Annu. Rev. Phytopathol. 1996. 34:435–55

GENETICS OF RESISTANCE TO WHEAT LEAF RUST[1]

J. A. Kolmer
Agriculture and Agri-Food Canada, Cereal Research Centre, 195 Dafoe Road, Winnipeg, Manitoba R3T 2M9, Canada

KEY WORDS: *Triticum aestivum*, *Puccinia recondita*, specific resistance

ABSTRACT

Leaf rust (caused by *Puccinia recondita* f. sp. *tritici*) is the most widespread and regularly occurring rust on wheat. Genetic resistance is the most economical method of reducing yield losses due to leaf rust. To date, 46 leaf rust resistance genes have been designated and mapped in wheat. Resistance gene expression is dependent on the genetics of host-parasite interaction, temperature conditions, plant developmental stage, and interaction between resistance genes with suppressors or other resistance genes in the wheat genomes. Genes expressed in seedling plants have not provided long-lasting effective leaf rust resistance. Adult-plant resistance genes *Lr13* and *Lr34* singly and together have provided the most durable resistance to leaf rust in wheat throughout the world. Continued efforts to isolate, characterize, and map leaf rust resistance genes is essential given the ability of the leaf rust fungus to overcome deployed resistance genes.

INTRODUCTION

Leaf rust of wheat (*Triticum aestivum* L.), caused by *Puccinia recondita* Roberge ex Desmaz. f. sp. *tritici* Eriks. & E. Henn, is found nearly wherever wheat is grown, and is the most regularly occurring of the three rusts found on wheat (15, 97). The wheat leaf rust fungus is adapted to a range of different climates, and the disease can be found in diverse wheat growing areas throughout the world (96). Wheat cultivars that are susceptible to leaf rust regularly suffer yield reductions of 5–15% (97) or greater, depending on the stage of crop development when the initial rust infections occur (15).

Genetic resistance is the most economical and preferable method of reducing yield losses due to leaf rust. Various wheat breeding programs throughout the world have had mixed results in producing cultivars with long-lasting, effective resistance to leaf rust. Spring wheat breeding programs in North America (61), Mexico (90), and Australia (72) have generally been very successful in producing cultivars that have had high levels of durable and effective resistance. In contrast, the winter wheats grown in the southern plains of the United States often lose effective resistance after only a few years of cultivation (68, 71).

Genetic resistance to leaf rust can be most fully utilized by knowledge of the identity of resistance genes in commonly used parental germplasm and released cultivars. Identification of the leaf rust resistance genes allows for efficient incorporation of different genes into germplasm pools, thus helping to avoid the release of cultivars that are genetically uniform. In this chapter, I wish to update and review various aspects of genetics of leaf rust resistance in wheat and attempt to relate this genetic information with the effectiveness and longevity of resistance.

DESIGNATION OF SPECIFIC *Lr* GENES

To date, 46 leaf rust resistance genes (*Lr*) have been isolated, mapped to specific chromosomes, and given official designations according to the standards set forth in the Catalogue of Gene Symbols for Wheat (74). Descriptions of genes *Lr1–Lr34* have been given in previous summaries (11, 54, 67, 96). Twenty-five of these *Lr* genes were isolated directly from hexaploid wheats (67, 96). The other genes were derived from lower-ploidy relatives of hexploid wheat within the tribe Triticeae in the Poaceae. The methodologies used in transferring resistance genes from related species to hexaploid wheat have been previously summarized (33, 54). Near-isogenic Thatcher lines for nearly all the designated leaf rust resistance genes were developed by PL Dyck and RG Anderson of the Agriculture and Agri-Food Canada Cereal Research Centre in Winnipeg.

Table 1 lists the genes designated after *Lr34*. Gene *Lr35* was originally derived from *Triticum speltoides* (53) and was transferred by backcrossing an amphiploid of *T. speltoides* $\times$ *T. monococcum* to the wheat cultivar Marquis. Resistance expressed by *Lr35* first becomes noticeable at the seond-leaf stage, and is fully expressed after the sixth-leaf stage. Gene *Lr36* was derived from *T. speltoides* and backcrossed into the wheat cultivar Neepawa (18). Gene *Lr37* was initially derived from VPM1, a wheat cultivar with resistance to eyespot derived from *T. ventricosa* (35). Resistance derived from VPM1 was designated *Lr37* and mapped to chromosome 2AS (4, 5). Gene *Lr38* was isolated from an *Agropyron intermedium* group 7 chromosome that had been transferred to

Table 1 Chromosome location, source, infection types[a], and test lines of leaf rust resistance genes *Lr35–Lr45*

Gene	Chromosome location	Source	Seedling infection type	Test line
Lr35	2B	*Triticum speltoides*	Adult-plant resistance	RL 6082
Lr36	6BS	*Triticum speltoides*	;1	2-9-2 E84018
Lr37	2AS	*Triticum ventricosa*	Adult-plant resistance[b]	RL 6081
Lr38	6DL	*Agropyron intermedium*	;	RL 6097
Lr39[c]	1DS	*Triticum tauschii*	;12[d]	—
Lr40[c]	1DS	*Triticum tauschii*	;12	—
Lr41	1D	*Triticum tauschii*	0;	KS90WGRC10
Lr42	1D	*Triticum tauschii*	;1−	KS92WGRC11
Lr43	7D	*Triticum tauschii*	0;	KS92WGRC16
Lr44	1B	*Triticum aestivum* spelta	;−3c	RL 6147
Lr45	2A	*Secale cereale*	;12	RL 6144 ST-1

[a]Infection type scale:

0 = no uredinia or flecks visible
0; = very faint hypersensitive flecks
; = hypersensitive flecks
1 = small uredinia surrounded by necrosis
2 = small uredinia surrounded by chlorosis
3 = moderate size uredinia without chlorosis
4 = large uredinia without chlorosis
c = chlorosis
\+ = slightly larger uredinia than expected for the infection type
− = slightly smaller uredinia than expected for the infection type

[b]At temperatures below 20°C, *Lr37* expresses a 2^{+}c infection type in seedlings (35).
[c]*Lr39* and *Lr40* are allelic or identical to *Lr21*.
[d]The most common infection type is listed first, followed by other infection types that were also observed.

a Heine IV background (47). The *A. intermedium* translocations were later mapped to 2AL, 1DL, 3DS, 5AS, and 6DL (46).

Gene designations for *Lr39* and *Lr40* were tentatively given for genes derived from *T. tauschii* (16). However, when hexaploid wheat lines with the two genes singly were crossed with the Thatcher line with *Lr21,* which was also derived from *T. tauschii,* no segregation was observed (16). Genes *Lr39* and *Lr40* are either different or identical alleles of *Lr21*. It will remain impossible to distinguish between either alternative until isolates of *P. recondita* with virulence to *Lr21* become available. Genes *Lr41, Lr42,* and *Lr43* were also transferred to hexaploid wheat from *T. tauschii* (16). Genes *Lr41* and *Lr43* segregated independently of the other genes derived from *T. tauschii,* and *Lr42* was linked to *Lr21*. Gene *Lr44* was derived from an accession of *T. aestivum* (spelt) (42). Gene *Lr45* was found in a Japanese wheat:rye derivative, and is located on chromosome 2A (76).

Other leaf rust resistance genes have been isolated and characterized, although these genes have not been mapped to chromosome locations and therefore have not been assigned *Lr* numbers. Dyck & Samborski (36) isolated *LrB* from the cultivar Brevit. Dyck & Jedel (30) examined the leaf rust resistance in the AE Watkins wheat collection, and isolated a gene designated as *LrW*. Dyck (26) later isolated an additional gene, *LrW2,* and speculated that certain accessions in the collection had adult plant resistance genes that had not been previously characterized. In the same collection of spelt wheats from which *Lr44* was isolated, Dyck & Sykes (42) determined that three of the accessions have an uncharacterized adult plant resistance gene. Dyck (27) also isolated a gene from *T. turgidum* ssp. *dicoccoides* that conditions a very low seedling infection type.

EXPRESSION OF RESISTANCE GENES

The Genetics of Host-parasite Interaction

The gene-for-gene relationship has been thoroughly studied in the *T. aestivum*–*P. recondita* pathosystem. Generally, for each resistance gene in the host, there is a corresponding locus in the pathogen with alternate alleles that condition virulence and avirulence (98, 99, 114, 115, 117). However, for some corresponding gene pairs (65) the interactions differ from the classical one-to-one relationship. For example, three different patterns have been found for inheritance of avirulence corresponding to resistance genes in allelic sets in wheat. The *Lr2* locus in wheat has three alleles, *Lr2a, Lr2b,* and *Lr2c*. Avirulence to all three alleles is conditioned by a single allele in *P. recondita,* although a dominant gene at an independent locus differentially inhibits the expression of avirulence to the three resistance alleles (38, 98) (Table 2). Isolates of the

Table 2 Infection types[a] produced by *Puccinia recondita* genotypes on alleles at the *Lr2* locus in wheat

P. recondita genotypes	*Lr2* alleles		
	Lr2a	*Lr2b*	*Lr2c*
P2[b]*–i2i2*	O[b]	O;	O;
P2p2 I[c]*2i2*	;2^-	22^+	3^+
p2p2 −−	3^+	3^+	3^+

[a]See footnote in Table 1 for infection type scale.
[b]Pathogenicity gene corresponding to *Lr2* locus in wheat.
[c]Gene inhibits expression of avirulence to *Lr2* locus in wheat.

fungus with very low infection type (0) to *Lr2a* also have low infection types (0;) to *Lr2b* and *Lr2c*. Isolates with intermediate infection types on *Lr2a* (2^-) have higher infection types (22^+–3^+) to the other two alleles. Isolates virulent to *Lr2a* (infection type 3^+) are also virulent to *Lr2b* and *Lr2c*. Isolates of *P. recondita* that are virulent to *Lr2a* and avirulent to *Lr2b* and *Lr2c* have never been found in either virulence surveys or genetic studies.

A gene in the cultivar Prelude inhibits the resistance expressed by *Lr3* to certain genotypes of *P. recondita*. Recombinant isolates of *P. recondita* were avirulent to Thatcher lines with *Lr3*, but were virulent to *Lr3* in a Prelude background (49, 50). Two other alleles were found at the *Lr3* locus, *Lr3ka* and *Lr3bg*. Two complementary genes in *P. recondita* conditioned virulence to *Lr3bg*, while a single gene conditioned virulence to *Lr3ka* (49, 50). The loci conditioning virulence/avirulence to *Lr3*, *Lr3ka*, and *Lr3bg* segregated independently.

Resistance genes *Lr14a* and *Lr14b* were also determined to be allelic (37). Each gene conditions a mesothetic avirulent response to different isolates of *P. recondita*. The corresponding virulences to *Lr14a* and *Lr14b* segregated independently. Gene *Lr22b* is found in the cultivars Thatcher and Marquis and conditions adult plant resistance to only a few phenotypes of *P. recondita* (20). Virulence to *Lr22b* segregated as a single gene (6). An alternate allele, *Lr22a* derived from *T. tauschii*, is highly effective in the adult plant stage to all North American isolates of *P. recondita* that have been tested (JA Kolmer, unpublished data).

Dominance relationships of avirulence/virulence genes in the pathogen and leaf rust resistance genes in the host are dependent on the respective genotypes. Kolmer & Dyck (60) examined the infection types obtained when segregating progenies of a selfed isolate of *P. recondita*, and near-isogenic Thatcher lines were tested in nine combinations of pathogen and host genotypes at seven corresponding gene loci (four examples shown in Table 3). Expression of resistance genes in the host ranged from completely dominant to recessive, depending on whether the pathogen was homozygous or heterozygous for avirulence. Likewise, expression of avirulence in the pathogen depended on whether the host was homozygous or heterozygous for resistance.

Background Effects

Cultivar background can affect the expression of resistance genes. Gene *Lr2b* in a Prelude background was partially dominant in crosses with Thatcher and completely dominant in crosses with Red Bobs (38). The *Lr2c* allele in Prelude was recessive in crosses with Thatcher and dominant in crosses with Prelude and Red Bobs. The *Lr2* alleles expressed most resistance in the Thatcher background and least resistance in Red Bobs (38). Similar differences were noted for *Lr3* in Thatcher and Red Bobs. Pretorius et al (88) noted background effect on the expression of *Lr22a*.

Suppressors of Resistance Genes

The transfer of resistance genes from related species of lower-ploidy into hexaploid bread wheat can be complicated by interactions between resistance genes and suppressor genes in the different genomes. Bai & Knott (3) crossed ten leaf rust resistant accessions of *T. turgidum* var. *dicoccoides* (AABB) with susceptible bread wheat (AABBDD) and durum wheats (AABB). The F_1 plants from crosses with the durum wheats expressed leaf rust resistance, while the F_1 plants from crosses with the hexploid wheats were susceptible. In the F_2

Table 3 Infection types[a] of *Puccinia recondita* genotypes and Thatcher near-isogenic wheat lines at four corresponding gene loci[b]

	P. recondita				*P. recondita*		
Wheat lines	*P3ka P3ka*[c]	*P3ka p3ka*	*p3ka p3ka*	Wheat lines	*P3P3*	*P3p3*	*p3p3*
Lr3ka Lr3ka[d]	;	;2^-	3^+	*Lr3 Lr3*	;	;1^-	3^+
Lr3ka lr3ka	22^-	2^+	3^+	*Lr3 lr3*	22^+	3^+	3^+
lr3ka lr3ka	3^+	3^+	3^+	*lr3 lr3*	3^+	3^+	3^+
	P. recondita				*P. recondita*		
Wheat lines	*P17P17*	*P17p17*	*p17p17*	Wheat lines	*P30P30*	*P30p30*	*p30p30*
Lr17 Lr17	;1 =	;1	3^+	*Lr30 Lr30*	;1^-	2^-	3^+
Lr17 lr17	22^-	3^+	3^+	*Lr30 lr30*	3^+	3^+	3^+
lr17 lr17	3^+	3^+	3^+	*lr30 lr30*	3^+	3^+	3^+

[a]See footnote in Table 1 for infection type scale.
[b]From Kolmer & Dyck (60).
[c]Pathogenicity loci in *P. recondita*.
[d]Resistance gene loci in wheat.

progenies from the hexaploid crosses, resistant plants had fewer D chromosomes (average 3.2) compared to susceptible plants (average 11.5). Chromosomes 2B and 4B carried genes for leaf rust resistance, and 1D and 3D carried suppressors of resistance.

Suppressors of leaf rust resistance have also been located to the A and B genomes. Innes & Kerber (51) intercrossed 12 leaf rust–resistant accessions of *T. tauschi* (DD) and determined the accessions had four leaf rust seedling resistance genes that had not been previously isolated from *T. tauschii*. Accessions were crossed by Tetra-Canthatch (AABB) to produce triploid (ABD) progeny, which were then doubled by a colchicine treatment to produce synthetic hexaploids. Two of the four leaf rust resistance genes derived from *T. tauschii* did not express resistance in the synthetic hexaploids. The suppression of seedling resistance in these lines, however, did allow the detection of three different adult-plant leaf rust resistance genes. The most likely explanation for the loss of seedling leaf rust resistance in the hexaploids is the presence of suppressor genes located on the A or B genomes.

Modifiers of resistance gene action may also be present in wheats that are used as recurrent parents in backcrossing programs. McIntosh & Dyck (75) determined that Thatcher has a gene that inhibited the expression of *Lr23* when tested with isolates of *P. recondita* from Canada, and partially inhibited the resistance when tested with isolates from Australia.

Temperature Effects

Dyck & Johnson (31) tested the response of 27 Thatcher near-isogenic lines for leaf rust resistance with four different isolates of *P. recondita* at temperatures between 10–25°C (Table 4). They found that optimal rust development occurred at 15–20°C; at 25°C the uredinia showed pronounced chlorosis, and at 10°C the infections developed very slowly and were restricted in size. Genes *Lr18, Lr14a, Lr30, Lr15,* and *Lr11* had lower infection types at low temperatures, and higher infection types at the higher temperatures. However, genes *Lr16, Lr17,* and *Lr23* had lower infection types at high temperatures, and had high infection types to various isolates at the low temperatures. Genes *Lr2a, Lr3ka,* and *Lr3* had less resistance at low temperatures to isolates that had intermediate infection types to the genes. Dyck & Johnson (31) concluded it was difficult in general to classify leaf rust resistance genes for temperature sensitivity based on their results. The temperature responses of the genes were found to be highly isolate dependent.

Pretorius et al (89) showed that the resistance of the adult-plant gene *Lr13* was expressed at 25°C in seedling plants to three isolates of *P. recondita* from Mexico, China, and Chile. However, the resistance was not expressed in seedlings to isolates from North America. Kolmer (unpublished data) found that isolates

Table 4 Infection types produced at four temperatures by a *P. recondita* isolate on Thatcher near-isogenic wheat lines[a]

Thatcher line	Temperature 10°	15°	20°	25°
Lr18	;1+[b]	1+	3−	3+
Lr14a	x	x	x+	3+
Lr30	1+	1+	1+	2+
Lr15	;1c	1c	;1+c	;1+c
Lr11	2	2+	2+	2++
Lr16	3+	2+c	2−*c*	2c
Lr17	3+	3	;1+	;1−
Lr23	3+	2++	;1	;1=
Lr2a	2++	2+	;2	;1+
Lr24	;1−	;1=	;	;1−
Lr3	3	x	;2	;1
Lr3ka	3	2+	1+	1

[a]See footnote in Table 1 for infection type scale.
[b]From Dyck & Johnson (31).

from North America have high infection types to seedlings with *Lr13* regardless of temperature, but many of the same isolates have low infection types to adult plants with *Lr13*. Use of high temperatures may not be reliable in determining virulence of isolates to *Lr13*. Other studies (86, 87, 105) have also examined the effects of temperature and growth stage on leaf rust infection types using the Thatcher near-isogenic lines.

Complementation and Gene Interactions

There is only one clear example of complementary genes conditioning resistance to wheat leaf rust. Singh & McIntosh (110, 111) found that *Lr27* and *Lr31* in the cultivar Gatcher conditioned resistance only when present together. The genes were also determined to be in Hope (*Lr27*) and Chinese Spring (*Lr31*). Complementation was observed in Chinese Spring substitution lines with chromosome 3B from Hope.

The gene-for-gene theory emphasizes relationships between individual genes in the host and parasite (45, 85). In combinations where more than one corresponding gene pair is involved, the gene pair with the lowest infection type should determine the infection type, since the low infection types are epistatic to high infection types. However, there are a number of examples in leaf rust resistance where lower than expected infection types have been observed in cultivars and lines with more than one resistance gene (19, 101). Schafer et al (101), Dyck (19), and Samborski & Dyck (100) defined gene interaction as the combination of two or more genes resulting in a higher level of resistance than that conferred by the individual genes.

The adult-plant resistance genes *Lr13* and *Lr34* are present, either singly or together, in nearly all hard red spring wheats bred for leaf rust resistance in North America (61). How these two genes interact together and with other resistance genes is important in determining the level of resistance in these cultivars. Germán & Kolmer (48) and Kolmer (57) intercrossed Thatcher near-isogenic lines with the *Lr34* line and the *Lr13* line and selected progeny lines that were homozygous for both genes. The infection types and field rust reactions (Table 5) were essentially the same whether each gene was combined with *Lr34* or *Lr13*. In seedling and adult-plant tests, genes *Lr13* and *Lr34* interacted to condition higher levels of resistance than expected when paired with other leaf rust genes that also conditioned some level of resistance when present singly. In seedling tests, the paired combinations of resistance genes with *Lr13* or *Lr34* had lower infection types than either gene singly when the additional seedling resistance conditioned an intermediate avirulent response to the *P. recondita* isolate (Table 5). The same relationship was observed when lines homozygous

Table 5 Infection types[a] produced by *Puccinia recondita* phenotypes[b] on Thatcher near-isogenic wheat lines with pairs of leaf rust resistance genes[c]

	P. recondita phenotype				
Thatcher line	CHB	MFB	PBD	TBD	Field
Lr2a	0;[c]	;	;2^{-}	3^{+}	90S[d]
Lr2a,13	0;	0;	0;	4	10R-20MR
Lr2a,34	0	0	0;	4	5-20M
Lr16	3	1	1	1	70MR
Lr16,13	1^{+}	;1^{-}	;1^{-}	;1	5R
Lr16,34	12^{-}	;1^{-}	;1^{-}	1	5VR
Lr17	1–	1	3^{+}	3^{+}	60MR
Lr17,13	;1	;1	3^{+}	3^{+}	5R
Lr17,34	;1^{-}	;1	3^{+}	3^{+}	5VR
Lr13	4	4	4	4	60MR
Lr34	3	3	3	3	5–20M
Thatcher	4	4	4	4	90S

[a]See footnote in Table 1 for infection type scale.
[b]See Long & Kolmer (67) for description of virulence phenotype code.
[c]From Germán & Kolmer (48) and Kolmer (57).
[d]Severity (% infection) and response:

VR = very resistant; hypersensitive flecks with no sporulation.
R = resistant; hypersensitive flecks and small uredinia with necrosis.
MR = moderately resistant; moderate size uredinia with necrosis.
MS = moderately susceptible; moderate size uredinia with chlorosis.
S = susceptible; large uredinia with necrosis or chlorosis.

for two genes were rated for resistance in field tests. Combinations of *Lr13* or *Lr34* with other effective resistance genes resulted in lines with superior leaf rust resistance relative to either parent.

ANALYSIS OF LEAF RUST RESISTANCE IN WHEAT CULTIVARS AND GERMPLASM

The isolation and characterization of specific leaf rust resistance genes has made it possible to determine exactly which resistance genes are present in commercial wheat cultivars and breeding lines. This information is extremely valuable in breeding programs where maintenance of leaf rust resistance is a high priority. Two methods have been commonly used to elucidate the leaf rust resistance genotypes of wheat cultivars: gene postulation and genetic analysis.

Gene Postulation

Gene postulation applies the principles of gene-for-gene specificity (45, 85, 65) to hypothesize which *Lr* genes may be present in host materials. This method uses the avirulent isolate/resistant host combination from the quadratic check (43) as the definitive combination. Low or incompatible infection types are expressed only when hosts with a specific resistance gene are challenged with a pathogen isolate that is avirulent to that gene. All other combinations result in high or compatible infection types. Using this as a basis, it is possible to hypothesize which resistance genes are present by testing the material with a diverse collection of isolates of *P. recondita* that have been characterized for avirulence/virulence using the near-isogenic series of Thatcher lines. Identity of *Lr* genes can be hypothesized by comparing the isolate/wheat cultivar combinations that result in low infection types with the isolate/near-isogenic line combinations that also result in low infection types. This method was initially developed by Loegering et al (66) and Browder (10).

The main advantage of gene postulation is that information regarding the possible identity of *Lr* genes can be obtained within four weeks if the tests are conducted using the primary leaves of seedling plants. Large numbers of cultivars and breeding lines can thus be evaluated in a relatively short period of time. However, there are restrictions that limit the usefulness of this method. Gene postulation is best suited for the identification of resistance genes that clearly express in seedling plants. As such, this method is not appropriate for the identification of resistance genes that are optimally expressed in adult plants. When more than one effective resistance gene is present in a cultivar or breeding line, the characteristic infection types of the individual genes are often altered due to interaction between the resistance genes. As seen previously, the adult-plant genes *Lr13* and *Lr34* often interact with seedling resistance genes in seedling plants to produce lower than expected infection types. Gene postulation is also highly dependent on the available collection of *P. recondita* virulence phenotypes. Critical combinations of virulences may not be available, and thus it may not be possible to identify all of the seedling genes present in the materials being studied.

Genetic Analysis

The number and identity of leaf rust resistance genes in wheat cultivars can be conclusively determined only by genetic analysis. In this method, the cultivar being studied is crossed with a susceptible parent and the F_1 plants are selfed to obtain F_2 populations, or are backcrossed to the susceptible parent to obtain BCF_1 plants. The F_2 or BCF_1 plants are then selfed to obtain F_3 or BCF_2 families, respectively. The number of segregating resistance genes can then be determined by inoculating the F_3 and BCF_2 families with specific rust races in seedling tests, and also evaluating the segregating families for adult-plant resistance in field tests using a representative mixture of *P. recondita* races. Evaluation of resistance based on segregation of F_3 and BCF_2 families is more reliable than using single F_2 plants, since more than a single plant is evaluated for infection type and severity. F_3 and BCF_2 families can also be tested simultaneously with different races. Segregation ratios obtained with different races can be used in identification of the resistance genes.

Genetic studies of leaf rust resistance at the Cereal Research Centre in Winnipeg have used the backcross method to isolate and characterize seedling and adult-plant resistance genes (1, 2, 22, 23). Major advantages of using BCF_2 populations compared to F_3 families are that smaller population sizes are required, and resistance genes can be isolated within families that are segregating in single gene ratios. In these families, plants with the lowest infection type can be progeny tested to obtain lines that are homozygous for resistance. Homozygous lines can be tested with a collection of isolates of *P. recondita* to determine if the resistance is a previously identified gene or an uncharacterized resistance gene. Isolation and characterization of single genes are more difficult using F_3 families in crosses with two or more segregating genes, since many families will have more than one gene. An additional advantage of the backcross method is that the segregating resistances can be evaluated in a background with 75% of the susceptible recurrent parent. This can be very helpful in evaluating adult-plants in field tests from crosses in which the two parents vary for maturity or vernalization response (23).

Common Leaf Rust Resistance Genes in Spring Wheats

Dyck et al (41) studied the genetics of leaf rust resistance in the Brazilian cultivar Frontana and in the cultivar Exchange from Purdue University. Adult-plant resistance genes designated as *Lr13* and *Lr12* were isolated from Frontana, and Exchange, respectively. However, progeny lines with *Lr13* or *Lr12* alone did not have the high level of resistance that was characteristic of Frontana or Exchange; a modifying gene was also needed to condition the original levels of resistance. Because of their high levels of resistance Frontana and Exchange were used as parents in hard red spring wheat breeding programs in Canada and the United States (44, 61). Gene *Lr13* was first used in North America in the cultivars Manitou and Chris in the mid 1960s and is likely present in most of the Canadian and US hard red spring wheats since developed.

Dyck & Samborski (40) identified genes *LrT2* and *LrT3,* which interacted for high levels of seedling resistance in a group of common wheat cultivars that included Frontana and Terenzio. *LrT2* was determined to be the modifying gene in Frontana and Exchange and was designated as *Lr34* (21). *Lr13* and *Lr34* are the most important resistance genes in the Canadian and US hard red spring wheats. Roelfs (95) has indicated that *Lr13* and *Lr34* may be present in many wheats worldwide that have displayed durable leaf rust resistance.

The North American hard red spring wheats also have a number of seedling resistance genes. Gene *Lr16* is found in a number of cultivars, and interacts with the adult-plant genes *Lr13* and *Lr34* for very high levels of resistance (48, 57). Seedling genes *Lr1, Lr2a, Lr3, Lr10,* and *Lr24* were determined to be in US spring wheats by intercrossing or by gene postulation (77, 93, 94, 116). These genes do not currently condition effective resistance; resistance expressed by these cultivars must be due to adult-plant resistance genes.

Wheat cultivars developed at the Agriculture and Agri-Food Canada Research Centre in Winnipeg have been genetically examined to determine their leaf rust resistance genotypes. Genotypes of recent cultivars include Columbus-*Lr13, Lr16* (100); Pasqua-*Lr11, Lr13, Lr14b, Lr30, Lr34* (24); Roblin-*Lr1, Lr10, Lr13, Lr34* (25); AC Domain-*Lr10, Lr16, Lr34* (JA Kolmer, unpublished data). The adult-plant resistance gene *Lr22a* and the seedling gene *Lr21,* both originally isolated from *T. tauschii* (32), are in the cultivars AC Minto-*Lr11, Lr13, Lr22a,* and AC Cora-*Lr13, Lr21,* respectively (JA Kolmer, unpublished data). Genotypes of other cultivars developed in Canada include Kenyon-*Lr13, Lr16* (22), Laura-*Lr1, Lr10, Lr34,* and Genesis and Biggar-*Lr13, Lr14a* (59). Of the seedling genes in these cultivars, only *Lr16* and *Lr21* currently condition effective resistance.

Leaf rust-resistant cultivars in Australia in the 1950s had *Lr14a* (69). Later, cultivars with combinations of *Lr3, Lr2a, Lr20, Lr23,* and *Lr27* were released. Genes *Lr1, Lr2a, Lr3, Lr13, Lr17,* and *Lr24* are currently found in Australian spring wheats (72). *Lr13* is very effective in Australia; combinations of seedling genes with *Lr13* confer resistance to all Australian phenotypes of *P. recondita*. Indian and Pakistani wheats were hypothesized by gene postulation to have combinations of *Lr1, Lr2a, Lr3, Lr10, Lr11, Lr14a, Lr17, Lr23, Lr26,* and *Lr27* and *Lr31* (91, 92, 109). *Lr34* was also thought to be present since several lines had adult-plant resistance.

Wheat lines released by the CIMMYT program are selected for high levels of adult-plant resistance (90). Singh & Rajaram (113) indicated that highly resistant CIMMYT cultivars had *Lr34* plus three additional uncharacterized adult-plant resistance genes. Gene postulation studies of CIMMYT lines (109, 112) indicated the presence of *Lr1*, *Lr3, Lr3bg*, *Lr10*, *Lr14a*, *Lr17*, *Lr19*, *Lr23*, *Lr26*, and the genes *Lr27* and *Lr31*. *Lr34* was hypothesized to be present in a number of lines due to its characteristic lower seedling infection type at cooler temperatures (105).

Many spring wheats developed in South America trace some of their leaf rust resistance to Americano 44d, a Uruguayan land race, and to Alfredo Chaves, a Brazilian land race (95). These cultivars possibly have *Lr13* and/or *Lr34*. Other wheats developed in South America have been valuable sources of leaf rust resistance. Dyck (67) isolated *Lr3* from Sinvalocho, *Lr3ka* from Klein Aniversario, *Lr3bg* from Bage, *Lr11* from El Gaucho, *Lr14b* from Maria Escobar and Rafaela, *Lr17* from Klein Lucero and Rafaela, and *Lr30* and *Lr34* from Terenzio. The Argentine cultivar Buck Manantial was determined to have *Lr3, Lr13, Lr16, Lr17,* and an unidentified adult-plant resistance gene (22).

Leaf rust resistance in the durum wheats has been examined only to a limited degree. Zhang & Knott (119) genetically determined that the cultivars Stewart 63 and Medora had two seedling resistance genes, and four other cultivars had a single gene. Dyck (29) isolated *Lr33* from Medora and Stewart and also from *T. turgidum* var. *dicoccoides* (27). Zhang & Knott (120) examined six cultivars for adult-plant resistance. The genes that conditioned resistance in seedling plants also conferred resistance in adult plants in field tests. Singh et al (108) examined nine CIMMYT cultivars for leaf rust resistance. Single resistance genes were in four cultivars and two genes in five cultivars. The nine cultivars had at least one gene in common.

Common Leaf Rust Resistance Genes in Winter Wheats

Leaf rust resistance in the winter wheats grown in the United States has been much shorter-lived than in the spring wheats. Less genetic work has also been done with leaf rust resistance in winter wheats compared to the spring wheats. Early hard red winter wheat cultivars such as Pawnee and Comanche had *Lr3,* which conditioned resistance to *P. recondita* race 9, the most common leaf rust race from 1930–1944 in the Great Plains of North America (15). The cultivars recently grown in Kansas and Nebraska are thought to contain various combinations of genes *Lr1, Lr3, Lr9, Lr10, Lr11, Lr14a, Lr16, Lr24,* and *Lr26,* based on gene postulation (10, 78, 79, 80). Parental breeding lines were determined to also have *Lr3ka, Lr17, Lr18,* and *Lr30* (79). McVey (78) examined 86 winter wheat cultivars from 26 countries and determined that genes *Lr1, Lr3, Lr10, Lr16, Lr24,* and *Lr26* were present. Genes *Lr10* and *Lr26* were the most common. Of the common seedling resistance genes in the hard red winter wheats, only *Lr9* and *Lr16* would currently condition effective levels of resistance in North America. However, virulence to these genes has been high in previous years when cultivars with these genes were grown (68). In recent years, cultivars with *Lr3ka, Lr11, Lr24,* and *Lr26* have been released in Kansas. These genes conditioned effective levels of resistance when first released; however, within 1–2 years isolates of *P. recondita* with the corresponding virulences were selected and rapidly increased in the Great Plains leaf rust population (56, 58, 68). Genes *Lr3* and *Lr26* are very common in European winter wheats (7, 118).

Adult-plant resistances have also been used in red winter wheat germplasm. Vigo had adult-plant resistance that was effective in Indiana from 1946–1957 (14). Knox was derived from a Vigo sib and a line with Chinese Spring resistance (13, 82). Dyck (23) determined that Chinese Spring had *Lr12* and *Lr34*. From 1961–1965, some of the resistance in Knox was eroded by changes in the *P. recondita* population (12). However, Knox continued to have a useful level of resistance until it was replaced by higher-yielding cultivars (62, 82). The erosion of resistance in Knox was probably due to the increase of *P. recondita* races with virulence to *Lr12*. The remaining adult-plant resistance in Knox was most likely due to *Lr34*.

The Brazilian cultivars Frondosa (*Lr13,*$^{+}$) and Fronteira (*Lr13,*$^{+}$) (95) are in the pedigrees of Atlas 50, Atlas 66, Coastal, Coker 47–27, Anderson, and Taylor (95). These cultivars may have *Lr13* in addition to *Lr34*. Bezostaja, a Russian winter wheat, was genetically determined to have *Lr13* and *Lr34* (PL Dyck, unpublished data), and the Texas cultivar Sturdy (70) had *Lr12* and *Lr34* (23).

ADULT-PLANT AND PARTIAL RESISTANCE TO WHEAT LEAF RUST

Terminology and Characteristics

Caldwell (12) was the first to characterize what has become known as partial, slow rusting, or general resistance in cereal crops. His description was based on the longevity and nonspecific nature of the slow rusting adult-plant resistance in the cultivar Knox. Parlevliet (83, 84), based on his experience with barley leaf rust, defined a number of terms that are commonly used in discussing this type of resistance. He defined partial resistance as a form of incomplete resistance in which the individual lesions are characterized by a susceptible infection type and which is conditioned by minor genes whose effects are too small to detect individually. Partial resistance was assumed to be more durable compared to resistance conditioned by single major resistance genes.

These definitions and characteristics used initially by Caldwell (12) and Parlevliet (83) have influenced subsequent research in slow rusting or partial leaf rust resistance in wheat. Ohm & Shaner (82) and Kuhn et al (62) compared the latent periods, uredinia sizes, and uredinia number/leaf area^2 between resistant slow rusting and susceptible wheats. In general, the slow rusting wheats had longer latent periods, fewer uredinia, and smaller uredinia sizes at 10–14 days after inoculation compared to the susceptible wheats. This characterization of slow rusting resembles the initial description (39) of the resistance conditioned by *Lr34*; fewer numbers of small- to moderate-sized uredinia throughout the leaves, with larger uredinia often near the base of the flag leaves. Dyck & Samborski (39) also noted a high degree of variability in the expression of *Lr34*; some plants with the gene could be easily distinguished as resistant, whereas others had rust reactions almost as high as susceptible check lines. Drijepondt & Pretorius (17) showed that adult plants with *Lr34* had significantly longer latent periods, fewer numbers of uredinia/leaf area^2, and significantly smaller sized uredinia compared to susceptible lines. Isolates with virulence to *Lr34* have not been detected in North America (JA Kolmer, unpublished data), so the resistance would appear to be nonspecific. The characteristics of resistance conditioned by *Lr34*, and the characteristics of slow rusting or partial resistance are identical. Many wheat cultivars characterized as having slow rusting or partial resistance (9, 82) have wheats related to either Chinese Spring or to Frontana in their pedigrees. It is very likely that adult-plant resistance genes *Lr12, Lr13,* and/or *Lr34* are present in these wheats.

Inheritance of Slow Rusting or Partial Resistance to Wheat Leaf Rust

Studies examining the inheritance of slow rusting or partial resistance to leaf rust in wheat (8, 9, 52, 63, 64) generally have had very similar results and conclusions. Segregation for latent period, or area under the disease progress curve, is found to occur in a continuous manner, indicating that two to three genes with small effects condition the resistance. Heritability estimates range from 0.5–0.9, indicating that slow rusting resistance can be selected in a breeding program. Since the resistance is conditioned by more than one gene, is apparently nonspecific, and pathogen reproduction is not totally curtailed, the slow rusting resistance is then stated to be of a more durable nature than resistance based on single genes. Since many of the wheats characterized for slow rusting or partial resistance were derived from known sources of *Lr34,* it is very likely that *Lr34* was segregating with possibly other genes in these studies. The incomplete, nonspecific resistance conditioned by *Lr34*, combined with the variable response of the gene within and between years, has probably lead many researchers to conclude that this was an example of slow rusting or partial resistance (sensu Parlevliet) that was conditioned by minor genes whose effects were too small to detect individually. Although *Lr34* does not condition an extremely low hypersensitive response in greenhouse or field tests, the gene has been isolated and mapped to a specific chromosome (21).

Lr34

Of the adult-plant genes that have been isolated and characterized, *Lr34* is probably the most important both in terms of widespread distribution and durability. This gene has been found in a number of wheats collected from diverse locations.

Dyck (19, 28) identified *Lr34* from wheats from Iran, China, Afghanistan, and Lebanon. Dyck (28) further identified *Lr34* from a number of wheats from Russia, Argentina, Tunisia, and France. Shang et al (102) found *Lr34* in wheats from Manuchuria and India.

Dyck (23) speculated that *Lr34* became widespread owing to its presence in commonly used parents such as Chinese Spring, which was introduced into South America shortly after 1900. The Argentine cultivar 38MA was developed from a cross between Barleta and a Chinese introduction that may have been Chinese Spring. 38MA appears in the pedigrees of many Argentine cultivars (55). South American wheats were subsequently used as resistance sources in wheat breeding programs in North America.

It is remarkable that *Lr34* has continued to condition an effective level of resistance despite being in cultivars that have been extensively grown for extended periods of time in many wheat growing areas throughout the world. There is no clear explanation for the longevity of *Lr34*'s effectiveness. For example, the wheat leaf rust fungus is present year-round in the wheat growing areas of South America. Wheats with *Lr34* have maintained effective levels of resistance in this region despite the large number of yearly uredinial generations that should give ample opportunity for isolates with virulence to this gene to increase within the *P. recondita* population.

Lr34 is tightly linked with, or is pleiotropic for resistance genes to stripe rust (*P. striiformis*) (73, 104) and barley yellow dwarf virus (BYDV) (106). Selection for *Lr34* would also select resistance to both stripe rust and BYDV. Selection for stripe rust resistance was most likely very important in breeding programs in South America. This may help to explain why *Lr34* is so common in wheats from that area.

Lr34 also contributes to stem rust (*P. graminis*) resistance in the North American hard red spring wheats. Dyck (25) showed that *Lr34* segregated with higher stem rust resistance in crosses with the cultivar Roblin. Dyck (21) previously noted that Thatcher near-isogenic lines with *Lr34* were always more stem rust resistant than the recurrent parent Thatcher. The stem rust resistance background of Thatcher (61) may be needed for the expression of the *Lr34* stem rust resistance. Since many Canadian and US hard red spring wheats are derived from Thatcher, the presence of *Lr34* in these wheats is an important component of their stem rust resistance.

Lr34 may also be present at more than one location in the wheat genome. Thatcher line RL 6077 has leaf rust resistance, stripe rust resistance (104), and leaf tip necrosis (103) similar to Thatcher line RL 6058 and other lines with *Lr34*. Dyck (21) suggested that RL 6077 probably has *Lr34*. RL 6077 and RL 6058 were intercrossed, and F_3 progeny lines segregated in a two-gene ratio for resistance. The stem and leaf rust resistance of five of the F_3 lines was slightly more effective than that of either of the parents (34). It is possible that these lines are homozygous for resistance from both RL 6077 and RL 6058.

Cytogenetic evidence from RL 6077/RL 6058 hybrids indicated that the *Lr34* gene in RL 6077 may be translocated onto another chromosome.

CONCLUSIONS

In most wheat growing areas of the world there are distressingly few genes that currently provide useful levels of leaf rust resistance. Virulences to seedling resistance genes may be low only because these genes have not been used in cultivars grown in that area. Use of these genes would quickly lead to the selection for the corresponding virulences in the *P. recondita* population, rendering the genes ineffective. The resistances that have been shown to be durable are almost inevitably conditioned by adult-plant resistance genes, often *Lr34*. However, based on past experiences with leaf rust, overreliance on *Lr34* would be unwise. There is no reason to assume that isolates of *P. recondita* with virulence to this gene will not eventually appear and quickly be selected in the pathogen population.

It is imperative that wheat and its related species continue to be genetically examined for the presence of new resistance genes to help maintain a diversity of effective resistance genes in released cultivars. In order to maintain progress in this area, new resistance genes should be isolated, genetically characterized relative to previously designated *Lr* genes, and incorporated into breeding programs. Research effort in this area is essential given the ability of *P. recondita* populations to overcome deployed resistance genes, and the paucity of effective genes that are currently at our disposal.

ACKNOWLEDGMENTS

I thank Dr. Peter Dyck for his outstanding contribution to wheat leaf rust genetics. This chapter was possible mainly because of his efforts.

Literature Cited

1. Anderson RG. 1961. The inheritance of leaf rust resistance in seven varieties of common wheat. *Can. J. Plant Sci.* 41:342–59
2. Anderson RG. 1963. Studies on the inheritance of resistance to leaf rust of wheat. *Hereditas* 2:144–55
3. Bai D, Knott DR. 1992. Suppression of rust resistance in bread wheat (*Triticum aestivum* L.) by D-genome chromosomes. *Genome* 35:276–82
4. Bariana HS, McIntosh RA. 1993. Cytogenetic studies in wheat. XV. Location of rust resistance genes in VPM1 and their genetic linkage with other disease resistance genes in chromosome 2A. *Genome* 36:476–82
5. Bariana HS, McIntosh RA. 1994. Characterization and origin of rust and powdery mildew resistance genes in VPM1 wheat. *Euphytica* 76:53–61
6. Bartos P, Dyck PL, Samborski DJ. 1969. Adult-plant leaf rust resistance in Thatcher and Marquis wheat: a genetic

analysis of the host-parasite interaction. *Can. J. Bot.* 47:267–69

7. Bartos P, Samborski DJ, Dyck PL. 1969. Leaf rust resistance of some European varieties of wheat. *Can. J. Bot.* 47:543–46
8. Bjarko ME, Line RF. 1988. Heritability and number of genes controlling leaf rust resistance in four cultivars of wheat. *Phytopathology* 78:457–61
9. Broers LHM, Jacobs TH. 1989. The inheritance of host plant effect on latency period of wheat leaf rust in spring wheat. II: Number of segregating factors and evidence for transgressive segregation in F3 and F5 generations. *Euphytica* 44:207–14
10. Browder LE. 1973. Probable genotype of some *Triticum aestivum* Agent derivatives for reaction to *Puccinia recondita* f. sp. *tritici*. *Crop Sci.* 13:203–6
11. Browder LE. 1980. A compendium of information about named genes for low reaction to *Puccinia recondita* in wheat. *Crop Sci.* 20:775–79
12. Caldwell RM. 1968. Breeding for general and/or specific plant disease resistance. In *Int. Wheat Genet. Symp., 3rd*, ed. KW Finlay, KW Shepherd, pp. 263–72. Sydney: Butterworth
13. Caldwell RM, Compton LE, Schafer JF, Patterson FL. 1954. Knox, a new leaf rust resistant and stem rust escaping soft winter wheat. *Phytopathology* 44:483–84
14. Caldwell RM, Schafer JF, Compton LE, Patterson FL. 1957. A mature-plant type of wheat leaf rust resistance of composite origin. *Phytopathology* 47:690–92
15. Chester KS. 1946. *The Nature and Prevention of the Cereal Rusts as Exemplified in the Leaf Rust of Wheat*. Waltham, MA: Chronica Botanica. 169 pp.
16. Cox TS, Raupp WJ, Gill BS. 1994. Leaf rust-resistance genes *Lr41*, *Lr42*, and *Lr43* transferred from *Triticum tauschii* to common wheat. *Crop Sci.* 34:339–43
17. Drijepondt SC, Pretorius ZA. 1989. Greenhouse evaluation of adult-plant resistance conferred by the gene *Lr34* to leaf rust of wheat. *Plant Dis.* 73:669–71
18. Dvorak J, Knott DR. 1990. Location of a *Triticum speltoides* chromosome segment conferring resistance to leaf rust in *Triticum aestivum*. *Genome* 33:892–97
19. Dyck PL. 1977. Genetics of leaf rust reactions in three introductions of common wheat. *Can. J. Genet. Cytol.* 19:711–16
20. Dyck PL. 1979. Identification of the gene for adult-plant leaf rust resistance in Thatcher. *Can. J. Plant Sci.* 59:499–501
21. Dyck PL. 1987. The association of a gene for leaf rust resistance with the chromosome 7D suppressor of stem rust resistance in common wheat. *Genome* 29:467–69
22. Dyck PL. 1989. The inheritance of leaf rust resistance in wheat cultivars Kenyon and Buck Manantial. *Can. J. Plant Sci.* 69:1113–17
23. Dyck PL. 1991. Genetics of adult-plant rust resistance in 'Chinese Spring' and 'Sturdy' wheats. *Crop Sci.* 24:309–11
24. Dyck PL. 1993. The inheritance of leaf rust resistance in the wheat cultivar Pasqua. *Can. J. Plant Sci.* 73:903–6
25. Dyck PL. 1993. Inheritance of leaf rust and stem rust resistance in 'Roblin' wheat. *Genome* 36:289–93
26. Dyck PL. 1994. Genetics of leaf rust resistance in 13 accessions of the Watkins wheat collection. *Euphytica* 80:151–55
27. Dyck PL. 1994. The transfer of leaf rust resistance from *Triticum turgidum* ssp. *dicoccoides* to hexaploid wheat. *Can. J. Plant Sci.* 74:671–73
28. Dyck PL. 1994. Genetics of resistance to leaf rust and stem rust on wheat. *Ann. Wheat Newslett.* 40:79–80
29. Dyck PL, Bartos P. 1994. Attempted transfer of leaf rust resistance from *Triticum monococcum* and durum wheat to hexaploid wheat. *Can. J. Plant Sci.* 74: 733–736
30. Dyck PL, Jedel PE. 1989. Genetics of resistance to leaf rust in two accessions of common wheat. *Can. J. Plant Sci.* 69:531–34
31. Dyck PL, Johnson R. 1983. Temperature sensitivity of genes for resistance in wheat to *Puccinia recondita*. *Can. J. Plant Pathol.* 5:229–34
32. Dyck PL, Kerber ER. 1970. Inheritance in hexaploid wheat of adult-plant leaf rust resistance derived from *Aegilops squarrosa*. *Can. J. Genet. Cytol.* 12:175–80
33. Dyck PL, Kerber ER. 1985. Resistance of the race-specific type. See Ref. 95a, 2:469–500
34. Dyck PL, Kerber ER, Aung T. 1994. An interchromosomal reciprocal translocation in wheat involving leaf rust resistance gene *Lr34*. *Genome* 37:556–59
35. Dyck PL, Lukow OM. 1988. The genetic analysis of two interspecific sources of leaf rust resistance and their effect on the quality of common wheat. *Can. J. Plant Sci.* 68:633–39
36. Dyck PL, Samborski DJ. 1968. Genetics of resistance to leaf rust in the common wheat varieties Webster, Loros, Brevit, Carina, Malakof and Centenario. *Can. J. Genet. Cytol.* 10:7–17

37. Dyck PL, Samborski DJ. 1970. The genetics of two alleles for leaf rust resistance at the *LR14* locus in wheat. *Can. J. Genet. Cytol.* 12:689–94
38. Dyck PL, Samborski DJ. 1974. Inheritance of virulence in *Puccinia recondita* on alleles at the *Lr2* locus for resistance in wheat. *Can. J. Genet. Cytol.* 16:323–32
39. Dyck PL, Samborski DJ. 1979. Adult-plant leaf rust resistance in PI 250413, an introduction of common wheat. *Can. J. Plant Sci.* 59:329–32
40. Dyck PL, Samborski DJ. 1982. The inheritance of resistance to *Puccinia recondita* in a group of common wheat cultivars. *Can. J. Genet. Cytol.* 24:273–83
41. Dyck PL, Samborski DJ, Anderson RG. 1966. Inheritance of adult-plant leaf rust resistance derived from the common wheat varieties Exchange and Frontana. *Can. J. Genet. Cytol.* 8:665–71
42. Dyck PL, Sykes EE. 1994. Genetics of leaf-rust resistance in three spelt wheats. *Can. J. Plant Sci.* 74:231–33
43. Ellingboe AH. 1984. Genetics of host-parasite relations: an essay. In *Advances in Plant Pathology,* ed. DS Ingram, PH Williams, 2:131–51. London: Academic Press
44. Ezzahiri B, Roelfs AP. 1989. Inheritance and expression of adult plant resistance to leaf rust in Era wheat. *Plant Dis.* 73:549–51
45. Flor HH. 1971. Current status of the gene-for-gene concept. *Annu. Rev. Phytopathol.* 9:275–96
46. Friebe B, Jiang J, Gill BS, Dyck PL. 1993. Radiation-induced nonhomologous wheat-*Agropyron intermedium* chromosomal translocations conferring resistance to leaf rust. *Theor. Appl. Genet.* 86:141–49
47. Friebe B, Zeller FJ, Mukai Y, Forster BP, Bartos P, Dyck PL. 1992. Characterization of rust-resistant wheat-*Agropyron intermedium* derivatives by C-banding, in situ hybridization and isozyme analysis. *Theor. Appl. Genet.* 83:775–82
48. Germán SE, Kolmer JA. 1992. Effect of gene *Lr34* in the enhancement of resistance to leaf rust of wheat. *Theor. Appl. Genet.* 84:97–105
49. Haggag MEA, Dyck PL. 1973. The inheritance of leaf rust resistance in four common wheat varieties possessing genes at or near the *Lr3* locus. *Can. J. Genet. Cytol.* 15:127–34
50. Haggag MEA, Samborski DJ, Dyck PL. 1973. Genetics of pathogenicity in three races of leaf rust on four wheat varieties. *Can. J. Genet. Cytol.* 15:73–82
51. Innes RL, Kerber ER. 1994. Resistance to wheat leaf rust and stem rust in *Triticum tauschii* and inheritance in hexaploid wheat of resistance transferred from *T. tauschii. Genome* 37:813–22
52. Jacobs TH, Broers LHM. 1989. The inheritance of host plant effect on latency period of wheat leaf rust in spring wheat. I: Estimation of gene action of effective factors in F1, F2 and backcross generations. *Euphytica* 44:197–206
53. Kerber ER, Dyck PL. 1990. Transfer to hexaploid wheat of linked genes for adult-plant leaf rust and seedling stem rust resistance from an amphiploid of *Aegilops speltoides* × *Triticum monococcum. Genome* 33:530–37
54. Knott DR. 1989. *The Wheat Rusts—Breeding for Resistance.* Berlin: Springer-Verlag. 201 pp.
55. Kohli MM. 1986. *Variedades de Trigo del Cono Sur de Sudamerica: Nombres, Progenitores; Genealogia y Origen.* Mexico, DF: CIMMYT. 68 pp.
56. Kolmer JA. 1989. Virulence and race dynamics of *Puccinia recondita* f. sp. *tritici* in Canada during 1956–1987. *Phytopathology* 79:349–56
57. Kolmer JA. 1992. Enhanced leaf rust resistance in wheat conditioned by resistance gene pairs with *Lr13. Euphytica* 61:123–30
58. Kolmer JA. 1994. Physiologic specialization of *Puccinia recondita* f. sp. *tritici* in Canada in 1992. *Can. J. Plant Pathol.* 16:61–63
59. Kolmer JA. 1994. Genetics of leaf rust resistance in three western Canada spring wheats. *Plant Dis.* 78:600–2
60. Kolmer JA, Dyck PL. 1994. Gene expression in the *Triticum aestivum-Puccinia recondita* f. sp. *tritici* gene-for-gene system. *Phytopathology* 84:437–40
61. Kolmer JA, Dyck PL, Roelfs AP. 1991. An appraisal of stem and leaf rust resistance in North American hard red spring wheats and the probability of multiple mutations in populations of cereal rust fungi. *Phytopathology* 81:237–39
62. Kuhn RC, Ohm HW, Shaner GE. 1978. Slow leaf-rusting resistance in wheat against twenty-two isolates of *Puccinia recondita. Phytopathology* 68:651–56
63. Lee TS, Shaner G. 1985. Transgressive segregation of length of latent period in crosses between slow leaf-rusting wheat cultivars. *Phytopathology* 75:643–47
64. Lee TS, Shaner G. 1985. Oligogenic inheritance of length of latent period in six slow leaf-rusting wheat cultivars. *Phy-*

topathology 75:636–43
65. Loegering WQ. 1985. Genetics of the pathogen-host association. See Ref. 95a, 1:165–92
66. Loegering WQ, McIntosh RA, Burton CH. 1971. Computer analysis of disease data to derive hypothetical genotypes for reaction of host varieties to pathogens. *Can. J. Genet. Cytol.* 13:742–48
67. Long DL, Kolmer JA. 1989. A North American system of nomenclature for *Puccinia recondita* f. sp. *tritici*. *Phytopathology* 79:525–29
68. Long DL, Roelfs AP, Roberts JJ. 1992. Virulence of *Puccinia recondita* f. sp. *tritici* in the United States during 1988–1990. *Plant Dis.* 76:495–99
69. Luig NH. 1985. Epidemiology in Australia and New Zealand. See Ref. 95a, 2:301–28
70. Marshall D. 1988. Characteristics of the 1984–1985 wheat leaf rust epidemic in central Texas. *Plant Dis.* 72:239–41
71. Marshall D. 1989. Virulence of *Puccinia recondita* and cultivar relationships in Texas from 1985 to 1987. *Plant Dis.* 73:306–8
72. McIntosh RA. 1992. Pre-emptive breeding to control wheat rusts. *Euphytica* 63:103–13
73. McIntosh RA. 1992. Close genetic linkage of genes conferring adult-plant resistance to leaf rust and stripe rust of wheat. *Plant Pathol.* 41:523–27
74. McIntosh RA. 1993. Catalogue of gene symbols for wheat. In *Proc. Int. Wheat Genet. Symp. 7th,* 1988, pp. 79–85
75. McIntosh RA, Dyck PL. 1972. Cytogenetical studies in wheat. VII. Gene *Lr23* for reaction to *Puccinia recondita* in Gabo and related cultivars. *Aust. J. Biol. Sci.* 25:765–73
76. McIntosh RA, Friebe B, Jiang J, The D, Gill BS. 1995. Cytogenetical studies in wheat XVI. Chromosome location of a new gene for resistance to leaf rust in a Japanese wheat-rye translocation line. *Euphytica* 82:141–47
77. McVey DV. 1989. Verification of infection-type data for identification of genes for resistance to leaf rust in some hard red spring wheat. *Crop Sci.* 29:304–7
78. McVey DV. 1992. Genes for rust resistance in International Winter Wheat nurseries XII through XVII. *Crop Sci.* 32:891–95
79. McVey DV, Long DL. 1993. Genes for leaf rust resistance in hard red winter wheat cultivars and parental lines. *Crop Sci.* 33:1373–81
80. Modawi RS, Browder LE, Heyne EG. 1985. Genes for low reaction to *Puccinia recondita* in Newton hard red winter wheat. *Crop Sci.* 25:13–16
81. Modawi RS, Browder LE, Heyne EG. 1985. Use of infection-type data to identify genes for low reaction to *Puccinia recondita* in several winter wheat cultivars. *Crop Sci.* 25:9–13
82. Ohm HW, Shaner GE. 1976. Three components of slow leaf-rusting at different growth stages in wheat. *Phytopathology* 66:1356–60
83. Parlevliet JE. 1985. Resistance of the non-race-specific type. See Ref. 95a, 2:501–25
84. Parlevliet JE. 1989. Identification and evaluation of quantitative resistance. In *Plant Disease Epidemiology,* ed. KJ Leonard, WE Fry, 2:215–48. New York: McGraw-Hill
85. Person C. 1959. Gene-for-gene relationships in host:parasite systems. *Can. J. Bot.* 37:1101–30
86. Pretorius ZA, Kemp GHJ. 1990. Effects of growth stage and temperature on components of resistance to leaf rust in wheat genotypes with *Lr26. Plant Dis.* 74:631–35
87. Pretorius ZA, Kloppers FJ, Drijepondt SC. 1994. Effect of inoculum density and temperature on three components of leaf rust resistance controlled by *Lr34* in wheat. *Euphytica* 74:91–96
88. Pretorius ZA, Rijkenberg FHJ, Wilcoxson RD. 1990. Influence of genetic background on the expression of wheat leaf rust resistance gene *Lr22a. Phytopathology* 80:579–84
89. Pretorius ZA, Wilcoxson RD, Long DL, Schafer JF. 1984. Detecting wheat leaf rust resistance gene *Lr13* in seedlings. *Plant Dis.* 68:585–86
90. Rajaram S, Singh RP, Torres S. 1988. Current CIMMYT approaches in breeding wheat for rust resistance. See Ref. 102a, pp. 101–18
91. Reddy MSS. 1980. Hypothetical genotypes for low reaction to *Puccinia recondita* in eight wheat cultivars of India. *Phytopathology* 70:392–93
92. Reddy MSS, Rao MV. 1980. Identification and cataloguing of genetic information for resistance to wheat leaf rust. *Euphytica* 29:769–75
93. Rizvi SSA, Buchenau GW. 1994. Tentative identification and verification of genes for leaf rust resistance in wheat cultivars of South Dakota. *Plant Dis.* 78:674–79
94. Rizvi SSA, Statler GD. 1982. Probable genotypes of hard red spring wheats for

resistance to *Puccinia recondita* f. sp. *tritici*. *Crop Sci.* 22:1167–70
95. Roelfs AP. 1988. Resistance to leaf and stem rusts in wheat. See Ref. 102a, pp. 10–22
95a. Roelfs AP, Bushnell WR, eds. 1995. *The Cereal Rusts.* Orlando, FL: Academic. Vol. 1, 2
96. Roelfs AP, Singh RP. 1992. *Rust Diseases of Wheat: Concepts and Methods of Disease Management.* Mexico, DF: CIMMYT, 81 pp.
97. Samborski DJ. 1985. Wheat leaf rust. See Ref. 95a, 2:39–59
98. Samborski DJ, Dyck PL. 1968. Inheritance of virulence in wheat leaf rust on the standard differential wheat varieties. *Can. J. Genet. Cytol.* 10:24–32
99. Samborski DJ, Dyck PL. 1976. Inheritance of virulence in *Puccinia recondita* on six backcross lines of wheat with single genes for resistance to leaf rust. *Can. J. Bot.* 54:1666–71
100. Samborski DJ, Dyck PL. 1982. Enhancement of resistance to *Puccinia recondita* by interactions of resistance genes in wheat. *Can. J. Plant Pathol.* 4:152–56
101. Schafer JF, Caldwell RM, Patterson FL, Compton LE. 1963. Wheat leaf rust resistance combinations. *Phytopathology* 53:569–73
102. Shang HS, Dyck PL, Samborski DJ. 1986. Inheritance of resistance to *Puccinia recondita* in a group of resistant accessions of common wheat. *Can. J. Plant Pathol.* 8:123–31
102a. Simmonds NW, Rajaram S, eds. 1988. *Breeding Strategies for Resistance to the Rusts of Wheat.* Mexico, DF: CIMMYT
103. Singh RP. 1992. Association between gene *Lr34* for leaf rust resistance and leaf tip necrosis in wheat. *Crop Sci.* 32:874–78
104. Singh RP. 1992. Genetic association of leaf rust resistance gene *Lr34* with adult plant resistance to stripe rust in bread wheat. *Phytopathology* 82:835–38
105. Singh RP. 1992. Expression of wheat leaf rust resistance gene *Lr34* in seedlings and adult plants. *Plant Dis.* 76:489–91
106. Singh RP. 1993. Genetic association of gene *Bdv1* for tolerance to barley yellow dwarf virus with genes *Lr34* and *Yr18* for adult plant resistance to rusts in bread wheat. *Plant Dis.* 77:1103–6
107. Singh RP. 1993. Resistance to leaf rust in 26 Mexican wheat cultivars. *Crop Sci.* 33:633–37
108. Singh RP, Bechere E, Abdalla O. 1993. Genetic analysis of resistance to leaf rust in nine durum wheats. *Plant Dis.* 77:460–63
109. Singh RP, Gupta AK. 1991. Genes for leaf rust resistance in Indian and Pakistani wheats tested with Mexican pathotypes of *Puccinia recondita* f. sp. *tritici*. *Euphytica* 57:27–36
110. Singh RP, McIntosh RA. 1984. Complementary genes for reaction to *Puccinia recondita tritici* in *Triticum aestivum*. I. Genetic and linkage studies. *Can. J. Genet. Cytol.* 26:723–35
111. Singh RP, McIntosh RA. 1984. Complementary genes for reaction to *Puccinia recondita* in *Triticum aestivum*. II. Cytogenetic studies. *Can. J. Genet. Cytol.* 26:736–42
112. Singh RP, Rajaram S. 1991. Resistance to *Puccinia recondita* f. sp. *tritici* in 50 Mexican bread wheat cultivars. *Crop Sci.* 31:1472–79
113. Singh RP, Rajaram S. 1992. Genetics of adult-plant resistance in Frontana and three CIMMYT wheats. *Genome* 35:24–31
114. Statler GD. 1977. Inheritance of virulence of culture 73–47 *Puccinia recondita*. *Phytopathology* 67:906–8
115. Statler GD. 1979. Inheritance of pathogenicity of culture 70–1, Race 1, of *Puccinia recondita tritici*. *Phytopathology* 69:661–63
116. Statler GD. 1984. Probable genes for leaf rust resistance in several hard red spring wheats. *Crop Sci.* 24:883–86
117. Statler GD, Jin Y. 1991. Inheritance of virulence of *Puccinia recondita* culture X43. *Can. J. Plant Pathol.* 13:33–37
118. Zadoks JC, Bouwman JJ. 1985. Epidemiology in Europe. See Ref. 95a, 2:329–69
119. Zhang H, Knott DR. 1990. Inheritance of leaf rust resistance in durum wheat. *Crop Sci.* 30:1218–22
120. Zhang H, Knott DR. 1993. Inheritance of adult plant resistance to leaf rust in six durum wheat cultivars. *Crop Sci.* 33:694–97