;1-cn	10;cn
BW708	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	1-0;cn	1cn	0;cn	0;cn	0;cn	33-	3-	3-2	12-cn	21cn	21cn	1-0;cn	0;1-cn	12cn
BW710	<i>Rph15</i>	1-0;cn	1-0;cn	10;cn	1-0;cn	0;1-cn	0;1cn	0;cn	0;cn	0;cn	3-2	33-	3-	12-cn	21cn	213-	1-0;cn	0;1-cn	0;1cn
BW711	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	3-2	3-	3-2	12-cn	21cn	213-	1-0;cn	0;1-cn	0;1-cn
BW712	<i>Rph15</i>			10;cn			1cn			0;cn			3			21cn			0;1cn
BW713	<i>Rph15</i>	1-0;c	10;cn	10;cn	1-0;cn	10;cn	10;cn	0;cn	0;cn	0;cn	3-2	3-2	3-	23-c	21cn	213-cn	1-0;cn	0;1-CN	0;1-cn
BW714	<i>Rph15</i>	12c	120;cn	12cn	1-cn	10;cn	0;1cn	0;cn	0;cn	0;cn	33-	33-	3-2	3-2	213-c	213-cn	1-0;cn	1-0;cn	10;cn
BW715	<i>Rph15</i>	1-0;cn	10;cn	1cn	1-cn	1-0;cn	1cn	0;cn	0;cn	0;cn	33-	33-	3	12-cn	21cn	23-cn	1-0;cn	0;1-cn	10;cn
BW716	<i>Rph15</i>	1-0;cn	10;cn	12cn	1-0;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	3-2c	3-2	3-2	12-cn	21cn	213-	0;1-cn	0;1-cn	10;cn
BW717	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	1-0;cn	1-0;cn	0;cn	0;cn	0;cn	3-2c	3-	3-2	12-cn	21cn	21cn	0;1-cn	0;1-cn	10;cn
BW718	<i>Rph15</i>	1-0;cn	120;cn	10;cn	1-0;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	3-	33-	3-	12-cn	21cn	23-cn	0;1-cn	0;1-cn	10;cn
BW720	<i>Rph15</i>	1-0;cn	10;cn	12cn	1-0;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	23-c	3-	3-	12-cn	21cn	23-1c	0;1-cn	0;1-cn	10;cn
BW721	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	3-3	3-	3-2	12-cn	21cn	23-1c	0;1-cn	0;1-cn	12c
BW722	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	0;1-cn	1cn	0;cn	0;cn	0;n	3-2	3-	3-2	12-cn	21cn	3-2	0;1-cn	1-0;cn	
BW723	<i>Rph15</i>	1-0;cn	12cn	21cn	12cn	10;cn	12cn	0;cn	0;cn	0;cn	3-2	3-	3-2	3-2	213-c	3-	12-cn	1-0;cn	21cn
BW724	<i>Rph15</i>	1-0;c	1-0;cn	21	1-0;cn	12cn	10;cn	0;1-cn	0;cn	0;cn	3	3-	33-	3-2	213-c	3-2	12-cn	1-0;cn	10;cn
BW725	<i>Rph15</i>	12	10;cn	1cn	213-	21cn	10;cn	0;1-cn	0;cn	0;cn	33-	3	3-	3-2	23-	3-2	21cn	1-cn	12cn
BW729	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-0;cn	1-0;cn	0;1-cn	0;cn	0;cn	0;n	23-c	21c	3-	23-cn	21cn	23-cn	0;1-cn	1-0;cn	10;cn
BW730	<i>Rph15</i>	1-0;cn	10;cn	12cn	1-0;cn	10;cn	10;cn	0;cn	0;cn	0;cn	3-2	3-	3-2	23-1cn	21cn	3-2	1-0;cn	0;1-cn	1cn
BW733	<i>Rph15</i>	1-0;cn	10;cn	1cn	1-0;cn	0;1-cn	10;cn	0;cn	0;cn	0;cn	3-3	3-2	33-	23-c	21cn	3-2	1-0;cn	0;1-cn	1cn
BW734	<i>Rph15</i>	1-0;cn	10;cn	1cn	1-0;cn	120;cn	1cn	0;cn	0;cn	0;cn	3-2	3-2c	3-2	3-2	213-cn	23-1	1-0;cn	0;1-cn	1-cn
BW697	<i>Rph15+?</i>	120;c	21c	0;12c	0;1-c	10;cn	0;1-cn	120;c	120;c	0;1c	3-2c	3-	33-	0;1c	0;1-	0;1-c	21c	10;	12c
BW691	<i>Rph15+?</i>	21c	10;cn	0;1-	10;c	0;1-c	0;1-	12c	10;c	0;1-	3-2c	23-c	23-1c	213-c	10;c	0;12c	10;c	120;c	0;1-c
BW709	<i>Rph15+?</i>	1-0;cn	1-0;cn	1cn	1-0;cn	1-0;cn	0;1-cn	0;cn	0;cn	0;cn	23-c	23-c	23-1c	12-cn	21cn	21cn	1-0;cn	0;1-cn	10;cn

¹Infection types were scored using the original 0-4 rating scale developed by Mains (1930) with the additional notations of "+" and "-" to indicate larger or smaller size variations of classically described uredinia (Levine and Cherevick, 1952) and "c" and "n" to indicate the presence of chlorotic or necrotic reactions (Moseman and Roane, 1959), respectively. Complex ITs were recorded when more than one IT was observed on the surface of a single leaf. For example, an IT of 213- indicates that all three pustule types exist on the same leaf and that the "2" type pustules were the most common, followed by the "1" type, with "3-" type pustules being the least common. Infection types from "0;" to "0;12" are classified as highly resistant ("R", green cells), infections types from "12" to "23-c" are classified as moderately resistant ("M", yellow cells), and infection types from "23-" to "33+" are classified as susceptible ("S", red cells) (Steffenson et al., 1993).

² BW lines are the 95 Bowman leaf rust resistance introgression lines.

³ The *Rph* gene retained in each leaf rust resistant introgression line.

Asterisks indicate the introgression line selected to represent that *Rph* gene in the differential host set.

Appendix Table 7. Raw infection types¹ for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.

Line	Gene Identity	Isolates of <i>Puccinia hordei</i>																	
		BR (Octal 11)			ARG231			Aus 220			Race 4			Ger 7			WGCC		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
Bowman	NA	3	33-	3	3	33-	3	3	33-	3	33+	3+	3	33+	33+	3	33-	33+	3
Sudan	<i>Rph1.a</i>	23-1	3-2c	3-2	23-1	3-21	3-0;	1-0;	0;cn.	0;1	0;1-	0;1c	0;1-	0;1-	0;1-	0;1	0;12	0;12	0;2
Bolivia	<i>Rph2.r+Rph6.f</i>	0;1-	00;1c	0;1	0;1-n	00;cn	0;1-	0;12	0;1c	0;1	0;1-	00;1-cn	0;	0;1	120;	0;1c	0;	0;1	0;1
Juliaca	<i>Rph2.l</i>	0;1		0;12	10;c		0;1-	23-1		0;1	0;1-		0;1	120;		213-	0;1-c		0;12c
Peruvian	<i>Rph2.b</i>	213-	0;1c	0;1	21	0;12c	0;12	23-1	0;12c	0;12	210;	0;12c	120;	213-c	210;	23-1	10;	210;c	0;12c
PI 531841	<i>Rph2.y</i>	0;1		0;1-	10;	0;1-c	0;n	120;c	0;1cn	0;n	0;1-1		0;1-	12c		120;c	0;1-		0;1-
Reka 1	<i>Rph2.t+Rph19.ah</i>			0;n	213-c	120;	0;n	23-c	0;1-n	0;1-	0;1-	1-0;c	0;1	0;1-	0;1-	120;c	0;13-	0;12c	0;12
Estate	<i>Rph3.c</i>	0;1-c	0;12c	0;	0;1-	00;1	00;	0;1-c	0;1-c	0;1	00;1-	0;1c	00;	00;1-	00;2	00;2	00;1-	0;12c	00;2
PC249	<i>Rph3.aa</i>	00;		00;1	00;n		00;	0;cn		00;	00;	00;n	00;	00;	00;	00;	00;n	0;2n	00;2
PI 466324	<i>Rph3+?</i>	0;1-	00;	0;1	0;1-	0;cn	10;	120;c	0;1c	0;1		00;2cn	00;	10;	0;1c	0;	0;1-	0;c	0;1c
Q21861	<i>Rph3</i>	1-0;	00;2	0;1	10;	00;2n	0;1-	120;	00;n	0;1-			0;1		00;n	23-	0;1-	0;2n	0;1c
Batna	<i>Rph4</i>	21		3-2	21c		213-	10;		0;1	10;		120;	3-2		3	210;		0;1c
Gold	<i>Rph4.d</i>	12	23-c	3-2c	21	23-c	23-1	0;1-	0;12c	0;1	10;	0;1c	120;	3-2	3-2	3-2	10;2	0;12	0;1
Lechtaler	<i>Rph4.d</i>	21	3-2	23-	21	210;c	23-1	0;1c	0;1-c	0;1c	0;1c	12c	0;12c	33-	33-	3-2	213-	120;c	0;1-c
Magnif 102	<i>Rph5.e</i>	0;1-n	0;1-cN	0;1n	11-cN	10;c	23-c	23-	3-2c	3	213-c	23-c	213-	1c	12c	120;c	10;2c	3-2	3
Bolivia	<i>Rph2.r+Rph6.f</i>	0;1-	00;1c	0;1	0;1-n	00;cn	0;1-	0;12	0;1c	0;1	0;1-	00;1-cn	0;	0;1	120;	0;1c	0;	0;1	0;1
PI 531990	<i>Rph3</i>	0;1-	00;1-	0;	0;1-	3	00;1	00;1-	0;1c	0;c	00;1-n	00;2c	00;c	00;1-	00;2	00;2	00;1-	0;12c	0;12c
Tunisian 17	<i>Rph5+Rph7</i>	00;1-	00;	00;		00;	00;	00;1-	00;	00;	00;	00;1	00;	00;	00;	00;	00;	00;1-	00;
Tunisia 25	<i>Rph5+Rph7</i>	00;1-		00;1	00;n		00;	00;1		00;	00;1-		00;	00;		00;	00;		00;
Tunisia 29	<i>Rph5+Rph7</i>	00;		00;			00;	00;		00;	00;		00;	00;1-		00;	00;1-		00;
Cebada Capa	<i>Rph7.g</i>	00;n	00;cn	0;n	0;1-c	0;1-cn	00;n	00;1-	00;	00;	0;1-	0;1-c	0;	0;1-	0;1-c	0;1-c	00;1-c	00;n	00;
Egypt 4	<i>Rph8.h</i>	210;n	0;1cn	0;1n	3-2	213-c	3-	23-c	0;12c	3-	3+3	3	3	0;1-cn	0;1n	0;1-c	3-2c	23-c	23-c
Hor2596	<i>Rph9.i</i>	0;1-cn	10;cn	0;1N	0;cN	0;1-cn	0;n	0;cN	0;1-cn	0;1c	0;1-n	0;1c	0;1-c	1-0;cN	10;c	0;1c	00;1-N	0;1c	0;1-c
PI 466247	<i>Rph9</i>	21	0;1c	213-	12	0;1c	213-		00;2n	00;1		00;n	00;1	00;	00;	00;2	21c	00;2n	0;12
Quinn	<i>Rph2.q+Rph5.e</i>	0;1-n	00;1	0;1	10;	0;12c	0;1	3-3	210;	0;1	0;1-		0;1	0;1-	120;c	0;1		0;12c	0;12c

Appendix Table 7 continued. Raw infection types¹ for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.

Line	Gene Identity	Isolates of <i>Puccinia hordei</i>																	
		BR (Octal 11)			ARG231			Aus 220			Race 4			Ger 7			WGCC		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
Triumph	<i>Rph9.z</i>	0;1-	0;1-cn	10;	1-0;c	0;1-cn	0;1	0;1-c	0;1-cn	0;1-c	10;c	10;c	0;1-c	0;1-c	0;cn	0;1-	11-c	0;1-cn	0;1-c
Quinn	<i>Rph2.q+Rph5.e</i>	0;1-n	00;1	0;1	10;	0;12c	0;1	3-3	210;	0;1	0;1-		0;1	0;1-	120;c	0;1		0;12c	0;12c
Clipper BC 8	<i>Rph10.o</i>	120;	10;c	213-	1-0;c	12c	213-	11-c	0;12c	10;c	213-c	23-	3-2	23-	23-	3-2	12c	23-c	0;1c
Clipper BC 67	<i>Rph11.p</i>	120;	213-c	21	1-0;c	21c	21c	11-c	3-2c	23-c	21c	21c	12c	21c	213-c	21c	21c	23-c	23-c
PI 531849	<i>Rph13.x</i>	0;1-	0;12	00;1	0;1-	0;1c	0;1	00;1-	00;1-n	0;1	0;1	0;1-c	120;	0;1-	0;1-	0;1-	0;1-	0;1-n	0;1
PI 531901-1	<i>Rph14.ab</i>			0;1n			23-	12c		21c			213-			0;cn			10;c
HS584	<i>Rph15+?</i>	1-1	0;1-cn.	0;1-	1-c	0;1-cn.	0;12	0;1-cn		0;n	1-0;cn	0;1-c	10;c	0;1-cn	1-cn	0;1-cn	1-0;cn	0;n	0;
PI 282610	<i>Rph15?</i>	11-	0;1-cn	0;1-	0;cn	0;cn	0;n	0;1-2cn	0;1-cn	00;	00;n	00;2n	00;	0;1-n	0;1-	00;1	00;1-n	0;1-cn	0;
PI 296841	<i>Rph15</i>	0;1-	0;cn.	0;cn	0;cn	0;cn.	0;n	0;cn	0;n.	0;n		0;cn	0;cn	0;cn	0;cn	0;cn		0;cn	0;n
PI 354923	<i>Rph15</i>	0;cn		0;cn	0;cn		0;1-cn			0;cn	1-n		1-cn			0;cn			0;cn
PI 354923	<i>Rph15</i>	0;cn	0;cn	0;cn	0;cn	0;cn	0;1-cn		0;cn	0;cn	21c	0;1-cn	1-cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;cn
PI 354926	<i>Rph15</i>	0;1-n	0;cn	0;	0;n	0;cn	00;	0;1-cn	0;cn	00;	00;1-n	00;n	00;	0;cn	0;cn	0;1-c	00;1-	00;1-n	0;
PI 354928	<i>Rph15</i>		0;cn	00;		00;1-	0;1-n		0;cn	0;	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;n
PI 354932	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	0;cn	0;cn	0;cn		0;cn	1-0;cn	0;cn
PI 354937	<i>Rph15</i>	0;1-cn	0;cn	0;cn	1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	1-0;cn	0;cn	0;cn
PI 354940	<i>Rph15</i>	1-0;cn		0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn
PI 355434	<i>Rph15</i>	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn
PI 355447	<i>Rph15</i>	1-c	0;1-cn	0;cn	1-cn	0;cn	0;1-	0;cn	0;cn	0;cn	0;1-cn	0;1-cn	0;1-cn	0;cn	1-cn	0;cn	1-0;cn	0;cn	0;cn
PI 391000	<i>Rph15</i>		0;cn	0;cn		0;cn	0;cn	0;cn	0;cn	0;cn	1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;cn
PI 391002	<i>Rph15</i>	1-0;	0;1-cn.	0;cn	0;1-cn	0;1-cn	0;cn	12c	0;1-c	0;1c	0;1-n	0;1cn	0;cn	1-0;	21c	1-cn		120;c	0;c
PI 391004	<i>Rph15+?</i>	0;1-n		0;1	1-0;cn		0;1-	210;c		0;1c	0;1-n	0;1cn	0;cn	0;1-c	120;c	0;1c	1-0;cn	210;c	0;c
PI 391007	<i>Rph15+?</i>	0;1-		0;	0;n		0;	00;1-n	00;n	0;	00;1-n		0;	0;1-n		0;c	00;n	0;1-cn	0;
PI 391024	<i>Rph15</i>	00;1-n		0;	0;1-n		0;	0;1-cn		0;	0;1-n	00;2cn	00;	0;1-cn	0;cn	0;1-c	0;1-n	0;1-n	0;
PI 391044	<i>Rph15</i>		0;1-cn	0;cn		0;cn	0;1-cn		0;cn	0;cn	12c	1cn	10;cn	0;1-cn	0;cn	0;1-cn	1-0;cn	0;cn	0;cn
PI 391045	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn		0;cn	0;cn	0;cn	0;cn	0;cn		0;cn	0;cn

Appendix Table 7 continued. Raw infection types¹ for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.

Line	Gene Identity	Isolates of <i>Puccinia hordei</i>																	
		BR (Octal 11)			ARG231			Aus 220			Race 4			Ger 7			WGCC		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
PI 391049	<i>Rph15</i>		0;cn	10;c		0;cn	23-	21c	0;12c	3-2	21c	10;cn	3	0;1-cn	0;cn	10;cn	0;1cn	0;cn	21c
PI 391069	<i>Rph15</i>	0;1-cn	0;cn	0;cn	1-0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	1-0;cn	10;cn	0;cn	0;1-cn	0;cn	0;cn	11-cn	0;1-cn	0;cn
PI 391072	<i>Rph15</i>		0;1-cn	0;cn		0;1-cn	0;cn		0;1-cn	0;cn	10;cn	10;cn	0;cn		0;cn	0;1-cn	0;1-cn	0;1-cn	0;cn
PI 391087	<i>Rph15</i>		0;cn	0;cn		0;cn	0;n		0;cn	0;cn	0;1-n	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;cn
PI 391089	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;1-cn	0;1cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn
PI 391121	<i>Rph15+?</i>	0;1-c	0;1-	0;1-c	0;1-c	0;1-c	0;1	0;1-c	0;1-c	0;	0;1-	0;12c	0;	0;1-	10;c	0;1-c	1-0;	0;1c	0;c
PI 405154	<i>Rph15+?</i>			0;	0;1-		0;	0;1-		0;	00;1-n		00;	00;n		00;			0;
PI 405179	<i>Rph15</i>		00;1-n	0;		00;1-n	0;		00;cn	0;	00;1-	0;12cn	0;	00;	0;cn	0;c	00;	0;1c	0;
PI 405194	<i>Rph15</i>		0;cn	0;n		0;cn	0;1-cn	0;cn	0;cn	0;cn	1-0;cn	0;1-cn	0;12c	0;1-	0;1-cn	0;cn	0;1cn	0;12cn	0;n
PI 405201	<i>Rph15</i>		0;cn	00;	0;1-cn	0;1cn	0;	0;1-cn	0;cn	0;	00;1-n	00;2n	00;n	00;1-n	0;cn	0;1-c	00;1-	0;cn	0;
PI 405210	<i>Rph15</i>	0;1-n	00;cn	00;	0;1-n	0;n	0;	0;cn	00;n	0;	00;1-n	10;cn	0;	00;1-n	0;cn	0;1-	00;1-	00;cn	0;
PI 405219	<i>Rph15</i>	00;n		0;	0;n		0;	0;1-cn	00;1-n	0;	00;n		00;	00;n		00;1-	00;1-		0;
PI 405220	<i>Rph15</i>	0;cn	0;cn	0;cn		0;cn	0;n		0;cn	0;cn	0;1cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn
PI 405227	<i>Rph15</i>	1-0;cn	0;cn	0;cn	0;n	0;cn	0;1-cn	0;cn	0;cn	0;cn	1-cn	0;1-cn	0;1-cn	0;cn	0;1-cn	0;cn	1-0;cn	0;cn	0;cn
PI 405233	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	0;1-cn		0;cn	1-0;cn		0;cn	1-0;c		1-0;cn			0;cn
PI 405235	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;1-cn	0;1c	0;1-cn	0;cn	0;1-cn	0;cn	1-0;cn	0;cn	0;cn
PI 405236	<i>Rph15</i>	1-c	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;1-cn	0;1-cn	0;cn	1-0;cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn
PI 405277	<i>Rph15</i>	1-0;	0;cn	0;cn		0;1-cn	0;1-cn		0;cn	0;cn	12c	0;1cn	12cn	0;1-cn	0;cn	0;1-cn	0;1-cn	0;cn	0;cn
PI 405289	<i>Rph15</i>	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;1c	0;cn	0;1-cn	0;cn	1-cn	0;cn	0;1c	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;cn
PI 405298	<i>Rph15</i>	1-	0;1-c	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;1-c	0;cn	0;cn		0;cn	0;cn
PI 405305	<i>Rph15</i>	0;1-	0;cn	10;	10;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;1-c	1-c	0;cn	10;c	0;1-cn	0;cn	0;1-
PI 405308	<i>Rph15</i>	1-0;c	0;1-n	0;1-cn	1-0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	1cn	0;1-c		1-cn	0;cn	0;1-cn	0;cn	0;cn
PI 405311	<i>Rph15</i>	1-0;cn		0;cn	1-cn		0;1-cn	0;cn		0;cn	0;1-cn	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn
PI 405332	<i>Rph15</i>	0;cn	0;1cn	0;cn		0;1c	0;n		0;1-cn	0;cn	213-c	0;cn	0;cn	0;1-cn	120;c	0;cn	21c	0;1-cn	0;cn
PI 405335	<i>Rph15</i>	00;1-cn	0;1-n	0;cn	12	0;cn	0;1-cn		0;cn	0;cn	10;c	0;cn	0;1cn	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;cn

Appendix Table 7 continued. Raw infection types¹ for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.

Line	Gene Identity	Isolates of <i>Puccinia hordei</i>																	
		BR (Octal 11)			ARG231			Aus 220			Race 4			Ger 7			WGCC		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
PI 405338	<i>Rph15</i>	0;1	0;cn	0;1cn	0;cn	0;cn	00;	0;cn	0;cn	00;	0;cn	00;2n	0;n	0;cn	0;cn	0;1	0;cn	0;cn	0;1
PI 405341	<i>Rph15</i>	0;1-cn	00;1-	0;		00;n	0;		00;cn	00;n	00;1-n	0;cn	00;	0;cn	0;cn	0;	0;1-n	0;cn	0;
PI 405354	<i>Rph15</i>		0;cn	0;cn	0;cn	0;cn	0;n	0;1-cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	00;1	0;cn	0;1-	0;12c	0;cn
PI 405399	<i>Rph15</i>	0;cn	0;cn	0;cn	1-0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1-n	0;cn	10;cn	0;cn	0;cn	0;1-cn	0;1-cn	0;cn	0;cn
PI 466245	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;cn	0;cn
PI 466470	<i>Rph15+?</i>	120;c	0;12	0;12		0;1c	0;1-		0;1-	0;	0;1	0;1c	0;1c	0;1-c	0;1-c	0;1c	0;1c	0;1c	0;c
PI 466483	<i>Rph15+?</i>	0;12	0;1	00;	120;c	0;1-c	0;1	0;1-c	0;1-c	00;	0;1c	0;1c	0;1c	0;1-c	0;1c	0;1c	0;1c	0;c	0;
Columbia	?	23-	3-21	3	3-2	3-21	3	33-	33+	3	33-	3	3	3-2	33+	3	33-	33+	3
Emir/H. bulbosum	?	00;n		00;	00;n		00;	0;n		00;	00;		0;	00;		00;	00;		00;
HS580	<i>Rph15</i>		00;1-n	0;1-cn	0;1-cn	00;n	0;1-cn	0;cn	00;n	0;cn	0;1-cn	00;2	10;cn	1-0;cn	0;cn	0;1-cn	1-cn	00;1-n	0;cn
PI 355445	<i>Rph15</i>		0;cn	0;1-		0;1-cn	0;cn	0;cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;1-cn	1-0;cn	0;cn	0;n
PI 405154	<i>Rph15+?</i>		00;1-			00;n			00;cn		00;n	00;1-		00;	0;1-cn		00;n	0;1n	
PI 405169	<i>Rph9</i>	21		10;	12	0;1	0;1	0;1c	10;c	0;cn	0;1-	0;1	0;c	0;1-c	0;1c	0;1-cn	0;1-c	0;1c	0;1-
PI 405215	<i>Rph15+?</i>		0;cn	0;cn		0;1-cn	0;cn		0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;1cn	0;cn	0;cn	0;cn	0;cn
PI 466373	<i>Rph15+?</i>	0;1-	0;cn	0;	0;1-	0;cn	0;n	0;1c	0;1-c	0;1	00;1n	00;cn	00;n	0;1-c	0;1c	0;cn	00;1-n	0;12c	0;1-
PI 531901-2	?			0;			00;			0;c	0;1-		0;			00;1			0;1c
Tunisia 33	<i>Rph7+?</i>	00;1-		00;1	00;		00;1			00;	00;		00;2			210;c			0;
Vada/H. bulbosum	?			10;c	120;c		10;			10;			0;1			0;1c			0;1-c

Appendix Table 7 continued. Raw infection types¹ for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.

Line	Gene Identity	Isolates of <i>Puccinia hordei</i>																	
		98-20			94-8			BRS 76-12			90-3			Neth 28			92-7		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
Bowman	NA	3+3	33+	3	33-	33+	3	3	3	3-	3+3	33+	3	33+	3	3-	3+3	33+	3
Sudan	<i>Rph1.a</i>	33-	33-	3-2	3-	33-	3	33-	33-	3-	33+	3	3	33-	3-	3	3-2	3-	3
Bolivia	<i>Rph2.r+Rph6.f</i>	23-	23-0;	213-	213-	23-0;	23-1	210;	3-20;c	0;12c	33+	3-	3	3-3	33-	33-	3	33-	3
Juliaca	<i>Rph2.l</i>	213-		23-1	213-		210;	210;		0;12	3-3		3	3-2		3	3		3
Peruvian	<i>Rph2.b</i>	3-21c	21c	23-1	3-2c	210;c	210;	213-c	120;c	120;c	320;	33+	3	33-	3-2	33-	33-	33-	3-
PI 531841	<i>Rph2.y</i>	213-	23-c	213-	21	33-	213-	10;	210;	0;1	3	3-	23-1	10;	12	210;	10;		210;c
Reka I	<i>Rph2.t+Rph19.ah</i>	3-2	3+	23-	23-1	33-	210;	3-2	33+	120;	3-3	33-	3	3-	33-	33-	3	33-	3
Estate	<i>Rph3.c</i>	0;	00;1c	00;	0;1-c	00;1-	00;1	33-	3-2	3-	0;12	23-1c	0;12c	213-c	2c	213-c	3-2c	23-c	3-2c
PC249	<i>Rph3.aa</i>		00;n	00;1			00;		33-	3-2c	0;1-		0;12c	3-		3-	3		3
PI 466324	<i>Rph3+?</i>	0;1	00;1-	0;12	00;1-c	0;1-	0;1-	0;1-c	00;1-n	0;1	213-	23-c	3-2c	0;1-	2c	3-2c	0;1	0;1c	0;12c
Q21861	<i>Rph3</i>	21	00;2	21	213-	00;2n	12	33-	33-	120;	23-1	0;1c	3-2	21		3-	33-	33-	3
Batna	<i>Rph4</i>	33-		3	3-2		3-	3-2		3	3-2	33-	3	10;c	21	210;	3+3	33-	3
Gold	<i>Rph4.d</i>	23-1	3-	3-2	3-2c	23-c	3	3-2	33-	3-2	33-	3	3-	0;12c	120;c	0;12	3-2	33-	3-
Lechtaler	<i>Rph4.d</i>	3-2c	3	3-2	3-2	33+	3-2	3-2	23-	3-	3-2	33-	3-2	0;1	10;	0;12c	33-	33-	3
Magnif 102	<i>Rph5.e</i>	12c	10;c	120;	10;c	0;1c	0;1	23-c	12c	23-c	23-1c	3-2c	33-	33-	33-	3	33-	33-	3
Bolivia	<i>Rph2.r+Rph6.f</i>	23-	23-0;	213-	213-	23-0;	23-1	210;	3-20;c	0;12c	33+	3-	3	3-3	33-	33-	3	33-	3
PI 531990	<i>Rph3</i>	00;n	00;1	00;1	0;1-c	00;1-c	00;2	3-2c	33-	3	210;	120;c	3-21	210;c	3-	33-	33-	33-	3-2
Tunisian 17	<i>Rph5+Rph7</i>	0;1-	0;1	00;1	00;	00;1	00;	0;1c	00;	00;1		30;	3-21	3-2	33-	3		33-	3
Tunisia 25	<i>Rph5+Rph7</i>	0;1-cn	00;1n	00;1	0;1-c	21c	0;1-	10;c	210;	00;	23-1c	33-	3-0;		33-	3-	3	33-	3-
Tunisia 29	<i>Rph5+Rph7</i>	00;	00;	00;	00;	00;n	00;	00;	0;n	00;	3-2c	23-	3-21		3-	3-		3	3
Cebada Capa	<i>Rph7.g</i>	33-	3-20;	3-0;	0;1-cn	0;1-cn	0;1-	0;1-c	00;cn	0;n	120;c	213-c	3	33-	33-c	3	33-	33-	3-
Egypt 4	<i>Rph8.h</i>	3-c	3-2c	3	3-2c	21c	3-2	33-	23-c	3-2	33+	3	33-	33-c	33+	33-	3	33+	3
Hor2596	<i>Rph9.i</i>	10;c	10;c	120;c	3-2c	23-c	3-2	10;c	0;1c	10;c	3-2	33+	3	120;c	10;c	210;c	23-1c	23-	3-
PI 466247	<i>Rph9</i>	120;c	12c	120;c	120;c	210;	23-1c	120;c	210;c	23-1c	3-2	3-	3-2	33-	33+	3	33+	33-	3-
Quinn	<i>Rph2.q+Rph5.e</i>	120;	00;1-	0;12	210;c	0;1-c	0;1	3-2	3-2	0;12	23-1	33-	3-21	3-2	33-	33-	33+	3	3-

Appendix Table 7 continued. Raw infection types¹ for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.

Line	Gene Identity	Isolates of <i>Puccinia hordei</i>																	
		98-20			94-8			BRS 76-12			90-3			Neth 28			92-7		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
Triumph	<i>Rph9.z</i>	10;c	120;c	0;12	3-2	33-	3-	0;1-c	0;1-c	0;1	33-	3	3	23-c	33+	3	3-2c	3	3
Quinn	<i>Rph2.q+Rph5.e</i>	120;	00;1-	0;12	210;c	0;1-c	0;1	3-2	3-2	0;12	23-1	33-	3-21	3-2	33-	33-	33+	3	3-
Clipper BC 8	<i>Rph10.o</i>	12c	10;	120;c	12c	10;c	10;	23-1c	120;c	23-1	23-c	23-c	3-2c	33+	3	3	33-	33+	3
Clipper BC 67	<i>Rph11.p</i>	21c	23-c	23-1	3-2c	23-c	213-	21c	21c	23-1	23-1c	23-c	3-2c	3-c	3-2c	3-2c	23-c	23-c	3-
PI 531849	<i>Rph13.x</i>	3-2	213-	23-1	23-c	213-	3-2	10;c	0;1	0;1	0;1c	0;1c	210;	0;1c	0;1c	120;	0;1	0;1c	12
PI 531901-1	<i>Rph14.ab</i>	10;		210;c	21		210;c	21c		10;c			3-			23-c			3-2
HS584	<i>Rph15+?</i>	10;cn	10;cn	0;1	1-0;cn	1-0;cn	10;cn	0;cn	0;cn	0;cn	2c	2c	3-2c	10;c	1c	210;c	10;c	2c	0;1c
PI 282610	<i>Rph15?</i>	21c	10;cn	0;1	10;c	10;cn	0;1-n	11-c	0;1-cn	0;1-	33+	3-	3	3-2	33-	21cn	3-2	33-	3-2
PI 296841	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;cn	0;cn	0;cn	0;cn	0;n	3-2	33-	33-	12-cn	21cn	21cn	1-0;cn	10;cn	0;1c
PI 354923	<i>Rph15</i>			0;1-cn		0;1-n	10;cn		0;cn	0;cn			3-c		21cn	3-2n			0;1-cn
PI 354923	<i>Rph15</i>	0;1-cn	0;1-cn	0;1-cn	1-0;cn	0;1-cn	10;cn	0;cn	0;cn	0;cn	3-2c	33-	3-c	23-cn	21cn	3-2n	1-0;cn	1-0;cn	0;1-cn
PI 354926	<i>Rph15</i>	0;1-cn	0;1-cn	0;1	0;1-c	0;1-c	0;1-	0;1-cn	00;cn	0;	213-c	12c	3-2	213-c	21c	213-c	10;c	0;1-cn	0;1
PI 354928	<i>Rph15</i>	0;1-cn	0;1-cn	0;cn	0;1-c	0;1-c	0;cn	0;cn	0;cn	0;	213-c	23-c	3-2	12-cn	21c	210;c	0;1-cn	10;c	0;1
PI 354932	<i>Rph15</i>	1-0;cn	10;cn	0;1-cn	1-0;cn	0;1-cn	10;cn	0;cn	0;cn	0;cn	33+	33-	3-2c	21cn	21cn	21cn	1-0;cn	0;1-cn	0;1-cn
PI 354937	<i>Rph15</i>	1-0;cn	10;cn	0;1cn	1-0;c	1-0;cn	0;1-cn	0;cn	0;cn	0;	3-	23-c	3-	23-1cn	21cn	21cn	1-0;cn	10;cn	0;1cn
PI 354940	<i>Rph15</i>	1-0;c		0;1-cn	1-0;c	0;1-cn	0;cn	0;cn	0;cn	0;cn	3	3-2c	3-	12-cn	21cn	12cn	1-0;cn	1	0;1-cn
PI 355434	<i>Rph15</i>	0;1-cn	10;cn	0;1cn	1-0;c	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	23-c	33-	3-c	12-cn	21cn	21cn	1-0;cn	1cn	10;cn
PI 355447	<i>Rph15</i>	1-0;c	0;1-cn	0;1-cn	1-0;	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	3-3	3-	3-2	23-cn	21cn	23-cn	1-0;cn	0;1-cn	0;1cn
PI 391000	<i>Rph15</i>	0;1-cn	0;cn	0;1-cn	1-0;c	0;1-cn	0;1-cn	0;cn	0;cn	0;cn	3-2c	3	3	12-cn	23-c	3-2n	1-0;cn	1-0;cn	0;1-cn
Pi 391002	<i>Rph15</i>	120;	0;1-cn	0;1c	0;1	10;	0;1-	10;c	0;1-c	0;c	213-c	213-	3-2	120;c	2c	210;c	0;1-c	10;	0;1c
Pi 391004	<i>Rph15+?</i>	10;c	0;1-cn	0;1c	10;	0;1-cn	0;1	0;1-c	10;c	0;1-	23-1c	3-2	213-	10;c	10;c	210;c	0;1-	0;1	
PI 391007	<i>Rph15+?</i>	0;1c	0;1-	0;1-cn	0;1c	0;1-	0;1-	0;cn	0;1-cn	0;	213-c	21c	23-c	213-	21	210;		10;n	0;1-
PI 391024	<i>Rph15</i>	0;1-c	0;1-cn	0;1	0;1-c	0;1-c	0;1	0;1-cn	00;cn	0;1-	23-1c	23-c	3-2	23-c	21cn	210;	1-0;cn	0;1cn	10;c
PI 391044	<i>Rph15</i>	1-0;cn	10;cn	10;cn	1-c	1-0;cn	10;cn	0;cn	0;cn	0;cn	3-2	3	3-	3-2cn	23-cn	3-2	1-0;cn	1cn	10;cn
PI 391045	<i>Rph15</i>	1-0;n	10;cn	0;cn	1-0;cn	0;1-cn		0;cn	0;cn	0;cn	3	3-	33-	21cn	21cn	21cn	1-0;cn	10;c	0;1-

Appendix Table 7 continued. Raw infection types¹ for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.

Line	Gene Identity	Isolates of <i>Puccinia hordei</i>																	
		98-20			94-8			BRS 76-12			90-3			Neth 28			92-7		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
PI 391049	<i>Rph15</i>	11-c	10;cn	23-	213-	1-0;cn	21	1-0;cn	10;c	10;c	33+	33-	3-	3-2c	21cn	3-	1-0;cn	10;cn	3-
PI 391069	<i>Rph15</i>	120;cn	10;cn	0;1cn	10;c	1-0;cn	0;1-cn	0;cn	0;cn	0;cn	33-	3-	33-	3-cn	23-cn	23-cn	1-0;cn	10;cn	0;12cn
PI 391072	<i>Rph15</i>	1-0;cn	0;1-cn	10;cn	10;c	0;1-cn	0;1cn	0;1-cn	0;1-cn	0;cn	3-2	33-	33-	12-cn	21cn	23-cn	1-cn	1-0;cn	10;cn
PI 391087	<i>Rph15</i>	1-0;cn	0;cn	0;cn	1-0;cn	0;cn	0;cn	0;cn	0;cn	0;n	3-2c	3-	3-2	11-cn	21cn	21cn	0;1-cn	0;1cn	0;1-c
PI 391089	<i>Rph15</i>	0;1-cn	0;cn	0;cn	0;1-cn	0;1-cn	0;1cn	0;cn	0;cn	0;n	23-c	3-	3-	213-c	21cn	21cn	0;1c	0;1c	0;12c
PI 391121	<i>Rph15+?</i>	120;	10;c	0;1c	1-0;c	0;1-cn	0;1	10;c	10;c	0;1-	3+3	3-3	3	0;1-	0;1-c	0;1c	0;1c	0;1-cn	10;
PI 405154	<i>Rph15+?</i>	0;1-cn		0;1	10;	0;cn	0;1-	0;1-cn	0;cn	0;		23-c	213-	210;	2	210;			0;1c
PI 405179	<i>Rph15</i>	1-0;cn	0;1-cn	0;1-	0;1-c	0;1-c	0;1-	0;cn	00;cn	0;	3-2c	213-c		21cn	21c	213-c	0;1c	10;c	2
PI 405194	<i>Rph15</i>	1-	0;1-cn	0;1-cn	12-c	10;cn	10;cn	0;cn	0;cn	0;cn	23-c	3-2c	3-2	12-cn	21cn	23-cn	12-cn	2c	0;1cn
PI 405201	<i>Rph15</i>	0;1-cn	0;1-	0;cn	0;1-c	0;1-c	0;1-	0;1-cn	0;cn	0;	3-2c	23-c	3-2	21cn	21c	210;c	10;c	10;c	0;12c
PI 405210	<i>Rph15</i>	0;1-cn	0;1c	0;1	0;1-c	0;1-c	0;1-	0;1-cn	0;cn	0;	120;c	3-2	3-2	23-c	21c	210;c	0;1c	10;c	0;1
PI 405219	<i>Rph15</i>	0;1c		0;1-	0;1-	0;1-c	0;1-	0;cn	0;cn	0;	3-2c	33-	23-1	23-cn	21c	210;c	0;1c	0;1-	0;1c
PI 405220	<i>Rph15</i>	0;1-cn	0;cn	0;cn	1-0;cn	0;cn	0;1-cn	0;cn	0;cn	0;n	3-2c	3-2	3-	12-cn	21cn	210;c	0;1c	0;1	0;1c
PI 405227	<i>Rph15</i>	0;1-cn	0;1-cn	0;1-cn	10;cn	0;cn	0;1-cn	10;c	10;c	0;cn	23-c	23-c	3-2	21c	21cn	23-c	1-0;cn	1-0;cn	10;cn
PI 405233	<i>Rph15</i>	1-0;		10;cn			10;cn	0;cn		0;cn	33+	23-c	3-	23-c	21cn	23-cn	1-0;cn	0;1-n	1-0;cn
PI 405235	<i>Rph15</i>	10;cn	10;cn	10;cn		10;cn	120;cn	0;cn	0;cn	0;cn	3-2c	23-c	33-	21cn	21cn	21cn	1-0;cn	0;1-cn	10;cn
PI 405236	<i>Rph15</i>	10;cn		10;cn	1-0;c	0;cn	0;1-cn	0;cn	0;cn	0;cn	23-1c	23-c	3-	12-cn	12cn	21cn	0;1c	0;1-cn	0;1-cn
PI 405277	<i>Rph15</i>	1-0;cn	10;cn	10;cn	12c	10;cn	1cn	0;1-cn	0;cn	0;cn	33-	3-	3-	23-cn	3	3-n	1-0;cn	1-0;cn	12cn
PI 405289	<i>Rph15</i>	1-c	0;cn	10;cn	21c	0;cn	0;1cn	0;cn	0;cn	0;cn	213-c	21c	3-2c	21	12c	21c	1-0;cn	0;1-c	0;1-cn
PI 405298	<i>Rph15</i>	1-0;cn	0;1-cn	0;cn	0;1-cn	1-0;cn	0;1-cn	0;1-cn	0;cn	0;cn	3-2	3-2	3-	12-cn	21cn	12cn	1-0;cn	10;c	0;1-cn
PI 405305	<i>Rph15</i>	1-0;c	0;1cn	0;1c	10;c	10;cn	120;c	0;1-cn	0;cn	0;cn	3-2	23-c	3-c	12-cn	21cn	23-cn	1-0;cn	1-0;cn	213-c
PI 405308	<i>Rph15</i>	1-0;cn	0;1-cn	0;1-cn	1-0;c	0;1-cn	0;cn	0;cn	0;cn	0;cn	33-	3-	3-	3-2cn	213-cn	3-2n	1-0;cn	0;1-cn	0;1cn
PI 405311	<i>Rph15</i>	1-0;cn	0;1-cn	0;1-cn	0;1-c	0;1-cn	0;1cn	0;cn	0;cn	0;cn	33-	3-2	33-	23-cn	23-cn	3-2n	1-0;cn	0;1cn	12
PI 405332	<i>Rph15</i>	10;c	0;1c	0;cn	120;c	0;cn	0;cn	0;cn	12c	0;n	3-2c	3-2	3-	12-cn	21c	21cn	210;		0;1c
PI 405335	<i>Rph15</i>	0;1-cn	0;1-cn	0;1-cn	1-0;cn	0;1-c	0;cn	10;c	0;cn	0;1c	3-2	3-2c	3-2c	12-cn	21cn	213-c	0;1-cn	1-0;c	213-c

Appendix Table 7 continued. Raw infection types¹ for barley cultivar Bowman and the 95 leaf rust resistance gene donors across three experiments.

Line	Gene Identity	Isolates of <i>Puccinia hordei</i>																	
		98-20			94-8			BRS 76-12			90-3			Neth 28			92-7		
		Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3	Rep1	Rep2	Rep3
PI 405338	<i>Rph15</i>	120;c	0;cn	0;1	0;1-cn	0;cn	0;1c	0;cn	0;cn	0;1c	33+	33-	3	210;cn	21cn	3-2c	1-0;cn	10;	23-1c
PI 405341	<i>Rph15</i>	0;1-cn	0;1-	0;1-	0;1-c	0;1-c	0;1-	0;cn	00;cn	0;	213-c	3-2	3-21	210;c	21c	210;	0;1-c	0;1c	120;
PI 405354	<i>Rph15</i>	0;1-cn	0;1-cn	0;cn	10;c	1-0;cn	0;cn	120;c	0;cn	0;n	3-	3-2	33-	12-cn	21cn	21cn	1-0;cn	1-0;cn	0;1c
PI 405399	<i>Rph15</i>	0;1-cn	0;cn	0;1-cn	2-1c	0;cn	0;1cn	0;cn	0;cn	0;cn	3+3	3-	33-	23-cn	21cn	23-cn	1-0;cn	0;1cn	0;1cn
PI 466245	<i>Rph15</i>	10;cn	0;1cn	0;1cn	1-0;c	0;1-cn	10;cn	0;cn	0;cn	0;cn	33+	3-3	3-	23-cn	21cn	3-	12-cn	1-0;cn	0;1cn
PI 466470	<i>Rph15+?</i>	213-c	120;c	3-2	210;	120;c	0;12	0;1-c	10;c	0;c	120;c	120;c	0;12c	0;1c	0;1-c	0;12c	0;1c	10;	0;1-
PI 466483	<i>Rph15+?</i>	11-c	23-1c	23-1	213-	2	210;c	0;1c	0;1-c	0;1-	210;c	120;c	120;c	0;1c	0;1-c	0;12c	0;1	0;1c	0;1c
Columbia	?	33-	3	3+	33-	3	3	213-	33+	3	33+	33-	33-	21c	213-	3-2	3-2	33-	3
Emir/H. bulbosum	?	00;n	00;	00;	00;n		00;	00;		00;	00;n	00;n	00;	00;	00;n	00;	00;	00;n	00;n
HS580	<i>Rph15</i>	1c	0;1-	0;1cn	11-	0;1-c	10;cn	1-0;cn	00;cn	0;cn	3	23-c	3-	23-cn	21cn	23-cn	0;1-cn	0;1-cn	0;1-cn
PI 355445	<i>Rph15</i>	1-0;cn	1-0;cn	0;1cn	1-0;c	10;cn	0;1-cn	0;1-cn	12c	0;	33-	33-c	3-	12-cn	21c	210;n	1-0;cn	10;c	0;1-cn
PI 405154	<i>Rph15+?</i>	1-0;cn	0;1-		0;1-c	0;1-cn		0;cn	00;c		23-1c	23-1c		213-c	21c		1-0;cn	10;c	
PI 405169	<i>Rph9</i>		10;c	213-	213-c	21c	120;c	12	33-	213-c			3-	33-	33-	3-		3	3-
PI 405215	<i>Rph15+?</i>	0;1-cn	0;cn	0;cn	1-0;cn	0;cn	0;cn	0;cn	0;cn	0;cn	3-2c	23-c	23-c	12-cn	21cn	12cn	0;1-c	0;1-cn	10;cn
PI 466373	<i>Rph15+?</i>	0;1-cn	00;1-	0;cn	10;c	00;1-c	0;cn	0;1-cn	00;1-	0;	23-c	21c	3-	210;c	21c	213-c	00;1	0;1	0;1-
PI 531901-2	?			00;			0;1			0;12			0;12c			213-c			3-2
Tunisia 33	<i>Rph7+?</i>	00;	00;1n	0;1	00;n		0;1			00;			0;3-		33-	3-			3-
Vada/H. bulbosum	?			213-			10;			10;			3-			3-		00;2	3-2

¹Infection types were scored using the original 0-4 rating scale developed by Mains (1930) with the additional notations of "+" and "-" to indicate larger or smaller size variations of classically described uredinia (Levine and Cherewick, 1952) and "c" and "n" to indicate the presence of chlorotic or necrotic reactions (Moseman and Roane, 1959), respectively. Complex ITs were recorded when more than one IT was observed on the surface of a single leaf. For example, an IT of 213- indicates that all three pustule types exist on the same leaf and that the "2" type pustules were the most common, followed by the "1" type, with "3-" type pustules being the least common. Infection types from "0;" to "0;12" are classified as highly resistant ("R", green cells), infections types from "12" to "23-c" are classified as moderately resistant ("M", yellow cells), and infection types from "23-" to "33+" are classified as susceptible ("S", red cells) (Steffenson et al., 1993).

Appendix Table 8. Raw infection types¹ of barley differential lines carryng the leaf rust resistance genes *Rph1*-24 in this and a previous study.

Isolate ²	Replication ³	Gene	Differential																									
				<i>Rph1</i>	<i>Rph2</i>	<i>Rph3</i>	<i>Rph4</i>	<i>Rph5</i>	<i>Rph6</i>	<i>Rph7</i>	<i>Rph8</i>	<i>Rph9</i>	<i>Rph10</i>	<i>Rph11</i>	<i>Rph12</i>	<i>Rph13</i>	<i>Rph14</i>	<i>Rph15</i>	<i>Rph16</i>	<i>Rph17</i>	<i>Rph18</i>	<i>Rph19</i>	<i>Rph20</i>	<i>Rph2+21</i>	<i>Rph22</i>	<i>Rph23</i>	<i>Rph24</i>	
BR (Octal 11)	Fetch et al. (1998)			10;c	210;	0;	3	0;n	0;n	0;n	12c	0;1c	231c	23c	0;nc	32	0;1	0;N										
	Exp1 2003 GH INC			21	21	0;1	23	0;1N	1C	0;cN	0;1cn	0;1cN	20;	12C	0;1Cn	120;n		0;N										
	Exp1 2015 GH INC			23C	0;1C	0;12	23C		21C	0;cn		0;Cn	0;1C	0;1N		10;cN	0;1n	0;CN										
	Exp1 NSGC			0;1N	0;1C	0;CN	213C	0;N		0;cN	0;2C				3													
	Exp1 2014 Field INC																	12	0;1c	00;n	0;1Cn	0;1C	0;1n		0;12c			
	Exp3 GH + Field INC			23-1	213-	0;1-c	12	0;1-n	11-c	00;n	210;n	0;1-cn	120;	120;	0;1-	0;1-	00;1-	0;1-cn	12	1-0;c	00;n	0;1-	10;c	1c	12	213-	213-	
	Exp3 NSGC			0;1-n	0;1	00;1-	12	0;1-	0;1-	00;1-n	210;n	0;1-cN	12	0;1-c	1-0;	10;	0;1	0;1-cn				0;1-n		0;1		21		
	Exp10 GH + Field INC			3-2c	0;1c	0;12c	23-c	0;1-cN	0;1c	00;cn	0;1cn	10;cn	10;c	213-c	0;1-cn	0;12	0;cn	0;1-cn	23-c	0;12c	00;	0;1-cn	0;1c	0;1c		23-1		
	Exp10 NSGC			0;1cn	0;1c	0;12	23-c	10;cn	0;12cn	00;cn	0;1cn	0;1cn			0;12cN	00;1	00;1cn					0;1-cn		00;1c		0;12		
	Exp 11 GH + Field INC			3-2	0;1	0;	3-2c	0;1n	0;1	0;n	0;1n	0;1N	213-	21	10;	00;1	10;	0;1-cn	2	10;c	00;	0;1n	0;1c	10;c	10;	0;12	33-	
ARG231	Fetch et al. (1998)			3	3	0;	3	3	3	0;n	3	213C	3	3	0;1nc	0;cn	12c	0;n										
	Exp1 2003 GH INC			210;c	0;1c	00;1	21C	21c	12C	0;cN		0;N	21c	2c		120;	2	0;N										
	Exp1 2015 GH INC			213c	0;1c	00;1	21c		21c			0;N	21c	1c	10;n	0;1	21	0;CN										
	Exp1 NSGC			0;2N	0;cn	00;n	2	12c		00;cN	23-				21													
	Exp1 2014 Field INC																	213c	0;cn	00;n	0;1cn	0;1c	0;1N		0;1			
	Exp3 GH + Field INC			23-1	21	0;1-	21	11-cN	11-	0;1-c	3-2	0;cN	1-0;c	1-0;c	1-0;c	0;1-	12	0;1-cn	120;	1-0;c	00;n	10;	0;1-c	0;1-	23-1	1-0;	23-1	
	Exp3 NSGC			12	0;1	00;1-	12	213-N	0;1-	00;n	3-2	0;cN	11-	0;1-	1-0;cN	0;1-	12	0;1-cn				10;		0;1-		1-0;		
	Exp10 GH + Field INC			3-21	0;12c	00;1	23-c	10;c	120;c	0;1-cn	213-c	0;1-cn	12c	21c	0;1-cn	0;1c	0;12c	0;cn	0;12	0;1-c	00;n	0;1c	0;1c	0;1-c		0;1cn		
	Exp10 NSGC			0;1-cn	0;1c	00;	120;c	120;c	0;1	0;cn	3-c	0;cN			0;1-cn	0;1-c	0;1-cn					0;1cn		0;cn		0;12c		
	Exp 11 GH + Field INC			3-0;	0;12	00;	23-1	23-c	213-	00;n	3-	0;n	213-	21c	0;1	0;1	213-	0;1-cn	210;	0;1-	00;	120;	0;1	0;1	120;	0;1	3-2	

Appendix Table 8 continued. Raw infection types¹ of barley differential lines carryng the leaf rust resistance genes *Rph1*-24 in this and a previous study.

Isolate ²	Replication ³	Gene	Differential																									
				<i>Rph1</i>	<i>Rph2</i>	<i>Rph3</i>	<i>Rph4</i>	<i>Rph5</i>	<i>Rph6</i>	<i>Rph7</i>	<i>Rph8</i>	<i>Rph9</i>	<i>Rph10</i>	<i>Rph11</i>	<i>Rph12</i>	<i>Rph13</i>	<i>Rph14</i>	<i>Rph15</i>	<i>Rph16</i>	<i>Rph17</i>	<i>Rph18</i>	<i>Rph19</i>	<i>Rph20</i>	<i>Rph2+21</i>	<i>Rph22</i>	<i>Rph23</i>	<i>Rph24</i>	
				<i>Sudan</i>	<i>Peruvian</i>	<i>Estate</i>	<i>Gold</i>	<i>Magnif 102</i>	<i>Bolivia New</i>	<i>Cebada Capa</i>	<i>Egypt 4</i>	<i>Hor 2596</i>	<i>Clipper BC 8</i>	<i>Clipper BC 67</i>	<i>Triumph</i>	<i>PI 531849</i>	<i>PI 584760</i>	<i>I95-282-2</i>	<i>HOR15560</i>	<i>81882/BS1</i>	<i>38P18/8/1/10</i>	<i>Prior</i>	<i>Flagship</i>	<i>Ricardo</i>	<i>182Q20/1/M1</i>	<i>Yerong</i>	<i>ND24260</i>	
WGCC	Fetch et al. (1998)			0;1n	10;2c	00;	0;1	213	213c	0;	32c	0;nc	32	23c	0;c	10;	21c	0;N										
	Exp1 2003 GH INC			10;2n	10;	0;1	10;c	23	23	0;n	21c	0;1c	21c	3-2		10;c	0;cN	0;n										
	Exp1 2015 GH INC			10;2n	213-	0;1	210;c		23	0;n		0;1N	12c	21c		0;1N	21	0;N										
	Exp1 NSGC			120;n	0;1c	00;1	10;c	3		0;cN	23c				3													
	Exp1 2014 Field INC																		10;c	0;N	00;n	32	10;	0;1c		21		
	Exp3 GH + Field INC			0;12	10;	00;1-	10;2	10;2c	21c	00;1-c	3-2c	00;1-N	12c	21c	11-c	0;1-	21	1-0;cn	0;1	1-0;c	00;	10;c	0;1c	1-0;	0;1	0;1-	33-	
	Exp3 NSGC			1-0;n	0;1-c	0;1-	0;1c	10;	0;	00;1-	33-	0;1-N	210;c	120;	10;c	0;1-c	12c	0;cn			10;c		0;1-					
	Exp10 GH + Field INC			0;12	210;c	0;12c	0;12	3-2	3-2	00;n	23-c	0;1c	23-c	23-c	0;1-cn	0;1-n	21c	0;cn	0;1c	0;1c	00;	33-	10;c	0;12		3-2		
	Exp10 NSGC			213-	0;1c		0;12	33-	0;12c	00;n	23-c	0;1c			0;1c	00;n	0;1c				33-		0;12c			3-2		
Exp 11 GH + Field INC			0;2	0;12c	00;2	0;1	3	3	00;	23-c	0;1-c	0;1c	23-c	0;1-c	0;1	10;c	0;cn	0;1	0;1-c	0;	3	0;1-c	0;1c	0;3-	0;12	23-		
Ger 7	Fetch et al. (1998)			0;1	32	0;	32	10;n	0;1c	0;	0;nc	10;C	3	23c	0;nc	0;n	0;1	0;nc										
	Exp1 2003 GH INC			0;1Cn	23	00;1	23-	0;1CN	1C	0;CN	0;CN	10;C	21	1c		10;Cn	0;cN											
	Exp1 2015 GH INC			0;1C	3-2	00;1	23-	0;1n	10;C			0;CN	23-	21c	0;cN	0;1cn	0;N	0;N										
	Exp1 NSGC			213	10;c	00;1	23-	10;		0;CN	0;CN				32													
	Exp1 2014 Field INC																		1C		00;n	1c	12c	213		3-2		
	Exp4 GH + Field INC			0;1-	213-c	00;1-	3-2	1c	12c	0;1-	0;1-cn	1-0;cN	23-	21c	0;1-c	0;1-	0;1-n	1-0;cn	0;1-	0;1-c	00;	12c	0;1-c	210;	21	23-	23-	
	Exp4 NSGC			213-	10;2	00;	23-	10;c	0;1	0;1-	0;1-cn	10;cN	3-2	00;	0;1-	0;1-	0;1-n	0;cn			120;		3-21			33-		
	Exp6 GH GH + Field INC			0;1-	210;	00;2	3-2	12c	21c	0;1-c	0;1n	10;c	23-	213-c	0;cn	0;1-	0;1n	1cn	0;1-c	0;1c	00;	12c	0;1c	3-20;		33+		
	Exp6 NSGC			213-	0;12c		3-	21c	23-0;	0;1-c	0;2cn	10;c			0;12cN	0;1-n	00;1				120;c		210;			3-2		
Exp12 GH + Field INC			0;1	23-1	00;2	3-2	120;c	120;c	0;1-c	0;1-c	0;1c	3-2	21c	0;1-	0;1-	0;1-cn	0;1-cn	0;cn	0;1-c	00;	210;c	0;c	120;	120;	3-2	3		

Appendix Table 8 continued. Raw infection types¹ of barley differential lines carryng the leaf rust resistance genes *Rph1*-24 in this and a previous study.

Isolate ²	Replication ³	Gene	Differential																									
				<i>Rph1</i>	<i>Rph2</i>	<i>Rph3</i>	<i>Rph4</i>	<i>Rph5</i>	<i>Rph6</i>	<i>Rph7</i>	<i>Rph8</i>	<i>Rph9</i>	<i>Rph10</i>	<i>Rph11</i>	<i>Rph12</i>	<i>Rph13</i>	<i>Rph14</i>	<i>Rph15</i>	<i>Rph16</i>	<i>Rph17</i>	<i>Rph18</i>	<i>Rph19</i>	<i>Rph20</i>	<i>Rph21</i>	<i>Rph22</i>	<i>Rph23</i>	<i>Rph24</i>	
Race 4	Fetch et al. (1998)			0;1	210;	0;	0;1	0;n	0;N	0;n	3	0;1C	3	32	0;nc	0;1c	3c	0;N										
	Exp1 2003 GH INC			10;c	0;12c	00;1	120;c	213c	23c	0;cN	32	21c	23-	23-	21c	0;12	2	0;cN										
	Exp1 2015 GH INC			0;1c	0;1	00;1	0;1c		1c			0;1cN	23-	23-	0;1cn	10;	21	0;cN										
	Exp1 NSGC			0;1n	0;1c	00;	10;c	213		0;cN	32				32													
	Exp1 2014 Field INC																		10;c	0;1	00;	120;	10;c	0;cN		120;		
	Exp4 GH + Field INC			0;1-	210;	00;1-	10;	213-c	23-1c	0;1-	3+3	0;1-n	213-c	21c	10;c	0;1	213-	1-0;cn	0;1-	1-0;c	00;n	0;1	0;1c	1-0;	2	0;1-	33+	
	Exp4 NSGC			0;1	0;1-	00;1-	10;	21	0;1-	00;1-n	3+3	10;N	3-2	21c	21cN	0;1-	213-c	0;1-cn			10;		0;1-			33+		
	Exp9 GH + Field INC			01c	0;12c	0;1c	0;1c	23-c	33-	0;1-c	3	0;1c	23-	21c	10;c	0;1-c	3-	0;1-cn	0;1-c	0;1-	00;	213-c	10;c	0;12c		0;12		
	Exp9 NSGC			0;12	0;1c	0;1c	0;12c	33-	00;1n	00;1-	3	0;1c			0;1-c	0;1-c	33-				120;		0;1c			0;12		
	Exp12 GH + Field INC			0;1-	120;	00;	120;	213-	213-	0;	3	0;1-c	3-2	12c	0;1-c	120;	23-1	0;1-cn	0;1-cn	10;	00;	213-c	0;1-c	0;1-n	0;12	0;12	3-2	
Aus 220	Fetch et al. (1998)			0;1	2310;Y	0;	0;1	3	3	0;	3c	0;1C	23c	32c	0;cn	0;	1c	0;n										
	Exp1 2003 GH INC			0;n	10;c	00;1	0;1c	3	3	00;n	2C	0;1C	2C	2C	0;cN	0;1n	10;c											
	Exp1 2015 GH INC			0;n	10;C	00;1	0;1		21			0;N	12c	2c	0;cN	0;N	0;1cN	0;N										
	Exp1 NSGC			0;cN	0;1C	12c	0;cn	33+		0;n	23c				32													
	Exp1 2014 Field INC																		0;1C	0;Cn	00;n	23-	0;cn	0;2c		213		
	Exp3 GH + Field INC			1-0;	23-1	0;1-c	0;1-	23-	3-2	00;1-	23-c	0;cN	11-c	11-c	0;1-c	00;1-	0;1-c	0;cn	0;1-	1-0;	00;	23-	1-0;	213-c	23-1	21c	3-2	
	Exp3 NSGC			3-2	120;	0;1-c	0;1-	3-2	0;12	00;n	23-c	0;cN	23-	10;c	0;1-c	00;n	10;c	0;cn				3-2		10;		21c		
	Exp10 GH + Field INC			0;cn.	0;12c	0;1-c	0;12c	3-2c	3-2	00;	0;12c	0;1-cn	0;12c	3-2c	0;1-cn	00;1-n	0;1c	0;cn	0;1-c	10;c	00;n	3-2	0;1c	0;1-c		23-0;		
	Exp10 NSGC			0;12c	0;1c		0;1-c	33-	0;12c	00;	23-c	0;1-c			0;1-cN	0;1-N	0;12c					33-		0;12c		0;12c		
	Exp 11 GH + Field INC			0;1	0;12	0;1	0;1	3	3	00;	3-	0;1c	10;c	23-c	0;1-c	0;1	23-1c	0;cn	0;cn	0;1c	00;	3-	0;1c	0;1	0;12	0;12	3-2	

Appendix Table 8 continued. Raw infection types¹ of barley differential lines carryng the leaf rust resistance genes *Rph1*-24 in this and a previous study.

Isolate ²	Replication ³	Gene	Differential	<i>Rph1</i>	<i>Rph2</i>	<i>Rph3</i>	<i>Rph4</i>	<i>Rph5</i>	<i>Rph6</i>	<i>Rph7</i>	<i>Rph8</i>	<i>Rph9</i>	<i>Rph10</i>	<i>Rph11</i>	<i>Rph12</i>	<i>Rph13</i>	<i>Rph14</i>	<i>Rph15</i>	<i>Rph16</i>	<i>Rph17</i>	<i>Rph18</i>	<i>Rph19</i>	<i>Rph20</i>	<i>Rph21</i>	<i>Rph22</i>	<i>Rph23</i>	<i>Rph24</i>
				Sudan	Peruvian	Estate	Gold	Magnif 102	Bolivia New	Cebada Capa	Egypt 4	Hor 2596	Clipper BC 8	Clipper BC 67	Triumph	PI 531849	PI 584760	195-282-2	HOR15560	81882/BS1	38P18/8/1/10	Prior	Flagship	Ricardo	182Q20/1/M1	Yerong	ND24260
98-20	Fetch et al. (1998)			3	3	0;c	3	0;n	0;1n	3>3n	3c	2C	21	23c	0;nc	3	1cn	0;Nc									
	Exp1 2003 GH INC			3-2	23	00;	213-	0;1cN	1c	3	33-c	12C	0;N	1c		21	1										
	Exp1 2015 GH INC			3	321	00;1	3-2		0;1c			0;1N	10;	12		23-1	10;	0;cN									
	Exp1 NSGC			3	0;12	00;1	2	1		3	32c				23												
	Exp1 2014 Field INC																		32	0;N	00;N	10;	0;N	21		213	
	Exp5 GH + Field INC			33-	3-21c	0;	23-1	12c	12c	33-	3-c	10;c	12c	21c	10;c	3-2	12c	1-0;cn	3-2	11-c	00;	213-	0;1c	213-c	213-	33-	3
	Exp5 NSGC			3-2	213-c	0;c	23-c	12-c	23-1		3c	10;c	23-c	23-c	11-c	23-	12c	0;1-cn				3-2		213-c		3-3	
	Exp8 GH + Field INC			33-	21c	00;1c	3-	10;c	10;c	3-20;	3-2c	10;c	10;	23-c	120;c	213-	10;c	1-0;cn	3	0;1c	00;	21c	10;c	21		33-	
	Exp8 NSGC			23-	120;		3-	10;c	3-20;	3-20;	3-2c	21c	3-2c		10;c	21c	12c					23-c		21c		3-2	
	Exp13 GH + Field INC			3-2	23-1	00;	3-2	120;	120;	3-0;	3	120;c	120;c	23-1	0;12	23-1	21	10;cn	213-	120;	00;	3-2	0;12c	3-21	210;	3	3-2
94-8	Fetch et al. (1998)			3	32c	0;	3	0;	0;n	0;	3c	23C	213c	32c	3	3	1c	0;Nc									
	Exp1 2003 GH INC			3-2	21c	00;	23-	0;cN	0;N	0;cN	23	3	1c	33-	33	2c	12c	0;cN									
	Exp1 2015 GH INC			33-	23-	00;cn	23	0;1	1cn	0;cN		3-2	1c	21c	32	213c	21c	0;cN									
	Exp1 NSGC			23	0;1	00;1	23-	0;cN		0;cN	33-				32												
	Exp1 2014 Field INC																		3-2	0;N	00;n	12	0;1n	21		3-2	
	Exp5 GH + Field INC			3-	3-2c	0;1-c	3-2c	10;c	10;c	0;1-cn	3-2c	3-2c	12c	3-2c	3-2	23-c	10;c	0;1-cn	23-1	1-0;cn	0;	213-c	10;c	23-1c	33-	33-	3-2c
	Exp5 NSGC			3-2	23-1c		3-2	10;cn	120;	0;1-c	3-2	3-2	3-	23-c	3-	3-2	120;c	1-0;cn				3-2c		210;c		3-	
	Exp8 GH + Field INC			33-	210;c	00;1-	23-c	0;1c	0;1c	0;1-cn	21c	23-c	10;c	23-c	33-	213-	10;c	0;1-cn	23-	0;1-c	00;	213-c	0;1-c	21c		33-	
	Exp8 NSGC			3-2	120;c		23-c	1-0;c	23-0;	0;cN	23-c	33-	33-		3-2	213-	12c					23-		210;c		33-	
	Exp13 GH + Field INC			3	210;	00;1	3	0;1	0;1	0;1-	3-2	3-2	10;	213-	3-	3-2	210;c	12cn	23-	0;1	00;	3-	0;12c	3-2	3-0;	3-2	3-2

Appendix Table 8 continued. Raw infection types¹ of barley differential lines carryng the leaf rust resistance genes *Rph1*-24 in this and a previous study.

Isolate ²	Replication ³	Gene	Differential	Differential																								
				Rph1	Rph2	Rph3	Rph4	Rph5	Rph6	Rph7	Rph8	Rph9	Rph10	Rph11	Rph12	Rph13	Rph14	Rph15	Rph16	Rph17	Rph18	Rph19	Rph20	Rph2+21	Rph22	Rph23	Rph24	
BRS 76-12	Fetch et al. (1998)			3	3	3	3	3	3	0;n	3	2C	3	32c	0;cn	0;1	213c	0;										
	Exp1 2003 GH INC		21	23-	3	23	32	21c	0;cN			12	0;1c	0;cN	0;1c	2c	0;N											
	Exp1 2015 GH INC		23	23-	23	10;c		12c	0;cN	3	0;CN	21	21c	0;cn	21cN	10;c	0;cN											
	Exp1 NSGC		213	23-	23-c	23-	2	0;cN	3	3				3														
	Exp1 2014 Field INC																	3-	10;n	00;n	23	1c	3-2		3			
	Exp5 GH + Field INC		33-	213-c	33-	3-2	23-c	23-c	0;1-c	33-	10;c	23-1c	21c	0;1-c	10;c	213-c	0;1-cn	23-	10;c	00;	12c	0;1-c	10;c	23-1	33-	33-		
	Exp5 NSGC		3-2	120;c	23-	3-2	213-	120;	0;1-c	3	10;c	3-2	21c	0;1-c	0;1c	210;c				3-2c		210;c		33-				
	Exp8 GH + Field INC		33-	120;c	3-2	33-	12c	213-c	00;cn	23-c	0;1c	120;c	21c	0;1-c	0;1	120;c	0;cn	3-2	10;c	00;	23-1c	10;c	210;c		33-			
	Exp8 NSGC		23-1c	120;c	23-1c	23-c	33-	3-20;c	0;n	33-	10;c	3-2		0;1-cN	0;1-c	10;c				23-1c		120;c		3-2				
	Exp13 GH + Field INC		3-	120;c	3-	3-2	23-c	23-c	0;n	3-2	10;c	23-1	23-1	0;1	0;1	210;c	0;cn	3-2	0;1-c	00;	23-1	0;1c	210;	3-20;	3-2	3-2		
Neth 28	Fetch et al. (1998)		3	3	23c	210;	3	3	3	3C	1C	3	3	3	0;	3	1N											
	Exp1 2003 GH INC		32	32	3-2c	10;	32	21	23	3	1c	3-2	23-c	21c	10;c	21												
	Exp1 2015 GH INC		3-2	3-2	213c	0;1		21	3-2		10;c	23	12c		10;c	21c	21c											
	Exp1 NSGC		32	3-2	12c	0;1	3		3 2	3			3															
	Exp1 2014 Field INC																	2	32	00;n	23	21c	23		3-2			
	Exp6 GH + Field INC		33-	33-	213-c	0;12c	33-	23-c	33-	33-c	120;c	33+	3-c	23-c	0;1c	120;c	12-cn	3-2	21c	00;n	33-	23-c	33-2c		33-	3		
	Exp6 NSGC		3-c		0;12c	3-	3-3	33+	33+	3+3	120;c	3+3c	3+3	3-2c	0;1c	210;c				3-3		33-c		33+				
	Exp7 GH + Field INC		3-	3-2	2c	120;c	33-	23-	33-c	33+	10;c	3	3-2c	33+	0;1c	12c	12cn	3-2	21c	00;	3-2	23-c	33-		3-			
	Exp7 NSGC		3-3	33-		0;1c	3-	33-	33-	33+	10;c	3-	23-c	33-	0;1c	21c				33-		33-		33-				
	Exp14 GH + Field INC		3	33-	213-c	0;12	3	3-2	3	33-	210;c	3	3-2c	3	120;	3-2c	23-1c	23-1c	23-1c	00;	3	213-c	33-	3-0;	3	3		

Appendix Table 8 continued. Raw infection types¹ of barley differential lines carryng the leaf rust resistance genes *Rph1*-24 in this and a previous study.

Isolate ²	Replication ³	Gene	Differential																									
				<i>Rph1</i>	<i>Rph2</i>	<i>Rph3</i>	<i>Rph4</i>	<i>Rph5</i>	<i>Rph6</i>	<i>Rph7</i>	<i>Rph8</i>	<i>Rph9</i>	<i>Rph10</i>	<i>Rph11</i>	<i>Rph12</i>	<i>Rph13</i>	<i>Rph14</i>	<i>Rph15</i>	<i>Rph16</i>	<i>Rph17</i>	<i>Rph18</i>	<i>Rph19</i>	<i>Rph20</i>	<i>Rph2+21</i>	<i>Rph22</i>	<i>Rph23</i>	<i>Rph24</i>	
90-3	Fetch et al. (1998)			3	32	2c	3	3	3	0;N	3C	3	32	32	3	10;N	3	3										
	Exp1 2003 GH INC			23	32	0;1	12	210;	23	0;1cN	32	3-2	21c	21c		10;C	23	23										
	Exp1 2015 GH INC			23	120;	0;1	23-	21	2c			3-2	21c	21c	33+	120;c	21c	32										
	Exp1 NSGC			23	120;	0;1	21c	10;cn		12c	33+				21c													
	Exp1 2014 Field INC																	23	21c	00;n	10;c	21	23c		23			
	Exp6 GH + Field INC			33+	320;	0;12	33-	23-1c	3-2	120;c	33+	3-2	23-c	23-1c	33-	0;1c	3-2c	33-	3-2	23-c	00;n	23-1c	3-2c	23-0;c		3	33+	
	Exp6 NSGC			33+	23-1c	0;12	3-2	23-1c	33-0;	11-0;cn	33+	33-	23-c	23-c	33-	0;1c	23-1c	3-2c				3-2c		3-21c		33-		
	Exp7 GH + Field INC			3	33+	23-1c	3	3-2c	3	213-c	3	33+	23-c	23-c	3	0;1c	33-	3	3-	23-c	00;	23-c	23-c	3-0;		33+		
	Exp7 NSGC			33-	3		33-	3-	3	213-c	33+	33-	23-c		33-	0;1c	23-					3-		3-0;		33-		
	Exp14 GH + Field INC			3	3	0;12c	3-	33-	3-	3	33-	3	3-2c	3-2c	3	210;	3	3	3	3-	0;	3	3	3	3-21	3	33-	
92-7	Fetch et al. (1998)			3	3	32	3	3	3	3	3c	32c	3	32	3	1c	32	0;Nc										
	Exp1 2003 GH INC			32	23-	23	21	23	21	32	32	12	23	213-	2	21	32											
	Exp1 2015 GH INC			23	32	23	32		213	33+	32	21	213	21c	3	10;c	21	0;cN										
	Exp1 NSGC			32	21	23-1	23-	3		32	3				32													
	Exp1 2014 Field INC																		3	12	00;	3-2	12	32		33-		
	Exp6 GH + Field INC			3-2	33-	3-2c	3-2	33-	23-c	33-	3	23-1c	33-	23-c	3-2c	0;1	210;c	1-0;cn	3-2	23-c	00;	3-	23-c	3-2		3	33+	
	Exp6 NSGC			33-	3-2c	33+c	3-	33-	33-	33+	3+3	33-	33-c		3-2c	0;1	21c	0;1-cn				33-		33-		3+3		
	Exp7 GH + Field INC			3-	33-	23-c	33-	33-	33-	33-	33+	23-	33+	23-c	3	0;1c	21c	0;1-cn	3-2	3-2	00;	33-	23-c	3-		3		
	Exp7 NSGC			3	3-2	3-	3-	3-	3	3	3	3-2	33-	2c	3-	10;c	21					33-		3-2		33-		
	Exp14 GH + Field INC			3	3-	3-2c	3-	3	3-	3-	3	3-	3-	3	12	3-2	0;1-cn	3-	21c	00;	3-	213-c	3-	3	3	33-		

Appendix Table 8 Footnotes.

¹ Infection types were scored using the original 0-4 rating scale developed by Mains (1930) with the additional notations of "+" and "-" to indicate larger or smaller size variations of classically described uredinia (Levine and Cherewick, 1952) and "c" and "n" to indicate the presence of chlorotic or necrotic reactions (Moseman and Roane, 1959), respectively. Complex ITs were recorded when more than one IT was observed on the surface of a single leaf. For example, an IT of 213- indicates that all three pustule types exist on the same leaf and that the "2" type pustules were the most common, followed by the "1" type, with "3-" type pustules being the least common. Infection types from "0;" to "0;12" are classified as highly resistant ("R", green cells), infections types from "12" to "23-c" are classified as moderately resistant ("M", yellow cells), and infection types from "23-" to "33+" are classified as susceptible ("S", red cells) (Steffenson et al., 1993).

² Twelve isolates of *Puccinia hordei* were characterized for their infection types (ITs) against the barley leaf rust differentials for *Rph1-24*.

³ The different experimental replications of these plant-plant pathogen interactions are listed. The previous study by Fetch et al. (1998) described the infection types of these 12 isolates against the barley leaf rust differentials for *Rph1-15*. The different experimental replicates in this study range from Experiment 1 (Exp1) to Experiment 14 (Exp14). Some replications are also identified as different seed sources grown from either greenhouse (GH) or field increased (INC) genetic stocks or from seed stocks provided by the National Small Grains Collection (NSGC) genebank.

Appendix Table 9. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW682	Rph1.a	1_0326	AK364110	Pyruvate dehydrogenase E1 component alpha subunit
BW682	Rph1.a	1_0326	MLOC_62638.1	Polyketide cyclase/dehydrase and lipid transport-like protein
BW682	Rph1.a	1_1059	AK368531	12-oxophytodienoate reductase 1
BW682	Rph1.a	2_1377	AK361947	Zinc finger (Ran-binding) family protein
BW682	Rph1.a	2_1377	MLOC_10639.1	NA
BW682	Rph1.a	2_1377	MLOC_10640.1	NA
BW682	Rph1.a	2_0563	MLOC_67779.1	Cullin-associated NEDD8-dissociated protein
BW743	Rph2.t	2_0845	MLOC_10278.1	Single myb histone 6
BW743	Rph2.t	2_0845	AK249399.1	Pentatricopeptide repeat (PPR) superfamily protein
BW743	Rph2.t	2_0980	MLOC_10278.1	Single myb histone 6
BW743	Rph2.t	2_0980	AK249399.1	Pentatricopeptide repeat (PPR) superfamily protein
BW743	Rph2.t	2_1391	MLOC_5116.4	Vacuolar fusion protein CCZ1 homolog
BW743	Rph2.t	2_1253	MLOC_54218.3	Protein kinase superfamily protein LENGTH=390
BW743	Rph2.t	2_1253	MLOC_54219.1	Tuftelin interacting protein
BW743	Rph2.t	2_0571	MLOC_3932.1	Ubiquitin-conjugating enzyme family protein-like
BW743	Rph2.t	2_0571	MLOC_3933.1	NA
BW743	Rph2.t	1_0955	MLOC_38116.1	NA
BW743	Rph2.t	1_0955	AK356629	L-galactose dehydrogenase
BW743	Rph2.t	1_1432	AK370125	Auxin responsive protein
BW743	Rph2.t	1_1432	MLOC_73035.1	Uridine kinase-like 5
BW743	Rph2.t	1_1432	MLOC_73034.1	NA
BW743	Rph2.t	1_1432	MLOC_73036.2	Leucine Rich Repeat family protein
BW743	Rph2.t	2_0841	MLOC_38191.2	NA
BW743	Rph2.t	2_0841	AK367062	UNC93-like protein MFSD11

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW743	Rph2.t	2_0697	MLOC_64255.1	Transmembrane protein
BW743	Rph2.t	1_0058	MLOC_63593.1	40S ribosomal protein S9
BW743	Rph2.t	1_0058	MLOC_63594.1	NA
BW743	Rph2.t	2_0179	MLOC_59475.1	V-type proton ATPase 16 kDa proteolipid subunit c1/c3/c5
BW743	Rph2.t	1_0157	AK251398.1	Eukaryotic translation initiation factor 5a-2, putative
BW743	Rph2.t	1_0177	NA	NA
BW743	Rph2.t	1_0240	MLOC_54831.1	Thioredoxin m
BW743	Rph2.t	1_0252	AK368598	Chaperone protein ClpB 1
BW743	Rph2.t	2_0332	AK248962.1	Ubiquitin activating enzyme 2
BW743	Rph2.t	1_0318	AK371168	Ubiquitin carboxyl-terminal hydrolase, putative
BW743	Rph2.t	2_0461	MLOC_19387.1	Aldehyde dehydrogenase
BW743	Rph2.t	2_0461	AK251412.1	Peroxidase superfamily protein
BW743	Rph2.t	2_0461	MLOC_19388.1	NA
BW743	Rph2.t	2_0524	MLOC_57983.5	Fe/S biogenesis protein nfuA
BW743	Rph2.t	2_0524	MLOC_57984.1	NA
BW743	Rph2.t	2_0524	MLOC_57982.1	NA
BW743	Rph2.t	2_0524	AK356301	Transmembrane 9 superfamily protein member 3
BW743	Rph2.t	2_0700	AK365226	RNA polymerase-associated protein RTF1, putative
BW743	Rph2.t	2_0700	AK366262	2,3-bisphosphoglycerate-independent phosphoglycerate mutase
BW743	Rph2.t	2_0708	MLOC_38118.1	NA
BW743	Rph2.t	2_0708	MLOC_38119.1	NA
BW743	Rph2.t	2_0737	MLOC_5324.1	Chalcone--flavonone isomerase
BW743	Rph2.t	2_1011	MLOC_40010.1	NA
BW743	Rph2.t	2_1040	AK370183	AP-2 complex subunit sigma-1

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW743	Rph2.t	1_0856	MLOC_57598.1	NA
BW743	Rph2.t	2_1215	MLOC_33142.1	NA
BW743	Rph2.t	2_1215	MLOC_33143.1	NA
BW743	Rph2.t	1_0913	AK373225	NA
BW743	Rph2.t	2_1260	AK251163.1	Sorting and assembly machinery component-like protein
BW743	Rph2.t	2_1260	MLOC_72013.1	Myb family transcription factor
BW743	Rph2.t	2_1350	MLOC_62403.4	NA
BW743	Rph2.t	2_1350	MLOC_62404.1	NA
BW743	Rph2.t	1_0995	MLOC_38005.1	Syntaxin, putative
BW743	Rph2.t	1_1469	AK358200	4-alpha-L-fucosyltransferase
BW743	Rph2.t	1_1506	MLOC_73610.1	NA
BW743	Rph2.t	1_1506	MLOC_73609.2	Saposin B domain-containing protein
BW743	Rph2.t	1_1260	MLOC_4554.1	ABSCISIC ACID-INSENSITIVE 5-like protein
BW743	Rph2.t	2_1318	MLOC_12555.1	Vacuolar sorting receptor protein
BW743	Rph2.t	2_1536	MLOC_69295.2	Ferritin
BW743	Rph2.t	2_0306	AK365203	Polyadenylate-binding protein, putative
BW743	Rph2.t	2_0283	AK251150.1	Protein phosphatase 2A regulatory subunit A
BW743	Rph2.t	2_0372	MLOC_6055.1	Ubiquitin-conjugating enzyme variant
BW743	Rph2.t	1_0671	AK374108	NA
BW743	Rph2.t	1_0671	MLOC_59580.1	Ferrochelatase
BW743	Rph2.t	2_0961	AK252957.1	ABC-1 domain protein
BW743	Rph2.t	1_0840	NA	NA
BW743	Rph2.t	1_1240	NA	NA
BW743	Rph2.t	2_1200	MLOC_52503.1	Cytochrome b5-like

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW743	Rph2.t	1_1159	MLOC_38219.1	Protein arginine N-methyltransferase 1
BW743	Rph2.t	1_1221	AK369379	Fatty acid desaturase, putative
BW743	Rph2.t	2_0265	AK365065	Trehalose-6-phosphate phosphatase
BW743	Rph2.t	2_0441	AK250045.1	Jasmonate ZIM-domain protein 2
BW743	Rph2.t	2_0441	AK365854	Thioredoxin-like protein
BW743	Rph2.t	2_1344	NA	NA
BW743	Rph2.t	1_1281	MLOC_11773.1	Trehalose-6-phosphate synthase
BW743	Rph2.t	2_0713	MLOC_68788.2	TBP-associated factor 2 LENGTH=1390
BW743	Rph2.t	2_0713	MLOC_68790.1	NA
BW743	Rph2.t	2_0713	MLOC_68789.5	NA
BW743	Rph2.t	2_1275	AK362143	TATA-binding related factor (TRF)
BW743	Rph2.t	2_1121	MLOC_34809.1	Cathepsin L-like cysteine proteinase
BW743	Rph2.t	2_1001	MLOC_59805.1	Golgi SNAP receptor complex member 1-1
BW743	Rph2.t	1_0127	MLOC_44755.2	Chlorophyll a-b binding protein 4
BW743	Rph2.t	1_0127	MLOC_44757.2	O-fucosyltransferase family protein
BW743	Rph2.t	1_0127	MLOC_44756.1	NA
BW743	Rph2.t	2_0236	MLOC_12614.1	Formate-tetrahydrofolate ligase
BW743	Rph2.t	2_1133	MLOC_5225.1	NA
BW746	Rph3.c	1_0182	MLOC_59546.1	V-type ATP synthase alpha chain
BW746	Rph3.c	2_1104	MLOC_12098.1	NA
BW746	Rph3.c	2_1104	AK364357	Mitochondrial substrate carrier family protein
BW746	Rph3.c	1_0078	MLOC_13526.1	NA
BW746	Rph3.c	1_0078	MLOC_13527.1	NA
BW746	Rph3.c	1_0797	MLOC_50091.1	Senescence-associated protein DH

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW746	Rph3.c	2_1160	MLOC_60883.1	NA
BW746	Rph3.c	2_1160	MLOC_60884.1	NA
BW746	Rph3.c	2_1160	MLOC_60880.1	NA
BW746	Rph3.c	2_1160	MLOC_60881.1	WD-repeat protein 57
BW746	Rph3.c	2_1160	MLOC_60885.1	NA
BW746	Rph3.c	2_1160	MLOC_60886.1	NA
BW746	Rph3.c	2_1160	MLOC_60882.1	NA
BW753	Rph4.d	2_0479	NA	NA
BW753	Rph4.d	1_0654	NA	NA
BW755	Rph5.e	2_0858	AK374872	RING-box protein 1a
BW755	Rph5.e	2_0858	MLOC_21379.2	1-deoxy-D-xylulose 5-phosphate reductoisomerase
BW756	Rph5.f	2_0858	AK374872	RING-box protein 1a
BW756	Rph5.f	2_0858	MLOC_21379.2	1-deoxy-D-xylulose 5-phosphate reductoisomerase
BW756	Rph5.f	2_0252	AK367001	NA
BW756	Rph5.f	1_1453	AK252051.1	Dephospho-CoA kinase
BW756	Rph5.f	1_1453	MLOC_61361.2	TPX2 (targeting protein for Xklp2) protein family
BW756	Rph5.f	2_0529	AK367229	Fructose-bisphosphate aldolase
BW756	Rph5.f	2_1398	AK248209.1	Glutamate decarboxylase
BW758	Rph7.g	2_0858	AK374872	RING-box protein 1a
BW758	Rph7.g	2_0858	MLOC_21379.2	1-deoxy-D-xylulose 5-phosphate reductoisomerase
BW758	Rph7.g	2_0159	MLOC_61066.1	NA
BW758	Rph7.g	2_0159	AK374643	NA
BW758	Rph7.g	2_0252	AK367001	NA
BW758	Rph7.g	1_1453	AK252051.1	Dephospho-CoA kinase, putative, expressed

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW758	Rph7.g	2_0529	AK367229	Fructose-bisphosphate aldolase
BW758	Rph7.g	2_1398	AK248209.1	Glutamate decarboxylase
BW758	Rph7.g	2_1027	MLOC_69438.1	Ethylene-responsive transcription factor
BW758	Rph7.g	2_0976	AK370400	NA
BW758	Rph7.g	1_0112	MLOC_64595.1	NA
BW758	Rph7.g	1_0112	AK249901.1	Bowman-Birk type trypsin inhibitor
BW758	Rph7.g	1_0886	AK366387	Mannose-6-phosphate isomerase
BW758	Rph7.g	2_0595	MLOC_36532.1	Ubiquitin-conjugating enzyme 22
BW759	Rph8.h	1_0216	AK372085	26S protease regulatory subunit 4
BW759	Rph8.h	2_1187	MLOC_7090.1	NA
BW759	Rph8.h	2_1187	AK369130	NA
BW759	Rph8.h	1_0787	MLOC_56743.1	Guanine nucleotide-binding protein-like 3
BW759	Rph8.h	1_0787	MLOC_56745.1	NA
BW759	Rph8.h	1_0891	MLOC_44215.2	Poly(A) polymerase-like protein
BW759	Rph8.h	1_0987	AK357120	Ovarian cancer-associated gene 2 protein-like
BW759	Rph8.h	1_0987	MLOC_34486.1	NA
BW760	Rph9.i	1_1507	MLOC_4808.1	NA
BW760	Rph9.i	1_1507	MLOC_4807.1	NA
BW760	Rph9.i	1_1507	MLOC_4809.1	NA
BW760	Rph9.i	1_1456	MLOC_4744.1	Aspartyl/glutamyl-tRNA(Asn/Gln) amidotransferase subunit C
BW760	Rph9.i	2_0003	MLOC_55532.1	GTP binding protein
BW760	Rph9.i	2_0003	MLOC_55533.1	NA
BW760	Rph9.i	1_0360	MLOC_37451.1	NA
BW760	Rph9.i	1_0360	AK250598.1	Chaperone protein dnaJ

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW760	Rph9.i	2_0653	MLOC_53946.1	NA
BW760	Rph9.i	2_0653	MLOC_53947.1	Pyruvate dehydrogenase E1 component beta subunit
BW760	Rph9.i	2_0653	MLOC_53948.1	expansin B4
BW760	Rph9.i	2_0653	MLOC_53949.2	Leucine-rich repeat receptor-like protein kinase
BW760	Rph9.i	2_1203	AK356347	Translation initiation factor eIF-2B delta subunit
BW760	Rph9.i	2_1325	MLOC_76072.2	Solute carrier family 22 member 1
BW760	Rph9.i	1_1375	MLOC_81731.1	Pentatricopeptide repeat-containing protein
BW760	Rph9.i	1_1375	MLOC_81732.1	NA
BW760	Rph9.i	1_0146	AK364599	Protein transport protein SEC23
BW760	Rph9.i	1_0146	MLOC_54787.1	Auxin responsive protein
BW760	Rph9.i	2_0259	MLOC_19607.1	NA
BW760	Rph9.i	2_1177	AK366333	Transmembrane emp24 domain-containing protein
BW760	Rph9.i	2_1247	MLOC_4610.1	NA
BW760	Rph9.i	2_1247	MLOC_4611.1	Protein kinase
BW760	Rph9.i	1_1090	AK373628	Transcription factor
BW760	Rph9.i	1_1090	MLOC_51340.2	Aluminum-induced protein-like protein
BW760	Rph9.i	1_0783	MLOC_66584.1	NA
BW760	Rph9.i	1_1080	AK364080	Cysteine proteinase
BW760	Rph9.i	1_0755	MLOC_16232.1	NA
BW760	Rph9.i	1_0755	MLOC_16233.2	Proteasome assembly chaperone
BW760	Rph9.i	1_0845	MLOC_56549.1	NA
BW760	Rph9.i	1_0845	MLOC_56550.1	Maternal effect embryo arrest 14
BW760	Rph9.i	2_1289	AK363763	Signal peptidase I
BW760	Rph9.i	2_1289	MLOC_63880.1	NA

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW760	Rph9.i	1_1532	AK367148	Molybdenum cofactor sulfurase
BW760	Rph9.i	2_0375	MLOC_65920.1	NADH-quinone oxidoreductase subunit I
BW760	Rph9.i	1_0292	AK371585	Succinate dehydrogenase subunit A
BW760	Rph9.i	1_1092	MLOC_10622.1	Citrate synthase
BW760	Rph9.i	1_1092	MLOC_10623.1	NA
BW760	Rph9.i	1_1092	MLOC_10625.1	NA
BW760	Rph9.i	1_1092	MLOC_10624.1	NA
BW760	Rph9.i	1_0104	MLOC_1273.1	60S ribosomal protein L44
BW760	Rph9.i	1_0104	MLOC_1275.1	NA
BW760	Rph9.i	1_0104	MLOC_1274.1	NA
BW760	Rph9.i	2_0676	MLOC_43074.1	NA
BW760	Rph9.i	2_0731	MLOC_6875.2	Reticulon-like protein B8
BW760	Rph9.i	2_0731	AK365628	NA
BW683	Rph10.o	1_0728	AK358661	Chitinase
BW683	Rph10.o	1_0281	MLOC_52444.1	Protein plastid transcriptionally active 16
BW683	Rph10.o	1_1191	MLOC_4753.2	Late embryogenesis abundant protein
BW683	Rph10.o	1_0335	MLOC_11772.1	RING finger and CHY zinc finger domain-containing protein 1
BW683	Rph10.o	1_1391	MLOC_56148.2	Putative inositol-1,4,5-trisphosphate phosphatase
BW683	Rph10.o	2_1305	MLOC_50950.1	NBS-LRR-like protein
BW683	Rph10.o	2_1305	MLOC_50949.1	NA
BW683	Rph10.o	2_0931	MLOC_63920.1	WD-40 repeat family protein
BW683	Rph10.o	1_1394	AK366039	ABC transporter ATP-binding protein
BW683	Rph10.o	1_1394	MLOC_5109.1	B3 domain-containing protein
BW683	Rph10.o	2_0115	AK376197	Maternal effect embryo arrest 22

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW683	Rph10.o	2_1358	MLOC_69131.1	NA
BW683	Rph10.o	2_1358	MLOC_69130.3	Zinc knuckle (CCHC-type) family protein
BW683	Rph10.o	2_1358	MLOC_69132.1	NA
BW684	Rph11.p	1_0107	MLOC_67524.1	Bidirectional sugar transporter N3
BW761	Rph9.z	1_1456	MLOC_4744.1	Aspartyl/glutamyl-tRNA(Asn/Gln) amidotransferase subunit C
BW761	Rph9.z	2_0003	MLOC_55532.1	GTP binding protein
BW761	Rph9.z	2_0003	MLOC_55533.1	NA
BW761	Rph9.z	2_0347	MLOC_5598.2	Methyl-CpG binding domain containing protein
BW761	Rph9.z	1_0360	MLOC_37451.1	NA
BW761	Rph9.z	1_0360	AK250598.1	Chaperone protein dnaJ
BW761	Rph9.z	1_0360	MLOC_37450.1	NA
BW761	Rph9.z	2_0653	MLOC_53946.1	NA
BW761	Rph9.z	2_0653	MLOC_53947.1	Pyruvate dehydrogenase E1 component beta subunit
BW761	Rph9.z	2_0653	MLOC_53948.1	Expansin B4
BW761	Rph9.z	2_0653	MLOC_53949.2	Leucine-rich repeat receptor-like protein kinase
BW761	Rph9.z	2_1203	AK356347	Translation initiation factor eIF-2B delta subunit
BW761	Rph9.z	2_0300	MLOC_35821.1	Ornithine aminotransferase
BW761	Rph9.z	1_0805	AK354421	NA
BW761	Rph9.z	1_0146	AK364599	Protein transport protein SEC23
BW761	Rph9.z	1_0146	MLOC_54787.1	Auxin responsive protein
BW761	Rph9.z	2_0259	MLOC_19607.1	NA
BW761	Rph9.z	2_0298	MLOC_60235.1	Histone H2A
BW761	Rph9.z	1_0705	MLOC_59423.4	NA
BW761	Rph9.z	1_0705	MLOC_59424.2	Tetratricopeptide repeat (TPR)-like superfamily protein

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW761	Rph9.z	2_1177	AK366333	Transmembrane emp24 domain-containing protein
BW761	Rph9.z	2_1247	MLOC_4610.1	NA
BW761	Rph9.z	2_1247	MLOC_4611.1	Protein kinase
BW761	Rph9.z	1_1090	AK373628	Transcription factor
BW761	Rph9.z	1_1090	MLOC_51340.2	Aluminum-induced protein-like protein
BW761	Rph9.z	1_0783	MLOC_66584.1	NA
BW761	Rph9.z	1_0095	MLOC_5287.1	UDP-glucose 6-dehydrogenase
BW761	Rph9.z	2_1241	MLOC_57253.1	receptor-like protein kinase 2
BW761	Rph9.z	1_1080	AK364080	Cysteine proteinase
BW761	Rph9.z	1_0755	MLOC_16232.1	NA
BW761	Rph9.z	1_0755	MLOC_16233.2	Proteasome assembly chaperone
BW761	Rph9.z	1_0845	MLOC_56549.1	NA
BW761	Rph9.z	1_0845	MLOC_56550.1	Maternal effect embryo arrest 14
BW761	Rph9.z	2_1289	AK363763	Signal peptidase I
BW761	Rph9.z	2_1289	MLOC_63880.1	NA
BW761	Rph9.z	1_1532	AK367148	Molybdenum cofactor sulfurase
BW761	Rph9.z	2_0375	MLOC_65920.1	NADH-quinone oxidoreductase subunit I
BW761	Rph9.z	1_0819	MLOC_39144.1	NA
BW761	Rph9.z	1_0819	MLOC_39140.1	NA
BW761	Rph9.z	1_0819	MLOC_39141.1	NA
BW761	Rph9.z	1_0819	MLOC_39143.3	Cytosolic purine 5-nucleotidase
BW761	Rph9.z	1_0819	MLOC_39142.1	NA
BW761	Rph9.z	1_0292	AK371585	Succinate dehydrogenase subunit A
BW761	Rph9.z	1_1092	MLOC_10622.1	Citrate synthase

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW761	Rph9.z	1_1092	MLOC_10625.1	NA
BW761	Rph9.z	1_1092	MLOC_10624.1	NA
BW761	Rph9.z	1_0104	MLOC_1273.1	60S ribosomal protein L44
BW761	Rph9.z	1_0104	MLOC_1275.1	NA
BW761	Rph9.z	1_0104	MLOC_1274.1	NA
BW761	Rph9.z	2_0568	AK370882	Aldehyde oxidase 1
BW761	Rph9.z	2_0676	MLOC_43074.1	NA
BW761	Rph9.z	2_0731	MLOC_6875.2	Reticulon-like protein B8
BW761	Rph9.z	2_0731	AK365628	NA
BW761	Rph9.z	2_0388	AK376464	YTH domain family protein
BW761	Rph9.z	2_0388	MLOC_68497.2	NA
BW761	Rph9.z	1_0363	AK368805	ABI3-interacting protein 2
BW761	Rph9.z	2_0104	MLOC_24232.1	NA
BW761	Rph9.z	2_0104	MLOC_24233.1	NA
BW761	Rph9.z	1_0217	MLOC_56767.1	NA
BW761	Rph9.z	1_0217	MLOC_56768.1	Eukaryotic translation initiation factor 3 subunit 9
BW761	Rph9.z	1_0589	MLOC_19781.1	NA
BW761	Rph9.z	1_0589	AK251250.1	NA
BW761	Rph9.z	1_0589	AK364297	DNA helicase family protein
BW761	Rph9.z	2_1355	AK359961	rRNA-processing protein EFG1
BW761	Rph9.z	1_1497	AK358288	P-loop containing nucleoside triphosphate hydrolases superfamily protein
BW761	Rph9.z	1_0901	MLOC_56328.1	NA
BW761	Rph9.z	1_0901	MLOC_56327.1	Non-specific lipid-transfer protein
BW761	Rph9.z	2_1041	AK252089.1	NA

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW761	Rph9.z	2_0078	MLOC_44136.1	NA
BW761	Rph9.z	2_0078	AK376547	NA
BW761	Rph9.z	1_0741	MLOC_61930.2	Cellulose synthase-5
BW761	Rph9.z	2_1024	AK358394	Phosphate transporter
BW761	Rph9.z	1_0902	AK358452	NA
BW761	Rph9.z	2_1452	AK363434	Early nodulin-like protein
BW761	Rph9.z	2_0876	MLOC_61508.1	NA
BW761	Rph9.z	2_0876	MLOC_61507.1	Mitochondrial Rho GTPase
BW761	Rph9.z	2_0876	AK367513	Remorin
BW761	Rph9.z	1_0336	AK366329	Development and Cell Death domain protein
BW761	Rph9.z	2_0988	MLOC_51705.1	NA
BW761	Rph9.z	2_0988	MLOC_51707.1	NA
BW761	Rph9.z	2_0988	MLOC_51706.2	CRAL/TRIO domain containing protein
BW761	Rph9.z	1_1464	MLOC_71880.1	Alkylated DNA repair protein AlkB
BW761	Rph9.z	1_1216	MLOC_55692.1	NA
BW761	Rph9.z	1_1216	AK249737.1	NA
BW761	Rph9.z	2_0546	AK252179.1	Phosphoesterase family protein
BW761	Rph9.z	2_0686	AK364134	Kinase family protein
BW761	Rph9.z	2_0686	MLOC_20051.2	Mitochondrial import inner membrane translocase subunit Tim10
BW761	Rph9.z	2_1012	MLOC_1045.1	NA
BW761	Rph9.z	2_0536	AK367079	Guanine deaminase
BW761	Rph9.z	2_0536	MLOC_13213.1	Pectin lyase-like superfamily protein
BW685	Rph13.x	1_1196	MLOC_52456.1	NA
BW685	Rph13.x	1_1196	MLOC_52455.4	NA

Appendix Table 9 continued. Annotated genes in the same contig as SNP markers residing in *Rph*-carrying introgressions.

BW Line ¹	Allele ²	BOPA_SNP ³	Gene on same contig ⁴	Annotation ⁵
BW685	Rph13.x	1_1196	MLOC_52458.1	NA
BW685	Rph13.x	2_0612	MLOC_37644.1	NA
BW685	Rph13.x	2_0612	MLOC_37645.1	Zinc finger CCCH domain-containing protein
BW685	Rph13.x	1_0842	MLOC_17018.1	Peptidyl-prolyl cis-trans isomerase
BW685	Rph13.x	2_0527	MLOC_60122.1	Hydroxyproline-rich glycoprotein-like
BW685	Rph13.x	2_0527	MLOC_60123.1	NA
BW685	Rph13.x	2_1428	MLOC_9078.1	RING/U-box superfamily protein
BW685	Rph13.x	2_1427	AK248786.1	Beta-glucosidase
BW685	Rph13.x	2_1427	MLOC_64032.1	NA
BW685	Rph13.x	2_1427	AK369539	NBS-LRR disease resistance protein homologue
BW685	Rph13.x	2_1272	MLOC_66627.2	Glycerol-3-phosphate dehydrogenase
BW685	Rph13.x	1_0681	MLOC_10046.2	Progesterone 5-beta-reductase
BW685	Rph13.x	1_0893	AK354147	Alkaline phytoceramidase
BW685	Rph13.x	1_0767	MLOC_114.1	NA
BW685	Rph13.x	1_0767	AK367356	Pentatricopeptide repeat-containing protein
BW685	Rph13.x	1_0767	MLOC_113.1	NA
BW685	Rph13.x	1_0343	MLOC_81141.1	NA
BW685	Rph13.x	1_0343	MLOC_81140.1	NA
BW686	Rph14.ab	2_0173	NA	NA
BW686	Rph14.ab	1_0342	MLOC_44617.2	Nucleolar protein 5
BW719	Rph15.ad	1_0399	MLOC_38808.1	GTPase, IMAP family member 4
BW719	Rph15.ad	1_0919	AK368803	Photosystem I reaction center subunit N
BW719	Rph15.ad	1_0919	MLOC_6378.3	60S ribosome subunit biogenesis protein NIP7
BW719	Rph15.ad	2_1153	MLOC_44144.1	CW-type Zinc Finger family protein
BW719	Rph15.ad	2_1153	MLOC_44146.1	Pentatricopeptide repeat-containing protein
BW719	Rph15.ad	2_1153	MLOC_44145.1	NA

Appendix Table 9 Footnotes.

- ¹. BW lines are the Bowman leaf rust resistance introgression lines whose introgressions carry annotated genes for defense.
- ². *Rph* introgressions carrying leaf rust resistance alleles and annotated defense genes.
- ³. BOPA SNP markers residing on the same genetic contig as annotated defense genes.
- ⁴. Annotated genes for defense residing on the same contig as Barley Oligonucleotide Polymorphic Assay (BOPA) SNP markers.
- ⁵. The listed gene annotation from the barley genome explorer provided by the Leibniz-Institut für Pflanzengenetik und Kulturpflanzenforschung (IPK, Gatersleben, Germany).