Plant Pathology (2001) **50**, 546–551

Simple inheritance of partial resistance to leaf rust in two wheat cultivars

J. A. Kolmer[a]*†‡ and J. Q. Liu[b]‡

[a]*USDA-ARS Cereal Disease Laboratory, University of Minnesota, St Paul, MN 55108; and* [b]*Pioneer Hi-Bred International, Johnston, IA 50131, USA*

The wheat cultivars Westphal 12A and BH1146 were characterized in previous studies as having partial resistance to the leaf rust pathogen *Puccinia triticina*. In the present study, genetic analysis showed that Westphal 12A has three genes that condition resistance in seedling plants, and the gene *Lr34*, which is optimally expressed in adult plants. The three seedling resistance genes in Westphal 12A may be new leaf-rust resistance genes. BH1146 was shown to have the adult plant resistance genes *Lr13* and *Lr34*. Many wheat cultivars with partial resistance to leaf rust may have *Lr13* and/or *Lr34*, or other previously described *Lr* genes.

Keywords: adult plant resistance, partial resistance, *Puccinia triticina*, slow-rusting resistance, *Triticum aestivum*

Introduction

Partial resistance to leaf rust (causal organism *Puccinia triticina*) in wheat (*Triticum aestivum*) has been characterized in an epidemiological sense by examination of components of the disease cycle that affect the rate of rust increase (Caldwell, 1968; Parlevliet, 1989). Latent period, in particular, was identified as an important component of partial resistance (Kuhn *et al*., 1978; Parlevliet, 1989). In wheat, latent period to leaf rust was inherited oligogenically in various cultivars (Lee & Shaner, 1985; Das *et al*., 1992; Shaner *et al*., 1997). In this approach, progeny lines in advanced segregating generations such as F_4 or F_6 were evaluated for latent period. The number of genes was estimated from the number of progeny lines that resembled the resistant or susceptible parents. The genes that conditioned latent period or slow rusting were not characterized individually, as progeny lines with single resistance genes from the resistant parent were not derived using this type of approach. Resistance based on epidemiological characteristics such as slow rusting or reduced latent period length has generally been assumed to be race-nonspecific, and thus to be durable.

Another approach for studying resistance to leaf rust in wheat has been the genetic isolation and characterization of single genes that condition infection type in seedling tests and the severity of rust infections in field tests (Dyck *et al*., 1966). In this method, F_1 plants are backcrossed to the susceptible parent and the backcross F_2 (BCF_2) families are examined for segregation of leaf-rust resistance in seedling and field tests. The number of genes conditioning resistance is estimated from the proportion of BCF_2 families that are homozygous susceptible, with rust severity and infection type equal to the susceptible parent. This crossing method allows the resistance genes to be evaluated in a relatively uniform genetic background as the BCF_2 families have a genetic background of 75% of the susceptible parent. Different leaf-rust resistance genes can be observed individually in the segregating BCF_2 families that differ for levels of leaf-rust resistance. Single plant selections from segregating BCF_2 families that differ for leaf-rust resistance can be used to establish progeny lines that have single adult plant resistance genes derived from the resistant parent. Adult plant resistance genes such as *Lr12*, *Lr13* and *Lr22b* condition specific resistance to leaf rust (Bartos *et al*., 1969; Kolmer, 1997). The adult plant resistance gene *Lr34* was shown to have many characteristics in common with genes that condition slow rusting or partial resistance (Drijepondt & Pretorius, 1989; Rubiales & Niks, 1995).

Broers & Jacobs (1989) studied the inheritance of latent period of leaf rust in the wheat cultivars Westphal 12A and BH1146. Based on segregating progeny lines in the F_5 generation, they concluded that Westphal 12A had three genes, and BH1146 two to three genes, for increased latent period. Van der Gaag & Jacobs (1997)

*To whom correspondence should be addressed.

†E-mail: jkolmer@cdl.umn.edu

‡This research was conducted while both authors were employed at the Agriculture and AgriFood Canada Cereal Research Centre, Winnipeg, Manitoba, R3T 2M9, Canada.

Accepted 28 April 2001.

tested F_8 lines and concluded that BH1146 carried at least five genes for increased latent period. In the present study, leaf-rust resistance in Westphal 12A and BH1146 was examined using an approach that would allow the isolation and characterization of individual resistance genes in the segregating progeny generations. The objective was to determine whether leaf-rust resistance in these slow-rusting or partially resistant wheat cultivars was due to the presence of previously identified genes such as *Lr13* and/or *Lr34*.

Materials and methods

Seeds of Westphal 12A and BH1146 were obtained from Th. Jacobs, Wageningen Agricultural University, the Netherlands. BH1146 was developed in Brazil from the pedigree of Ponta Grossa 1//Fronteira/Mentana. The origin of Westphal 12A is unclear. Jacobs & Broers (1989) indicated that Westphal 12A was derived from a landrace in Harage, Ethiopia. However, the International Wheat Information System (IWIS) database compiled by CIMMYT (Fox *et al*., 1997) indicated that a wheat cultivar designated Westphal 12 was derived from the pedigree Logoa-Vermelha//Frontana/Trintecino in Brazil, and was released in 1969.

BH1146 and Westphal 12A were used as pollen parents, and crossed with the leaf-rust-susceptible cultivar Thatcher (Tc). The F_1 plants were used as pollen parents and crossed with Tc. Westphal 12A, BH1146 and BCF_2 families of Tc × Westphal 12A were evaluated for seedling resistance in glasshouse tests with selected isolates of *P. triticina* (Long & Kolmer, 1989). BCF_2 families of Tc × BH1146 were not tested as seedlings, as BH1146 did not have any seedling resistance. Fifteen to 20 seedlings of each BCF_2 family were tested with each isolate of *P. triticina* used. Plants for the seedling tests were seeded in clumps in 25 × 30 cm fibre flats filled with a sand–peat–soil mix. Plants were grown at 20 ± 2°C with 8 h supplemental fluorescent light per day. Nine to 10 days after seeding, the primary leaves were inoculated by atomizing urediniospores suspended in a nonphytotoxic light mineral oil. Inoculated plants were incubated at 100% RH for 16 h at 20°C. Infection types (IT) on primary leaves were rated 12 days after inoculation using a scale of 0–4 (Long & Kolmer, 1989). Infection types of $0–2^+$ were considered resistant, and infection types 3–4 as susceptible. The BCF_2 families were classified as either segregating or homozygous susceptible. Goodness of fit to segregation ratios in BCF_2 families was determined using χ^2 tests (Steel & Torrie, 1980).

To evaluate adult plant resistance, ≈50 seeds of each BCF_2 family were planted in 2 m rows in a field rust nursery. Susceptible spreader rows of wheat were inoculated with a mixture of *P. triticina* isolates prevalent in the eastern prairie region of Canada (Kolmer & Liu, 1997; Kolmer, 1998). Westphal 12A, BH1146 and Thatcher lines near-isogenic for leaf-rust resistance were evaluated for leaf-rust severity and response in the field nursery. Rust ratings were recorded at the early milk stage (Zadoks growth stage 73–74) when the susceptible check (Thatcher) had a severity and response rating of 70% susceptible (70S). To determine the identity of the adult plant resistance genes in Westphal 12A and BH1146, single heads from resistant plants were selected from BCF_2 families. The selected BCF_2 heads were thrashed individually, and BCF_3 plants derived from a BCF_2 single seed were grown to maturity in a glasshouse. BCF_4 lines derived from BCF_3 plants were evaluated for adult plant resistance in glasshouse tests and also in the field rust nursery. In the glasshouse, flag and penultimate (flag−1) leaves were inoculated with *P. triticina* isolates that were virulent or avirulent to Tc*Lr13*. In the following year's rust nursery, BCF_4 lines from both crosses were compared with Tc*Lr13* and Tc*Lr34* for leaf-rust severity and response. Westphal 12A and BH1146 were also crossed with Tc*Lr13* and Tc*Lr34*, and the progenies were tested in the rust nursery. Approximately 600 F_2 plants from each of the four crosses were evaluated as adult plants for leaf-rust resistance in the field rust nursery.

Results

In seedling tests, Westphal 12A had low infection types

Table 1 Seedling infection types[a] and field responses to isolates of *Puccinia triticina* of wheat cultivars Westphal 12A, BH1146 and Thatcher wheat lines with *Lr13* and *Lr34*

	Isolate									
Cultivar	BBB 1-59	KBJ 139-1	MBQ 43-2	MBR 47-1	MFM 6-1	MFQ 53-2	MJB 10-3	TCT HG-3	TJB 50-1	Field severity
Westphal 12A	3+	4	4	3+	; c	; c	4	;22+	4	TR[b]
BH1146	4	4	4	4	4	4	4	3	4	TR−10 MR
Tc*Lr13*	4	4	4	4	4	4	4	4	4	80 MR S
Tc*Lr34*	3	3	3	3	3	3	3	3	3	20–70 M

[a]Infection types according to Long & Kolmer (1989). ;, fleck; c, chlorosis; ;c, flecks with chlorosis.

[b]TR, trace level of uredinia; MR, small uredinia with necrosis; MS, moderate size uredinia with chlorosis; M, mixture of small and large uredinia; S, large uredinia without necrosis or chlorosis.

Table 2 Segregation of leaf rust resistance in Thatcher × Westphal 12A and Thatcher × BH1146 BCF_2 families in seedling and field tests

Puccinia tricitina isolate	Number of families		Expected	
	Segregating	Susceptible	Ratio	χ^2
Tc^2 × Westphal 12A				
Seedling tests				
MFM 6-1	56	24	3 : 1	1·07
TCT HG-3	37	43	1 : 1	0·45
Field mixture[a]	55	16	3 : 1	0·23
			7 : 1	6·53
Tc^2 × BH1146				
Field mixture	65	12	3 : 1	3·64
			7 : 1	0·67

[a]Mixture of *P. triticina* isolates from Canada.

of ;c (chlorotic flecks) to isolates MFM 6-1 and MFQ 53-2, and infection type ;22^+ to TCT HG-3 (Table 1). Westphal 12A had high infection types of 3^+ to 4 to the other six *P. triticina* isolates. BH1146 had high infection types of 3^+ to 4 to all nine isolates. Both cultivars were highly resistant to leaf rust in field tests, with reactions of TR (trace level of uredinia) for Westphal 12A and TR-10 MR (trace level to 10% small uredinia with necrosis) for BH1146.

In seedling tests, the BCF_2 families of Tc × Westphal 12A with isolate MFM 6-1 segregated for at least two genes, as the number of segregating and of homozygous susceptible families approximated a 3 : 1 ratio (Table 2). One of the genes must condition a low-infection type of ;1 (flecks with small necrotic uredinia), as 37 of the segregating families had this infection type, while the other gene must condition a low infection type of ;2 (flecks with small chlorotic uredinia), as 19 segregating families had this infection type (Table 3). Based on infection types to the *P. triticina* isolates, the two genes that conditioned resistance to MFM 6-1 could not be identified as previously characterized *Lr* genes (McIntosh *et al.*, 1995). To isolate TCT HG-3, the BCF_2 families segregated for a single gene that conditioned infection type ;$2–2^+$ as the number of segregating to homozygous susceptible families approximated a 1 : 1 ratio (Table 2; Table 3). The single gene that conditioned seedling resistance to isolate TCT HG-3 could not be identified as a previously identified *Lr* gene. The BCF_2 families that segregated for two genes for resistance to MFM 6-1 were independent of those families that segregated for a single gene for resistance to TCT HG-3 according to the contingency table test (Table 3).

Table 3 Infection types and numbers of BCF_2 families of Thatcher × Westphal 12A in contingency test of independence for segregation of seedling resistance to isolates MFM 6-1 and TCT HG-3 of *Puccinia triticina*

	TCT HG-3		
	Segregating 22^+-4	Homozygous susceptible 3^+4	χ^2
MFM 6-1			
Segregating ;$1-2^+$-4	18	19	
Segregating ;$2-2^+$-4	11	8	
Homozygous susceptible 3^+-4	8	16	2·72

Table 4 Infection types and numbers of BCF_2 families of Thatcher × Westphal 12A in contingency table test of independence for segregation of seedling resistance to isolate MFM 6-1 and field resistance to *Puccinia triticina*

	Field resistance		
Seedling infection type	Segregating	Homozygous susceptible	χ^2
MFM 6-1			
Segregating ;$1-2^+$-4	25	8	
Segregating ;$2-2^+$-4	12	4	
Homozygous susceptible 3^+-4	18	6	0·006

In the field test, the number of segregating and homozygous susceptible BCF_2 families approximated a 3 : 1 ratio, indicating that two genes from Westphal 12A conditioned resistance to the mixture of *P. triticina* isolates (Table 2). The two genes derived from Westphal 12A that conditioned resistance to isolate MFM 6-1 did not condition field resistance, as the BCF_2 families that segregated for resistance to MFM 6-1 were independent of the families that segregated for field resistance (Table 4). Of the BCF_2 families that were homozygous susceptible to MFM 6-1, 18 segregated for field resistance and six were homozygous susceptible, which also indicated that two genes conditioned field resistance. The BCF_2 families that segregated for resistance to isolate TCT HG-3 also segregated for resistance to the field mixture of *P. triticina* isolates (Table 5). The single gene that conditioned resistance to TCT HG-3 was one of the two genes that conditioned resistance in the field test. Of the BCF_2 families homozygous susceptible to TCT HG-3, 21 segregated for resistance in the field test and 16 were homozygous

Table 5 Infection types and numbers of BCF_2 families of Thatcher × Westphal 12A in contingency test of independence for segregation of seedling resistance to isolate TCT HG-3 and field resistance to *Puccinia triticina*

	Field resistance		
Seedling infection type	Segregating	Homozygous susceptible	χ^2
TCT HG-3			
Segregating ;22^+-4	33	0	
Homozygous susceptible 3^+-4	21	16	18·51

Table 6 Adult plant infection types and field resistance to *Puccinia triticina* of Thatcher × Westphal 12A and Thatcher × BH1146 BCF_4 lines

Line	Isolate			Field	Gene present[a]
	TDB 22–1	MBRJ 10–2	MBDS 12–3		
Tc^2 × Westphal 12A					
323	3^+	3^+	3^+	10 M	*Lr34*
Tc^2 × BH1146					
387	3^+	3^+	3^+	20-70 M	*Lr34*
409	;22$^-$	3^+	3^+	80 MR S	*Lr13*
Thatcher *Lr13*	2	4	4	80 MR S	
Thatcher *Lr34*	23	23	23	5-40 M	
Thatcher	4	4	4	90 S	

[a]M, mixed small and large uredinia; MR, small uredinia with necrosis; S, large uredinia without necrosis or chlorosis.

for resistance: this indicated that the second gene that conditioned resistance in the field test did not condition resistance in seedlings. The BCF_4 line 323, derived from a single plant selection from a BCF_2 family homozygous susceptible to both isolates MFM 6-1 and TCT HG-3, had a field leaf rust severity and reaction similar to Tc*Lr34* (Table 6). Approximately 600 F_2 adult plants from the cross of Tc*Lr34* × Westphal 12A were all resistant in a field test to the mixture of *P. triticina* isolates. Five susceptible plants were found in the F_2 progeny of Tc*Lr13* × Westphal 12A, which indicated that *Lr13* was not in Westphal 12A. A total of four leaf-rust resistance genes was found in Westphal 12A. Two genes conditioned seedling resistance to isolate MFM 6-1. A single gene conditioned resistance to isolate TCT HG-3, and also resistance to the field mixture of *P. triticina* isolates. A fourth gene, which conditioned adult plant resistance to the mixture of *P. triticina* isolates, was probably *Lr34*.

The number of segregating or homozygous susceptible BCF_2 families in field tests from Thatcher × BH1146 fit both 3 : 1 and 7 : 1 ratios, which indicated that at least two – possibly three – genes conditioned field resistance to the mixture of *P. triticina* isolates (Table 2). The BCF_4 line 387, derived from a single plant selection from a BCF_2 family that segregated for resistance, had a field leaf-rust reaction and severity similar to Tc*Lr34* (Table 6). Line 387 had infection type of 3^+ to isolates TDBJ 22-1, MBRJ 10-2, and MBDS 12-3, which was slightly higher than the 2–3 infection type these isolates had to Tc*Lr34*. The BCF_4 line 409, derived from a single plant selection from a BCF_2 family that segregated for resistance, had a field leaf rust severity and reaction similar to Tc*Lr13*. Line 409 also had adult plant infection types to isolates TDBJ 22-1, MBRJ 10-2 and MBDS 12-3 very similar to Tc*Lr13*. Approximately 600 F_2 plants from each of Tc*Lr13* × BH1146 and Tc*Lr34* × BH1146 were all leaf-rust resistant, which indicated that BH1146 has *Lr13* and *Lr34*.

Discussion

The wheat cultivars Westphal 12A and BH1146 were shown to have leaf-rust resistance genes that could be isolated and characterized individually. Both Westphal 12A and BH1146 probably have the adult plant resistance gene *Lr34*. Westphal 12A also has a gene that conditioned an intermediate infection type to specific leaf-rust isolates in seedling plants and an intermediate level of field resistance, and two other genes that conditioned seedling resistance, but not field resistance. Broers & Jacobs (1989), using data from F_3 and F_5 lines from crosses with the susceptible parent Little Club, estimated that Westphal 12A had three genes that prolonged the latent period, and BH1146 two to three genes. Broers & Jacobs (1989) also indicated that *Lr13* may be present in BH1146, in addition to the genes that affected latent period. In regard to the number of genes that conditioned field resistance, the results of this study are in general agreement with Broers & Jacobs (1989), as Westphal 12A was determined to have two genes that conditioned field resistance, and BH1146 also had at least two genes. Resistance gene *Lr13* was also identified in progeny lines derived from BH1146.

Van der Gaag & Jacobs (1997) further examined F_8 lines of BH1146 × Little Club for latent period distribution. Four to seven genes for prolonged latent period in BH1146 were estimated based on only one of 75 F_8 lines having a latent period equal to BH1146, and two lines having latent periods equal to the cultivar Morocco, used as the susceptible standard. However, if Little Club is used as the susceptible standard, then 57 of the F_8 lines had a latent period longer than Little Club, and 18 lines had latent periods equal to or shorter than Little Club. This grouping of segregating lines fits a 3 : 1 ratio, which indicated that BH1146 has two genes for prolonged latent period relative to the susceptible parent Little Club.

Rubiales & Niks (1995) characterized BH1146, Tc*Lr34*, Tc*Lr13* and Thatcher for infection type, latent period and infection frequency (number of uredinia per leaf area). Tc*Lr13* had chlorotic flecks with few susceptible-type uredinia. Thatcher and BH1146 both had large uredinia without necrosis or chlorosis. Tc*Lr34* had a significantly longer latent period than Thatcher, but did not differ significantly from BH1146

when tested with the same leaf-rust isolates. BH1146 also did not differ significantly from Thatcher or Tc*Lr13* for latent period. Thatcher and BH1146 did not differ significantly for uredinia per leaf area, while Tc*Lr34* and Tc*Lr13* had significantly lower uredinia per leaf area. Based on differences in uredinia per leaf area and histological observations, Rubiales & Niks (1995) concluded that the resistance expressed in BH1146 was not due to *Lr13* or *Lr34*.

Using an approach that allowed the isolation and characterization of single resistance genes, it was determined that adult plant resistance to leaf rust in Westphal 12A was conditioned by *Lr34* and a previously unidentified gene which also conditioned resistance in seedlings to specific *P. triticina* isolates. In BH1146, *Lr13* and *Lr34* conditioned adult plant resistance. From both crosses, progeny lines were derived that had resistance levels similar or identical to *Lr34*, and F_2 progenies from crosses with Tc*Lr34* did not segregate for susceptible plants, which indicated that *Lr34* was present in both Westphal 12A and BH1146. Progeny lines derived from BH1146 had infection types and rust severities similar to Tc*Lr13*, and F_2 progenies from a cross with Tc*Lr13* did not segregate for susceptible plants, which indicated that BH1146 has *Lr13*. If Westphal 12A originated from Brazil, the pedigree given by the IWIS database (Fox *et al*., 1997) includes Frontana, which has both *Lr13* and *Lr34*. Fronteira, which is in the pedigree of BH1146, has been postulated to have *Lr13* (Roelfs, 1988). Roelfs *et al*. (1992) indicated that BH1146 has *Lr13* and *Lr34*. The leaf-rust resistance conditioned by *Lr13* and *Lr34* has similar characteristics to the partial resistance conditioned by BH1146, as determined by Drijepondt & Pretorius (1989). Adult plants with *Lr13* often have 22^+ (small to moderate size uredinia with chlorosis) infection type to isolates that produced 3^+ to 4 infection types in seedling plants (Kolmer, 1997). For *Lr34*, Rubiales & Niks (1995) cited increased resistance at low temperatures; the absence of hypersensitive response; long latent period; and smaller uredinia size as characteristics that this gene shares with partial resistance. Rubiales & Niks (1995) also determined that Tc*Lr34* had a significantly higher infection frequency than BH1146. This might be caused by differences in cultivar backgrounds, as a resistance gene may be expressed differently in various genetic backgrounds (Dyck & Samborski, 1968; Pretorius *et al*., 1990). It is also possible that adult plants of BH1146 and Tc*Lr34* at the same growth stage would be sufficiently different in terms of plant age to affect the infection frequency. As the two wheat lines differ for maturity, flag leaves at the same growth stage would differ for age. BH1146 and Thatcher did not differ significantly for latent period or infection frequency (Rubiales & Niks, 1995), yet Thatcher was highly susceptible to leaf rust and BH1146 was very resistant in field tests in Canada. Latent period or other measures of partial resistance may not always be predictive of adult plant leaf-rust resistance in wheat. The presence or absence of leaf-rust resistance genes in wheat should be determined by direct genetic analysis, as indirect methods such as measurement of latent period may not be adequate.

The premise of partial, slow rusting, or horizontal resistance, has been that epidemiological components of the disease cycle which affect the rate of pathogen increase confer a nonspecific resistance. As this partial or slow-rusting resistance is nonspecific, it is usually implied to be a durable type of resistance. Adult plants with *Lr34* were shown to condition an intermediate level of resistance to all *P. triticina* isolates that have been tested (Kolmer, 1997), thus having an incomplete, apparently nonspecific resistance. In North America, wheat cultivars with *Lr34* have displayed at least moderate levels of resistance to leaf rust, as this gene was introduced in wheat germplasm in the mid 1960s (Kolmer *et al*., 1991). Genotypes with *Lr34* can also express variable amounts of leaf-rust severity as adult plants in glasshouse and field tests. In an inheritance study of latent period with advanced inbred lines (Van der Gaag & Jacobs, 1997; Shaner *et al*., 1997), the intermediate and variable level of resistance conditioned by *Lr34* could be interpreted as being conditioned by more than one gene. Many of the wheats that have been characterized as having partial or slow-rusting resistance to leaf rust may actually have *Lr34* as a major component of their resistance. Genetic analysis of leaf-rust-resistant wheat cultivars should be conducted in such a manner as to allow a direct comparison of results with previous studies. Only by their isolation and characterization can resistance genes be conclusively identified and assessed as potential new sources of leaf rust resistance.

References

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. *Canadian Journal of Botany* **47**, 267–9.

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.

Caldwell RM, 1968. Breeding for general and/or specific plant disease resistance. In: Finlay KW Shepherd KW, eds. *Third International Wheat Genetics Symposium*. Sydney, Australia: Butterworth, 263–74.

Das MK, RajaramS, Mundt CC, Kronstad WE, 1992. Inheritance of slow-rusting resistance to leaf rust in wheat. *Crop Science* **32**, 1452–6.

Drijepondt SC, Pretorius ZA, 1989. Greenhouse evaluation of adult-plant resistance conferred by the gene *Lr34* to leaf rust of wheat. *Plant Disease* **73**, 669–71.

Dyck PL, Samborski DJ, 1968. Genetics of resistance to leaf rust in the common wheat varieties Webster, Loros, Brevit, Carina, Malakof and Centenario. *Canadian Journal of Genetics and Cytology* **10**, 7–17.

Dyck PL, Samborski DJ, Anderson RG, 1966. Inheritance of

adult-plant leaf rust resistance derived from the common wheat varieties Exchange and Frontana. *Canadian Journal of Genetics and Cytology* **8**, 665–71.

Fox PN, Magana RI, Lopez C, Sanchez H, Herrera R, Vicarte V, White JW, Skovmand B, Mackay MC, 1997. *International Wheat Information System (IWIS)*, Version 2. Mexico, D.F.: CIMMYT.

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 and number of effective factors in F1, F2, and backcross generations. *Euphytica* **44**, 197–206.

Kolmer JA, 1997. Virulence in *Puccinia recondita* f. sp. *tritici* isolates from Canada to genes for adult-plant resistance to wheat leaf rust. *Plant Disease* **81**, 267–71.

Kolmer JA, 1998. Physiologic specialization of *Puccinia recondita* f. sp. *tritici* in Canada in 1996. *Canadian Journal of Plant Pathology* **20**, 176–81.

Kolmer JA, Liu JQ, 1997. Physiological specialization of *Puccinia recondita* f. sp. *tritici* in Canada in 1995. *Canadian Journal of Plant Pathology* **19**, 166–70.

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**, 237–9.

Kuhn RC, Ohm HW, Shaner GE, 1978. Slow leaf-rusting resistance in wheat against twenty-two isolates of *Puccinia recondita*. *Phytopathology* **68**, 651–6.

Lee TS, Shaner G, 1985. Oligogenic inheritance of length of latent period in six slow leaf-rusting wheat cultivars. *Phytopathology* **75**, 636–43.

Long DL, Kolmer JA, 1989. A North American system of nomenclature for *Puccinia recondita* f. sp. *tritici*. *Phytopathology* **79**, 525–9.

McIntosh RA, Wellings CR, Park RF, 1995. *Wheat Rusts: An Atlas of Resistance Genes*. Australia/Dordrecht, the Netherlands: CSIRO/Kluwer Academic.

Parlevliet JE, 1989. Identification and evaluation of quantitative resistance. In: Leonard KJ, Fry WE, eds. *Plant Disease Epidemiology*, **Vol. 2**. New York, USA: McGraw-Hill, 215–48.

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.

Roelfs AP, 1988. Resistance to leaf and stem rusts in wheat. In: Simmonds NW, Rajaram S, eds. *Breeding Strategies for Resistance to the Rusts of Wheat*. Mexico, D.F.: CIMMYT, 10–22.

Roelfs AP, Singh RP, Saari EE, 1992. *Rust Diseases of Wheat: Concepts and Methods of Disease Management*. Mexico, D.F.: CIMMYT.

Rubiales D, Niks RE, 1995. Characterization of *Lr34*, a major gene conferring nonhypersensitive resistance to wheat leaf rust. *Plant Disease* **79**, 1208–12.

Shaner G, Buechley G, Nyquist WE, 1997. Inheritance of latent period of *Puccinia recondita* in wheat. *Crop Science* **37**, 748–56.

Steel RD, Torrie JH, 1980. *Principles and Procedures of Statistics*. New York, NY: McGraw-Hill.

Van der Gaag DJ, Jacobs T, 1997. Inheritance of host plant effect on latent period of wheat leaf rust in single-seed descent F_8 lines. *Euphytica* **97**, 67–72.