Leaf Rust of Cultivated Barley: Pathology and Control

Robert F. Park,[1] Prashant G. Golegaonkar,[1,2] Lida Derevnina,[1,3] Karanjeet S. Sandhu,[1] Haydar Karaoglu,[1] Huda M. Elmansour,[1] Peter M. Dracatos,[1] and Davinder Singh[1]

[1]Plant Breeding Institute Cobbitty, The University of Sydney, Narellan, NSW 2567, Australia; email: robert.park@sydney.edu.au

[2]Present address: Monsanto India Ltd., Hebbal, Bangalore 560092, India

[3]Present address: Genome Center, University of California, Davis, California 95616

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Abstract

Leaf rust of barley is caused by the macrocyclic, heteroecious rust pathogen *Puccinia hordei*, with aecia reported from selected species of the genera *Ornithogalum*, *Leopoldia*, and *Dipcadi*, and uredinia and telia occurring on *Hordeum vulgare*, *H. vulgare* ssp. *spontaneum*, *Hordeum bulbosum*, and *Hordeum murinum*, on which distinct parasitic specialization occurs. Although *Puccinia hordei* is sporadic in its occurrence, it is probably the most common and widely distributed rust disease of barley. Leaf rust has increased in importance in recent decades in temperate barley-growing regions, presumably because of more intensive agricultural practices. Although total crop loss does not occur, under epidemic conditions yield reductions of up to 62% have been reported in susceptible varieties. Leaf rust is primarily controlled by the use of resistant cultivars, and, to date, 21 seedling resistance genes and two adult plant resistance (APR) genes have been identified. Virulence has been detected for most seedling resistance genes but is unknown for the APR genes *Rph20* and *Rph23*. Other potentially new sources of APR have been reported, and additivity has been described for some of these resistances. Approaches to achieving durable resistance to leaf rust in barley are discussed.

INTRODUCTION

Rust: the disease that results from the interaction between a rust fungus and a host plant. Referring to the fungus only as rust is incorrect

The genus *Hordeum* belongs to the tribe *Triticeae* of the *Poaceae*, the largest monocot family. It comprises 32 species and 45 taxa, of which only *Hordeum vulgare* underwent domestication (136). Barley is considered to be one of the founding species of modern agriculture. It was domesticated approximately 10,000 years ago from the wild progenitor *H. vulgare* spp. *spontaneum*, most probably in the western part of the Fertile Crescent (8). A whole-genome sequence was recently completed for *H. vulgare*, with the size estimated at 5.1 gigabases (54).

Barley is an important cereal crop, ranking fourth in world food production after maize, rice, and wheat. In 2012, total barley production was 133 million metric tons from an area of 50 million hectares (31). It is also one of the hardiest cereal crops, growing in a wide variety of environments that include extremes of latitude and altitude to which other crops are not adapted (47). Because of its greater tolerance to soil salinity, barley can be grown in areas that are unsuitable for wheat (48). The major barley production areas are Europe, the Mediterranean fringe of North Africa, Ethiopia, the Middle East, Russia, China, India, Canada, the United States, and Australia. Barley is an important source of animal feed and brewing malts and is also important for human consumption.

The rust fungi (Phylum Basidiomycota, Teliomycetes, Pucciniales) are cosmopolitan in distribution and parasitize a wide range of plants, including ferns and conifers, and most families of dicotyledon and monocotyledon angiosperms. In nature, the rust fungi are highly specialized biotrophic plant pathogens with the capacity to change and acquire virulence to resistant cultivars, to build up rapidly, and to spread rapidly over great distances (70, 146).

Worldwide, cultivated barley can be affected by four rust diseases: leaf (brown) rust caused by *Puccinia hordei*, crown rust caused by *Puccinia coronata* var. *hordei*, stripe rust caused by *Puccinia striiformis* f. sp. *hordei*, and stem rust caused by *Puccinia graminis*. In Australia, stem rust in barley is caused by three special forms (formae speciales) of *P. graminis*: the form virulent on wheat and triticale (*P. graminis* f. sp. *tritici*), the form virulent on cereal rye (*P. graminis* f. sp. *secalis*), and a putative somatic hybrid between these two formae speciales, referred to as Scabrum rust (93). Among these diseases, leaf rust is the most important and can be the most devastating (92). This disease has increased in importance in recent decades in temperate barley growing regions, presumably because of more intensive agricultural practices. In this review, we provide an overview of leaf rust of barley, covering the pathogen and its host, its economic importance, and approaches to control with a particular focus on genetic control and future prospects.

BIOLOGY, NOMENCLATURE, AND LIFE CYCLE

Leaf rust (dwarf leaf rust, or brown rust) of barley is caused by the pathogen *Puccinia hordei* G. Otth. (syn. *Puccinia rubigo-vera* var. *simplex* Körn., *Puccinia simplex* Eriks. et Henn., and *Puccinia anomala* Rostr.). The uredinia of *P. hordei* occur mainly on the upper but also on the lower side of leaf blades and also form on leaf sheaths of barley (**Figure 1**), appearing as small orange-brown pustules up to 0.5 mm in size, which are scattered and may be surrounded by chlorotic halos or green islands (**Figure 2**). In the case of severe infection under high inoculum load, stems, glumes, and awns can also be infected. Later in the season, particularly on leaf sheaths but also on stems, heads, and leaf blades, blackish-brown telia are formed usually in stripes and covered by the epidermis (**Figure 3**). Occasionally, telial formation is observed on seedlings under greenhouse conditions.

P. hordei is a macrocyclic, heteroecious rust pathogen. Aecia have been reported from selected species of the genera *Ornithogalum*, *Leopoldia*, and *Dipcadi*, all within the Liliaceae, and uredinia and telia occur on *H. vulgare*, *H. vulgare* ssp. *spontaneum* (2, 66), *Hordeum bulbosum*, and *Hordeum murinum* (2). Although the pathogen occurring on *H. murinum* was considered to be a separate

Figure 1

Leaf rust of barley caused by *Puccinia hordei* (image courtesy of Professor Robert F. Park).

species, *P. hordei murini*, cross inoculation studies on both the telial and aecial hosts by Anikster (1, 3) led to the conclusion that it should be regarded as *P. hordei*. In these studies, teliospore samples from all four *Hordeum* taxa infected *Ornithogalum eigii* and *Ornithogalum trichophyllum* (3) and *Ornithogalum brachystachys*, *Dipcadi erythraeum*, and *Leopoldia eburnea* (1). Studies using uredinial isolates established from the four hosts demonstrated the existence of distinct parasitic specialization, with isolates from *H. vulgare* and *H. vulgare* ssp. *spontaneum* infecting only these host species, and isolates from *H. bulbosum* and *H. murinum* infecting only the host from which they were originally isolated (1, 3). A subsequent study of ITS (internal transcribed spacer) sequences confirmed that isolates of *P. hordei* from all four *Hordeum* taxa were very closely related (128). On the basis of the range of host species infected in the telial and aecial stages of the pathogen life cycle, Anikster (3) concluded that *P. hordei* has undergone biogenic radiation by expansion from the primary telial host to a wider range of aecial hosts.

Figure 2

Leaf rust of barley showing green islands around pustules (image courtesy of Professor Robert F. Park).

Figure 3

Telia of *Puccinia hordei* formed on leaf sheaths (image courtesy of Dr. Karanjeet Sandhu).

The importance of the alternate host in the life cycle varies globally. It is unimportant in places such as Europe where the growth of *Ornithogalum* does not coincide with the germination of teliospores (4). In contrast, in Israel, where *Ornithogalum* species and wild *Hordeum* species coexist, the alternate host is essential for the survival of the pathogen and for the generation of pathogenic variability (2). In Australia, *Ornithogalum umbellatum* occurs in localized parts of the Yorke Peninsula of South Australia (138; **Figure 4**). In this region, conditions are suitable for germinating basidiospores to infect the alternate host, producing both pycniospores and aeciospores in abundance (138). Despite this, observations on the annual epidemic development of the disease and pathotypic composition over the past 25 years suggest that the telial stage is more important in the survival of the pathogen in Australia (92; R.F. Park, unpublished results).

ECONOMIC IMPORTANCE

Alternate host: one or the other of the different hosts infected by heteroecious rust fungi

Although the economic importance of barley leaf rust depends on the region in the world and differs from year to year (90), it appears that it has increased in recent years (19, 22, 46, 90). Although its incidence is sporadic, it is probably the most common and widely distributed rust disease of barley, occurring throughout all the barley growing areas of North Africa, Europe, New Zealand, Australia, the eastern and Midwestern United States, and some parts of Asia, where severe yield losses occur in susceptible varieties, particularly in areas where crops mature late

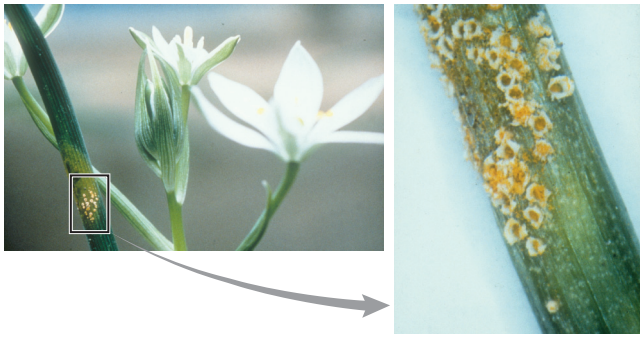

Figure 4

Aecia on *Ornithogalum umbellatum* (images courtesy of Dr. Hugh Wallwork, South Australian Research and Development Institute).

(11, 13, 15, 92, 101, 119, 149, 151). Significant yield losses have been reported from New Zealand (5), Australia (20), North America (46, 81), the Czech Republic (29), the United Kingdom (56, 83), Ethiopia (118), and South Africa (135). Murray & Brennan (86) estimated that in Australia the disease causes economic losses of AUD$9 million per year. Barley leaf rust survey data from the United Kingdom indicate that between 2001 and 2005, the disease caused national yield losses of £2.4 million a year (at £100 per ton) despite chemical treatment (30). In Australia, barley leaf rust epidemics were first reported in New South Wales in the 1920s (142). However, between the 1920s and 1970s little documentation of the occurrence of *P. hordei* was made. A move toward more intensive barley production and early and extended crop planting, coupled with cultivar susceptibility, is believed to have contributed to increased levels of barley leaf rust in Australia, with major epidemics observed in Queensland (1978, 1983, 1984, 1988), South Australia (1988), northern New South Wales, and Tasmania (1990) (20).

Although total crop loss does not occur, under epidemic conditions, yield reductions of up to 62% have been reported in susceptible varieties (15, 20, 46, 132). On the basis of disease severity, it was predicted that for each 1% increment in leaf rust on the flag leaf, penultimate leaf, or whole plant, there is a potential yield loss of 0.77% (83), 0.40%, (69), and 0.60%, respectively. Griffey et al. (46) estimated grain yield losses of 31 kg/ha (0.42%) for each 1% increment of leaf rust severity on the upper two leaves at the early dough stage of plant development. Lim & Gaunt (75) established that leaf rust epidemics occurring after growth stage 75 had little effect on grain yield, although they reported yield losses of 20% when disease was present at early growth stages. Decreased kernel weight resulting in yield reduction has also been reported for late season epidemics (81).

The major damage from *P. hordei* is from loss of photosynthetic area and reduced root and shoot growth, which lead to a stunted plant, shriveled kernels, and reduction in fertile tiller numbers, markedly reducing grain yield and seed quality of the crop (15). Shriveled seeds also result in reduced protein content in the grain, thus affecting marketing for malting purpose (22, 46). Newton et al. (89) reported adverse effects of leaf rust on malting quality due to reduced percentage of heavy grade kernels.

GENETIC RESISTANCE

The first study of the inheritance of resistance to *P. hordei* in barley was undertaken by Waterhouse (142). Since then, studies of seedling (major or all stage) resistance, partial resistance,

Table 1 The source and chromosomal locations of designated genes conferring resistance to *Puccinia hordei* in barley

Gene[a]	Source	Species of origin	Location (chromosome)	Typical low infection type[b]	References
Rph1	Oderbrucker	*Hordeum vulgare*	2H	;1N	134, 131, 143
Rph2	Peruvian	*H. vulgare*	5HS	;12–N	10, 35, 124
Rph3	Estate	*H. vulgare*	7HL	0;C	59, 124
Rph4	Gold	*H. vulgare*	1HS	;12–	134, 131
Rph5	Magnif 104	*H. vulgare*	3HS	;N	80, 114, 124, 131, 134
Rph6	Bolivia	*H. vulgare*	3HS	2C	114, 124, 153
Rph7	Cebada Capa	*H. vulgare*	3HS	0;N	13, 45, 96, 124
Rph8	Egypt 4	*H. vulgare*	N/A[c]	2CN	129
Rph9	HOR2596	*H. vulgare*	5HL		9, 130
Rph10	Clipper BC8	*H. vulgare* ssp. *spontaneum*	3HL	12–	34
Rph11	Clipper BC67	*H. vulgare* ssp. *spontaneum*	6HL	2+	34
Rph12	Triumph	*H. vulgare*	5HL	;2 = N	9, 59, 140
Rph13	PI 531849	*H. vulgare* ssp. *spontaneum*	5HL	;1–N	58, 60
Rph14	PI 584760	*H. vulgare*	2HS	;12–N	41, 58
Rph15	PI 355447	*H. vulgare* ssp. *spontaneum*	2HS	;+N	14, 147
Rph16	HS078/HS084	*H. vulgare* ssp. *spontaneum*	2HS	0;NC	55
Rph17	81882/BS1	*H. bulbosum*	2HS	12C	105, 108
Rph18	38P18/8/1/10	*H. bulbosum*	2HL	0;	107
Rph19	Prior	*H. vulgare*	7HL	;1N	95
Rph20	Flagship	*H. vulgare*	5HS	N/A	42, 51, 76
Rph21	Ricardo	*H. vulgare*	4H	12+C	116
Rph22	182Q20	*H. bulbosum*	2HL	;C	63
Rph23	Yerong	*H. vulgare*	7HS	N/A	121

[a] *Rph5/Rph6* (153), *Rph9/Rph12* (9), and *Rph15/Rph16* (147) were reported to be allelic.
[b] See **Table 4**.
[c] The chromosomal location of *Rph8* has not been determined.

and adult plant resistance (APR) have characterized specific loci conditioning resistance [given the designation *Rph* (previously *Pa* for *P. anomala*)] as well as QTLs (quantitative trait loci). To date, 21 loci conferring hypersensitive seedling resistance (*Rph1* to *Rph19*, *Rph21*, and *Rph22*) and 2 APR genes (*Rph20* and *Rph23*) (51, 92, 95, 116, 121) from *H. vulgare*, *H. vulgare* subsp. *spontaneum*, and *H. bulbosum* have been designated (**Table 1**). An allele designation system was proposed by Franckowiak et al. (35) in which a letter is assigned to loci in cultivars with different alleles at an *Rph* locus. Several of the *Rph* genes designated to date appear to comprise two or more alleles (see below).

Adult plant resistance (APR): resistance that becomes effective at post-seedling growth stages. Often referred to as minor gene resistance

Seedling Resistance

The first study of the genetics of resistance to leaf rust in barley was published in 1927, when Waterhouse studied the inheritance of resistance to leaf rust in six Australian barley cultivars:

Californian feed, O.A.C. 2, Cape, Manchuria, Minn. II 21.15, and Minn. 21.17. He showed that the resistance in all was due to monogenic dominant genes. In a second study, Waterhouse (143) demonstrated that six barley cultivars carried resistance genes at the same locus. Watson & Butler (145) showed that the genes for resistance to leaf rust in Minn. II 21.15 and No. 22 were different and not allelic, and designated the genes *Pa1* and *Pa2*, respectively. Oderbrucker, a differential genotype used by Waterhouse (143), carried a gene at the same locus as Minn. II 21.15 (145). Henderson (49) designated two genes *Pa* and *Pa1*. He showed that the varieties Weider, Bolivia, Purple Nepal, Modia, Morocco, Barley 305, Ricardo, and Marco had a common single gene (*Pa*; now *Rph2*) for resistance to leaf rust, whereas the variety Estate had gene *Pa1* (*Rph3*).

Results of several studies have suggested that *Rph2* is a complex locus comprising many alleles (35). Roane (112) conducted a series of genetic studies to determine the number of loci conditioning leaf rust reaction in nine North American differential varieties and identified four loci, designated A, B, C, and D. Reka 1 and Bolivia possessed the A locus, whereas Quinn possessed both the A and the B loci. Oderbrucker, Speciale, and Sudan possessed locus C, and locus D was present in the differential genotypes Gold and Lechtaler. Resistance to leaf rust in barley has also been described by several other workers (85, 124, 155); however, the relationships between the genes identified in these studies were not resolved.

A series of experiments was conducted by Roane & Starling (113, 114) to resolve the genetic relationships between seedling resistance genes that had been identified by previous workers. On the basis of genetic relationships, they described a series of genes, *Pa1* to *Pa6*, in the barley differential set based on the results of reaction to an isolate (race 4, isolate 57–19) of *P. hordei*. The genes were given the designation *Pa* because at that time *P. hordei* was referred to as *P. anomala*. Following the adoption of the name *P. hordei*, Moseman (84) suggested changing the gene symbols *Pa1–Pa6* to *Rph1–Rph6*. Bolivia was shown to carry two loci, i.e., *Rph2* and *Rph6* (109). Zhong et al. (153) separated the *Rph6* locus of Bolivia using *P. hordei* pathotype ND8702. The locus was positioned on chromosome 3HS and shown to be allelic to the *Rph5* locus of Magnif 104 (153). Resistance gene *Rph7* was identified in the cultivar Cebada Capa (25, 62, 124). This gene was considered to be at the same locus as *Rph5* (114). Johnson (62), however, indicated that Cebada Capa carried a dominant gene that differed to all genes from *Rph1* to *Rph6*. This gene was designated as *Pa-y* and was thought to be similar to the dominant gene present in Forrajera Klein, La Estanzuela, and H2212. Frecha (36, 37) studied linkage relationships between *Pa5* and *Pa-y*. He reported that the *Pa5* resistance locus of Quinn was closely linked to the *Pa-y* resistance locus of Forrajera Klein, with a recombination value of approximately 8%. However, genetic analysis of resistance in Cebada Capa, La Estanzuela, H2212, and Forrajera Klein suggested that they all carried *Rph7* (96). Yahyaoui et al. (152) reported new sources of resistance to *P. hordei* in the Tunisian landraces Tu17, Tu27, and Tu34. Genetic analysis and allelism tests between Tu17 and a stock carrying *Rph7* suggested that the gene carried by Tu17 is an allele of *Rph7* (14). The symbols *Rph8* (129) and *Rph9* (17, 130) were allocated to loci conferring resistance against *P. hordei* in Egypt 4 and Hor2595 (CI 1243), respectively. It was speculated that *Rph9* might be similar to the resistance found in the German cultivar Trumpf (also known as Triumph) (141). Further tests with different isolates suggested that *Rph9* and Triumph exhibited different infection types. A genetic analysis of Triumph indicated that the resistance was governed by three genes (two dominant and one recessive) (140). In another study, a single resistance gene was identified in Triumph and designated *Rph12* (59). The genetic relationship between *Rph9* and *Rph12* was subsequently resolved by Borovkova et al. (9), who proved that *Rph12* and *Rph9* are allelic. A third allele at this locus was recently described by Dracatos et al. (27).

Feuerstein et al. (34) described two leaf rust resistance loci derived from *H. vulgare* ssp. *spontaneum* that had been backcrossed into cv. Clipper. These loci were different from other reported

Seedling resistance: resistance that is effective at all growth stages; often referred to as major gene resistance

Race (pathotype, strain): a group of isolates that share common pathogenic attributes, which may or may not be genetically identical

Rph genes and were designated *Rph10* and *Rph11*. Jin et al. (58) studied inheritance of leaf rust resistance in four barley accessions (PI 531840, PI 531841, PI 531849, and PI 584760) as well as their allelic and linkage relationships with other *Rph* genes. The resistance in each accession was governed by a single locus. Incomplete dominant inheritance was observed in accessions PI 531841 and PI 584760, whereas a completely dominant inheritance was observed in PI 531840 and PI 531849. Allelism tests between PI 531841 and PI 531840 suggested that the same resistance locus was present in both and that it was allelic to *Rph2*. The linkage relationships with other *Rph* genes indicated that the locus providing resistance in PI 531841 and PI 531840 was linked with *Rph5* with recombination frequencies of 33.8 ± 3.8% and 17.0 ± 3.5%, respectively. This contrasts with the results of molecular mapping of *Rph5* and *Rph2* that showed *Rph5* was located on the short arm of barley chromosome 3H (80) and that *Rph2* was located on the short arm of chromosome 5H (10, 35). The resistances in PI 531849 and PI 584760 were not allelic to previously identified loci. New allele symbols, *Rph13* and *Rph14*, were therefore given to the resistances in PI 531849 and PI 584760, respectively. Jin et al. (61) identified several potential sources of resistance to *P. hordei* in the *H. vulgare* ssp. *spontaneum* accessions PI 354937, PI 355447, PI 391024, PI 391069, PI 391089, PI 466245, and PI 646324. Genetic studies of these accessions demonstrated a common single locus governing resistance against *P. hordei*. The locus was not allelic to previously identified loci and was given the new allele symbol of *Rph15* (14). Ivandic et al. (55) reported a new gene in two accessions of *H. vulgare* ssp. *spontaneum*. The gene was effective against a wide range of *P. hordei* pathotypes, including several from Israel, Morocco, and the United States that were virulent on *Rph*7. This gene was designated *Rph16*, and it was mapped to chromosome 2HS. Recent molecular and allelism studies revealed that *Rph15* and *Rph16* are allelic (147). Derevnina et al. (24) undertook a comprehensive analysis of the allelic relationship among genes described on chromosome 2H: *Rph14*, *Rph15*, *Rph17*, and *RphHOR1063*, *RphZhu4*, and *RphHOR15560* (all three reported to map same region as *Rph16*). Their analysis suggested that resistance genes in Zhu 4 and HOR 1063 were allelic to both *Rph14* and *Rph15*, while *Rph14* and *Rph15* were possibly independent. *Rph17* and *RphHOR15560* were shown to be independent of each other and of *Rph14* and *Rph15*. Because the resistance *RphZhu4* and *RphHOR1063* were shown to be allelic to *Rph14*, the designations *Rph14.am* and *Rph14.an*, respectively, were proposed.

Pickering and colleagues (103, 107, 108) intercrossed a colchicine-induced autotetraploid *H. bulbosum* (accession HB2032) with diploid *H. vulgare* (cv. Emir), and the resulting partially fertile triploid hybrid was backcrossed to Emir. The recombinants obtained by this method were assessed for resistance to leaf rust. Two introgressions of *H. bulbosum* chromatin conferred resistance to leaf rust. The resistance loci in the stocks were designated *Rph17* and *Rph18*. An unknown resistance gene present in the differential cultivar Reka 1 (129) and several other Australian cultivars also present in Prior (18) were characterized by Park & Karakousis (95). This locus was designated *Rph19* and was mapped on chromosome 7HL. It was shown to be linked with *Rph3* with a recombination distance of 28 ± 4.3 cM.

Apart from the *Rph8* locus, all designated seedling leaf rust resistance genes have been assigned to a chromosome or a specific chromosomal region (**Table 1**). Various methods have been used to characterize genes providing resistance to leaf rust in barley, including trisomic analysis (131, 134), linkage with morphological markers (59), linkage with isozyme markers (34), and linkage with DNA-based markers (147).

Although the loci *Rph6*, *Rph8*, *Rph10*, *Rph11*, *Rph14*, *Rph15*, *Rph16*, *Rph17*, *Rph18*, and *Rph22* have been described only from single genetic stocks, all other loci have been reported in a range of barley genotypes through either tests of allelism, gene postulation from multipathotype testing, or marker analyses (11, 18, 22, 23, 29, 43, 90, 96, 117).

Partial Resistance

Parlevliet & Ommeren (100) proposed the term partial resistance to describe resistance to leaf rust in certain barley genotypes. They differentiated partial resistance from seedling resistance and APR, indicating that the host is susceptible at all growth stages but the infection frequency, latent period, rate of spore production, and period of spore production may vary. Selection for partial resistance is often difficult in field plots, as all genotypes show a susceptible reaction (100).

Neervoort & Parlevliet (88) studied the components of partial resistance to leaf rust in eight barley cultivars. They observed substantial variation among the cultivars for each component. Among these components, latent period was found to be the most crucial factor in partial resistance. In a further study, Parlevliet (97) reported that the latent period was governed by many genes that were additive in nature. On the basis of latent period, several Western European cultivars were shown to have variable levels of partial resistance to *P. hordei* (99). Histological studies of partial resistance in the barley cultivar Vada demonstrated early abortion of hyphal growth of fungal spores at adult plant growth stages, in contrast to seedling growth stages (98).

The partial resistance of Vada and the line TR306 has undergone detailed genetic analyses. Spaner et al. (122) found three QTLs conferring resistance in a cross between Harrington and the resistant line TR306. These QTLs were located on 5H, 2H, and 6H, and explain 45% of the total phenotypic variation. Qi et al. (110) conducted a molecular analysis of the partial resistance of Vada at seedling and adult plant growth stages using a high-density amplified fragment length polymorphism (AFLP) marker linkage map of a population derived from a cross between Vada and the susceptible line L94. Three QTLs, *Rphq1*, *Rphq2*, *Rphq3*, were effective at the seedling stage, whereas four QTLs, *Rphq2*, *Rphq3*, *Rphq4*, and *Rphq5*, were effective at adult plant growth stages. Two QTLs (*Rphq2* and *Rphq3*) were consistently detected at both seedling and adult plant growth stages.

Race specificity for partial resistance was demonstrated by Qi et al. (109). They identified an additional four QTLs for a long latent period in cultivar Vada when tests for long latent period were conducted using two pathotypes of *P. hordei*. Out of the four QTLs, *Rphq*7 was effective at the seedling stage, whereas *Rphq8*, *Rphq9*, and *Rphq10* were effective at adult plant growth stages. An additional three QTLs were described from a cross between L94 and the partially resistant barley line 116-5, derived from a cross between Cebada Capa and L94. Only two QTLs, *Rphq2* and *Rphq3*, which were mapped to 2HL and 6HS, were consistently effective in both studies at all growth stages against both races (109, 110). Interestingly, molecular mapping using the population Vada by IB-87 identified only two QTLs responsible for resistance against *P. hordei* (7), which were mapped on 2HL and 6H. Fufa & Hundie (38) further evaluated the performance of four QTLs [*Rphq13* (7H), *Qch2* (2H), *Rphq10* (4H), and *Rphq3* (6H)] originally mapped in studies of Qi et al. (110) under natural infection and epidemic development in different environments of Ethiopia. Their studies concluded that all tested QTLs were effective under natural epidemic development across environments except *Rphq3*. Backes et al. (7) suggested a close relationship between the quantitative and qualitative types of resistance due to colocalization of QTLs and resistance gene analogs (RGAs). This has been observed on a number of occasions in various host-pathogen relationships (67, 74). Molecular mapping of several other QTLs in barley has resulted in them being localized on previously mapped qualitative resistance genes. This has been reported for powdery mildew (6), net blotch disease (111), stripe rust (133), and leaf rust (68, 133). These contrasting results on quantitative and qualitative resistance against pathogens warrant further analysis and demonstrate the value of knowing the genotypes of host and pathogen in interpreting data applied to map-based genetic analysis.

Adult Plant Resistance

APR is only expressed during post-seedling growth stages. It has been well characterized and utilized in wheat to control rust diseases, with some APR genes conferring high levels of resistance (e.g., *Lr12*) and others conferring intermediate levels of resistance (e.g., the pleiotropic *Lr34/Yr18/Sr57* locus). Golegaonkar et al. (41) showed that many barley cultivars reported to carry partial resistance also carried APR, and it appears that these terms have at times been used interchangeably despite the fact that genotypes such as Vada display clear resistance in the field rather than the susceptibility associated with partial resistance.

Although numerous QTLs have been identified for both partial resistance at the seedling stage and APR to *P. hordei*, in only a few instances have genetic stocks for such QTLs been cataloged and markers developed for marker-assisted selection (MAS). In barley, only two genes conferring APR to *P. hordei* have been characterized and designated (*Rph20* and *Rph23*) on chromosome 5HS and 7HS, respectively, based on consistent detection in diverse field nurseries over multiple seasons and QTL mapping analysis (42, 51, 121).

Detailed integrated greenhouse and field testing of barley germplasm from Australia, China, Germany, Spain, and Uruguay with specific pathotypes of *P. hordei* allowed the identification of APR in 213 barley genotypes (23, 41, 117). Screening of the genotypes with PCR-based markers closely linked to *Rph20* (*bPb0837*) and *Rph23* (*EBmac0603*) indicated that some 93% carried one or more uncharacterized APR genes with or without *Rph20* and *Rph23* (28). On the basis of the presence of the linked markers *EBmac0603* and *bPb0837*, both *Rph20* and *Rph23* were postulated as being present in the German cultivars "Lenka" and "Volla," and in the Australian cultivar "Macquarie." Genetic analyses established that gene *Rph23* (121) and an uncharacterized APR gene in the Chinese cultivar "Zhoungdamei" (23) acted in an additive manner when each was present with *Rph20*.

A recent study was able to identify new sources of APR and existing seedling resistance genes using an association mapping approach (154). The utilization of novel QTLs for APR to *P. hordei* in breeding programs will depend on fine-mapping population development amenable to effective phenotypic assessment, genetic stocks of resistance donor sources, and marker development. For pyramiding APRs in barley, MAS can provide the most rapid advances to breeders; once identified, diagnostic markers can be applied without the requirement of rigorous field testing under diverse environmental conditions that often effect the expression of APR.

PATHOGENIC VARIATION

Surveys of pathogenic variability in cereal rust pathogens, including *P. hordei*, are conducted in many parts of the world, and typically involve identifying pathotypes present in rust samples collected from crops, volunteer (self-sown) cereals, rust susceptible grass species, and experimental plots (including breeders' plots and rust trap nurseries) in greenhouse assays of virulence using genotypes carrying different resistance genes (differential genotypes).

Differential Genotypes and Pathotype Identification

Working in the United States, Mains (78) was the first to demonstrate the existence of races (pathotypes) within *P. hordei*. Subsequent studies in countries, including Australia (142), Canada (12), Germany (50), Argentina (52), and Portugal (26), identified additional pathotypes, but because the host genotypes used in each study differed, it was not possible to compare the pathotypes detected. Levine & Cherewick (73) demonstrated that 9 of 16 differential genotypes used in these

Table 2 Nine barley genotypes identified by Levine & Cherewick (73) to differentiate isolates of *Puccinia hordei* and to allocate Unified (UN) Race designations

Genotype	Accession	Notes	*Rph* locus
Speciale	C.I. 7536	Hey's *Hordeum vulgare speciale*	*Rph1*
Reka 1	C.I. 5051	Waterhouse's Reka 1 and Hey's Australische Recka	*Rph1*, *Rph19*
Sudan	C.I. 6489	Hey's Sudan or Aegyptische Sudan	*Rph1*
Bolivia	C.I. 1257	Mains' Bolivia, C.I. No. 1257	*Rph2*, *Rph6*
Oderbrucker	C.I. 940	Mains' Oderbrucker, C.I. No. 940	*Rph1*
Quinn	C.I 1024	Mains' Quinn, C.I. No. 1024	*Rph2*, *Rph5*
Egypt 4	C.I. 6481	Ronsdorf's Aegyptische 4 zeilige	*Rph8*
Gold	C.I. 1145	Mains', also Brown's, Gold, C.I. No. 1145	*Rph4*
Lechtaler	C.I. 6488	Hey's Lichtis Lechtaler	*Rph4*

studies were of most use in differentiating isolates of *P. hordei* from North America, Europe, and Australia. A dichotomous key based on these nine differentials was used to identify 52 variants of *P. hordei* that coded as unified (UN) races. Later studies showed that some of these differentials share the same resistance gene (**Table 2**).

Clifford (16) proposed the use of 10 standard differential genotypes to monitor virulence in *P. hordei* and an octal system as first described by Gilmour (39) to designate pathotypes (**Table 3**). The differential set used in Australia is based on these 10 genotypes, but with Estate replacing Ribari and with differentials for *Rph10*, *Rph11*, *Rph12*, and *Rph19* added (92). To obtain a unique octal notation, differential genotypes are assigned a fixed linear order and grouped into sets of three. A binary number is initially assigned to each differential genotype, allowing the allocation of binary triplet numbers for each set of three differential genotypes. This system has been adopted widely, and in Australia, the suffix P+ or P− is added to each octal designation to indicate virulence or avirulence, respectively, for the resistance gene *Rph19*, present in the differential cultivar Prior. Additional differential genotypes representing newly designated and temporarily designated *Rph* loci are used in some cases (**Table 3**).

Virulence has been reported for most seedling *Rph* genes. Cotterill et al. (19) reported that most characterized genes were ineffective against pathotypes identified in Australia during 1966 to 1995, and only *Rph3* and *Rph7* were considered to be suitable for protecting Australian barley cultivars from the disease. In a more recent study, Park (92) reported that in addition to *Rph3* and *Rph7*, the newly described genes *Rph11*, *Rph14*, *Rph15*, and *Rph18* were also effective under Australian conditions with prevailing pathotypes. However, pathotypes virulent to *Rph3* were detected in New Zealand (21) and have since been detected in Australia (R.F. Park, unpublished results). Although *Rph7* has provided resistance against leaf rust in Europe, virulence for *Rph7* has been identified in Israel (40), Morocco (101), and North America (125). Virulence for *Rph11* and *Rph14* has also been found frequently in many parts of the world (32), and virulence to *Rph15* was reported by Sun et al. (127).

Infection Types

Stakman et al. (123) developed a scale to assess rust diseases on seedlings on the basis of infection type. The original scale was developed for rating leaf rust and stem rust of wheat at seedling growth stages, and has now been adapted for several other rust diseases including *P. hordei*. Waterhouse (142) first described an infection type scale for *P. hordei*, and this was later refined by Levine &

Table 3 Differential genotypes used to allocate octal designations to pathotypes of *Puccinia hordei*, along with supplemental differentials used in Australia

Host genotype	Resistance gene(s)	Differential set	Octal value
Sudan	*Rph1*	International	1
Peruvian	*Rph2*	International	2
Estate	*Rph3*	International	4
Gold	*Rph4*	International	10
Magnif 104	*Rph5*	International	20
Bolivia	*Rph2* + *Rph6*	International	40
Cebada Capa	*Rph7*	International	100
Egypt 4	*Rph8*	International	200
Abyssinian	*Rph9*	International	400
Clipper BC8	*Rph10*	International	1000
Clipper BC67	*Rph11*	International	2000
Triumph	*Rph12*	International	4000
Gus	None	Australian supplemental	–
Berg	*Rph1*	Australian supplemental	–
Reka 1	*Rph2* + *Rph19*	Australian supplemental	–
Ricardo	*Rph2* + *Rph21*	Australian supplemental	–
Quinn	*Rph2* + *Rph5*	Australian supplemental	–
PI 531849	*Rph13*	Australian supplemental	–
PI 584760	*Rph14*	Australian supplemental	–
PI 355447	*Rph15*	Australian supplemental	–
Prior	*Rph19*	Australian supplemental	–
Q21861	*RphQ*	Australian supplemental	–
Cantala	*RphCantala*[a]	Australian supplemental	–

[a]Shown to be a third allele at the *Rph9/Rph12* locus (27).

Cherewick (73). On the basis of these two scales, Park & Karakousis (95) proposed the scale shown in **Table 4**. The letters c and n are included to indicate greater than normal chlorosis or necrosis, respectively. The symbols − and = indicate infection types that are lower than normal, and + and ++ indicate infection types higher than normal. Infection types of 3+ or higher are considered as compatible (i.e., virulent pathogen/susceptible host). Typical low infection types for each class are illustrated in **Figure 5**. To capture a phenotype fully, it is often necessary to use a combination of two or more descriptors (e.g., ;12−).

Origins of Pathogenic Variability

Pathogenicity: the pathogen character determined by the interaction with a host, the contrasting phenotypes of which are avirulence and virulence

Although it is known that alternate host species contribute to the epidemiology of *P. hordei* in several regions, few studies have looked at their role in generating new pathotypes. Studies of isolates derived from alternate host species have established that new virulence combinations are generated via sexual recombination in Israel (2). In Australia, five distinct pathotypes were established from seven isolates established from aeciospores collected from natural infection of *O. umbellatum* in 1990 (138).

Few long-term studies of pathogenic variability in *P. hordei* have been conducted. Following periodic monitoring of the pathogenicity of *P. hordei* in Australia beginning in the 1920s

Table 4 Major infection type classes used to score leaf rust responses on barley at seedling growth stages

Infection type	Host response	Description
0	Immune	No visible symptoms
;	Very resistant	Hypersensitive flecks
1	Resistant	Minute uredinia surrounded by mainly necrotic tissue
2	Resistant to moderately resistant	Small- to medium-sized uredinia surrounded by chlorotic and/or necrotic tissue, often appearing as green islands
3	Moderately resistant to moderately susceptible	Medium to large uredinia with surrounding chlorosis
4	Susceptible	Large uredinia, without chlorosis or necrosis
X	Resistant	Mesothetic, heterogeneous infection types similarly distributed over the leaf, cholorosis and/or necrosis usually present

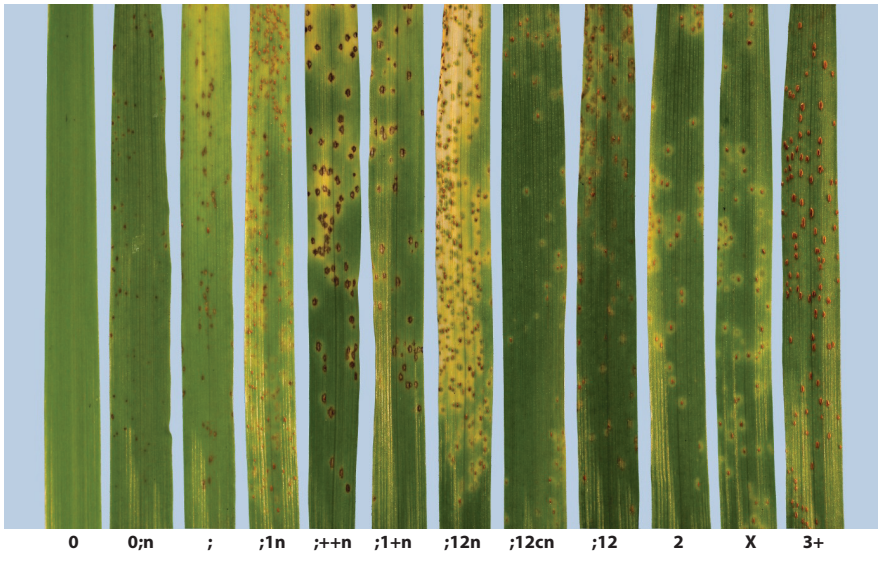

Figure 5

Range of seedling infection types for the *Puccinia hordei–Hordeum vulgare* interaction (image by Dr. Davinder Singh). Each infection type is based on a 0 to 4 scale with the addition of the symbols; and X, where 0 (the immune reaction) corresponds to no visible symptoms;; (very resistant) to hypersensitive flecks; 1 (resistant) to minute uredinia surrounded mainly by necrotic tissue; 2 (resistant to moderately resistant) to small- to medium-sized uredinia surrounded by chlorotic and/or necrotic tissue, often appearing as green islands; 3 (moderately resistant to moderately susceptible) to medium to large uredinia with surrounding chlorosis; 4 (susceptible) to large uredinia, without chlorosis or necrosis; and X (resistant) to mesothetic, heterogeneous infection types similarly distributed over the leaf, with cholorosis and/or necrosis usually present. The letters c and n are included to indicate greater than normal chlorosis or necrosis, respectively. The symbols − and = are added to indicate infection types that are lower than normal, and + and ++ to indicate infection types higher than normal. Infection types of 3+ or higher are considered as compatible (i.e., virulent pathogen/susceptible host).

(77, 142, 144, 145), regular surveys commenced in 1992 (92). These studies have provided convincing evidence of long-distance migration within Australia, sexual recombination, and mutation as generating genetic variability.

During the period of 1992–2001, a significant shift in the composition of populations occurred across four cereal-growing regions of Australia with virulence for resistance gene *Rph12*. Pathotype 4610P+, virulent on *Rph12*, was first detected in Tasmania in 1991. This pathotype was subsequently detected in all regions except Western Australia (WA). A further seven pathotypes virulent on *Rph12* were identified after the initial detection of *pt*. 4610P+. Two pathotypes virulent on *Rph12* were detected in WA in 1997 and 2001 (i.e., 5610P+ and 5453P−). The increase in virulence for *Rph12* in all cereal-growing regions was believed to be due to the cultivation of barley cultivars with this gene. The first Australian barley cultivar with *Rph12* was Franklin, released in 1989 and initially cultivated in Tasmania and later in several mainland states, including South Australia (SA) and Victoria (Vic). The cultivars Tallon, Lindwall, Fitzgerald, and Gairdner carry *Rph12*. Tallon and Lindwall were released in the northern part of the eastern cereal-growing region in 1991 and 1997, respectively. Similarly, Fitzgerald and Gairdner were released in WA in 1997 and were grown not only in that region but also in some parts of eastern Australia. The rapid increase of *Rph12* virulence in all regions during 1992–2001 demonstrates clearly how quickly pathogen populations can adjust to the selective force of host populations. The results presented here strongly implicate migration of pathotype 4610P+, either from Tasmania to SA or from SA to Tasmania, and subsequently to Vic, New South Wales (NSW), and Queensland (Qld). The detection of pathotype 5610P+ in WA in 1997, followed by its detection in SA and Vic in 1998 and in NSW and Qld in 1999, further demonstrated the widespread movement of urediniospores within Australia, including exchange between the western and eastern cereal-growing regions.

The origins of most pathotypes virulent for *Rph12* detected in Australia from 1992 to 2001 are unclear. Detailed comparative tests of these pathotypes demonstrated considerable variability between most of them, and with the possible exceptions of pathotypes 4610P+ and 4653P+ (+*Rph13*), which may have developed by mutation from pathotypes 210P+ and 4652P+ (+*Rph13*), respectively, it appears unlikely that most arose by simple mutation and that they may have arisen via sexual recombination on *O. umbellatum*. All of the *Rph12* virulent pathotypes first detected in eastern Australia were isolated initially from either Tasmania (4610P+, 4653P+, 5673P+, and 4652P+), Vic (5653P+), or SA (5452P+), and in view of the free movement of inoculum between these regions, it is possible that all originated from *O. umbellatum* in SA. Alternatively, *O. umbellatum* may occur more widely than is currently thought, or some or all of the new pathotypes may have originated outside Australia.

The development of virulence for *Rph3* in Australia most likely arose via simple mutation. This resistance gene was first deployed in Australia in the cultivar Yarra in 2005, followed by Fitzroy in 2007. Virulence for *Rph3* was first detected in March 2009 in northeastern Australia in pathotype 5457P+, which was detected again in surveys from 2010 to 2013 but only in eastern Australia. Gene *Rph3* was subsequently deployed in Western Australia in cultivar Bass, released in 2012, and this was soon followed by the detection of pathotype 5457P−, virulent for *Rph3*, in September 2013. Comparative studies indicated that both pathotypes with virulence for *Rph3* most likely arose via independent mutational acquisitions of virulence for *Rph3*, first in eastern Australia (from pathotype 5453P+) and second in WA (from pathotype 5453P−) (R.F. Park, unpublished results).

DNA Markers

Molecular markers have been applied widely to fungal plant-pathogen populations to characterize genetic diversity and assess phylogenetic relationships, and they are used to discriminate between

isolates and pathotypes. A study by Sun et al. (127) of pathogenic and AFLP variability among 45 isolates of *P. hordei* from 13 countries found molecular variation within and between the 28 pathotypes identified. The overall level of correlation between pathogenic and molecular diversity was low, but the reasons for this were unclear.

On the basis of a 73.2-Mb draft sequence of the *P. hordei* genome, Karaoglu & Park (64) identified more than 600 potential microsatellite [simple sequence repeat (SSR)] markers, of which, 76 proved to be highly polymorphic when tested across 19 Australian isolates of *P. hordei*. Preliminary studies with five of these markers showed that pathotypes 5453P−, 5453P+, 5457P−, and 5457P+ shared the same genotype, supporting the hypothesis that all are closely related and that the latter three are derived from the first via simple mutation (H. Karaoglu & R.F. Park, unpublished results). Overall, the draft sequence contained 21,179 SSR repeat loci, with an average of one SSR every 3.5 kb of DNA (64). The SSRs identified in *P. hordei* displayed longer repeat lengths than those seen in *P. graminis* f. sp. *tritici* and *P. striiformis* f. sp. *tritici* (H. Karaoglu, unpublished results). SSR repeats reflect a balance between expansion and contraction, and some researchers suggest that long repeat lengths in SSRs are recent in origin and are biased toward an increase in repeat length (65).

MANAGEMENT OF LEAF RUST

A variety of control measures exist to manage and/or suppress rust pathogens. All current management methods fall into three broad categories: cultural, chemical, and genetic resistance, which have been employed either singly or in combination. Walters et al. (139) advocated an integrated approach to control leaf rust in barley, including all three practices. They also realized the existence of barriers in the adoption of integrated management approaches, from growers and end users, such as acceptance of variety mixtures.

Cultural Practices

Cultural practices are primarily intended to reduce sources of inoculum by growing resistant cultivars, applying quarantine restrictions, and removing volunteer plants and alternate hosts (70). Often a simple procedure, such as altering sowing date, can effectively avoid and limit exposure to inoculum (126). The success of cultural control practices depends on understanding the biology of the pathogen and the response of the host to infection, which will then facilitate efficient management decisions (91). The principal cultural approach to control leaf rust is destruction of the green bridge either through grazing, cultivation, or application of herbicides to reduce over-seasoning inoculum.

Fungicides

Although many of the seed-dressing or in-furrow fungicides registered for the control of other barley diseases have been shown to be effective in preventing early leaf rust infection under experimental conditions, they are not currently registered for this use in some regions. Nagy et al. (87) applied fungicides (spyroxamine, tebuconazole, triadimenole, and trifloxystrobin) against foliar diseases of barley, including leaf rust, and observed a 7.9% yield increase with one foliar treatment and an 18.2% increase with two treatments in comparison to the untreated plots. Gonzales et al. (44) demonstrated that Epoxiconazole 125 SC 500 (500 mL/ha) and Tebuconazole 250 EW (450 mL/ha) were effective in controlling leaf rust on barley in Mexico. Walters et al. (139) considered fungicides as the key component in disease control strategies in barley cropping

systems but at the same time cautioned about the risks of fungicide resistance development and the implications for the major chemical classes in relation to current pathogen risks. Although chemical control methods are effective, they can be expensive and depending on weather conditions and length of growing season, multiple sprayings may be required (70). In areas such as Western Europe, where intensive cereal management is practiced and yields are high, chemical use may be justified; however, in regions such as Australia where barley production is centralized in low rainfall environments, requiring low input costs to be profitable, chemical control is uneconomic (70, 148). Owing to these issues, there is a tendency to reduce the use of fungicides and to minimize the effects of disease through the deployment of resistance genes through genetic breeding (82, 148).

Resistance Breeding

The deployment and utilization of host genetic resistance is the most economical, effective, and ecologically sustainable approach to controlling rust diseases (70, 82), including barley leaf rust (42, 90, 94). Although resistance in barley to *P. hordei* is widely available (90), durability, diversity, and effectiveness are important if disease control is to be effective. Information about resistance is supported by the knowledge of the epidemiology of the pathogen, of host/pathogen genetics, and of the environment (82). Understanding the effectiveness of resistance genes is vital for the durability and diversity aspects of resistance.

Apart from *Rph20* and *Rph23*, which are considered race nonspecific (42, 51, 121), most of the characterized *Rph* genes have limited value for plant breeding because nearly all have been completely overcome by adaptation of the pathogen (20, 29, 59, 125). The prevalence of pathotypes with virulence for major *Rph* genes is of great concern to plant breeders and pathologists because some of these genes (such as *Rph3*, *Rph7*, and *Rph9*) were considered the most effective and have been used widely in barley breeding programs worldwide (11, 13). Globally, only a few of these major genes have been deployed in commercial barley cultivars (11, 119, 120, 146): *Rph2*, *Rph3*, *Rph4*, *Rph7*, and *Rph9.z* (formally *Rph12*) have been used in Europe (29); *Rph2*, *Rph6*, and *Rph7* in the United States (125); *Rph2*, *Rph3*, *Rph4*, *Rph7*, *Rph9.z*, and *Rph19* in Australia (18); and *Rph3* and *Rph9.z* in New Zealand (21). Resistance conferred by these major genes has often failed to provide long-term disease control, and the deployment of single major hypersensitive genes in cultivars grown over a broad area can potentially lead to serious epidemics (11).

Golegaonkar et al. (42, 43) showed that in barley, many cultivars reported to carry partial resistance also carry APR. In wheat, although APR genes are often polygenic, monogenic APR genes also exists (*Sr2*, *Yr18*, *Yr29*, *Lr34*, *Lr46*) (82). Whereas major/monogenic resistance is often equated to race-specific resistance and nondurability, minor gene polygenic resistance is considered race nonspecific and durable. The dramatic loss of sources for effective major resistance against barley leaf rust has increased the importance of polygenic minor gene or APRs in breeding programs (68, 121). However, such resistance is difficult to characterize, select for, and utilize in breeding programs. This may be the reason that out of 22 mapped resistance genes, only two are partial APR genes and they have not been deliberately used in breeding programs.

To further diversify the genetic base of resistance, geneticists/breeders often mine supplementary gene pools. A large amount of genetic variation exists in the wild relatives of cultivated barley (57, 137) because of the coevolution of the host and pathogen, and a number of wild species in the genus *Hordeum* are of potential importance for barley breeding (115). These wild relatives offer diverse sources of unique alleles and novel resistance genes for barley improvement that can be utilized via interspecific hybridization (115). Since the application and establishment of embryo rescue techniques, wide crosses have become very successful (57). As pointed out by von Bothmer

et al. (136), the utilization of germplasm from wild *Hordeum* species has been largely limited in barley breeding for two main reasons. First, most wild *Hordeum* species are distantly related to cultivated barley, which makes interspecific crosses difficult to achieve, and second, the diploid constitution of cultivated barley means it is sensitive to genetic imbalances and disturbances and cannot tolerate as much genetic manipulation as polyploid cereal species (150). Several barriers therefore need to be overcome before successful transfer of material can be achieved: e.g., pre- and post-fertilization barriers, such as pollen tube-stylar incompatibility resulting in low seed setting; endosperm degeneration in the developing hybrid seed; chromosome instability; low chromosome pairing and crossing over between homoeologs; reduced recombination; and linkage drag and hybrid infertility (102–104, 106). However, despite these limitations, recombinant plants have been produced (14, 33, 34, 55, 58, 105, 108).

MAS offers an alternative to phenotype-based selections for rust resistance genes using closely linked and/or perfect markers. The use of tightly linked markers can efficiently facilitate the pyramiding of major resistance genes in a short period of time (72). It enables the breeder to screen large segregating populations in a short time and little space. Molecular markers can be applied to very early population generations before quality and agronomic (field) data are collected (71). However, for successful incorporation, these markers should cosegregate or map close to the target gene, which ensures low recombination frequencies occur and allows for a close estimation of genetic distance. Although molecular markers for at least 12 of the known *Rph* genes are available, a majority of them are not ideal for perfect MAS because they are not close enough or tightly linked to the target *Rph* gene or are first-generation molecular markers (restriction fragment length polymorphism, random amplified polymorphic DNA, or AFLP) and hence lack efficiency in MAS because they are either too expensive, too difficult to apply, or not repeatable. The reliability and diagnostic capabilities of these markers need to be validated and ensured before they can be used for MAS. Mammadov et al. (79) evaluated and validated the reliability and diagnostic capabilities of several molecular markers for barley leaf rust resistance genes *Rph5* and *Rph7* and recommended that sequence-tagged site markers TC2863-12.4 and ABG70 as well as SSR marker AY642926- CA11 are the most reliable for use in MAS. Hickey et al. (51) developed a closely linked marker *bPb-0837* for APR gene *Rph20* that has been successfully used in MAS. More recently, Singh et al. (121) optimized a PCR-based marker *Ebmac 0603* closely linked to APR gene *Rph23* and recommended its use in MAS. Although good advancement has been made at identifying molecular markers closely linked to *Rph* genes, there is no diagnostic marker available to date that is perfectly reliable for MAS.

SUMMARY POINTS

1. Leaf rust of barley is caused by the macrocyclic, heteroecious rust pathogen *P. hordei*, with aecia reported from selected species of the genera *Ornithogalum*, *Leopoldia*, and *Dipcadi*. Uredinia and telia occur on *H. vulgare*, *H. vulgare* ssp. *spontaneum*, *H. bulbosum*, and *H. murinum*, on which distinct parasitic specialization occurs.
2. Although sporadic, leaf rust is probably the most common and widely distributed rust disease of barley, and it has increased in importance in recent decades.
3. Although total crop loss does not occur, under epidemic conditions yield reductions of up to 62% have been reported in susceptible varieties.
4. To date, 21 seedling resistance genes and two APR genes have been identified.

5. Virulence has been detected for most seedling resistance genes but is unknown for the APR genes *Rph20* and *Rph23*.
6. It is likely that achieving durable resistance to leaf rust in barley will involve the use of resistance gene combinations that include both seedling and APR genes.
7. The availability of a genome sequence for *H. vulgare* will accelerate efforts to isolate resistance genes and develop linked markers.

FUTURE ISSUES

1. Three loci conferring durable resistance to all three rust pathogens of wheat and to mildew have been identified in wheat (*Pm46/Lr67/Yr46/Sr55*, *Pm38/Lr34/Yr18/Sr57*, and *Pm39/Lr46/Yr29/Sr58*). Experience to date suggests that these pleiotropic APR genes have some intrinsic durability and that combinations of multiple effective resistance genes contribute to durability by lowering the chance of the development of virulence-matching gene combinations. Should any such pleiotropic loci be found in barley, they will be extremely useful in developing improved cultivars with durable rust and mildew resistance.
2. Tightly linked high-throughput DNA markers are needed for more *Rph* genes to permit MAS and greater efficiency in assembling gene combinations. The recent availability of a whole-genome sequence for barley (54) will greatly expedite this process.
3. The ability to incorporate multiple resistance genes by transformation will also become possible as more genes are cloned. Relatively few resistance genes or QTLs in barley have been cloned, and these include genes *Rpg1* and *Rpg5*, which confer resistance to stem rust. Genetic resources, such as the recent barley genome sequence release, and the availability of bacterial artificial chromosome libraries will permit rapid progress in gene cloning efforts in barley for resistance genes to *P. hordei*. The subsequent validation of candidate genes through agrobacterium-mediated transformation is dependent on the availability of a susceptible barley genotype with high transformation efficiency. Golden Promise is one of the only cultivars with high transformation efficiency and is susceptible to certain *P. hordei* pathotypes; however, there is uncharacterized resistance to some Australian *P. hordei* pathotypes (R.F. Park, unpublished results). An important consideration with transgenic approaches is the potential for suppression of resistance genes. For example, recent studies in wheat have shown that the mildew susceptibility allele *Pm3C* and three resistance alleles (*Pm3a*, *Pm3b*, and *Pm3f*) all suppress *Pm8*, which is derived from rye (53). Although this example may be an exception, clearly any strategy based on combining cloned resistance genes will need to take this into consideration.
4. Although MAS has clearly increased the precision of predicting phenotypes from genotypic data and has accelerated genetic gain in barley breeding programs, translating to disease-resistant varieties, the efficiency is not as pronounced for quantitative traits. Recent and rapidly evolving advancements in next-generation sequencing technologies have dramatically reduced cost per unit (DArT and SNP marker platforms) while increasing the precision of genotypic and trait-dissection analysis in crop species such as barley. Genomic selection could potentially improve the gains previously observed by MAS. A

key to the success of genomic selection is that it incorporates information from large-marker data sets in the prediction model, thereby avoiding biased marker effect estimates and capturing more of the variation due to small effect QTLs. This makes genomic selection highly suitable for pyramiding APR to *P. hordei* into elite germplasm.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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