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Wheat leaf and stem rust in the United States

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Abstract. Leaf rust, caused by *Puccinia triticina*, is a common and widespread disease of wheat in the US. On an annual basis, over 50 races of the leaf rust fungus are detected. There are at least 5 major groups of genetically distinct *P. triticina* isolates in the US based on allelic variation at microsatellite loci. Distinct regional race populations of *P. triticina* are found in the US, due to the widespread use of race-specific leaf rust resistance (*Lr*) genes in different market classes of wheat. In the south-eastern States where soft red winter wheats are grown, races with virulence to *Lr9*, *Lr11*, and *Lr18* are predominant. In the southern Great Plains region where hard red winter wheats are grown, races with virulence to genes *Lr9*, *Lr17*, *Lr24*, and *Lr26* are common. In the northern Great Plains region where hard red spring wheats are grown, races with virulence to *Lr2a* and *Lr16* are common. Due to the wide dispersal of *P. triticina*, some races are found in all regions of the US. Highly effective durable resistance to leaf rust has been difficult to achieve due to the high degree of virulence variation in the *P. triticina* population and the rapid selection of races with virulence to effective *Lr* genes in wheat cultivars. Hard red spring wheat cultivars with genes *Lr16*, *Lr23*, and *Lr34* have been highly resistant for more than 10 years in Minnesota and the Dakotas. Stem rust, caused by *P. graminis* f. sp. *tritici*, has not been a common disease of wheat in the US since the last major epidemics in the 1950s. The low levels of stem rust infections in the US can be attributed to the increasing use of highly resistant winter and spring wheat cultivars, which has greatly reduced the overall level of stem rust urediniospores. Eradication of the alternate host, *Berberis vulgaris*, has reduced the number of races and slowed the emergence of new races. Resistance genes *Sr2*, *Sr6*, *Sr17*, *Sr24*, *Sr31*, *Sr36*, and *SrTmp* are common in the winter wheats. Genes *Sr6*, *Sr9b*, *Sr11*, and *Sr17* are common in the spring wheats. Spring wheat cultivars may also have adult plant stem rust resistance derived from cv. Thatcher. Many of the winter and spring wheats are susceptible to the new stem rust race from East Africa; however, cultivars with resistance to this race can be found in each of the major wheat classes.

Additional keywords: race specific resistance, specific virulence, *Puccinia triticina*, *Puccinia graminis* f. sp. *tritici*.

Introduction

Leaf and stem rust of wheat are regularly occurring and important diseases of wheat in the US. In past years, major stem rust epidemics occurred in wheat, and leaf rust continues to cause annual yield losses in wheat production. Extensive virulence surveys of both leaf and stem rust in the US are conducted annually at the USDA-ARS Cereal Disease Laboratory to detect the most prevalent races in different geographical areas, and to detect new races that have virulence to commonly grown wheat cultivars. Advanced wheat breeding lines and cultivars from wheat improvement programs throughout the country are tested with the most common rust races and also with races that have important combinations of virulence to leaf and stem rust resistance genes in wheat cultivars. Annual reports on the occurrence and distribution of wheat leaf rust and wheat stem rust, identification of leaf and stem rust races in the US, and data from testing wheat germplasm for rust resistance are posted at the USDA-ARS Cereal Disease Laboratory website (www.cdl.umn.edu).

Wheat leaf rust

Leaf rust, caused by *Puccinia triticina* Eriks., occurs nearly wherever wheat is cultivated, and is the most common and widely distributed disease of wheat in the US. Leaf rust infections can survive the winter over a large area of the U.S. from the southern Gulf Coast of Texas to Georgia, the Atlantic seaboard of South Carolina and North Carolina, and to the Great Plains states of Oklahoma and Kansas (Roelfs 1989). Uredinial infections of leaf rust that survive the summer on volunteer wheat in farm fields and roadside ditches are the source of inoculum for infections that become established on autumn-planted wheat in the southern US. Many of the soft red winter wheat cultivars that are grown in the south-eastern States, and hard red winter wheat cultivars that are grown in the Great Plains States are susceptible to leaf rust. This allows a large population of *P. triticina* to become established on winter wheat on an annual basis in the southern US.

Leaf rust infections on winter wheat can often be found along the southern Gulf Coast and Atlantic seaboard in February (Fig. 1). Urediniospores are carried by the southerly winds into wheat production areas in the south-eastern States, Ohio Valley, and southern Great Plains. By mid May leaf rust is usually widespread throughout the soft red winter wheat of the south-eastern States and in the hard red winter wheats of the southern Great Plains. Initial infections of leaf rust on winter wheats in the northern Great Plains of Minnesota and the Dakotas are usually

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Fig. 1. Wheat production areas in the US where leaf rust and stem rust of wheat occur.

detected in the latter half of May (Fig. 1). Leaf rust can also occasionally overwinter on winter wheat in this area. The initial leaf rust infections on spring wheat in the northern Great Plains are usually detected in the first 2 weeks of June. By the end of July, leaf rust is widespread on winter and spring wheat in the northern Great Plains. The progress and spread of wheat leaf rust in the US is recorded and updated regularly during the wheat growing season by electronic bulletins issued by the USDA-ARS Cereal Disease Laboratory in St. Paul, MN.

Yield losses due to leaf rust can be substantial. The final amount of loss will depend on the crop development stage when the initial infections occur, and the relative resistance or susceptibility of the wheat cultivar. Greater yield losses result when the initial infections occur early in the growing season, especially before the jointing and tillering stages. Infections after heading when grain filling is progressing will cause less crop loss. An early study with susceptible soft red winter wheat cultivars (Caldwell *et al*. 1934) showed losses ranging from 15 to 28% due to leaf rust. Most of the losses were due to a reduction in the number of kernels per head and a reduction in the kernel weight. Leaf rust was the major cause of a 25–30% yield loss in hard red winter wheat in Oklahoma in 1938 (Chester 1939). In Kansas, from 1993 to 2005, losses due to leaf rust have averaged nearly 3%, ranging from 11.0% in 1993 to trace levels of loss in 1996 and 2002 (USDA-ARS Cereal Disease Laboratory website).

In the US many different leaf rust resistance genes are present in the various market classes of wheat. In the soft red winter wheats grown in the south-eastern States, genes *Lr9*, *Lr10*, *Lr11*, *Lr18*, and *Lr26* are present in a large number of cultivars (Kolmer 2003) (Fig. 1). In the hard red winter wheats of the southern and central Great Plains region, genes *Lr3ka*, *Lr9*, *Lr17*, *Lr24*, *Lr26*, and *Lr41*, have been used in cultivars grown in the last 20 years (McVey and Long 1993). In the northern Great Plains region where hard red spring wheat cultivars are grown, cultivars with *Lr2a*, *Lr10*, *Lr16*, *Lr23*, and *Lr34* are common (Oelke and Kolmer 2004). The continual release of wheats with differing resistance genes in the different wheat classes has placed constant selection pressure on the *P. triticina* populations in these regions.

The continuous use of wheat cultivars with different resistance genes has led to a highly diverse population of *P. triticina* in the US. Generally, 40–60 races of *P. triticina* are characterised annually in the US on the 16 Thatcher line differentials in the currently used race nomenclature (Long and Kolmer 1989; Kolmer *et al*. 2006). In 2004, 50 races were identified in the US. The large populations of *P. triticina* that overwinter regularly in the southern US are reservoirs for mutations leading to new virulence phenotypes, which are selected by host resistance genes. Since avirulent *P. triticina* isolates are often heterozygous for virulence alleles (Kolmer 1992; Kolmer and Dyck 1994), a single mutation of an avirulence allele would be sufficient for the isolate to gain virulence. In 2004 the most common race in the south-east region, MCRK (29%), had virulence to genes *Lr10*, *Lr11*, *Lr18*, and *Lr26* (Kolmer *et al*. 2006) (Table 1). Selection for this combination of virulence occurred only in the south-eastern region since this race was not found in either the southern or northern Great Plains regions. The second most common race in the south-east, TLGJ (10%), had virulence to genes *Lr9*, *Lr10*, and *Lr11*. This race occurred at lower frequencies in the southern and northern Great Plains. In the southern Great Plains, race MBDS (18%), with virulence to gene *Lr17*, was one of the 2 most common races, and a highly related race, MCDS, with virulence to *Lr17* and *Lr26*, occurred at 9%. Race TNRJ, with virulence to *Lr9*, *Lr10*, *Lr11*, *Lr24*, and *Lr41*, also occurred at 18% in the southern Great Plains. In the northern Great Plains, TBBG (26%), with virulence to *Lr2a* and *Lr10*, was the most common race, followed by MCDS (13%), TNRJ (11%), and THBJ (7%), with virulence to *Lr2a*, *Lr10*, and *Lr16*.

The regional differences in virulence of *P. triticina* can also be seen in the overall frequencies of virulence (Table 2). Virulence to *Lr9* was highest in the south-east and southern Great Plains, where winter wheats with this gene are grown. Similarly, virulence to *Lr2a* and *Lr16* was highest in the northern Great Plains where spring wheats with these 2 genes are grown. Virulences to genes *Lr11*, *Lr18*, *Lr24*, *Lr26*, and *Lr41* also show regional differentiation, with the highest frequencies in the regions where cultivars with these genes are most commonly grown. Previous surveys have shown strong regional differentiation of *P. triticina* populations in the US (Long *et al*. 1998, 2000). The minimum and maximum frequencies of virulence to some of the resistance genes from 1990 to 2006 has varied from 0% to >90% in the different regions (Table 2).

In the Central Great Plains States of Kansas and Nebraska, virulence phenotypes of *P. triticina* have changed very rapidly in response to host resistance genes in the hard red winter wheats which are grown there (Fig. 2). In 1988 virulence to *Lr11* was at 10% and had increased to >70% by 1995. Virulence to *Lr26* was <10% in 1987, and had increased to nearly 50% in 1991. Virulence to *Lr3ka* was <5% in 1992, and had increased to nearly 60% in 1997. Virulence to *Lr17* was <10% in 1995 and by 2001 had increased to nearly 90%. Virulence to *Lr24* varied from 0 to 59% from 1978 to 1990, and declined to <10% in 2001. By 2006 virulence to *Lr24* had again increased to >70%. Virulence to *Lr9* was <5% from 1985 to 1999, and increased to >30% in 2004. Virulence to *Lr1* was <10% in 1981 and increased to 100% by 1996.

Table 1. Distribution of common races of *Puccinia triticina* in the US in 2004

Races are identified by high or low infection type to near-isogenic Thatcher lines of wheat with leaf rust resistance genes *Lr1*, *Lr2a*, *Lr2c*, *Lr3*, *Lr9*, *Lr16*, *Lr24*, *Lr26*, *Lr3ka*, *Lr11*, *Lr17*, *Lr30*, *LrB*, *Lr10*, *Lr14a*, *Lr18*. The 4-letter code nomenclature is described in Kolmer and Long (1989), and Kolmer *et al*. (2004)

Race	Virulence (*Lr* genes)	South-east[A]	North-east[B]	Ohio Valley[C]	Southern Plains[D]	Central Plains[E]	Northern Plains[F]
MBDS	*1*, *3*, *17*, *B*, *10*, *14a*	7%	7%	12%	18%	9%	6%
MCDS	*1*, *3*, *26*, *17*, *B*, *10*, *14a*	3%	29%	50%	9%	12%	13%
MCRK	*1*, *3*, *26*, *3ka*, *11*, *30*, *10*, *14a*, *18*	29%	21%	5%	0%	1%	0%
TBBJ	*1*, *2a*, *2c*, *3*, *10*, *14a*	8%	0%	0%	9%	9%	3%
TBBG	*1*, *2a*, *2c*, *3*, *10*	0%	0%	3%	3%	3%	26%
TBDS	*1*, *2a*, *2c*, *3*, *17*, *B*, *10*, *14a*	2%	0%	0%	3%	12%	7%
TCDS	*1*, *2a*, *2c*, *3*, *26*, *17*, *B*, *10*, *14a*	4%	29%	5%	0%	16%	5%
THBJ	*1*, *2a*, *2c*, *3*, *16*, *26*, *10*, *14a*	2%	0%	0%	5%	2%	7%
TLGJ	*1*, *2a*, *2c*, *3*, *9*, *11*, *10*, *14a*	10%	0%	2%	3%	0%	1%
TNRJ	*1*, *2a*, *2c*, *3*, *9*, *24*, *3ka*, *11*, *30*, *10*, *14a*	5%	0%	0%	18%	8%	11%
Total no. of races		21	5	12	21	25	22
Total no. of isolates		101	14	42	177	172	242

[A]South-eastern US States.[B]North-eastern US States.[C]States in the Ohio Valley.[D]Texas, Oklahoma.[E]Kansas, Nebraska. [F]Minnesota, South Dakota, North Dakota.

Table 2. Virulence (frequency, %) of *Puccinia triticina* isolates in the US in 2004, and minimum and maximum virulence frequencies (%) from 1990 to 2006, to Thatcher wheat lines near-isogenic for different leaf rust resistance genes

Gene	South-east[A]		North-east[B]		Ohio Valley[C]		S. Plains[D]		Cen. Plains[E]		N. Plains[F]	
	2004	1990–2006	2004	1990–2006	2004	1990–2006	2004	1990–2006	2004	1990–2006	2004	1990–2006
Lr1	99	85–100	100	72–100	93	75–100	84	85–100	83	86–100	62	78–100
Lr2a	54	21–64	43	0–44	17	6–73	61	4–61	71	5–71	79	15–80
Lr2c	54	22–65	43	21–80	17	6–73	61	4–61	71	5–71	79	15–80
Lr3	100	93–100	100	40–100	100	93–100	100	93–100	99	86–100	100	89–100
Lr9	17	6–46	0	0–44	2	0–26	31	0–32	12	0–20	14	0–14
Lr16	3	0–4	0	0–10	5	0–11	5	0–15	5	2–7	18	0–49
Lr24	10	3–56	0	0–60	7	0–59	34	6–71	19	3–70	15	5–70
Lr26	60	3–59	93	0–93	64	9–84	14	14–49	40	3–43	26	8–72
Lr3ka	49	2–76	21	19–93	7	0–90	22	0–58	9	0–73	12	0–83
Lr11	67	24–96	21	10–100	19	0–90	25	7–75	9	0–84	13	2–95
Lr17	27	0–36	64	0–80	67	0–89	44	1–88	53	0–91	34	0–63
Lr30	49	2–76	21	0–93	7	0–84	22	0–46	9	0–73	12	0–67
LrB	18	–	64	–	67	–	39	–	53	–	33	–
Lr10	94	64–100	100	71–100	97	66–100	100	80–100	99	86–100	99	62–100
Lr14a	94	–	100	–	95	–	89	–	94	–	67	–
Lr18	35	8–71	21	8–93	10	6–38	1	0–12	1	0–8	0	0–18
Lr41	5	–	0	–	0		18	–	8	–	11	–
No. of isolates	101		14		42		177		172		242	

–, Differential line not included until 2001. [A]South-eastern US States. [B]North-east US States.[C]States in the Ohio Valley. [D]Texas, Oklahoma. [E]Kansas, Nebraska. [F]Minnesota, South Dakota, North Dakota.

New races have also been introduced to the major wheat-producing regions of the US from other areas of North America. Races MBDS and MCDS, in addition to virulence to *Lr17*, differed from other isolates in the Great Plain region for virulence to genes *Lr3bg* and *LrB*, and by avirulence to *Lr28*. These races also differed greatly for AFLP phenotypes (Kolmer 2001*b*) when compared to other races in the Great Plains. The combined virulence and molecular data indicated that these races were introduced to the southern-central Great Plains region, most likely from either Mexico or the Pacific North-west region. Isolates with virulence similar to MBDS and MCDS have been described in Mexico (Singh 1991). Numerous introductions of *P. triticina* have most likely occurred since wheat cultivation began in North America in the 17th century. Distinct groups of *P. triticina* races in Canada were distinguished by using RAPD (Kolmer *et al*. 1995) and AFLP (Kolmer 2001*b*) markers. Isolates that were identical or closely related for virulence were also identical or closely related for molecular phenotype with both RAPD and AFLP markers. Recently developed microsatellite (SSR) markers (Szabo and Kolmer 2007) also grouped races of *P. triticina* from the US in the same manner as the RAPD and AFLP markers. In 2003, races

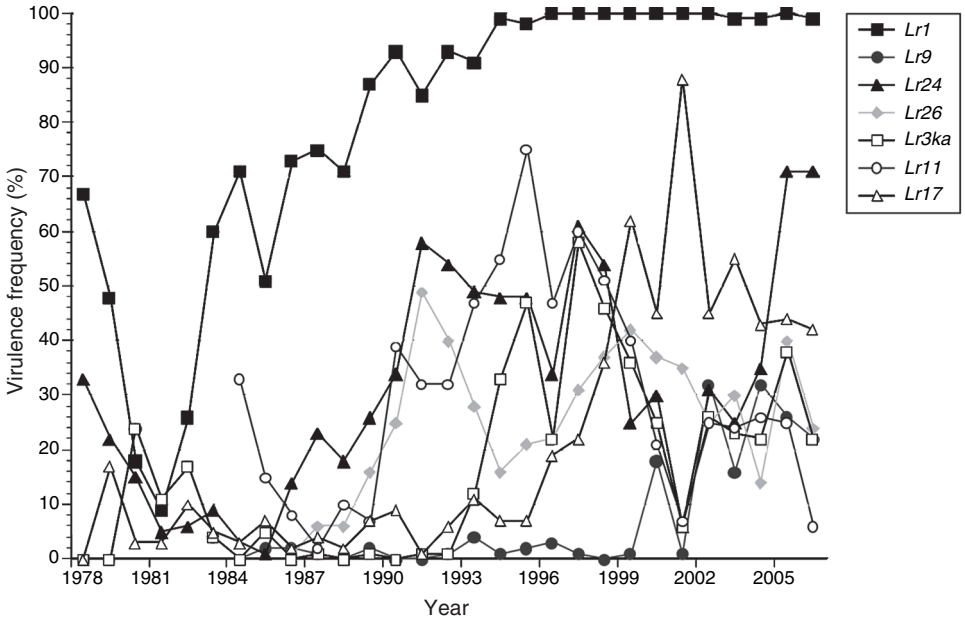

Fig. 2. Frequency (%) of *Puccinia triticina* isolates with virulence to leaf rust resistance genes in the hard red winter wheat region of Kansas and Nebraska from 1978 to 2006.

of *P. triticina* with high virulence to durum wheat were detected in southern California (Ordoñez and Kolmer 2007). Virulence characterisation and SSR genotype data (Ordoñez *et al*. 2005) indicated that these isolates were nearly identical to isolates from Mexico that also had high virulence to durum wheats (Singh *et al*. 2004). Isolates with high virulence to durum wheat were most likely introduced to California from Mexico. The general association between virulence and molecular variation in *P. triticina* would be expected, since the alternate host for *P. triticina*, *Thalictrum speciosissimum*, is not native to North America. Thalictrum species native to North America are resistant to basidiospore infection by *P. triticina* (Saari *et al*. 1968), making it unlikely that the sexual stage contributes to the epidemiology of the disease.

Different strategies have been used to develop leaf rust resistant wheat cultivars in the various wheat market classes that are grown in the U.S. In the soft red and hard red winter wheats, race-specific seedling genes have been often used. Seedling genes originally derived from common hexaploid wheat have not provided durable resistance, as cultivars with *Lr1*, *Lr2a*, *Lr3*, *Lr3ka*, *Lr10*, *Lr11*, and *Lr17* have lost resistance after the rapid emergence of virulent races. Seedling resistance genes derived from lower ploidy relatives of wheat have fared no better in conferring long lasting resistance. Gene *Lr9*, derived from *Triticum umbellulata* (Shaner *et al*. 1972), was initially used in soft red winter wheat in the 1970s and initially gave complete immunity to leaf rust. However, within a few years, races with virulence to *Lr9* appeared and soon became widespread in the eastern U.S. Gene *Lr18*, derived from *T. timopheevi*, has also been used in soft red winter wheat, and leaf rust races with virulence to this gene are easily found in the eastern U.S. Genes *Lr24* and *Lr26*, derived from *Thinopyrum elongatum* and *Secale cereale*, respectively, have been used in the hard red and soft red winter wheat. Races with virulence to these genes were soon found after the release of cultivars with these genes. Leaf rust races in the US with virulence to *Lr41*, derived from *T. tauschii*, and *Lr50*, derived from *T. timopheevi*, were identified even before cultivars with these genes were released.

Another strategy has been to select for genes that optimally express resistance at the adult plant stage of wheat development. Some adult plant genes, such as *Lr12* and *Lr13*, both derived from common wheat (Dyck *et al*. 1966) condition race-specific resistance and have not provided durable resistance. Some isolates of races MBDS and MCDS also have virulence to adult plant genes *Lr35* and *Lr37* (Kolmer *et al*. 2003). These genes, which were derived from *T. tauschii*, and *T. ventricosa*, respectively, had not been previously used in cultivars grown in the Great Plains region.

Many US hard red spring wheats have the adult plant resistance gene *Lr34* (Roelfs 1989). The source of this gene was the Brazilian cultivar Frontana (Dyck *et al*. 1966). The first US spring wheat with *Lr34* was cv. Chris, released in 1966 by the Minnesota Agricultural Experiment Station, which was derived by backcrossing the Frontana leaf rust resistance into Thatcher. Cultivar Era, released in 1971, was another important wheat from Minnesota with *Lr34* (Ezzahiri and Roelfs 1989) derived from Frontana. Era was used extensively as a parent in the Minnesota wheat project. Other recent hard red spring wheat cultivars in the US also have effective adult plant resistance that is most likely due to *Lr34* (Oelke and Kolmer 2004). This gene conditions non-specific resistance to leaf rust, and cultivars with *Lr34* have at least a moderate level of resistance in field plots. Although many spring wheat cultivars with *Lr34* have been grown since the 1970s, no isolates in the US with complete virulence to this gene have been found in the recent surveys. The hard red winter wheat Sturdy was determined to have *Lr34* (Dyck 1991); however, *Lr34*

is not commonly found in hard red winter wheat or in soft red winter wheat germplasm (J. A. Kolmer, unpublished data).

Cultivars Alsen, Norm, and Knudson were among the most leaf rust resistant hard red spring wheats in a recent study (Oelke and Kolmer 2004). Alsen, released by the North Dakota Agricultural Experiment Station in 2000, with resistance to fusarium head blight derived from Sumai 3 and moderate resistance to leaf rust has been widely grown in North Dakota and Minnesota. Norm, released by the Minnesota Agricultural Experiment Station in 1992, has been highly resistant to leaf rust since release. Knudson, developed by Agri-Pro in 2002, with improved resistance to fusarium head blight and very high resistance to leaf rust has been widely grown in Minnesota and North Dakota. Genetic studies determined that Knudson had leaf rust resistance genes *Lr3*, *Lr10*, *Lr13*, *Lr16*, *Lr23*, and *Lr34* (Kolmer and Oelke 2006); Alsen had genes *Lr2a*, *Lr10*, *Lr13*, *Lr23*, and *Lr34*; Norm had genes *Lr1*, *Lr10*, *Lr13*, *Lr16*, *Lr23*, and *Lr34* (Oelke and Kolmer 2005) (Table 3). Genes *Lr23* and *Lr34* are in Alsen, Norm, and Knudson. The interaction of these 2 genes, combined with *Lr16* in Knudson and Norm, accounts for the high levels of leaf rust resistance in these cultivars. In field plots the Thatcher lines with *Lr16*, *Lr23*, and *Lr34* all have some effective leaf rust resistance (Table 3) in comparison with Thatcher. When combined together in wheat cultivars, these genes interact for significantly higher levels of resistance, as has been noted for *Lr34* (German and Kolmer 1992).

Gene *Lr23* is temperature-sensitive for expression of leaf rust resistance in seedling plants (Dyck and Johnson 1983). The Thatcher line with *Lr23*, at ambient greenhouse temperatures of 18–25°C, when inoculated with a single leaf rust isolate may produce highly variable infection types ranging from small uredinia surrounded by chlorosis to large uredinia lacking chlorosis. Wheat lines with this gene are not included in the regular virulence survey differential sets because of this temperature sensitivity. At higher temperatures of 25°C in growth cabinets, many US leaf rust isolates will produce a very low infection type to the Thatcher line with *Lr23*. In field plots, the Thatcher line with *Lr23* is fairly resistant (Table 4). Leaf rust races with virulence to *Lr16* are currently present in the northern Great Plains region. However, the Thatcher *Lr16* line still had resistance in field plots. The Thatcher line with *Lr34* also had lower rust severity when compared with Thatcher in the field plots. Genes *Lr1*, *Lr2a*, *Lr3*, *Lr10*, and *Lr13* no longer provide effective resistance and are unlikely to contribute to resistance in these cultivars.

The future development of wheat germplasm in the US with high levels of durable leaf rust resistance will depend on the ability to select genotypes that have combinations of effective resistance genes such as *Lr16* and *Lr23*, with the adult plant resistance genes such as *Lr34*. Other genes that condition adult plant resistance may also be present in the US hard red spring wheat cultivars, since some of these cultivars had low infection types as adult plants that could not be explained by the presence of the postulated seedling resistance gene(s) or by *Lr34* (Oelke and Kolmer 2004). Additional sources of non-specific resistance,

Table 3. Leaf rust ratings of the wheat cultivars Alsen, Norm, and Knudson, and Thatcher lines with single leaf rust resistance genes in field plots at St. Paul, MN

Severity (% leaf area) estimated using the modified Cobb scale. T, trace level of uredinia; field reaction: R, small uredinia surrounded by necrosis; MR, moderate size uredinia surrounded by necrosis and chlorosis; M, mixture of large and small uredinia; MS, moderate size uredinia surrounded by chlorosis; S, large uredinia without chlorosis

Wheat line		Leaf rust rating
Alsen		5 MR
Norm		T R
Knudson		T R
Thatcher		50–80 S
Thatcher *Lr1*	RL 6003	50–80 S
Thatcher *Lr2a*	RL 6016	50–80 S
Thatcher *Lr3*	RL 6002	50–80 S
Thatcher *Lr10*	RL 6004	50–80 S
Thatcher *Lr13*	RL 4031	50–80 S
Thatcher *Lr16*	RL 6005	50–70 MS
Thatcher *Lr23*	RL 6012	5–10 R MR
Thatcher *Lr34*	RL 6058	5–10 M

Table 4. Races of wheat stem rust (*Puccinia graminis* f. sp. *tritici*) in the US in 2004

Virulence of stem rust races—*Sr* genes: QFCS—*5, 21, 8, 9g, 17, 9e, 9b, 10;* MCCF—*5*, *7b*, *9g*, *17*, *10*, *Tmp*; MCCD—*5*, *7b*, *9g*, *17*, *10*; TPMK—*5*, *21*, *9e*, *7b*, *11*, *8*, *9g*, *36*, *17*, *9b*, *10*, *Tmp*; QCCN—*5*, *21*, *9g*, *17*, *9a*, *10*; TTTT—*5*, *21*, *9e*, *7b*, *11*, *6*, *8*, *9g*, *36*, *9b*, *30*, *17*, *9a*, *9d*, *10*, *Tmp*

Area	State	No. of collections	No. of isolates	No. of isolates of stem rust races					
				QFCS	MCCF	MCCD	TPMK	QCCN	TTTT
Great Plains	TX	1	1	1					
	OK	1	1	1					
	KS	3	3	3					
	NE	3	2	1			1		
	s.d.	3	3	3					
	ND	7	6	6					
Midwest	MO	1	1	1					
	IN	1	1		1				
	MN	15	19	15	2	1			1
Pacific NW	WA	1	2					2	
US Total		36	39	31	3	1	1	2	1

as found in the CIMMYT germplasm (Singh *et al*. 2000), may complement and enhance leaf rust resistance in US wheats.

Wheat stem rust

Major epidemics of wheat stem rust, caused by *Puccinia graminis* f. sp. *tritici*, occurred in the US from 1900–1950s. During this time, stem rust was by far the most important pathogen of wheat (Leonard 2001), and stem rust resistance was a high priority in wheat-breeding programs throughout the country. Since the late 1950s, however, stem rust has decreased in importance as a disease of wheat in the US. The decline of stem rust has been attributed to the eradication of the alternate host common barberry (Campbell and Long 2001) and the increased and widespread use of winter and spring wheats that are highly resistant to stem rust.

Unlike leaf rust, the areas where stem rust can overwinter at the uredinial stage are limited to the Gulf Coast and southern Texas, on autumn-planted susceptible winter wheat cultivars and volunteer plants (Roelfs 1989). Stem rust is generally detected in late April in the southern US, much later than either leaf rust or stripe rust. In July, stem rust is detectable on trap plots of susceptible winter and spring wheat, barley, and wild grasses in the northern Great Plains. In the past, major stem rust epidemics in the central and northern Great Plains and the Midwest could be explained in most cases by a large population of *P. graminis* f. sp. *tritici* on susceptible winter wheat cultivars in the southern States. Since almost all of the current hard red winter wheats are resistant to stem rust, there is much less opportunity for the rust to infect and overwinter in the south, resulting in very small population sizes of *P. graminis* f. sp. *tritici*.

With the widespread eradication of barberry throughout most of the wheat producing regions of the US and increased cultivation of highly resistant wheat cultivars, there has been a large decline in the number of wheat stem rust races (Groth and Roelfs 1987). In recent virulence surveys, only 3–5 races were found on wheat in the northern US (McVey *et al*. 2002). Removal of the alternate host has eliminated sexual recombination in *P. graminis*, a major source of races with new combinations of virulence. This also eliminated aeciospores as a local inoculum source. Urediniospores transported long distances from overwintering sites in the southern US are now the sole inoculum sources for stem rust in the US. In recent years, barberry plants have been found in several of the original eradication sites in south-eastern Minnesota (Peterson *et al*. 2005). Although this is not a major area of wheat production, it is worrisome that seeds from these barberry plants may be carried by birds and become re-established in the wheat-growing areas of the Great Plains and Midwest. It will be important to monitor this population of barberry for aecial infections that may lead to the emergence of new stem rust races.

Race TPMK (Roelfs and Martens 1988) was the most common stem rust race in the US since the late 1950s, after the decline of race TMB (race 15B according to the Stakman differential system), which caused such devastating epidemics during 1950–1954 (Kolmer 2001*a*). Race TPMK is virulent to the stem rust resistance in the hard red winter wheat Triumph (*SrTmp*). The widespread use of Triumph-derived resistance in hard red winter wheat cultivars contributed to the increase of TPMK. Selection of wheat breeding lines for resistance to TPMK in the central and northern Great Plains led to the widespread use of *Sr6* in hard red winter and spring wheat cultivars. The predominant wheat cultivars growing in the Great Plains and the Midwest are resistant to TPMK due to *Sr6* and other resistance genes. As a result, TPMK declined in the US stem rust population starting in the late 1980s. In recent years, this race has rarely been identified in survey samples collected across the US (Table 4).

In 2000, a stem rust isolate with additional virulence to *Sr6*, *Sr9b*, *Sr30*, and *Sr9a*, designated as race TTTT, was identified for the first time in the US (Jin 2005). This race has also been found in subsequent years, but in very low frequencies. This race does not resemble any other race groups known to occur in North America, but is similar in virulence to some South American races. On a set of wheat cultivars, race TTTT did not have broader virulence compared to TPMK. Most spring wheat cultivars grown in the northern Great Plains are resistant to this race, but the identities of the *Sr* genes conferring resistance to TTTT in the spring wheat cultivars are unknown. Resistance gene *SrWld1* derived from cv. Waldron might be a part of the *Sr* gene complex present in most spring wheat cultivars. Preliminary testing of current cultivars indicated that only 26% of the hard red winter wheat cultivars grown in the Great Plains States, and 22% of the soft red winter wheat cultivars grown in the Midwest and south-eastern States, are resistant to race TTTT. Resistance in the hard red winter wheat is primarily due to *Sr24* and the resistance derived from the 1AL.1RS translocation in Amigo. Resistance to TTTT in the soft red winter wheat is mostly due to *Sr31* and *Sr24*.

Since 2003, race QFCS has been the predominant stem rust race in the US (Jin 2005) (Table 4). This race has been common in the wheat stem rust population in the US since the late 1980s. Race QFCS has virulence on genes *Sr5*, *Sr8a*, *Sr9a*, *Sr9d*, *Sr9g*, *Sr10*, *Sr17*, and *Sr21*. Several hard red winter and soft red winter wheat cultivars are susceptible to QFCS in seedling tests, but the majority of winter wheat cultivars are resistant. All of the hard red spring wheat and durum wheat cultivars in the northern Great Plains are resistant to race QFCS. This race may survive and overwinter on wild grasses, particularly *Hordeum jubatum*, which is susceptible to stem rust.

Several effective stem rust resistance genes, *Sr6*, *Sr24*, *Sr31*, *Sr36*, *SrTmp*, and the resistance derived from the 1AL.1RS translocation in Amigo are present either singly or in combinations in the hard winter wheat cultivars grown in the Great Plains and in the soft red winter wheat cultivars grown in the Midwest and south-eastern US. Gene *Sr24* occurs in several hard red winter wheat cultivars, and to a lesser degree in soft red winter wheat cultivars and spring wheat cultivars, likely due to the complete association with *Lr24* (McIntosh *et al*. 1995). Stem rust isolates virulent on *Sr24* have not been identified in North America; thus, this gene will continue to serve as a valuable source of stem rust resistance. Gene *SrTmp*, derived from cv. Triumph 64, is found primarily in cultivars grown in the southern and central Great Plains States. Races TPMK, MCCF, and TTTT are virulent to *SrTmp*; however, almost all other races are avirulent to wheat cultivars with this gene.

Several of the hard red winter wheat cultivars appear to have the stem rust resistance derived from the 1AL.1RS translocation

from Amigo wheat. This translocation was thought to be independent of the 1BL.1RS translocation derived from Petkus rye (McIntosh *et al*. 1995), which carries the rust resistance genes *Sr31*, *Lr26*, and *Yr9*. A stem rust resistance gene on 1RS in Amigo was reported (The *et al*. 1992), but the authors could not determine whether this gene was *Sr31* based on the stem rust isolates they used. A recent study with isolates virulent on *Sr31* indicated that the resistance on the 1AL.1RS translocation is not due to the presence of *Sr31* (Jin and Singh 2006). The 1B.1R wheat–rye translocation with *Sr31* is found mostly in the soft red winter wheat cultivars.

Almost all hard red spring wheat cultivars bred in the north central Great Plains are highly resistant to stem rust. Some of the cultivars may have the adult plant resistance gene *Sr2* derived from Yaroslav emmer (McFadden 1930) and adult plant stem rust resistance derived from cv. Thatcher (Nazareno and Roelfs 1981; Kolmer *et al*. 1991). Seedling resistance genes *Sr6*, *Sr9b*, *Sr11*, and *Sr17* are also common in the spring wheat cultivars. Thatcher, with adult plant stem rust resistance derived from Iumillo durum, is common in the pedigrees of many hard red spring wheats in the US and Canada. The adult plant stem rust resistance in Thatcher is enhanced by the presence of *Lr34* (Dyck 1987). Thatcher lines with *Lr34* are highly resistant in field nursery plots, while cv. Thatcher is only moderately resistant, and can appear to be susceptible under heavy inoculum pressure. The presence of *Lr34* allows the expression of additional adult plant stem rust resistance genes in Thatcher.

Stem rust race TTKS, found recently in East Africa (Pretorius *et al*. 2000; Wanyera *et al*. 2006) was the first stem rust race to have virulence to genes *Sr31* and *Sr38*. This race was also virulent to genes *Sr9b* and *Sr6*, which are in many US wheat cultivars. In seedling tests many hard red winter wheat cultivars, soft red winter wheat cultivars, and hard red spring wheat cultivars that are grown in the US are susceptible to this new race. If this stem rust race were to become established in the US winter wheat crop in the south-eastern States and southern Great Plains, it could result in the re-emergence of stem rust as a major disease of wheat in the US. Fortunately, several wheat cultivars with genes *Sr36* and *SrTmp*, and resistance on the 1AL.1RS translocation, are resistant to race TTKS. Wheat lines with these genes, and other sources of resistance, can be used in breeding projects to develop germplasm with resistance to this and other stem rust races.

Conclusions

Wheat leaf rust and stem rust continue to present challenges to the development of wheat cultivars with highly effective durable resistance. With the continual threat of new races being introduced to North America, and new virulent races arising by mutation, it is imperative to continue to characterise the virulence and molecular variation of these ‘shifty enemies’ (Stakman 1947). With the development of new molecular markers it should be possible to more accurately determine the migration patterns of these pathogens, which might eventually lead to more rational deployment strategies to lengthen the longevity of race-specific resistance genes. Gene combinations that confer durable resistance in widely grown wheat cultivars should be identified and utilised in order to develop germplasm with long-lasting rust resistance.

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