Seedling Resistance Genes to Leaf Rust in Soft Red Winter Wheat

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ABSTRACT

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Seedling and adult-plant resistance have been used to manage leaf rust, caused by *Puccinia triticina*, but there is little information on resistance genes in contemporary cultivars and advanced breeding lines of soft red winter wheat (*Triticum aestivum*). Lack of information on the genetic basis for resistance leads to uncertainty about durability of resistance and makes pyramiding resistance genes more difficult. The objective of this study was to determine the genetic basis for race-specific seedling resistance to leaf rust among the 116 contemporary lines from the 1998–99 Arkansas Wheat Cultivar Performance Test and the Uniform Eastern and Southern Soft Red Winter Wheat Nurseries. To postulate the presence of genes for leaf rust resistance (*Lr* genes), seedlings of each line and 24 isolines in a Thatcher background were evaluated for infection type in growth chambers at 22/18°C (day/night) or constant 17 or 18°C using 22 races of *P. triticina*. A computer program was used to analyze infection type data and facilitate identification of *Lr* genes. Genes *Lr1*, *2a*, *2c*, *3*, *3ka*, *9*, *10*, *11*, *14a*, *18*, *20*, *23*, *24*, and *26* were identified among the lines tested. Genes *Lr3*, *10*, and *11* were the most common. Genes *Lr15*, *28*, and *30* were postulated as possibly present in some lines but were not likely to be important among the lines. Genes *Lr16*, *17*, *21*, *32*, *36*, *38*, and *39* were not detected. Fifty-four lines had one or more unidentified *Lr* genes that were not included in the set of 24 isolines. Only four lines (Agripro Marion, APD94-5282, NC94-7197, and VA97W-375) were resistant to all races used in this study, and these were postulated to have the combination of *Lr9*, *24*, and *26*.

Additional keywords: gene-for-gene specificity

Leaf rust, caused by *Puccinia triticina* Eriks. (syn: *Puccinia recondita* Rob. ex. Desm. f. sp. *tritici*), is probably the most important and widespread disease of wheat (*Triticum aestivum* L.) (23,33). Leaf rust is adapted to a range of climates and occurs wherever wheat is grown (10). Yield loss estimates from leaf rust across all states that predominantly grow soft red winter wheat (Alabama, Arkansas, Florida, Georgia, Illinois, Indiana, Kentucky, Louisiana, Mississippi, Missouri, North Carolina, Ohio, South Carolina, Tennessee, and Virginia) averaged 1.2% (range 0.3% to 1.9%) from 1997 to 2001 (16). The estimated losses from leaf rust during this period were more than 555,000 metric tons, representing a loss of more than \$61,000,000 to growers. Without resistant cultivars, the loss would have been much greater.

P. triticina has diverse virulence and the ability to overcome resistance genes. New races typically overcome resistance in winter wheat cultivars in a shorter period than resistance in spring wheat cultivars grown in the United States (12). The use of resistance against *P. triticina* has often

been a single gene at a time (30) and has largely depended on replacing resistance genes upon detection of virulent races (33). However, resistance that is based on a single gene can easily be overcome by a change in virulence at a single locus in the pathogen (9). With a limited number of resistance genes in hexaploid wheat (22,36), and with the difficulties and associated problems of transferring genes from related species through traditional breeding, combinations of genes are believed to provide longer lasting resistance to leaf rust than single genes (30).

Genetic interactions between wheat and *P. triticina* have been shown to operate in a gene-for-gene relationship (32). Gene postulation applies the principles of gene-for-gene specificity to determine the most probable resistance genes present in host lines (12). Resistance genes can be identified based on the infection type expressed on the host when inoculated with a series of pathogen races with known virulence and avirulence on individual resistance genes (24). Lines that are susceptible to one or more races of the pathogen are the most amenable to gene postulation. Gene postulation is the most widely used method to identify *Lr* genes, and several researchers have used this approach to identify *Lr* genes in various populations of wheat lines. Statler (42) postulated *Lr1*, *2a*, *2c*, *10*, *17*, and *18* among 25 hard red spring wheats. Modawi et al. (27) identified *Lr3a*, *9*, *10*, and *24* among 30 winter wheat culti-

vars. McVey (25) postulated *Lr1*, *3*, *10*, *16*, *24*, and *26* among 86 winter wheat cultivars obtained from international winter wheat nurseries from 26 countries. McVey and Long (26) postulated *Lr1*, *2a*, *3*, *3ka*, *9*, *10*, *11*, *14a*, *16*, *17*, *18*, *24*, *26*, and *30* among 86 hard red winter wheat lines. Singh (38) identified *Lr1*, *3*, *13*, *16*, *17*, *23*, *26*, and *34* among 26 Mexican wheat cultivars. Singh and Rajaram (40) postulated *Lr1*, *3*, *3bg*, *10*, *13*, *14a*, *16*, *17*, *19*, *23*, *26*, and *27+31* among 50 Mexican cultivars. Singh et al. (39) postulated *Lr1*, *3*, *3bg*, *10*, *13*, *14a*, *16*, *23*, and *26* among 61 spring and 102 facultative and winter wheat cultivars from China. Singh et al. (37) postulated *Lr1*, *3a*, *10*, *13*, *17b*, *20*, *26*, and *37* among 70 cultivars from the United Kingdom. Gene *Lr26* has been associated with wheat lines that have the 1BL.1RS translocation from rye (43).

Of 125 soft red winter wheat cultivars summarized by Roelfs et al. (31), 111 were considered old and the remaining 14 that overlapped in this study were considered contemporary. Genes *Lr1*, *2a*, *3*, *9*, *10*, *11*, *13*, *18*, *24*, *26*, *3bg*, *14a*, *14b*, *16*, *19*, *20*, and *31* were postulated in the old soft red winter wheat, of which the last seven were present in less than 2% of the cultivars.

The expression of some *Lr* genes is dependent on postinoculation temperatures. Browder (1) classified genes *Lr11*, *14a*, and *18* as highly sensitive and *Lr3ka* as moderately sensitive to temperature. Dyck and Johnson (4) reported better expression of *Lr11*, *14a*, and *18* at temperatures below 20°C; expression of *Lr3ka* varied among races but not with temperature. Genes *Lr11* and *18* were most effective at 14 to 18°C in greenhouse studies (40), and Park et al. (28) detected *Lr18* at 18 to 20°C.

Expression of some *Lr* genes has been shown to differ depending on whether the pathogen isolate was homozygous or heterozygous for avirulence. Kolmer and Dyck (15) reported that avirulence for genes *Lr2a*, *2c*, *3ka*, *11*, and *30* was partially (incompletely) dominant because isolates of *P. triticina* that were heterozygous for avirulence produced higher infection types than isolates that were homozygous for avirulence when tested on homozygous resistant wheat lines. Heterozygosity for avirulence to *Lr* genes appears to be common among field collections of *P. triticina*. Samborski and Dyck (34) found that among four races (1, 9, 15, 161), only race 9 was homozygous at all loci tested. Races 1, 15, and 161 segregated at a number of loci for avirulence when these races were selfed. Likewise,

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with the *P. triticina* population in eastern Canada, frequencies of virulence to *Lr3ka*, *11*, *17*, and *30* increased in aecial populations compared with uredial populations, indicating heterozygosity for avirulence (11).

Resistance to leaf rust in hard red winter wheat lines at the seedling stage is likely due to some combinations of race-specific seedling genes, probably *Lr1*, *3*, *3ka*, *9*, *10*, *11*, *14a*, *16*, *17*, *18*, *24*, *26*, and *30* (12,25,26). However, less work has been

done on the genetic basis for leaf rust resistance in winter wheat than in spring wheat (12). Lack of information on the genetic basis for resistance leads to uncertainty about durability of resistance and makes pyramiding resistance genes more

Table 1. Entries and pedigrees for the 1998–99 Arkansas Cultivar Performance Test and Uniform Southern and Eastern soft red winter wheat nursery

Entry	Pedigree	Entry	Pedigree
Agripro Foster	KY 83-60/Tyler//KY 83-75	NC95-25305	C39/3/FL302/C8320//Cc(Pm1)
Agripro Hickory	Hunter/Caldwell	NC95-25707	C8629/3/KS8338422//Stella/Caldwell
Agripro Mallard	Wheeler/Caldwell	NK Coker 9025	AL850046/Coker 86-23
Agripro Marion	VA82-52-64/AP-SW85-50009	NK Coker 9152	Coker 86-27/Coker 9733//Pioneer 2555
Agripro Mason	Cardinal//MN74143/Oligoculm/3/Coker 9232	NK Coker 9184	Coker 9803/Coker 9835
Agripro Patton	SW85*94/IN82104B1-3-2	NK Coker 9543	Coker 916//Oasis/Coker 762
Agripro Shelby	FL302/7/C81132/5/SO.Belle/4/Riley/Stoddart// Top/3/Arthur/6/Coker 983	NK Coker 9663	IN71761A4-31-5-48/FL 302
Agripro Shiloh	Coker 833/Becker	NK Coker 9704	Coker 9803/Coker 983
AGS2000	Pioneer 2551/PF84301//FL302	NK Coker 9835	Coker 85-20/Pioneer 2550
AP-D94-5282	AP-E86-5258/AP-SW85-5009	OH 536	P71761A4-31-5-33/MD55-286-21
APD95*8811-1	Coker 9835/APE87-1785	OH 552	P71761A4-31-5-33/MD55-286-21
APD95*8811-2	Coker 9835/APE87-1785	OH599	PS-840026/OH416
APD95-7763	Wakefield/Coker 9877	OK91P648-41	2555 sib/Vona//2180
Armor 3235	Pioneer 2550/Keiser	P86364RA1-4-2-7	INW9241//Clark/IL79-1458
AR 584A-3-1	FL 302//Coker 833/Hunter	P88204RB1-2-1-6	P86982C2/P844030H1//P831800A1/Grant
BL931167	Pioneer Exp.9021/FL791G63	Pioneer 2548	Hadden*2/3/GA 1123//N10-B/Tenmarq/4/ MoW6582/Redcoat/5/Coker 68-15/4/Etiolo de Chotsy//Thorne/Clarkan/3/Pawnee/IN3848A5
BL940026	Coker 762//Coker 75-30//Coker 80-28	Pioneer 2684	PL W9057B/Caldwell//Hunter
Buckshot's DS9193	Not available	Pioneer 26R24	Aurora/Tyler//2553/2550 sib/3/Cok 983/4/Cok 87-13
Caldwell	Benhur sib*2/Siete Cerros	Pioneer 26R46	FL7927-G14//2555*3.Coker 80-28
Cardinal	Logan*2/3/VA63-5-12/Logan//Blueboy	Pioneer 26R61	Omega 78/Pio S76/4/Arthur71/3/stadler//Redcoat/ Wsc.1/5/ Coker 747/6/2555sib
Delta Grow 3300	Not available	Pioneer XW672	2548/3/2555*3//2555 sib/KU81H1640/5/McN1003/ Caldwell/4/ IN4946A4/MoW7510//Cok71/3/2550 sib
Delta King 1551W	Not available	Pocahontas	Wheeler*2/C39//Saluda
Delta King 9027	Not available	Quantum 708	Not available
Delta King 9051	Not available	Roane	VA71-54-147/Coker 68-15//IN65309C7-18-2-3-2
Delta King 9121	Not available	Roberts	Coker 86-3/Gore
Dixie 2000	Not available	Sabbe	Corin/3/FL302//Coker 833/Hunter
Dixie 911	Not available	SS36803	831127-3//821264*3/79102
EK 103	Not Available	S9412192	FL7925-G47-J10/FL8062-E4-H7-J1
EK 156	Not available	SC921285	SL4/67-78/7/Tift72-60/Arthur 71//Gabota/3/ LourinLL/4/ Tift72-60/Arthur 71/5/Beau/6/C76- 22/8/Brule
EK 114	Not available	SC921299	SL4/67-78/7/Tift72-60/Arthur 71//Gabota/3/ LourinLL/4/ Tift72-60/Arthur 71/5/Beau/6/C76- 22/8/Brule
F322W	Not available	SS550	Coker 9803/Freedom
FFR 522W	P71761/Coker 797	Stine 455	Not available
FFR 558W	Not available	Stine 488	Not available
FL8868	FL302-H9 backcross line/GA781176 (McN1003/Ck 762)	Stine 950455	Not available
Florida 302	Coker 65-20//P4946A4-18-2-10-1/Hadden/3/ Vogel/5/ Anderson//P4946A4-18-2-10-1/Hadden	T104	T814*2/T408
G41332	IL84-3010/T812	T105	T814*2/T408
G41333	IL84-3010/T812	T106	T814*2/T408
G60232	IL84-3511/T812	T109	T85/T814
GA881404E56	C983/4/Aepoglom//C762/FL302/3/C762	Terra E218	Not available
GA88622E51	Gore//831466/GA100	Terra SR 204	Not available
GA90524E35	Coker 9835//FL302/Gore	Terra SR216	Not available
HT98-10033	Feland/Becker//Chay/Stella	Terral TV 8555	Not available
HT98-10291	Tyler*2/C65-20/Atr	Terral TV 8768	Not available
IL91-15911	IL84-3511/Caldwell	Terral TV8825	Not available
IL94-2894	IL85-3132-1 (McN1003/Caldwell)/PSRW 71	Terral TVX8670	Not available
IL94-6727	IL87-3721 (Cardinal/Howell)/2*IL85-3132-1 (McN1003/Caldwell)	TW94315	Casey/Century
Jackson	Saluda/Coker 762	TX87-20	Caldwell/Sioux land
Jaypee	Arthur 6/AR 39-3 (Doublecrop//Forlani/Garibaldo)	TX91-13	FL8172-G98-L5/Gore
KY86C-127-3	NA-SW76-59/NA-SW78-111 = composite cross//Knox 62/SRW 14-74	USG 3209	Saluda/4/Massey*2/2/Massey*3/BALKAN//Saluda
LA 87167D8-10-2	GA8787:FR81-19/FL302//Coker 983	USG 3408	Not available
LA8513B1-7-B-1-4-2	FL302/Coker 762	USG Exp 97-4	Not available
LA90144B16-3-2	Pioneer 2548/Coker 9835	VA96-54-326	SC861562/Coker 9803
LA90412F14-1-4	..ms/3/Ck983/4/..ms/3/N76-261/5/SC810779/6/ OP-ms/7/ Coker 797/8/Coker 9835	VA97W-375	Coker 9803/Freedom
LA9070G45-3-3-1	Savannah/CEP75203//FL85363-G21-6	Wakefield	Composite of Arthur//Doublecrop//Abe/VA68-24- 42/3/and Oasis/VA68-24-42//BYC113836/8* Chancellor F3 Derivatives
M95*2883	Magnum/P101//OH394	Wilfarm 488	Not available
M95*3330	Roland/R269-74//HE270-15/3/Howell		
M95*3349	S76/N80738//Pioneer 2555		
Madison	Abe//Blueboy/VA 71-54-147/3/VA 72-54-14		
MO94-317	AP Traveller/Pioneer 2555		
NC94-7197	Coker 83-23/Bradford//CC(PM1)/3/Coker 8320/4/SXL/C39		

difficult. The objective of this study was to determine the genetic basis for race-specific resistance to leaf rust among 116 contemporary cultivars and advanced breeding lines of soft red winter wheat.

MATERIALS AND METHODS

The 116 lines from the 1998–99 Arkansas Wheat Cultivar Performance Test and Uniform Eastern and Southern Soft Red Winter Wheat Nurseries were selected as representative of contemporary soft red winter wheat cultivars and breeding lines at the beginning of this study (Table 1). Twenty-two races of *P. tritricina* (Table 2) were selected to identify *Lr* genes in the lines. Isolates of *P. tritricina* were obtained from the USDA-ARS, Cereal Disease Laboratory, St. Paul, MN, the Cereal Research Centre Agriculture and Agri-Food in Winnipeg, Canada, and from field collections in Arkansas.

To assure that each pathogen isolate was homogeneous, all isolates used in this study originated from a single-pustule transfer. To produce ample quantities of urediospores, each isolate was increased on wheat seedlings. Approximately 15 wheat seedlings were grown in 8.5-cm square pots filled with potting mix (6 parts peat moss, 4 parts vermiculite, 2 parts perlite, 3 parts Roxanna silt loam, and 3 parts sand). Seedlings were fertilized at emergence and before inoculation with Peters 20-20-20 NPK fertilizer at the rate of 1 g/liter. To enhance spore production (20) and to prevent further leaf formation

(13), seedlings were treated with 20 ml of 0.1% maleic hydrazide per pot when they were approximately 8 cm tall. When primary leaves were fully expanded, seedlings were inoculated with urediospores suspended in Soltrol 170 mineral oil (Chevron-Phillips Chemical Co., Houston, TX). Inoculated seedlings were placed in a dew chamber for 16 to 20 h at 20°C and then transferred to a greenhouse at 20 to 24°C or a growth chamber at 22/18°C (day/night) with a 12-h photoperiod. Seedlings inoculated with each isolate were maintained in a separate, clean isolation chamber to avoid cross contamination. Urediospores were collected with a vacuum spore collector, dried in a desiccator, and kept at 5°C for short-term storage or at –80°C for long-term storage.

To determine the avirulence/virulence formula, each isolate was inoculated on a set of 24 isolines in a Thatcher (Tc) background. Procedures were as described above except that isolines were not treated with maleic hydrazide and inoculated seedlings were incubated in a growth chamber at 22/18°C with a 12-h photoperiod. Isolines were grouped into sets (set 1: *TcLr1*, 2a, 2c, 3; set 2: *TcLr9*, 16, 24, 26; set 3: *TcLr3ka*, 11, 17, 30; set 4: *TcLr10*, 18, 14a, 23; set 5: *TcLr15*, 20, 21, 28; and set 6: *TcLr32*, 36, 38, 39), and reactions on the first four sets were used to determine the race designation for each isolate as described by Long and Kolmer (17). Host response was measured by recording the infection type on the first leaf. Infection

types were rated according to the 0 to 4 scale of Stakman et al. (41), where 0 = no macroscopic symptoms, ; = hypersensitive necrotic or chlorotic flecks, 1 = small uredia surrounded by necrosis, 2 = medium uredia surrounded by necrosis or chlorosis, 3 = medium uredia without necrosis or chlorosis, 4 = large uredia without necrosis or chlorosis, + = uredia somewhat larger than normal for the infection type, and – = uredia somewhat smaller than normal. A range of infection type was recorded using more than one infection type with the predominant infection type listed first. Infection types 3 and 4 were considered high (host was susceptible/race was virulent), and others were considered low (host was resistant/race was avirulent). Each race by wheat isolate combination was tested at least twice.

To determine reactions of soft red winter wheat lines, five to seven seedlings from each of the 116 lines and 24 isolines were grown in 8.5-cm square pots with four lines per pot. Wheat cultivars Morocco and Ernie were included in each test as susceptible controls. Plants were grown, inoculated, and incubated in a dew chamber as described previously. Inoculated plants were then incubated in growth chambers at 22/18°C. Infection types were rated 12 to 14 days after inoculation. Each race by wheat line combination was tested at least twice.

To facilitate determining the absence or presence of *Lr* genes, infection type data were analyzed using a computer program

Table 2. Infection type^a produced by the 23 races of *Puccinia tritricina* used in this study on seedlings of 24 Thatcher isolines with a single *Lr* gene at 22/18°C day/night and 12-h photoperiod except as footnoted

Race	Isolate	<i>Lr</i> gene									
		1	2a	2c	3	9	16	24	26	3ka	11
BBBB	–	;	1	;	;1	;	;	;	;	1;	1;
CBGS	85 CBG-677	0	0;	;1	;	;	1	;1	;	1	
CBTD	89 200	;	;	;1	;	;	1	;1	;		
DBBC	1991 MI	;1	;0		;	;1	1	;	;1	11+	2; ^b
KBGQ	–	;				;	1	;	;	1	
LBBQ	98FL119-1DLL		1;	;	12	;	1	;1	;	2+	1 ^b
LBCS	–		;	;1	;	;1	1	;	;1	;1	22+
MBMN	W1935		;	;		;	1	;	;1		24 ^b
MBRR	243 AR		;	;		;	1	;	;		
MCDN	1998 MN		2	2		;	1	1		1	24 ^b
MCRN	130 AR		;	;		;	1	;			
MFMP	94 KS 406		;	;		;	1				24 ^b
MGBN	492-99		2	2+		;		;1	;1	2	2 ^b
MGMN	1994 MN		;	2		;		;	;		2 ^b
PDLM	27-2-2	;2				0;	;1		;		;1 ^b
PNMT	94 LA 1		;				1		1		24 ^b
SBDB	94 PA 82				;	;	1	;	;	;	24 ^b
TBPM	19-3-2					0;	11+	;	;		;, ^b
TCRN	222 AR					;	1	1			
TDRP	486-99					;	1		1		
TFRP	98 OK 235					0	1				
TLGM	99 AR M3						11+	;	;	1	
TLRT	99 AR B1						1	;1	1		

^a 0 = no macroscopic symptoms, ; = hypersensitive necrotic or chlorotic flecks, 1 = small uredia surrounded by necrosis, 2 = medium uredia surrounded by necrosis or chlorosis, 3 = medium uredia without necrosis or chlorosis, 4 = large uredia without necrosis or chlorosis, + = uredia somewhat larger than normal, – = uredia somewhat smaller than normal. A range of infection type is indicated by more than one infection type, with the predominant infection type listed first. Blanks represent high infection types.

^b Tested at 17°C and a 12-h photoperiod.

^c Tested at 18°C and a 12-h photoperiod.

written specifically for this purpose (Y. A. Wamishe, K. C. Thompson, and E. A. Milus, *unpublished*). Based on the gene-for-gene concept, lines susceptible to a particular race cannot have an *Lr* gene for which the race is avirulent. For each line, step 1 of the program summarized results from races that were virulent on the line to definitively exclude *Lr* genes from the line, and this exclusion resulted in a relatively short list of *Lr* genes that could be present in the line. Step 2 of the program utilized data from races that were avirulent on the line, and the output listed the low infection types produced on the line and the isolines with *Lr* genes that were not excluded in step 1. Of the *Lr* genes not excluded in step 1, a gene was considered present if the low infection type produced on the line by one or more races matched the low infection type on the corresponding isolate. Otherwise, the gene was considered possibly present. Epistatic effects prevented the definitive inclusion or exclusion of genes considered possibly present. If the low infection type produced on the line was lower than that on any of the isolines in step 2, then the line was considered to have an unidentified *Lr* gene; i.e., the gene was not in the set of 24 isolines.

To correctly determine the reaction of genes *Lr11* and *Lr18* that are more effective at low temperature, races that appeared to be avirulent on *Lr11* or *Lr18* at 22/18°C day/night in preliminary tests were retested twice on the isolines and wheat lines at 17 or 18°C, respectively. Races TBPM and PDLM that were identified as homozygous

avirulent for *Lr11* by Kolmer and Dyck (15) were obtained late in the study from J. Kolmer (USDA, ARS, Cereal Disease Laboratory, St. Paul, MN) and used to confirm the presence of *Lr11* at 17°C. Infection types obtained from these tests were used to postulate genes *Lr11* and *Lr18*.

To obtain an indication as to whether avirulence for an *Lr* gene was fully or partially dominant, the range of low (avirulent) infection types on each Thatcher isolate in this study was examined.

To confirm the absence of *Lr* genes in the lines postulated to have no resistance genes based on inoculations with 22 races, seedlings of these lines were inoculated with race BBBB that was avirulent on all 24 isolines. Two lines that had an intermediate infection type with race BBBB were suspected to have *Lr14a*. These lines and appropriate isolines were inoculated with races avirulent on *Lr14a* and incubated at 18°C with a 12-h photoperiod to determine if these lines had *Lr14a*.

To determine if *Lr26* was always associated with 1BL.1RS status and vice versa, the presence of *Lr26* as determined by postulation was compared to 1BL.1RS status (8).

RESULTS

The low infection types produced by races on the isolines with *Lr* genes (Table 2) agreed with low infection types for these genes as summarized by McIntosh et al. (24). Therefore, data obtained for lines (Table 3) can be used to accurately determine the presence or absence of these *Lr*

genes in soft red winter wheat lines. Races differed for combinations of virulence on genes *Lr1*, *2a*, *2c*, *3*, *3ka*, *9*, *10*, *11*, *14a*, *15*, *16*, *17*, *18*, *20*, *23*, *24*, *26*, *28*, and *30*, making it possible to postulate these genes. All races were avirulent on *Lr21*, *32*, *36*, *38*, and *39*, and these genes were not detected in any of the lines.

Of the 116 lines tested, 13 lines had no detectable seedling *Lr* genes, 49 had one or more known *Lr* genes, 35 had one or more known *Lr* genes and one or more unidentified *Lr* genes, and 19 had one or more unidentified *Lr* genes (Table 4). Considering the *Lr* genes postulated to be present (excluding the unidentified genes), 44 different combinations of *Lr* genes were identified among the 116 lines.

Four lines (Agriprio Marion, APD94-5282, NC94-7197, and VA97W-375) were resistant to all races used in this study. The low infection types produced on these lines with races avirulent on *Lr26* matched for *Lr9*, *24*, and *26*. Races MFMP and TFRP that were virulent on both *Lr24* and *26* indicated the presence of *Lr9*, and race TLGM that was virulent on both *Lr9* and *26* indicated the presence of *Lr24*. Race PNMT that was virulent on both *Lr9* and *24* displayed low infection type that matched for *Lr26*. These lines were postulated to have a combination of *Lr9*, *24*, and *26* by matching infection types and using the 1BL.1RS translocation as a marker for *Lr26*. The combination of *Lr9*, *24*, and *26* was epistatic over all other *Lr* genes, and therefore, none of the other *Lr* genes could be eliminated. For clarity, however, these

Table 2. (continued)

<i>Lr</i> gene													
17	30	10	18	14a	23	15	20	21	28	32	36	38	39
1	;1	1;	1	22 ⁺	;1	1	2+	1	;2	1;	1	;	1
;	1				2+			2		1	1	;	;
		;	2 ⁺		1			1		1	2	;	;1
1+;	1	1	2 ⁺	2 ⁺		2+	2+	2	2+	;1	;1	;	;1
;	1				2+			2		;	;	;	;
1	1			22 ⁺	1	2	2	1		;	1	;	1;
;1					22+			12;		;1	;1	;	;1
1;			21 ^c		1;			;		;1	;1	;1	2;
1				2 ⁺		2	2+	1	2+	;1	1	;	;1
	1		2 ^c		1		;1	1		2	1	;	1;
1			2 ^c		1		;2	1		;1	1;	;	1
1								1		1;	1	;	;1
1	1		2 ⁺		1	2	2	;		;1	1	;	1
2			2 ^c		1	1	;	1+	2+	1	1	;	;
;	1+		2 ^b	22 ⁺				1;		;1	1+	;	;
;								;		1	1	;	2
	;	;	1 ^c	2 ⁺	;	2+	;	1	2+	12	1	;	1
			;1 ^b	22 ⁺				;1		;1	2	0	;1
;			2 ^c		1	2	;	1		1	1	;0	1
;			2 ⁺					2		1	1	;	1
1			2 ⁺					1		;1	;1	;	;
1	2		2 ^c	2 ⁺			2+	1	2+	2	1	;	;1
;								1		1	;1	0	;1

Table 3. Infection types produced by 23 races of *Puccinia triticina* on 116 soft red winter wheat lines

Line	Race									
	BBBB	CBGS	CBTD	DBBC	KBGS	LBBQ	LBCS	MBMN	MBRR	MCDN
Agripro Foster	NT ^a	0	;	;	;	;	;	1	1	1
Agripro Hickory	NT	3	3	;	3	3—	3	;1	4	;
Agripro Mallard	NT	3	3	3	3	3	3	3	3	3
Agripro Marion	NT	;	;	;	;	;	— ^c	;	1	1
Agripro Mason	NT	;	;	;	;	;	;	;	;	;1
Agripro Patton	NT	0	;	;	;	;	1	;	;	1
Agripro Shelby	NT	3	;	1	3	4	1;	3	3	3
Agripro Shiloh	NT	0	;	;	;	1	1	;	;	;
AGS 2000	NT	;	;	;	;	;	;	1	1	1
APD94-5282	NT	;	;	;	;	;	;	;	1	1
APD95*8811-1	NT	0	;	;	;	;	;	;	;	;
APD95*8811-2	NT	;	;	;	;	;	;	;	;	;
APD95-7763	NT	;	;	;	;	;	;	;	;	;
AR584A-3-1	NT	3	;	;	;	3	;1 ^b	3	3	2+ ^b
Armor 3235	NT	;	;	;	2+	3	3	3	3	3
BL931167	NT	;	;	;	;	;	;	;	;	1
BL940026	NT	;	;	;	;	;	;	;	;	;
Buckshot's DS9193	NT	;	;	;	;	;	;	;	;	1
Caldwell	3	3	3	3	3	3	4	3	3	3
Cardinal	NT	3	1	1;	3	3	1	3	3	1 ^b
Delta Grow 3300	NT	3	3	3	3	3	3	3	3	3
Delta King 1551W	NT	3	2+	1	3	2+	;1	0; ^b	3	22+
Delta King 9027	NT	3	2+ ^b	1 ^b	3	3	3	3	3	3
Delta King 9051	NT	3	2+ ^b	3	3	3	4	3	1 ^b	2 ^b
Delta King 9121	NT	; ^b	3	3	3	4	4	3	3	3
Dixie 2000	NT	;	;	;	;	;	;	;	;	;
Dixie 911	22+	3	3	2+	3	3	4	3	4	3
EK103	NT	3	3	3	3	3	; ^b	3	4	3
EK114	NT	3	3	;1	3	3—;	1	0; ^b	3	2+
EK156	NT	3	3	3	3	3	;0 ^b	3	3	3
F322W	NT	3	3	3	3	4	;1 ^b	3	3	3
FFR 522W	NT	;	;	;	;	;	;	;	;	;
FFR 558W	NT	;	;	;	;	;	;	;1	1	1
Florida 302	NT	3	;	;	3	3	2	3	3	3
Florida 8868	NT	;	;	;	;	;	;	;	;	;
G41332	3	3	3	3	3	4	4	3	3	4
G41333	3	3	3	3	3	4	3	3	3	4
G60232	1; ^b	3	3	3	3	3	—	3	3	3
GA881404E56	NT	;	;	;	3	;	;	;	;	;
GA88622E51	NT	;	;	;	;	;	2+	;	;	;
GA90524E35	NT	;	;	;	;	;	;	;	;	;
HT98-10033	NT	;	;	;	;	3	;2+	3	3	3
HT98-10291	NT	;	;	;	;	;	;	;	;	;
IL91-15911	3	3	3	3	3	3	3	3	3	3
IL94-2894	NT	;	;	;	;	3	1 ^b	3	3	3
IL94-6727	NT	3	3	3	3	3	2+ ^b	1 ^b	3	4
Jackson	NT	;	;	;	;	2+	;1	;1 ^b	4	2+
Jaypee	NT	2+ ^b	;	;	3	2+	;	;1 ^b	3	;0 ^b
KY86C-127-3	NT	;	;	;	;	;	;	1	;	1
LA 87167D8-10-2	NT	2 ^b	;	;	3	3—	;1	22+	3	1
LA8513B1-7-B-1-4-2	NT	;	;	;	;	;	;	;	;	;
LA90144B16-3-2	NT	;	;	;	;	;	;	;	;	;
LA90412F14-1-4	NT	;	;	;	;	;	;	;	;	;
LA9070G45-3-3-1	NT	;	;	;	;	;	;	;	;	;
M95*2883	NT	3	2+	;	3	3—;	2+	1+ ^b	3	2+
M95*3330	NT	3	2+ ^b	1+ ^b	3	3	3	3	3	3
M95*3349	NT	3	2+	;	3	3—	;2	3	3	3
Madison	NT	3	;	1	3	2	;1	2+	3	2+
MO94-317	NT	3	2+	;	3	3	;1 ^b	3	3	3
NC94-7197	NT	;	;	;	;	;	2+	;	1	;
NC95-25305	NT	3	1 ^b	2+	;1 ^b	2+	3	1 ^b	2	;1 ^b
NC95-25707	NT	;	;	;	;	;	;	;	;	;
NK Coker 9025	NT	0	;	;	3	;	;1	;	;	;1
NK Coker 9152	NT	;	1	;	;	;	;	;	;	;
NK Coker 9484	NT	;	;	;	3	;	;1	;	;	21
NK Coker 9543	NT	3	2+	1	3	3—	1	3—	3	2+
NK Coker 9663	NT	;	;	;	;	;	;	;	;	;
NK Coker 9704	NT	3	;	1	3	3	1	3	3	3
NK Coker 9835	NT	;	;	;	;	;	;	;	;	;

^a Not tested.^b A resistant reaction that could not be matched to any of the *Lr* genes in the set of isolines, indicating the presence of an unidentified gene.^c Missing data.

Table 3. (continued)

Race												
MCRN	MFMP	MGMN	MGBN	PDLN	PNMT	SBDB	TBPM	TCRN	TDRP	TFRP	TLGM	TLRT
2	2	;	1	NT	1	1	NT	;	1	3	;	1
2+	0	;1	12	;	3-	0	;	2+	3	2+	3	3
3	1 ^b	3	3	3	3	3	3	3	3	3	3	3
;2	;	;	;	NT	;	;1	NT	1	;	;	;	;
;	0	;	;	NT	2	;	NT	;	;	0	3	3
;1	2	;	;	NT	;	1	NT	1	1	3	;	;
3	4	3	3	3	3	;1	3	3	4	3	4	3
;	;	;	;	NT	;	;	NT	;	;	0	3	3
;2	2	;	;	NT	1	;	NT	1	1	3	;	;
1	1	;	;1	NT	1	1	NT	;	1	1	;	;
;	;	;	;	NT	3	;	NT	;	2	0	4	3
;	;	;	;	NT	1	;	NT	;	1	;	2	3
;	0	;	;	NT	3	;	NT	;	0;	0	;	1
3	3	3	3	3	2 ^b	;	3	3	3	4	4	3
3	3	3	3	NT	4	3	NT	3	4	4	4	3
;	;	;	;	NT	3	;	NT	;	2	0	3	3
;	0	;	;	NT	1;	1	NT	;	0	0	4	3
;	0	;	;	NT	;	;	NT	;	;	0	4	3
3	3	3	3	NT	3	3	NT	3	4	3	4	3
3	4	3	3	NT	3	1	NT	3	3	4	3	3
3	3	3	3	NT	3	3	NT	4	4	4	4	3
3	2	1	1	;1	1 ^b	3-	;	4	4	3	4	3
3	4	3	3	3	3	2 ^b	3	3	4	4	4	3
3	3	3	3	3	3	3	3	3	4	3	3	3
3	2 ^b	3	3	2 ^b	3	3	3	3	3	3	3	3
;	2	;	;	NT	2+	;	NT	;	;	0	3-	3
3	3	3	3	3	3	2+	3	3	4	4	3	3
3	3	1 ^b	3	3	3	3	3	3	3	3	4	3
3	1 ^b	1	0; ^b	;1	1 ^b	3-	;	3	4	3	4	3
3	3	3	3	3	3	3	3	3	4	4	4	3
3	3	3	3	3	3	3	3	3	4	3	4	3
;	;	;	;	NT	2+	;	NT	;	;	0	2+	3
-	3	;	;	NT	1	1	NT	1	1	3	;	1
3	3	3	3	3	3	;	3	3	3	3	4	4
;	0;	;	;	NT	2+	;	NT	;	3	0 ^b	4	3
3	3	3	3	NT	3	3	NT	3	3	3	4	3
3	3	3	3	NT	3	3	NT	3	3	3	4	3
3	3	3	3	NT	3	3	NT	3	4	3	4	3
;	0;	;	0	;	1	0	2+	3	;, ^b	3	4	3
;	1	1	1;	NT	2+	;	NT	;	3	3	;	;
;	0	;	;	NT	1	;	NT	;	;	0	4	3
3	3	3	3	NT	3	2	NT	3	3	3	4	3
;	3	;	2+	NT	3	;	NT	;	4	1 ^b	;	1
3	4	3	3	NT	3	3	NT	3	4	4	4	3
3	4	3	3	NT	3	3	NT	3	3	4	4	3
3	3	3	3	3	3+	3	3	3	3	3	4	3
3	;12 ^b	;2+ ^b	12	;1	1 ^b	;	;	3	3	3	3	3
3	;1 ^b	11+	1	;1	3-	;	;	3	3	3	1 ^b	1 ^b
2+	2+	;	1	NT	1	1	NT	;1	1	3	;	;
3	1	2;	;	;	1 ^b	;	;	1; ^b	1 ^b	1 ^b	1 ^b	1 ^b
;	0	;	;	NT	;1	;	NT	;	0	0	4	3
;	0	;	;	NT	;1	;	NT	;	0	1	2+	3
;	0	;	;	NT	;	;	NT	;	;	;	2+	3
;	;	;	;	NT	2+	;	NT	;	;	0	4	3
3	0 ^b	-	3-	;1	2	2+	;1	3	3	4	3	3
3	3	3	3	3	3	3	3	3	3	3	4	3
3	3	3	3	NT	3+	1;	NT	3	3	3	4	3
3	; ^b	;2+ ^b	; ^b	;1	2+	;	;	3	3	3	3	3
3	4	3	3	NT	3	1	NT	3	3	4	4	3
1	;	;	;	NT	1	;	NT	1	1	;	;	0
2	3- ^b	;	;	3	1 ^b	;	3	1	1 ^b	1 ^b	2	1 ^b
;	;	;	;	NT	2+	;	NT	;	;	;	4	3
;	0;	;	;	2+	1	;	3	3	0	3	4	3
;	;1	;	1;	;	1	;	;	;	3	;1 ^b	;	;
;	;	;	2+	;	;	1	;	3	1 ^b	3	4	;, ^b
3	;	1	1+	;1	1	2;	;1	3	4	1	3	3
;	;	;	;	NT	2+	;	NT	;	;	0	4	3
3	3	3	3	NT	3	;	NT	3	3	3	3	3
;	0	;	;	NT	;	;	NT	;	;	0	2+	3

(continued on next spread)

Table 3. (continued from previous spread)

Line	Race									
	BBBB	CBGS	CBTD	DBBC	KBGS	LBBQ	LBCS	MBMN	MBRR	MCDN
OH536	NT	3	2	;	3	3	2+	3	1 ^b	2; ^b
OH552	NT	3	2+	1	3	2	2+	3	3	3
OH599	NT	3	1	;	3	0	1	0 ^b	3	;
OK91P648-41	NT	3	2 ^b	1 ^b	;	3	1 ^b	3	3	3
P86364RA1-4-2-7	NT	;	;	;	2+	;	;	1	3	1;
P88204RB1-2-1-6	NT	2	3	3	3	3	;	2+	3	3
P92227C5-1-1	NT	1	;	;	3	3	1 ^b	3	3	3
Pioneer 2548	NT	3	2+	1	3	3-	1	0; ^b	3	2+
Pioneer 2684	NT	3	1	;	;	3-	;	;	3	1;
Pioneer 26R46	NT	;	;	;	;	3-	;	;	3	2+
Pioneer 26R61	NT	;	;	;	;	;	;	;	1	2
Pioneer XW672	NT	2+	2+	1	1	;	;	;	1 ^b	3-
Pioneer 26R24	1; ^b	3	3	3	3	3	4	3	3	3
Pocahontas	NT	3	3	2;	3	;	1	;	3	2+
Quantum 708	NT	;	;	;	;	;	;	;	;	1
Roane	NT	3	3	3	3	4	3	3	4	3
Roberts	NT	3	3	;	1	3	2+	;	3	0; ^b
S9412192	NT	;	;	;	;	;	3-	1	;	;
Sabbe	NT	;	;	;	;	;	;	;	;	1
SC921285	NT	1 ^b	3	3	3	3	;	1 ^b	1 ^b	2 ^b
SC921299	NT	2 ^b	2 ^b	2 ^b	3	2 ^b	4	1 ^b	3	3
SS36803	NT	3	2+	1+	3	2+	;	;	3	;
SS550	NT	;	;	;	;	;	;	;	1	1
Stine 455	2+	3	3	2+	3	3	4	3	3	3
Stine 488	3	3	3	3	3	3	4	3	4	3
Stine 950455	NT	3	1	1	3	3-	;	2+	3	2+;
T104	3	3	3	3	3	3	4	3	3	4
T105	3	3	3	3	3	4	4	3	3	4
T106	3	3	3	3	3	3	3	3	3	4
T109	NT	;	2+	1	3	22+	1	1+	3	;
Terra E218	3	3	3	3	3	3	4	3	3	3
Terra SR204	3	3	3	3	3	3	3	3	3	3
Terra SR216	3	3	3	3	3	3	3	3	4	3
Terral TV 8555	NT	;	;	;	;	;	;	;	;	;
Terral TV 8768	NT	;	;	;	;	;	;	;	;	1
Terral TV 8825	NT	;	;	;	;	;	;	;	;	;
Terral TVX8670	NT	;	;	;	;	;	;	;	;	1
TW94315	;	3	3	3	3	3	3	3	3	3
TX87-20	NT	2 ^b	1	2	3	1	2+	1 ^b	3	1 ^b
TX91-13	NT	;	;	;	;	;	;	;	2	;
USG3408	NT	;	;	;	;	2+	2	3-	3	2
USG EXP 97-4	NT	3	3	;	3	3	;	3	3	3
USG3209	NT	;	;	;	;	;	;	1	1	2
VA96-54-326	NT	3	1	2+	3	3	2+	3	3	;
VA97W-375	NT	;	;	;	;	;	;	;	2	;
Wakefield	NT	3	1	1	3	3	;	3	4	3
Wilfarm 488	3	3	3	3	3	3	3	3	3	3

genes were not indicated as possibly present for these lines in Table 4.

Of the *Lr* genes identified as present among the 116 wheat lines, *Lr11* was the most frequent (34.5%), followed by *Lr10* and 3 (25.8 and 23.3%, respectively) (Table 5). Genes *Lr9*, 26, 2a, and 24 were present in 19.8 to 12.9% of the lines, and genes *Lr20*, 14a, 2c, 3ka, 23, 18, and 1 were present in 0.9 to 7.7% of the lines. Of the genes identified as possibly present, *Lr10* was the most frequent (37.9%), followed by *Lr1* and 3 (35.3% each). Genes *Lr3ka*, 14a, 20, 30, 28, 2c, 23, and 15 were possibly present in 18.1 to 29.3% of the lines, and genes *Lr18*, 24, 11, and 2a were possibly present in 1.7 to 12.1% of the lines. Gene *Lr10* was the most frequently identified gene (63.7%) when results were summarized across the present and possibly present categories. Genes *Lr9* and 26 were only postulated to be present and

never as possibly present. On the other hand, *Lr15*, 28, and 30 were postulated to be only possibly present.

Of the 116 lines, 54 lines were found to have unidentified *Lr* genes (Table 3). The number of resistant reactions attributed to unidentified *Lr* genes ranged from 1 to 12 among lines with unidentified *Lr* genes. Lines SC921299 and SC921285 had the most resistant reactions attributed to unidentified *Lr* genes: 10 and 12, respectively. All 23 races were able to detect unidentified genes, and the number of resistant reactions attributed to unidentified *Lr* genes ranged from 1 to 21 among the races. Races MBMN, PNMT, and MFMP detected the most resistant reactions attributed to unidentified *Lr* genes, 17, 19, and 21, respectively. Lines with identical responses to the races likely have the same unidentified *Lr* gene. For example, Pioneer 26R24, G60232, and TW94315 were sus-

ceptible to all races tested except for resistant reactions caused by race BBBB; EK 156, F322W, IL94-2894, and MO94-317 had resistant reactions caused by race LBCS; and OH 552 and Stine 950455 had resistant reactions caused by races PNMT and MFMP.

Gene *Lr14a* was postulated in only two lines, Dixie 911 and Stine 455 (Table 4). These two lines were susceptible to all races used in this study with the exception of BBBB, which produced an intermediate infection type (22+) at 22/18°C. Subsequent tests with races avirulent on *Lr14a* at constant 18°C indicated the presence of *Lr14a*.

Infection types produced by all races avirulent on isolines with *Lr1*, 9, 10, 16, 24, or 26 were never higher than IT 1+ (Table 2) when inoculated onto these isolines. Races avirulent on *Lr11* or *Lr18* produced infection types that ranged from ;1

Table 3. (continued)

Race												
MCRN	MFMP	MGMN	MGBN	PDLN	PNMT	SBDB	TBPM	TCRN	TDRP	TFRP	TLGM	TLRT
3	;b	2+b	2+b	3	;b	1	3	3	4	3	3	3
3	;1 ^b	3	3	3	1	;	3	3	3	3	3	3
3	2	1	2+	1;	2+	1	;1	3	3	4	4	3
3	3	3	1 ^b	3	1 ^b	3	3	3	2+b	4	4	3
3	2	;b	;b	;	1 ^b	1	;	3	3	3	;	3
3	3	1 ^b	3	3	3	3	3	3	2 ^b	4	4	3
3	3	3	3	NT	3	3	NT	3	3	4	4	3
3	1 ^b	1	1	;1	;b	1	;1	3	3	3	3	3
;	0 ^b	;	1	0;	;b	;	;	1	1 ^b	2+	1	1+b
3	0	1	;	;	;	;	;	3	4	3	3	3
3	2+	;	;	NT	;	1	NT	3	1	3	;	1
3	1 ^b	;1	1	;1	1 ^b	2+	;	3	3	2 ^b	3	3
3	3	3	3	NT	3	3	NT	3	4	3	3	3
3	1+2	1	1	;1	12 ^b	;0	;1	3	3	3	4	3
1	3	;	1	NT	2+	;	NT	1	1	3	;	1
3	3	1 ^b	3	0 ^b	3	3	;b	3	3	3	3	3
3	0 ^b	;b	;b	;	1 ^b	0 ^b	;	3	3	4	3	3
;2+	3	;	1;	NT	1	;	NT	1	1	4	;	1
;	;	;	2+	NT	2+	;	NT	3	3	3	4	3
1 ^b	3	2+b	1 ^b	3	3	1 ^b	3	3	1 ^b	1 ^b	1 ^b	3
1 ^b	;b	3	1 ^b	3	3+	1 ^b	3	3	3	1 ^b	3	3
1 ^b	;1 ^b	1	2	;1	;1 ^b	2+	;	3	3	4	1 ^b	3
3	3	;	1	NT	1	1	NT	1	2+	3	;	1
3	3	3	3	3	3	2+	3	3	3	3	4	3
3	3	3	3	NT	3	3	NT	3	4	3	4	3
3	1 ^b	1+	1	;1	1 ^b	3-	;	3	4	3	4	3
3	3	3	3	NT	3	3	NT	3	3	3	4	3
3	3	3	3	NT	3	3	NT	3	3	3	4	3
3	4	3	3	NT	3	3	NT	3	3	3	4	3
3	;b	1	1	;1	1 ^b	2+	;	3	3	3	4	3
3	3	3	3	NT	3	3	NT	3	4	3	4	3
3	4	3	3	NT	3	3	NT	3	4	3	4	3
3	4	3	3	NT	3	3	NT	3	4	3	4	3
;	3	;	;	NT	1	;	NT	;	1	3	;	;
2	1	;	;	NT	1;	1	NT	1	1	1	;	1
;	3	;	2+	NT	2+	1	NT	;	4	4	;	1;
3	0 ^b	1	2+	;1	;	1	;1	1 ^b	1	1 ^b	1	1
3	3	3	3	NT	3	3	NT	3	3	3	3	3
;b	1 ^b	1	1	;1	1 ^b	1	;	1 ^b	3	1 ^b	3	3
;2+	-	2+	1;	NT	;2+	;1	NT	;	1	1	;	;
3	1 ^b	1	1;	;1	12	1	;	3	4	4	4	3
3	4	3	3	3	3	;b	3	3	4	4	3	3
;1	1	;	;	;	;	1;	;	3	1	1 ^b	;	;
3	4	1 ^b	3	3	3	1	3	3	3	3	3	3
;2	;1	;	;	0;	;	;	0;	1	1	1	;	1
3	3+	;b	3	3	4	;	4	3	3	3	3	3
3	4	3	3	NT	3	3	NT	3	4	4	4	3

to 2+ but were clearly avirulent on the appropriate isoline(s). However, races PDLN and TBPM that were homozygous avirulent for *Lr11* produced infection type ;1 or lower on *TcLr11*.

DISCUSSION

Results of this study indicated that at least 14 named *Lr* genes and several unidentified *Lr* genes are responsible for race-specific seedling resistance to leaf rust among 116 contemporary soft red winter wheat cultivars and breeding lines. Genes *Lr11*, *10*, and *3* were the most commonly identified (34.5, 25.8, and 23.3% of the lines, respectively), and these genes have also been commonly identified (29.7, 18.0, and 16.2%, respectively) among 111 older soft red winter wheat cultivars as summarized by Roelfs et al. (31). Genes *Lr2a*, *9*, *24*, and *26* were identified in more than 10% (13.8, 19.8, 12.9, and 15.5%, respec-

tively) of the lines in this study, and in 10.8, 18.9, 6.3, and 9%, respectively, in the older soft red winter wheat cultivars (31). Virulence to these genes has been common in North America (18,19,20).

Combinations of *Lr* genes contributed greatly to leaf rust resistance among the 116 lines in this study. Forty-four different combinations of *Lr* genes (excluding the group with only unidentified *Lr* genes) were identified (Table 4). Some of these combinations still provide protection against prevalent races of *P. tritici* in the field. The combination of *Lr9*, *24*, and *26* provided resistance to all races used in this study. Given that existing races can overcome combinations of *Lr9* and *26*, *Lr9* and *24*, and *Lr24* and *26*, it seems likely that a race capable of overcoming the combination of *Lr9*, *24*, and *26* will occur eventually.

The unidentified genes could be known *Lr* genes such as *Lr3bg*, *14b*, or *27+31* that

were not in the sets of isolines, adult-plant resistance genes such as *Lr13*, *34* that are expressed somewhat in the seedling stage (2-4,6,29), undescribed *Lr* genes, or undescribed alleles of known *Lr* genes. Inclusion of isolines for *Lr3bg*, *13*, *14b*, *27+31*, *34*, and *13+34* in this study may have allowed some of these *Lr* genes to be identified or excluded.

The 1BL.1RS translocation was a reliable marker for *Lr26*. All lines reported to have the 1BL.1RS translocation (8) were postulated to have *Lr26*, and all lines postulated to have *Lr26* had the 1BL.1RS translocation. The only inconsistency for postulating *Lr26* was found in P86764RA1-4-2-7, which was subsequently determined to have mixed seeds for the 1BL.1RS translocation (B. Graybosch, *personal communication*) and for reaction to races avirulent to *Lr26*. Using the 1BL.1RS translocation as a marker for

Lr26 facilitated postulating *Lr* genes for the four lines that were resistant to all races.

Genes *Lr3ka*, 18, and 23 were identified to be present in low frequencies (3.4, 6.9, and 3.4%, respectively). Virulence was common to *Lr23*, and this gene was not considered useful (24). Although virulence to *Lr3ka* and 18 is common in North

America (14,18), these genes had been considered useful in Australia (24) and as interacting with adult-plant resistance gene *Lr34* in North America (7).

Gene *Lr14a* was postulated to be present only in Dixie 911 and Stine 455. This gene was reported to be difficult to detect due to its moderate reaction and hence epistatic effect of other genes with lower infection

types (24). However, it appeared that Dixie 911 and Stine 455 had no other seedling genes that could be epistatic over *Lr14a*. Virulence was common to *Lr14a*, and this gene has not been considered useful (14,18,24).

Genes *Lr2a* and 2c were identified as present in 13.8 and 3.4% of the lines, respectively, in this study. Gene *Lr2a* has

Table 4. Seedling *Lr* genes in contemporary soft red winter wheat cultivars and advanced breeding lines

Seedling <i>Lr</i> genes ^a	Line
No detectable <i>Lr</i> genes	
None	Caldwell, Delta Grow 3300, G41332, G41333, Stine 488, T104, T105, T106, Terra SR204, Terra SR216, Terra E218, Wilfarm 488, IL91-15911
Known <i>Lr</i> genes	
1	Armor 3235
1, 2a, 2c, 3, and/or 10, 11	Sabbe
1, 10	HT98-10033
1, 3, 10, 11	Pioneer 26R46
2a, 9 (1, 2c, 3, 10, 11, 15, 23)	Agripro Shiloh, BL940026, Buckshot's DS91, GA90524E35, LA8513B1-7-B-1-4-2
2a, 9, 18 (1, 2c, 3, 3ka, 10, 11, 14a, 15, 20, 23, 28, 30)	APD95*8811-2, LA90144B 16-3-2, LA904122F14-1-4
2a, 9, 18, or 11 (1, 2c, 3, 3ka, 10, 14a, 15, 20, 23, 28, 30)	NK Coker 9835
2a, 2c, 3, and/or 10	NK Coker 9025
2a, 2c, 24 (1, 3, 3ka, 10, 14a, 15, 20, 23, 28, 30)	GA88622E51
3, 10	M95*3349
3 and/or 10, 11	NK Coker 9543
3ka, 18, 26 (1, 3, 10, 15, 20, 23, 28, 30)	Terral TV 8768
3ka, 26 (1, 3, 10, 14a, 15, 28, 30)	SS550
9 (1, 2c, 3, 10, 15, 23)	APD95*8811-1, BL931167
9, 11 (1, 2a, 2c, 3, 10, 15, 23)	Agripro Mason, LA9070G45-3-3-1, NC95-25707, NK Coker 9663
9, 11, 18 (1, 2a, 2c, 3, 3ka, 10, 14a, 15, 20, 23, 28, 30)	Dixie 2000, FFR 522W
9, 24 (1, 2c, 3, 3ka, 10, 14a, 15, 18, 20, 23, 28, 30)	APD95-7763
9, 26 (1, 2c, 3, 3ka, 10, 14a, 15, 18, 20, 23, 24, 28, 30)	TX91-13
9, 24, 26 ^(b)	Agripro Marion, AP-D94-5282, NC94-7197, VA97W-375
10	Agripro Shelby, Florida 302, NK Coker 9704
11, 18	Agripro Hickory
11, 23, 26 (1, 2a, 2c, 3, 3ka, 10, 14a, 15, 20, 24, 28, 30)	Agripro Foster, AGS2000, KY86C-127-3
11, 24, 26 (1, 2a, 2c, 3, 3ka, 10, 14a, 15, 20, 23, 28, 30)	Agripro Patton
11, 26 (1, 3, 3ka, 10, 14a, 28, 30)	Pioneer 26R61
14a	Dixie 911, Stine 455
24 (1, 3, 10, 14a, 28)	Terral TV 8825
24, 26 (1, 3, 3ka, 10, 14a, 15, 20, 23, 28, 30)	FFR 558W, Quantum 708, S9412192, Terral TV 8555
Known <i>Lr</i> genes plus one or more unidentified genes	
1, +	IL94-2894
1, 2a, 3, and/or 10, 23, + (2c, 11, 15)	Florida 8868
1, 3, and/or 10, 11, +	Jackson
1, 3, 11, + (10)	USG 3408
1, 3ka, 11, + (3, 10, 30)	P86364RA1-4-2-7
2a, 3, and/or 10, 11, + (2c)	GA881404E56
2a, 2c, 3, and/or 10, 11, +	NK Coker 9184
2a, 11, 26, + (1, 2c, 3, 3ka, 10, 14a, 28, 30)	USG3209
2a, 11, 24, + (1, 2c, 3, 3ka, 10, 14a, 15, 20, 23, 28, 30)	NK Coker 9152
3, 10, 11, +	Jaypee, Madison, LA87167D8-10-2, OH599, Stine 950455, T109, TX87-20, M95*2883, Pioneer 2548
3, 10, 11, 18, +	Pioneer 2684
3, 10, +	Cardinal
3, 11, +	EK 114, OH 552, Pocahontas
3, 11, + (10)	Delta King 1551W
3, 11, + (1, 10)	Pioneer XW672
3 and/or 10, 11, +	SS36803
3ka, 24, + (1, 3, 10, 14a, 15, 20, 28, 23, 30)	HT98-10291
3ka, 11, 26, + (1, 3, 10, 14a, 15, 28, 30)	Terral TVX8670
10, +	AR 584A-3-1, OH 536, MO94-317, VA96-54-326, Wakefield
11, +	Roberts
20, + (15, 28)	NC95-25305
One or more unidentified genes	
+	Agripro Mallard, Delta King 9027, Delta King 9051, Delta King 9121, EK 103, EK 156, F322W, G60232, M95*3330, OK91P648-41, P92227C5-1-1, IL94-6727, P88204RB1-2-1-6, Pioneer 26R24, Roane, SC921285, SC921299, TW94315, USG Exp 97-4

^a Genes without parentheses were determined to be present by matching the infection type on the line with the infection type on the corresponding Thatcher isolate. Genes within parentheses were determined to be possibly present if the low infection type produced on the line by one or more races did not match the low infection type on the corresponding isolate. Epistatic effects prevented the definitive inclusion or exclusion of genes considered possibly present. + = Unidentified gene, i.e., the gene was not in the set of 24 isolines. "3 and/or 10" means either *Lr3* or *Lr10* or both were present.

^b All the remaining 21 *Lr* genes could be possibly present due to epistatic effects of the other three genes.

been used largely in the north central United States (17) and in the southern United States (21), but it has been considered as having limited value if used alone (24). Since 1993, the frequency of virulence to *Lr2a* was much lower than that to *Lr1*, 3, 10, and 11 (18,19). Gene *Lr2c* was detected neither in hard red winter wheat (26) nor in older soft red winter wheat (31). However, in 2000, virulence to 2c was common in soft red winter wheat growing regions in the United States (18), and the virulence frequency to *Lr2c* was higher than to *Lr2a*, and therefore, *Lr2c* was considered to be less useful than *Lr2a* (5,18,24).

Although genes *Lr15*, 20, and 28 were identified as possibly present in several lines in this study, these genes are not likely to be important components of the genetic basis for leaf rust resistance in soft red winter wheat. Genes *Lr15*, 20, and 28 have never been postulated in wheat cultivars. *Lr20* has only been postulated in one line (NC95-25305) in this study, one older soft red winter wheat cultivar (Converse [31]), and a few cultivars from the United Kingdom (37). Furthermore, virulence for these genes is common (14,24), and the genes provide only moderate resistance to most of the avirulent races (Table 2).

Genes *Lr16* and 17 that were postulated among hard red winter wheat lines (26) were not detected in soft red winter wheat lines in this study. Of the older soft red winter wheat summarized by Roelfs et al. (31), *Lr17* was not present and *Lr16* was only in T63. Although virulence to *Lr16* and 17 is prevalent in North America, *Lr16* was reported to interact with *Lr13* to give enhanced resistance (35). Kolmer (14) did not find virulence to *Lr16* or 17 in *P. triticina* isolates from the South Atlantic region, and Long et al. (18) detected no virulence to *Lr16* in 2000 in soft red winter wheat growing regions in United States.

Isolines with genes *Lr21*, 32, 36, 38, and 39 were resistant to all races used in this study. These genes have not been widely exploited (24) and could be useful in soft red winter wheat.

Among soft red winter wheat lines that were common to this study and the summary by Roelfs et al. (31), results on the

postulated genes largely agreed. For example, Roelfs et al. (31) postulated *Lr26* in AP-D94-5282, VA97W-375, AGS2000, Agripro Foster, TX91-13, and SS550. In this study, however, other genes were identified in addition to *Lr26* (Table 4). There were disagreements with Roelfs et al. (31) for *Lr* genes in Agripro Hickory (*Lr2a*, 18) and OH 599 (*Lr26*). In this study, there was no indication for the presence of *Lr2a* in Agripro Hickory, but several races supported the presence of *Lr11*. Likewise, there was no indication for the presence of *Lr26* in OH 599. OH 599 and lines such as SS36803 that were reported to have the 1AL.1RS translocation (8) did not display the low infection type that matches *Lr26*.

Florida 302, which was described to have *Lr2a*, 10, and 11 by McIntosh et al. (24), and *Lr10+* by Roelfs et al. (31), was postulated to have only *Lr10* in this study. Only races avirulent on *Lr10* displayed low infection types (Table 3). All races avirulent on *Lr11*, including the two known homozygous avirulent races, produced susceptible reactions. None of the races avirulent on *Lr2a* indicated presence of *Lr2a* in Florida 302. These results were confirmed with two additional sources of Florida 302 seed.

Heterozygosity for avirulence is common among isolates of *P. triticina* (34), and it is likely that some isolates used in the current study were homozygous for avirulence and others were heterozygous. This heterozygosity coupled with partial dominance for avirulence may explain the wide range of avirulent infection types produced on isolines with certain *Lr* genes. The only genetic study of dominance for avirulence in *P. triticina* (15) determined that avirulence for genes *Lr2a*, 2c, 3ka, 11, and 30 was partially dominant and that virulence for genes *Lr3* and 17 was fully dominant. These conclusions are supported by infection type data in the current study (Table 2). Avirulent infection types on isolines with *Lr3* and 17 were relatively uniform, indicating that homozygous and heterozygous isolates probably produced similar infection types, indicating avirulence is fully dominant. Avirulent infection types on isolines with *Lr2a*, 2c, 3ka, 11, and 30 ranged from ; to 2 or 2+, indicating

that homozygous isolates likely are responsible for the lowest infection types and heterozygous isolates likely are responsible for the intermediate infection types. This relationship was confirmed with two isolates (PDLM and TBPM) known to be homozygous avirulent for *Lr11*. Avirulence for genes *Lr1*, 9, 10, 14, 16, 24, and 26 appeared to be fully dominant because all avirulent races produced similar infection types on isolines with these genes. Avirulence for genes *Lr15*, 18, 20, and 23 appeared to be partially dominant because avirulent races produced a range of infection types. Although genetic studies would be required to confirm whether avirulence to these genes was fully or partially dominant, comparing infection types on each isolate appeared to provide an indication of whether avirulence was fully or partially dominant. It would be advantageous to use races that are homozygous avirulent to postulate *Lr* genes that interact with avirulence genes that are known or suspected to be partially dominant. This is the first report to implicate partial dominance for avirulence as a contributing factor to the range of avirulent infection types produced by particular races on particular isolines and lines in gene postulation studies.

The combination of partial dominance for avirulence and temperature sensitivity are likely responsible for the large range of low infection types (; to 3-) and the difficulty of postulating *Lr11* as reported by McIntosh et al. (24). Results of this study indicated that *Lr11* is relatively easy to postulate by using homozygous avirulent isolates at low (17°C) constant temperature. However, additional races that are homozygous avirulent for *Lr11* are needed in order to overcome the epistatic effects of other resistance genes and allow definitive postulation for *Lr11* in more wheat lines. Similarly, the combination of partial dominance for avirulence and temperature sensitivity of *Lr18* probably was responsible for the range of infection types produced on *TcLr18*. Tests at low temperature could have been simplified by choosing only one low temperature, and 17°C likely would work well for detecting all three (*Lr14a*, 11, and 18) temperature-sensitive genes.

Table 5. Number and percentage of the 116 soft red winter wheat lines that were postulated to have specific leaf rust resistance genes

		<i>Lr</i> gene ^a																
		<i>1</i>	<i>2a</i>	<i>2c</i>	<i>3</i>	<i>3ka</i>	<i>9</i>	<i>10</i>	<i>11</i>	<i>14a</i>	<i>15</i>	<i>18</i>	<i>20</i>	<i>23</i>	<i>24</i>	<i>26</i>	<i>28</i>	<i>30</i>
Present ^b	No.	9	16	4	27	4	23	30	40	2	0	8	1	4	15	18	0	0
	%	7.7	13.8	3.4	23.3	3.4	19.8	25.8	34.5	1.7	0	6.9	0.86	3.4	12.9	15.5	0	0
Possibly present ^c	No.	41	14	31	41	21	0	44	10	24	34	2	24	32	4	0	30	29
	%	35.3	12.1	26.7	35.3	18.1	0	37.9	8.6	20.6	29.3	1.7	20.6	27.6	3.4	0	25.9	25
Total ^d	No.	50	30	35	68	25	23	74	50	26	34	10	25	36	19	18	30	29
	%	43.1	25.9	30.1	58.6	21.5	19.8	63.7	43.1	22.3	29.3	8.6	21.5	31	16.3	15.5	25.9	25

^a Resistance genes *Lr16* and 17 were not identified as present or possibly present, and genes *Lr21*, 32, 36, 38, and 39 could not be postulated.

^b *Lr* genes identified by matching infection type on wheat lines with the infection type on the corresponding isolate.

^c *Lr* genes could not be excluded from a line but could not be identified by matching infection types on the corresponding isolate because of epistatic interactions among *Lr* genes.

^d Sum of present and possibly present.

Approximately 97% of the contemporary soft red winter wheat cultivars and breeding lines in this study were vulnerable at the seedling stage to one or more of the races of *P. trititica*, and only four lines were resistant to all races used. Nearly 33% of the lines in this study were identified with three or more *Lr* genes compared with only 9% in older soft red winter wheat lines (31). Compared with older soft red winter wheat lines, contemporary soft red winter wheat lines had more combinations of *Lr* genes, suggesting soft red winter wheat breeders have been stacking genes to obtain resistance to leaf rust.

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