Chapter 5

Diseases Which Challenge Global Wheat Production—the Wheat Rusts

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SUMMARY

(1) Wheat rusts have been important throughout the history of wheat cultivation and are currently important diseases that are responsible for regularly occurring yield losses in wheat. New races of wheat rusts have recently emerged worldwide, complicating efforts to develop rust resistant cultivars.

(2) Wheat leaf rust, caused by the rust fungus *Puccinia triticina* Eriks., is the most common and widespread rust of wheat worldwide. Although the alternate host is not present in North America, *P. triticina* is highly diverse for virulence as many races are found annually.

(3) Many leaf rust resistance genes no longer provide effective resistance since virulent leaf rust races have been selected by resistant cultivars. Resistance gene *Lr34* has provided non-race-specific resistance for many years in wheat cultivars grown worldwide. Gene *Lr46* and other characterized sources of adult-plant partial resistance have also provided durable resistance to leaf rust. Wheat cultivars with high levels of resistance can be developed by combining *Lr34*, *Lr46*, and other effective resistance genes.

(4) Wheat stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., is an important disease in areas where wheat grows and matures in cool temperatures. The occurrence of wheat stripe rust has increased in the southern–mid Great Plains and the southeastern states of the US. Although an alternate host has never been found, *P. striiformis* f. sp. *tritici* is highly diverse as many different races are found worldwide.

(5) Race-specific stripe rust resistance genes have been rendered ineffective due to the increase of virulent races. Wheat cultivars with adult-plant resistance genes such as *Yr18* that condition non-race-specific partial resistance or genes that condition high-temperature adult-plant resistance (HTAP) have had long-lasting resistance to stripe rust. Wheat cultivars with durable resistance to stripe rust can be developed by using combinations of these resistance genes.

(6) Wheat stem rust, caused by *Puccinia graminis* Pers.:Pers. f. sp. *tritici* Eriks. & E. Henn., is potentially a highly destructive disease of wheat. The alternate host, *Berberis vulgare*, has largely been eradicated from North America, reducing the number of stem rust races. Major epidemics of wheat stem rust occurred periodically from 1900 to 1954 in North America, causing severe yield losses. The widespread cultivation of stem rust resistant winter wheat and spring wheat since the 1950s has greatly reduced the population size of *P. graminis* f. sp. *tritici*, resulting in many fewer stem rust infections annually.

(7) Wheat cultivars with the adult-plant non-race-specific resistance gene *Sr2* have had durable resistance to stem rust. Stem rust races with virulence to *Sr31* and *Sr38* have recently been found in eastern Africa. These races are virulent to many CIMMYT cultivars with *Sr31* and also have virulence to many US wheat cultivars. Gene *Sr2* and other sources of resistance can be used to develop cultivars with resistance to these stem rust races.

INTRODUCTION

The rusts of wheat are among the most important and common diseases of wheat in the US and worldwide. Rust has afflicted wheat for thousands of years as references to wheat rust can be found in the Bible and the classical literature of ancient Greece and Rome (Chester 1946). In the early 20th century widespread epidemics of wheat rusts provided the impetus for early advances in genetics of disease resistance in plants, epidemiology of plant pathogens, and genetics of host–parasite interactions. Today the rust diseases continue to cause regular yield losses worldwide, threatening the sustainable production of wheat. The continuing evolution of virulent rust races in response to the release of rust resistant wheat cultivars poses a constant challenge to wheat researchers.

WHEAT LEAF RUST

Distribution and epidemiology

Leaf rust, caused by *Puccinia triticina* Eriks., is the most common and widely distributed of the three rust diseases of wheat. In the US, leaf rust is commonly found on soft red winter wheat grown in the southeastern states and Ohio Valley region, on hard red winter wheat from Texas to South Dakota, and on hard red spring wheat in South Dakota, North Dakota, and Minnesota. Leaf rust also occurs on spring wheat that is grown in California, and to a lesser extent on winter and spring wheat in Oregon, Washington, and Idaho. Worldwide, leaf rust is a major continent-wide disease in the western prairies of Canada; the South American region of Argentina, Chile, Brazil, and Uruguay (German et al., 2007); the Central Asia region of northern Kazakhstan and Siberia; southern and central Europe; the Middle East; and parts of the Indian Subcontinent (Roelfs et al., 1992). Leaf rust is less widespread and occurs at a local level in eastern and western Australia, China, eastern Africa, and in South Africa.

Leaf rust is characterized by the uredinial stage of small round, brown to orange pustules that occur on the upper and lower leaf surfaces, and less frequently on the leaf sheaths (Color Plate 9). The pustules remain discrete, without coalescing. The uredinia are capable of producing up to 3,000 urediniospores per day (Roelfs et al., 1992) if the host plant tissue remains healthy. The urediniospores are 20 μm in diameter, echinulate, dikaryotic ($n+n$), wind-disseminated, and deposited in rain events on host plants in the immediate vicinity and potentially also on hosts hundreds of kilometers distant. The urediniospores germinate on wheat plants, producing the specialized infection structures of appressoria, germ tube, and penetration peg (Harder 1984) that allow the fungus to penetrate the host stomata. Further specialized structures, the substomatal vesicle and haustoria, are produced which allow the fungus to obtain nutrients from host mesophyll cells without killing them. Infectious hyphae of the fungus spread throughout the mesophyll layer. Leaf rust infections are initially visible as faint flecks on leaf surfaces 3–4 days after inoculation. Uredinia erupt and break through the epidermal leaf surface 8–10 days after initiation of the infection process. The clonally produced urediniospores can cycle indefinitely on wheat hosts.

Resistance responses to leaf rust are characterized by small uredinia surrounded by necrosis, or by an abundance of hypersensitive flecks produced in response to infection (Color Plate 9). Non-hypersensitive resistance is characterized by fewer and small uredinia compared to a susceptible response. As the uredinia age, teliospores are formed in the uredinia. Teliospores are dark, 16 μm wide, and thick walled and have two dikaryotic cells. The pycniospores and aeciospores, which are spore stages associated with sexual reproduction on alternate hosts, are not produced on the wheat host.

Leaf rust infections occur in the US during September to November on fall-planted winter wheat from Texas to South Dakota, and from the Gulf Coast states to North Carolina. Leaf rust infected volunteer winter wheat plants that survive the summer are the inoculum source for

the fall-planted winter wheat crop. The optimal conditions for infection are temperatures near 20 °C with free moisture on the leaf surface for at least 8 hours, and 25 °C is the optimal temperature for growth. Overnight periods of dew formation are optimal for germination of urediniospores. The infection process can occur at temperatures from 2 to 30 °C; however, longer periods of dew are required at the lower temperatures. Temperature and moisture conditions in the fall months throughout much of the winter wheat region of the US allow *P. triticina* to become established over a large geographical area.

Throughout the US winter wheat region leaf rust inoculum sources are a combination of (i) infections that survive the winter either as urediniospores or mycelia and (ii) windblown urediniospores carried in the southerly winds from infected winter wheat to the south. The relative importance of overwintering infections and exogenous sources of inoculum will vary based on conditions in the fall that affect leaf rust infection, temperatures during the winter that allow leaf rust to survive, and the maturity of the crop. Wheat cultivars with later maturity will be more affected by leaf rust infections from exogenous sources. Severe epidemics result from the combination of overwintering leaf rust infections, windblown urediniospores deposited in rain events, regular dew periods, and temperatures greater than 25 °C during the time when winter wheat is breaking dormancy and resuming growth and development.

In south Texas and along the Gulf Coast, winter temperatures often exceed 20 °C, with only infrequent freezes. In this region where *P. triticina* regularly overwinters, leaf rust can reach high severity levels in March and April (Roelfs 1989). From Oklahoma to eastern Virginia, winter temperatures usually do not exceed 20 °C and freezing temperatures are common. In this area leaf rust overwinters in isolated pockets, resulting in foci of leaf rust infections that are apparent in the spring when temperatures are regularly above 20 °C and leaf rust is rapidly increasing. Leaf rust severities in this region usually reach maximum levels in April and mid-May. In the area from Kansas north to South Dakota and east to the Ohio Valley, winter low temperatures range from −10 to −20 °C, and high temperatures rarely exceed 15 °C. Leaf rust can survive in winter wheat in this region as mycelium resulting from infections during the fall months. The overwintering survival of leaf rust in this region is also dependent on adequate snow cover to protect the wheat leaves from freeze damage during periods of extreme low temperatures. Leaf rust severities are at maximum levels in May in Kansas to June in South Dakota and the Ohio Valley states.

In Minnesota, South Dakota, and North Dakota, leaf rust infection on the spring wheat is usually first observed in mid-to-late June, with maximum severity levels in mid-to-late July. Daytime temperatures in the summer in this area are often greater than 25 °C, with frequent rain events and dew periods. All leaf rust infections on spring wheat originate from windblown urediniospores from winter wheat growing in the southern Great Plains or from local fields of winter wheat. Leaf rust epidemics in the spring wheat region are most severe when initial infections occur at the tillering stage, which allows additional generations of urediniospores to infect the crop.

Origin and historical importance

Puccinia triticina was introduced to North America with the first agricultural settlements and wheat cultivation in the early 17th century (Chester 1946). The origin of *P. triticina* is likely the Fertile Crescent region of southwest Asia and the Middle East (Wahl et al., 1984), which is also the origin of diploid, tetraploid, and hexaploid wheat. The most susceptible alternate host of *P. triticina* is *Thalictrum speciosissimum* (= *Thalictrum flavum*), which is native to southern Europe, western Asia, and Turkey. It is likely that the center of origin of *P. triticina* is a region where the alternate and telial hosts overlap, which would be southwest Asia.

In early rust research in the US, the importance of leaf rust was not recognized since all yield

losses due to rust in wheat were then attributed to stem rust (caused by *P. graminis* Pers.:Pers. f. sp. *tritici* Eriks. & E. Henn.). A common attitude was that *P. triticina* caused little or no damage to wheat (Chester 1946). This mistaken opinion was most likely because the disease only affected the leaves, did not cause grain shriveling, and was not visible when the grain was harvested. Replicated yield loss studies in field plots with sulfur or fungicide treatments determined that leaf rust was a major cause of yield loss in wheat.

Mains (1930) examined the importance of leaf rust on yield in the 1920s and determined that the commonly grown soft red winter wheat cultivars that were susceptible to leaf rust suffered losses that ranged from 25% to over 90%. Even a resistant cultivar had losses over 10% due to the premature death of heavily infected flag leaves. Caldwell et al. (1934) determined that seven winter wheat cultivars which varied from resistant to susceptible suffered losses from 15% to 28% due to leaf rust infections. In the early hard red winter wheats Johnston (1931) showed that resistant and susceptible cultivars suffered maximum losses of 22% and 55%, respectively, due to leaf rust. In 1938 leaf rust caused a yield loss of 25%–30% statewide in Oklahoma (Chester 1939). In hard red spring wheat in Canada, losses due to leaf rust were over 50% in susceptible cultivars and from 12% to 28% in resistant cultivars (Peturson et al., 1945; Samborski and Peturson 1960). Chester (1946) developed a predictive curve for estimating yield loss due to leaf rust based on the growth stage in which wheat was defoliated, using data from 68 yield studies. Losses ranged from an average of 10% if defoliation occurred in the dough stage of grain development to an average of 95% if defoliation occurred in the jointing stage. In a test with isogenic spring wheat lines that differed by only a single leaf rust resistance gene, Dyck and Lukow (1988) showed a 22% difference in yield between the resistant and susceptible lines. Martin et al. (2003) determined that isolines of winter wheat that had resistance genes *Lr41* and *Lr42* had a 63% and 26% yield increase compared with lines that lacked these genes. Herrera-Foessel et al. (2006) estimated yield losses over 50% due to leaf rust in durum wheat (*T. turigidum* ssp. *durum*) in Mexico.

Leaf rust continues to cause regular losses in present-day wheat cultivars. In 2007 yield loss due to leaf rust in the hard red winter wheat crop in Kansas was estimated to be 14% (Kansas Department of Agriculture, Topeka, Kansas). Khan et al. (1997) developed a yield loss model for southern US soft red winter wheat that predicted a 1% yield loss for every 1% increase in rust severity at the milky-ripe stage of grain development. Leaf rust resistance is a high priority in wheat germplasm developed at the International Maize and Wheat Improvement Center (CIMMYT). Wheat production in Africa, South America, and Asia where CIMMYT wheat germplasm is grown has suffered 1%–20% losses due to leaf rust (Marasas et al., 2004). CIMMYT has estimated a 27 : 1 benefit-to-cost ratio for development of leaf rust resistant wheat cultivars. This same study also determined that breeding for leaf rust resistance in wheat was economically justified even if yield losses in areas with yields of 4 t ha^{-1} were only 0.2%–0.8%.

Effects on grain and flour quality

In the study by Caldwell et al. (1934) 75% of the yield loss to leaf rust was due to reduced number of kernels per head. Susceptible winter wheat cultivars with leaf rust infection had 14%–17% fewer kernels than the same cultivars treated to control rust. In their study the weight of individual kernels was also 6%–7% lower in the leaf-rust-infected treatments, and kernel weight was reduced from 1.5% to 12%, depending on the resistance level of the cultivar. Martin et al. (2003) showed that kernel weight decreased by 9%–14% in susceptible winter wheat lines compared with their resistant isolines. Other studies with susceptible and resistant spring wheat types showed a larger effect, with a 6%–39% reduction in kernel weight (Waldron 1936; Peturson et al., 1945). Leaf rust infections that occur before flowering will result in fewer kernels per head, while infections that occur during grain filling will result in lighter kernels (Chester 1946). Thus different yield components may

be affected by leaf rust in winter wheat compared with spring wheat.

Leaf rust infection generally reduces the protein content in harvested grain (Caldwell et al., 1934; Peturson et al., 1945, 1948; Dyck and Lukow 1988), although in some tests protein content was unaffected or increased. In spring wheat heavy leaf rust infection generally resulted in flour with increased loaf volume (Peturson et al., 1945) and farinograph absorption (Dyck and Lukow 1988). Color of harvested grain and flour was also affected by leaf rust. In spring wheat yellow pigmentation of the flour was increased (Peturson et al., 1945); in winter wheat, increased yellow pigmentation of the grain was noted (Caldwell et al., 1934). Everts et al. (2001) determined that leaf rust affected the softness equivalent parameter and may reduce flour yield of soft red winter wheat. Reduced flour yield due to leaf rust was also determined in some tests with spring wheat (Peturson et al., 1945).

Taxonomy, life cycle, and host range

Leaf rust on wheat was originally placed in the highly complex species of *P. rubigo-vera* by Winter (1884). Leaf rusts with telial hosts on grasses and alternate hosts in the Boraginacea were placed into this single species. Eriksson (1899) described the leaf rust on wheat as a single species, *P. triticina*. Jackson and Mains (1921) determined that the most compatible alternate host for leaf rust on wheat was *Thalictrum flavuum* (= *Thalictrum speciosissimum*), which is in the Ranunculaceae. Mains (1932) preferred to group leaf rust of wheat within the complex group of *P. rubigo-vera*. Based on nondiscrete spore morphology and host range, Cummins and Caldwell (1956) also chose to place leaf rust of wheat within a complex species, *P. recondita*, with alternate hosts in Boraginacea and also Ranunculacea. In North America most rust workers referred to leaf rust on wheat as *P. recondita* f. sp. *tritici*, while in Europe leaf rust on wheat was placed in the more narrowly defined species of *P. triticina* Eriks. (Savile 1984) based on small and consistent differences in spore morphology.

D' Oliveira and Samborski (1966) conducted infection experiments of telial grass hosts with aeciospores derived from naturally infected plants of *Thalictrum speciosissimum* and *Anchusa* spp. and other genera in the Boraginacea in Portugal. They showed that the leaf rusts that differed for infection on *Thalictrum speciosissimum*, and hosts in the Boraginacea, also differed for telial hosts. Anikster et al. (1997) showed that the leaf rust from common wheat (*T. aestivum* ssp. *aestivum*), wild emmer wheat (*T. turgidum* ssp. *dicoccoides*), and durum wheat belonged to a distinct group with the alternate host *Thalictrum speciosissimum*. A second distinct group of leaf rusts with alternate hosts in the Boraginacea and telial hosts of wild wheats and rye was also described. The two groups could not be successfully crossed using either *Thalictrum speciosissimum* or alternate hosts in the Boraginacea. Based on these results, it was apparent that the leaf rust on wheat is a distinct species from the other leaf rusts on wild wheats and rye. *Puccinia triticina* Eriks. has since been used by most workers to describe leaf rust on wheat.

Puccinia triticina is a macrocyclic rust (Webster 1980) with five distinct spore stages on two taxonomically distinct hosts. The teliospores on wheat leaf tissue germinate to produce haploid basidiospores that are clear and hyaline. The basidiospores infect the alternate host, producing haploid pycnia that appear as circular yellow pustules on the upper leaf surface. The pycnia produce haploid pycniospores, which are carried by insects or rain to other pycnia. Fertilization in the pycnial structures occurs with the transfer of nuclei from pycniospores to flexuous hyphae in compatible combinations of opposite mating types. After fertilization dikaryotic aecia develop on the underside of the leaf beneath the pycnial infections. Aecial cups are produced in the aecia, from which dikaryotic aeciospores are released and wind-disseminated to infect wheat or other telial hosts. Infections from aeciospores result in production of urediniospores. Sexual reproduction in *P. triticina* has been observed in Portugal (d' Oliveira and Samborski 1966), where one of the alternate hosts, *Thalictrum speciosissimum* is commonly found. However, throughout most of the world

the disease is spread by the asexual cycling of dikaryotic urediniospores on wheat, as suitable alternate hosts are usually not present. In North America the native *Thalictrum* spp. are resistant to basidiospore infection (Jackson and Mains 1921; Saari et al., 1968). Infected plants of *Thalictrum* with aeciospores that were pathogenic to wheat have been reported in northern Kazakhstan. *Isopyrum fumarioides* has been noted as an alternate host of *P. triticina* in Siberia (Chester 1946).

Worldwide the primary host of *P. triticina* is common hexaploid wheat. Leaf rust caused by *P. triticina* has also been observed on tetraploid durum and emmer wheats in Europe, South America, Israel, Ethiopia, and Mexico (Ordoñez and Kolmer 2007b), and diploid *Aegilops speltoides* (Yehuda et al., 2004) in Israel. *Puccinia triticina* is also present on wild goatgrass, *Ae. cylindrica*, in the southern Great Plains of the US. For all of these nonhexaploid wheat hosts, only certain races or virulence phenotypes of *P. triticina* were pathogenic to these hosts, indicating a high degree of telial host specificity in *P. triticina*. Infections of *P. triticina* have not been noted in natural stands of wild wheat relatives such as *Ae. sharonensis*, *T. timopheevi*, *Ae. tauschii* (syn. *T. tauschii*), or *T. monococcum*, but infections can be obtained on these species in inoculated greenhouse tests. A different species of leaf rust, designated as *P. tritici-duri* with *Anchusa* spp. as the alternate host, occurs on durum wheat in Morocco (Viennot-Bourgin 1941; Ezzahiri et al., 1992).

Genetic variation in *P. triticina*

Virulence variation

Annual nationwide surveys of leaf rust virulence phenotypes have been conducted in Canada since 1931 (Johnson 1956) and in the US since 1926 (Johnston et al., 1968). The wheat cultivars Malakof with *Lr1*, Webster (*Lr2a*), Carina (*Lr2b*, *LrB*), Loros (*Lr2c*), Brevit (*Lr2c*, *LrB*), Hussar (*Lr11*), Democrat (*Lr3*), and Mediterranean (*Lr3*) were designated as the International Standard set of leaf rust differentials and were used in the early race identification studies. Virulence phenotypes of *P. triticina* are currently identified in the US by testing single-pustule isolates for virulence to near-isogenic lines of 'Thatcher' wheat with genes *Lr1*, *Lr2a*, *Lr2c*, *Lr3*, *Lr9*, *Lr16*, *Lr24*, *Lr26*, *Lr3ka*, *Lr11*, *Lr17*, *Lr30*, *LrB*, *Lr10*, *Lr14a*, *Lr18*, *Lr21*, *Lr28*, and winter wheat lines with *Lr41* and *Lr42* (Long and Kolmer 1989; Kolmer et al., 2007b). *Puccinia triticina* and wheat interact in a gene-for-gene manner (Samborski and Dyck 1968). For each *Lr* gene in wheat there is a corresponding locus in *P. triticina* with alleles that condition avirulent responses in the presence of host resistance genes and alternate alleles that condition virulent responses in the presence or absence of resistance genes (Kolmer and Dyck 1994). The range of seedling infection types in the Thatcher isogenic lines is shown in Color Plate 9b. In the US, up to 70 different virulence phenotypes are identified annually (Kolmer et al., 2007a), with the three most common phenotypes accounting for 25%–30% of isolates. Similar surveys of virulence phenotypes in *P. triticina* are conducted in Canada (McCallum and Seto-Goh 2006), in Australia at the Plant Breeding Institute at Cobbity, and in France (Goyeau et al., 2006).

The high degree of virulence variation in *P. triticina* in North America is directly related to the presence of susceptible hosts and the continual use of race-specific leaf rust resistance genes in the different classes of wheat. In the southern US many winter wheat cultivars that are initially resistant become susceptible to leaf rust due to the emergence and increase of virulent leaf rust races. The susceptible winter wheat cultivars allow a very large population of *P. triticina* to become established over a wide geographical area in the fall and survive during the winter. Mutations to virulence to leaf rust resistance genes are a recurrent event in such a large population. Since isolates of *P. triticina* are highly heterozygous for virulence alleles (Samborski and Dyck 1968; Kolmer 1992), a single mutation in an avirulent isolate would be sufficient to gain virulence to a resistance gene.

The wheat cultivars Renown with gene *Lr14a* (1937) and Pawnee with *Lr3* (1943) were the first cultivars with race-specific leaf rust resistance genes to be released in Canada and in the US, respectively. Previous to the release of these cultivars, leaf rust Race 9 (International Standard race designation), which is avirulent to both genes, was the most common race throughout both Canada (Johnson 1956) and the US (Johnston et al., 1968). Race 9 declined in frequency during the 1940s and is currently only rarely found on common wheat in North America, occurring almost exclusively on *Ae. cylindrica*. Isolates with virulence to *Lr3* and *Lr14a* rapidly increased in Canada and the US. Subsequently the release of winter and spring wheat cultivars with additional race-specific *Lr* genes has resulted in a highly diverse *P. triticina* population.

The cultivar Mediterranean with *Lr3* is a major ancestor to soft red winter wheat. Genes *Lr9*, *Lr10*, *Lr11*, *Lr12*, *Lr18*, *Lr24*, and *Lr26* have been present in soft red winter wheat cultivars that are grown in the southern and eastern US (Kolmer 2003). The presence of these genes over time has selected leaf rust isolates with corresponding virulences in the southern and eastern US. Many of the early hard red winter wheats were derived from crosses with 'Hope' (*Lr14a*), Pawnee (*Lr3*), and Mediterranean (*Lr3*). Currently, hard red winter wheat cultivars in the southern to central Great Plains region have genes *Lr9*, *Lr16*, *Lr17*, *Lr24*, *Lr26*, *Lr41*, and *Lr42*, and possibly *Lr34*. Hard red spring wheat cultivars in the northern Great Plains have genes *Lr1*, *Lr2a*, *Lr10*, *Lr13*, *Lr16*, *Lr23*, and *Lr34* (Oelke and Kolmer 2004). The selection and increase of leaf rust races with virulence to these genes in the different wheat classes has resulted in distinct regional populations of *P. triticina* virulence phenotypes in the US. In 2005 (Kolmer et al., 2007b) the frequency of isolates with virulence to genes *Lr2a* and *Lr16* were highest in the north central spring wheat region; virulence to genes *Lr11*, *Lr18*, and *Lr26* was highest in the southern soft red wheat region, and virulence to *Lr24* and *Lr41* was highest in the hard red winter wheat region.

Molecular variation

Genetic variation in populations of *P. triticina* has also been examined using various types of genetic markers. Molecular markers have the attribute of being neutral and thus not directly selected, as virulence to specific resistance genes is. Molecular markers can be used to assess the underlying genetic variation among isolates within and between populations, providing further insight into the genetic relationships between different populations. In Canada phenotypes of *P. triticina* that were identical or closely related for virulence, had identical or highly related random amplified polymorphic DNA (RAPD) phenotypes (Kolmer et al., 1995). Major groups of *P. triticina* isolates could be determined based on either virulence polymorphism or RAPD polymorphism since there was a significant correlation between the two types of markers. Isolates of *P. triticina* from international collections were also grouped into distinct groups based on continental region and virulence and RAPD polymorphism (Kolmer and Liu 2000). Park et al. (2000) showed that multiple isolates of the same virulence phenotype of *P. triticina* from different countries in western Europe also had identical RAPD phenotypes. The relationship between virulence phenotype and molecular polymorphism is maintained since *P. triticina* reproduces throughout the world almost exclusively by asexual urediniospores. In an experimental population of *P. triticina* derived from aeciospores, the disequilibria between individual virulence and RAPD markers was often eliminated or reduced (Liu and Kolmer 1998b).

In 1996 isolates of *P. triticina* with virulence to *Lr17* began to increase in the Great Plains region of the US (Long et al., 2000) and Canada (Kolmer 1998). These isolates were selected by the winter wheat cultivar Jagger with *Lr17*, which has been widely grown throughout Texas, Oklahoma, Kansas, and Nebraska. The isolates with *Lr17* virulence were unique in that they were also virulent to *Lr3bg* and *LrB*, and avirulent to *Lr28*. By 2001 these isolates had become widespread in almost all wheat growing regions of North

America and were the most common virulence phenotypes in the US. Further analysis with (amplified fragment length polymorphism AFLP) markers (Kolmer 2001a) indicated that the isolates with *Lr17* virulence had very distinct molecular phenotypes compared to all other isolates in North America. This indicated that the isolates with *Lr17* virulence were most likely introduced to the Great Plains region from either Mexico or the Pacific Northwest and were not derived by mutation from the previously existing population. New virulence phenotypes of *P. triticina* were also introduced to Australia in the mid-1980s (Park et al., 1995). In recent years a virulence phenotype of *P. triticina* with virulence to many durum cultivars has been found in France, Spain, Mexico, Argentina, and Chile (Singh et al., 2004; Ordoñez and Kolmer 2007b). This virulence phenotype may have had a single origin and subsequently spread to the other durum producing regions.

Recently locus-specific microsatellite or simple sequence repeat (SSR) markers have been developed for *P. triticina* (Duan et al., 2003; Szabo and Kolmer 2007). These markers can be used to determine molecular genotypes of *P. triticina* since heterozygotes can be distinguished from homozygotes. The SSR markers have been used to differentiate *P. triticina* populations in Central Asia (Kolmer and Ordoñez 2007) and to describe genetic diversity in *P. triticina* populations in France (Goyeau et al., 2007). These locus-specific markers will be extremely valuable for assessing genetic variation in *P. triticina* and patterns of migration between populations in different continental regions.

Leaf rust resistance in wheat

Race-specific resistance

The tremendous amount of genetic variation for virulence in *P. triticina* populations combined with the ability of urediniospores to be wind-disseminated over thousands of kilometers has made breeding for stable leaf rust resistance in wheat a continually challenging task. Time and again wheat cultivars with a single race-specific gene for leaf rust resistance have been quickly rendered susceptible because of the selection and increase of virulent leaf rust races. In the southeastern states since the mid-1970s, *Lr9* derived from *Ae. umbellulata*, *Lr11* derived from Hussar wheat, and *Lr1* derived from various common wheats, have been widely used in soft red winter wheat cultivars and have selected phenotypes of *P. triticina* with virulence to these genes (Fig. 5.1a). Currently *Lr1* and *Lr11* do not provide effective resistance, and cultivars with *Lr9* are moderately resistant, but this resistance would quickly erode if cultivars with *Lr9* were grown over a larger area.

In Texas and Oklahoma isolates with virulence to *Lr1* quickly increased in the late 1970s and early 1980s after the release of cultivars with this gene (Fig. 5.1b). Virulence to *Lr24* appeared shortly after the release of the hard red winter wheat cultivar Agent with *Lr24* in 1971. By the mid-1970s virulence to *Lr24* was common in the winter wheat region of the Great Plains. In the mid-1980s the cultivar Siouxland with *Lr24* and *Lr26* was widely grown from Texas to South Dakota. Isolates with virulence to *Lr24* and *Lr26* increased up to the early 1990s. Starting in 2002 isolates with virulence to *Lr24* increased again due to widespread cultivation of 'Jagalene', with *Lr24*. Although *Lr24* was originally derived from *Ae. elongatum*, and *Lr26* from *Secale cereale*, the nonwheat origin of both genes did little to enhance their durability of resistance. The cultivar Jagger, released in the mid-1990s with *Lr17*, selected isolates with virulence to this gene, as these reached nearly 90% of isolates in Texas and Oklahoma in 2001. Isolates of *P. triticina* with virulence to *Lr41*, derived from *Ae. tauschii*, were found even before winter wheat cultivars with this gene were released in the late 1990s in the southern Great Plains. Isolates with virulence to *Lr41* have increased such that cultivars with this gene ('Thunderbolt', 'Overley', and 'OK Bullet') are now susceptible to leaf rust.

Selection of isolates for virulence to specific resistance has also occurred in the spring wheat region of Minnesota, North Dakota, and South Dakota, even though leaf rust does not frequently overwinter in this area. Cultivars with *Lr1* and

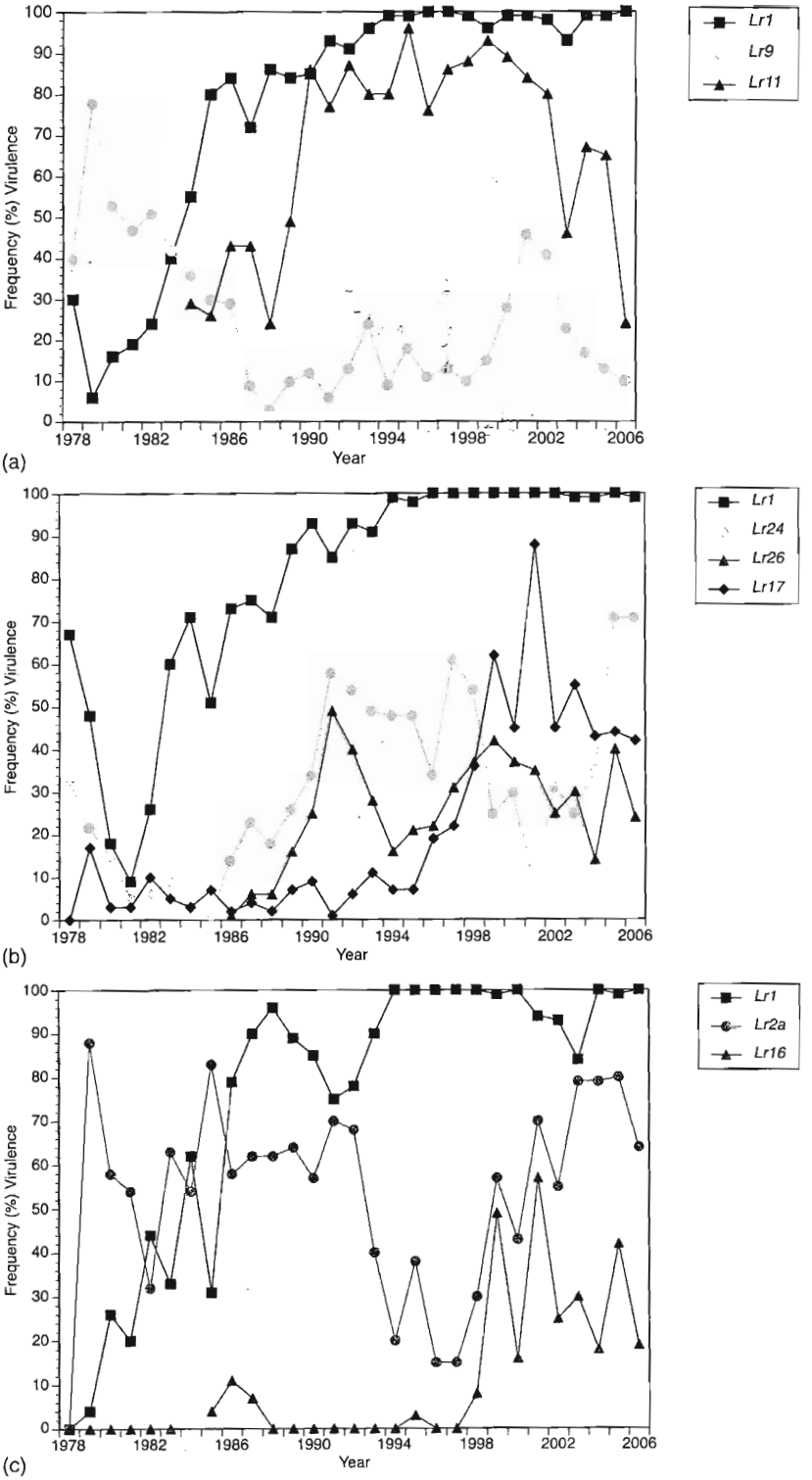

Fig. 5.1 Changes in frequency (%) of *Puccinia triticina* isolates with virulence to specific leaf rust resistance genes in different wheat growing regions of the US. (a) Soft red winter wheat region—southeastern states. (b) Hard red winter wheat region—Texas and Oklahoma. (c) Hard red spring wheat region—Minnesota, North Dakota, and South Dakota.

Lr2a were widely grown in this region starting in the mid-1970s. Isolates with virulence to both genes increased rapidly and were over 65% in the early 1990s (Fig. 5.1c). Virulence to *Lr16* has increased from the mid-1990s since many current spring wheat cultivars have this gene. In Australia cultivars with either combination of genes *Lr13*, *Lr23*, and *Lr34* or genes *Lr1*, *Lr13*, and *Lr23* are currently resistant to leaf rust (Bariana et al., 2007). Cultivars with various combinations of *Lr13*, *Lr24*, *Lr34*, and *Lr37* are considered moderately susceptible to leaf rust.

Leaf rust resistance genes up to *Lr60* have been designated (McIntosh et al., 2007). Genes *Lr1* (Cloutier et al., 2007), *Lr10* from common wheat (Feuillet et al., 2003), and *Lr21* from *Ae. tauschii* (Huang et al. 2003) have been sequenced. The three genes have NBS-LRR regions typical of resistance genes with isolate specificity. Genes *Lr1* and *Lr10* are widely ineffective, and *Lr21* has provided effective resistance in spring wheat cultivars in the US and Canada. Isolates with virulence to *Lr21* would be expected to increase if this gene was used in a winter wheat cultivar in the US. Many of the other *Lr* genes for which virulent isolates of *P. triticina* have not been found have also not been widely used in wheat improvement programs.

Durable leaf rust resistance in wheat

The development of wheat cultivars with high levels of effective durable resistance will depend on genes that confer nonspecific resistance or gene combinations that have proven to be effective over time. The cultivar Frontana released in Brazil in 1946 has been a valuable source of durable nonspecific leaf rust resistance. Dyck et al. (1966) backcrossed leaf rust resistance from Frontana into Thatcher. Backcross lines with the adult-plant gene *Lr13* were characterized, yet none of the lines was as resistant as Frontana because an additional gene was needed to recover the original resistance in Frontana. Dyck and Samborski (1982) characterized gene *LrT2* in a group of wheat cultivars that included 'Terenzio' and Frontana. Later, *LrT2* was determined to be the additional gene in Frontana, designated as *Lr34*, and mapped to chromosome 7DS (Dyck 1987). Singh and Rajaram (1992) determined that Frontana also carries other genes besides *Lr34* that condition adult-plant leaf rust resistance. Frontana was used as a leaf rust resistant parent in spring wheat programs in Minnesota and also at CIMMYT. The Minnesota cultivar Chris was derived from crosses with Frontana and released in 1966 as the first spring wheat in the US to have *Lr34*. The CIMMYT cultivars Penjamo 62, Lerma Rojo, and Nainari 60 also had *Lr34*.

Wheat lines and cultivars with *Lr34* optimally express leaf rust resistance in the adult-plant stage. Isolates of *P. triticina* with complete virulence to lines with *Lr34* have not been found in North America (Kolmer et al., 2003, McCallum and Seto-Goh 2006), despite the presence of wheat cultivars with *Lr34* for over 40 years. In field plots lines with only *Lr34* can have moderate to high levels of leaf rust severity, although these can usually be distinguished from completely susceptible lines if leaf rust readings are made when known susceptible lines are at near-terminal severity (Color Plate 9a). Lines with *Lr34* can also express resistance in seedling plants at cooler temperatures (Singh 1992b; Pretorius et al., 1994). The presence of *Lr34* enhances the response of other effective resistance genes in the same wheat genotype (German and Kolmer 1992). The presence of *Lr34* is also associated with a distinctive leaf-tip necrosis (Singh 1992a) that can vary between genotypes and environments. Wheat cultivars with other *Lr* genes combined with *Lr34* are often more resistant than lines with only *Lr34* or the other genes singly. Spring wheat cultivars with combinations of *Lr13*, *Lr16*, and *Lr34* were highly resistant in Canada (Samborski and Dyck 1982; Liu and Kolmer 1997) and the US (Ezzahiri and Roelfs 1989).

Diagnostic molecular markers closely linked to *Lr34* have been developed (Bossolini et al., 2006; Lagudah et al., 2006) that will greatly simplify selection of breeding materials with *Lr34*. In a survey of wheat classes in the US using the *Lr34* marker *csLV34*, the allele associated with the presence of *Lr34* was completely absent in soft

Table 5.1 US wheat cultivars tested for the presence of *csLV34* alleles and *Lr34*.

Class	Cultivar	*csLV34*[a]	*Lr34*[b]	Class	Cultivar	*csLV34*	*Lr34*
Soft red winter	Fultz	–		Hard red spring	Thatcher	–	–
	Monon	–			Chris	+	
	Knox62	–			Era	+	+
	Arthur	–			Waldron	–	
	Caldwell	–	–		Marshall	+	
	CK 9663	–	–		Wheaton	+	+
	Pioneer 26R61	–	–		Butte 86	-	
	Saluda	–			Grandin	+	+
	McCormick	–			Norm	+	+
	NC Neuse	–			Russ	-	
	Pocahontas	–			Oxen	+	
	SS550	–			BacUp	–	
					Keene	–	
					HJ98	–	
Hard red winter	Triumph	–			Reeder	–	
	Bison	–			Alsen	+	+
	Scout 66	–			Briggs	+	+
	Siouxland	–			Steele	–	
	Victory	–			Oklee	+	
	Sturdy	+	+		Glenn	–	
	Ogallala	+					
	Duster	+		Pacific Northwest spring wheat	Alpowa	–	
	Santa Fe[c]	+			Alturas	–	
	Fuller[c]	+			Hank	-	
	2137	–			Hyak	–	
	Overley	–			Jefferson	–	
	Deliver	+			Madsen	–	
	Endurance	–			Nick	–	
	Cutter	–			Scarlet	–	
	TAM 107	–			Stephens	–	

Source: Adapted from Kolmer et al. (2008).
[a]+ = allele associated with *Lr34*; – = allele associated with lack of *Lr34*.
[b]+ = genetic analysis indicated presence of *Lr34*; – = *Lr34* not present; blank indicates not tested.
[c]Winter wheat cultivars derived from 'Jagger' may lack *Lr34* yet have the *csLV34* allele associated with *Lr34*.

red winter wheat (Table 5.1). The allele associated with *Lr34* was most common in spring wheat cultivars bred for the northern Great Plains, was present at low frequency in older and current hard red winter wheat cultivars, and was not present in cultivars from the Pacific Northwest.

The CIMMYT cultivar Pavon 76 has provided an additional source of nonspecific adult-plant leaf rust resistance. The adult-plant resistance gene *Lr46* in Pavon 76 is on chromosome 1BL (Singh et al., 1998). Gene *Lr46* is likely present in CIMMYT germplasm that has been selected for adult-plant resistance to leaf rust. Cultivars and germplasm with combinations of *Lr34*, *Lr46*, and additional adult-plant resistance can be highly resistant, approaching complete immunity (Singh et al., 2000). Development of diagnostic molecular markers associated with *Lr46* (Rosewarne et al., 2006) will aid in selection of lines with this adult-plant resistance.

Combinations of adult-plant resistance genes with *Lr* genes effective in seedlings can also provide good levels of durable leaf rust resistance. The Minnesota spring wheat cultivar Norm released in 1992 has remained highly resistant to leaf rust. Norm was determined to have genes *Lr1*, *Lr10*, *Lr13*, *Lr16*, *Lr23*, and *Lr34* (Oelke and Kolmer 2005). Genes *Lr1*, *Lr10*, and *Lr13* are

now widely ineffective, but isogenic Thatcher lines with *Lr16*, *Lr23*, and *Lr34* have effective resistance in field plots when compared to the completely susceptible Thatcher (Oelke and Kolmer 2004). Gene *Lr23* is highly temperature-dependent in expression of resistance. In ambient-greenhouse temperatures of 15–25 °C, lines with this gene expressed variable infection types ranging from moderate to large uredinia to small uredinia surrounded by necrosis (Dyck and Johnson 1983). At 25 °C in growth cabinets, lines with *Lr23* expressed very low hypersensitive infection types to US isolates of *P. triticina*. Although *P. triticina* isolates with *Lr16* have been detected in the spring wheat region of the US, cultivars with this gene still have some resistance in field plots (Oelke and Kolmer 2004). 'Knudson', released by AgriPro-Coker in 2002, has also been very resistant and was determined to have *Lr3*, *Lr10*, *Lr13*, *Lr16*, *Lr23*, and *Lr34* (Kolmer and Oelke 2006). Spring wheat genotypes with combinations of *Lr16*, *Lr23*, and *Lr34* have shown good levels of resistance that has not been significantly eroded by virulence changes in the *P. triticina* population.

Additional genes from wheat germplasm that have shown good levels of durable resistance have also been characterized. Barcellos et al. (2000) determined that the Brazilian cultivar Toropi had two genes that conditioned adult-plant leaf rust resistance which were also associated with leaf-tip necrosis. Mishra et al. (2005) determined that the Indian cultivar C 306 had a single adult-plant resistance gene associated with leaf-tip necrosis that was independent of *Lr34*. The landrace-derived cultivars from Uruguay, 'Americano 25e' and 'Americano 44d', were shown to have unique adult-plant resistance genes that were not *Lr34* (Kolmer et al., 2007c). The Canadian spring wheat cultivar AC Taber was determined to have an effective adult-plant resistance gene other than *Lr13* or *Lr34* (Liu and Kolmer 1997). Navabi et al. (2003) estimated two to four effective adult-plant resistance genes were present in five CIMMYT lines.

Quantitative trait loci (QTLs) that affect resistance in adult plants have also been mapped to chromosome regions. Xu et al. (2005a,b) identified QTLs that affected final rust severity, infection rate, infection duration, and latent period on chromosomes 2B, 7BL, and 2DS in the soft red winter wheat germplasm line CI 13227. The Swiss cultivar Forno was determined to have a major QTL for adult-plant resistance on chromosome 1BS and minor regions for resistance on 2DL, 3DL, 4BS, and 5AL (Schnurbusch et al., 2004). These additional sources of adult-plant resistance can be used in wheat improvement programs to diversify germplasm for effective leaf rust resistance.

Association with other disease resistance genes

An intriguing aspect of adult-plant leaf rust resistance in wheat is an association with resistance to other diseases of wheat. Lines with *Lr34* also have adult-plant, nonspecific resistance to stripe rust (caused by *P. striiformis* Westend. f. sp. *tritici* Eriks.) (McIntosh 1992; Singh 1992c). The stripe rust resistance associated with *Lr34* has been designated as *Yr18*. The *Lr34* locus also exhibits a pleiotropic effect on barley yellow dwarf virus reaction (Singh 1993). Spielmeyer et al. (2005) showed that lines segregating for adult-plant resistance to leaf and stripe rust due to *Lr34/Yr18* also had adult-plant resistance to powdery mildew. The 7DS chromosomal region of *Lr34* may condition a generalized nonspecific response that acts against biotrophic wheat pathogens. The adult-plant resistance gene *Lr46* is also associated with nonspecific stripe rust resistance, which has been designated as *Yr29* (Williams et al., 2002). Navabi et al. (2005) also showed that adult-plant resistance genes other than *Lr34/Yr18* conditioned resistance to both leaf rust and stripe rust. Since adult-plant resistance to leaf and stripe rust was highly associated, selection of germplasm with resistance to both rust diseases could be accomplished by testing for resistance to only one disease.

The Minnesota spring wheat cultivar Thatcher was released in 1935 on the basis of resistance to stem rust and good breadmaking quality characteristics (Hayes et al., 1936). Many subsequent

spring wheat cultivars in Canada and the US (Kolmer et al., 1991) have had stem rust resistance derived from Thatcher. Dyck (1987) determined that isogenic lines of Thatcher with *Lr34* had better seedling and adult-plant resistance to stem rust than Thatcher. The Canadian wheat cultivars Roblin (Dyck 1993) and Pasqua (Liu and Kolmer 1998a) have some Thatcher in their pedigrees and also have *Lr34*. In crosses derived from both Roblin and Pasqua, progeny lines with *Lr34* have been associated with higher stem rust resistance. A stem rust resistance suppressor on chromosome 7DL (Kerber and Green 1980) is present in Thatcher and other Thatcher-derived lines. Kerber and Aung (1999) determined that Thatcher lines with *Lr34* had the same stem rust response as did Thatcher lines nullisomic for 7DL that lacked the suppressor. Hence *Lr34* appeared to inactivate the stem rust resistance suppressor.

Gavin-Vanegas et al. (2007) determined that progenies derived from a Thatcher line with *Lr34* crossed with a stem rust susceptible line segregated for two effective stem rust resistance genes in adult plants in the absence of *Lr34*, and segregated for three genes when all progeny lines were fixed for *Lr34*. In this study segregation of resistance to stem rust races that had high infection types to seedlings of Thatcher, but low infection types to Thatcher lines with *Lr34*, was strongly correlated with segregation of stem rust resistance in adult plants to a mixture of stem rust races. The presence of *Lr34* allowed the expression of additional stem rust resistance gene(s) in Thatcher that were most likely derived from 'Iumillo' durum.

Thatcher and the cultivar Chris had low seedling infection types to the stem rust isolate Ug-99, which has appeared in eastern Africa and is highly virulent to many US and CIMMYT wheat cultivars (Jin and Singh 2006). The stem resistance in Chris is most likely due to the presence of stem rust resistance derived from Thatcher that is enhanced by the presence of *Lr34*. Thatcher lines with *Lr34* show good resistance to Ug-99 in field plots in Kenya. Since so few current wheat cultivars have effective resistance to Ug-99, the Thatcher stem rust resistance enhanced by *Lr34* may be an important future source of stem rust resistance.

Leaf rust resistance in durum wheat

Cultivated durum wheat is generally highly resistant to the *P. triticina* isolates found on common wheat (*T. aestivum* L.). Genes *Lr14a* derived from 'Yaroslav' emmer (*T. turgidum* ssp. *dicoccum*) and *Lr23* derived from 'Gaza' durum are present in common wheat. Genes *Lr10* and *Lr33* (Dyck 1994) may also be present in durum wheat. Genetic studies of leaf rust resistance in durum wheat to *P. triticina* isolates from common wheat have often indicated the presence of one to two seedling resistance genes that were expressed in a recessive or dominant manner (Statler 1973; Zhang and Knott 1990, 1993). Phenotypes of *P. triticina* that are virulent to durum cultivars were described in Mexico (Singh 1991). Using isolates collected from durum wheat, Singh et al. (1993) determined that a collection of CIMMYT durum cultivars varied for seedling resistance and adult-plant resistance genes. The durum cultivar Altar C84 and three other durum cultivars had a single gene that conditioned seedling resistance, in addition to two adult-plant resistance genes.

A severe epidemic of leaf rust on durum wheat occurred during 2001–2003 in northwest Mexico (Singh et al., 2004). New phenotypes of *P. triticina* had emerged that were highly virulent to Altar C84. Increased levels of leaf rust infections were also noted in France (Goyeau et al., 2006) and Spain (Martinez et al., 2005). Isolates of *P. triticina* from durum wheat collected during 2002–2004 in France, Spain, Mexico, Argentina, and Chile were highly similar for virulence to the *Lr* genes in the Thatcher isogenic lines, a collection of durum cultivars (Ordoñez and Kolmer 2007b), and for molecular SSR variation (Ordoñez and Kolmer 2007a), suggesting a recent common origin. Singh et al. (2004) identified CIMMYT durum germplasm that was resistant to the new *P. triticina* durum virulent phenotypes in Mexico. The resistant lines were also resistant in the other

countries where higher infections of leaf rust on durum wheat had occurred.

Herrera-Foessel et al. (2005) described up to five genes in nine CIMMYT durum lines that conditioned resistance to the *P. triticina* race that was virulent to Altar C84. Herrera-Foessel et al. (2007) determined that one of these genes mapped to the *Lr3* locus on chromosome 6B, and a second resistance gene was closely linked to the *Lr3* locus.

WHEAT STRIPE RUST

Distribution and epidemiology

Stripe rust is an important disease of wheat worldwide. The disease has been reported in more than 60 countries and has caused yield losses in Africa, Asia, Australia, New Zealand, Europe, North America, and South America (Stubbs 1985; Chen 2005). In the US the disease has been most common in states west of the Rocky Mountains since the late 1950s but has become increasingly frequent in the eastern and midwestern states since 2000 (Chen 2005). In recent years significant losses in wheat due to stripe rust have been reported in California, Oregon, Washington, Idaho, Montana, Colorado, Texas, Oklahoma, Kansas, Nebraska, South Dakota, Louisiana, Arkansas, Missouri, Alabama, and Georgia.

Stripe rust infection can occur at any growth stage when green plant tissue is available. The first visible symptom of infection appears as chlorotic spots that resemble viral symptoms. Uredinia of stripe rust are yellow to orange in color; thus the disease is commonly called yellow rust. Stripe rust uredinia, 0.3–0.5 mm by 0.5–1.0 mm, are much smaller than uredinia of stem rust and leaf rust. Uredinia can form on both sides of leaves but are more abundant on the upper surface (Color Plate 10). Uredinia can also form on leaf sheaths, glumes, awns, and on immature green kernels. Uredinia form in patches around infection sites on seedlings and are arranged in stripes between leaf veins on adult plants. Depending on the level of plant resistance, uredinia can be surrounded by chlorosis or necrosis on seedling leaves. On adult-plant leaves of highly susceptible plants, uredinia continue to develop in stripes from the initial infection sites, without necrosis or chlorosis. On resistant cultivars, necrotic stripes develop from the initial infection sites. Responses on resistant wheat genotypes vary from no visible symptom to various sizes or lengths of necrotic patches or stripes with varying amounts of sporulation (Color Plate 10b). Uredinia erupt to release urediniospores. Each uredinium can produce thousands of urediniospores over a period of days. Urediniospores are spherical, 15–20 µm in diameter, and echinulate.

Urediniospores are dispersed mainly by wind but also can be spread by insects, animals, and humans. A minimum of three hours of dew formation on the plant surface is needed for urediniospores to germinate and infect plants (Rapilly 1979). The optimum temperatures for spore germination are 10–12 °C, and the minimum temperature for germination is just above 0 °C (Newton and Johnson 1936). Urediniospores do not germinate well when the temperature is above 20 °C. Germ tubes penetrate into plant tissue through stomata without forming appressoria as in other wheat rusts (Marryat 1907; Allen 1928). After a germ tube passes through a stoma, it forms a substomatal vesicle, from which branched hyphae grow intercellularly. Haustoria are formed from the hyphae and grow into host cells. The uredinia are produced from intercellular hyphae and emerge on the plant surface. Under optimum temperature conditions (13–16 °C), it takes 12–13 days from initial infection to sporulation of new uredinia (Hungerford 1923). Stripe rust will continue to produce new uredinia and urediniospores in stripes further up and down the leaf from the initial site of infection.

Urediniospores are the sole initial inoculum source for the stripe rust pathogen. Initial inoculum can be local and/or from outside a region depending upon climatic conditions that influence survival of stripe rust during the summer or winter. In the Pacific Northwest of the US and the adjacent area of British Columbia and Alberta in Canada, the stripe rust fungus is able to survive during the summer and winter most years. In this

region, urediniospores can be found at almost any time of year, especially west of the Cascade Mountains where the mild winters and cool summers are favorable to stripe rust survival. The cool night temperatures and dry conditions during summer in the major wheat growing areas east of the Cascade Mountains allow urediniospores to retain viability for extended periods. Urediniospores that are produced in late summer and early fall from spring wheat fields, volunteer plants, and grasses infect the winter wheat crops. The fungus overwinters in plant tissue as mycelia and often as viable urediniospores on plants (Hungerford 1923). According to Rapilly (1979), stripe rust can survive temperatures as low as −10 °C. Snow cover provides favorable conditions for both the wheat plants and stripe rust to survive the winter. Heavy local survival of the stripe rust pathogen over the winter will lead to early epidemic development in the spring. Stripe rust forecasting models based on December and January temperatures have been successfully used in the Pacific Northwest to predict stripe rust epidemics (Coakley et al., 1982, 1984; Line 2002; Chen 2005).

Stripe rust can also overwinter in regions with cold winters. Stripe rust can be endemic in the Gallatin Valley and Flathead Lake area in Montana (Sharp and Heln 1963) where the fungus has overwintered. In 2006, stripe rust severity was unusually high in Alberta but light in the US Pacific Northwest, indicating winter survival of the rust in Alberta. In North Dakota and Minnesota stripe rust generally does not overwinter. However, in 2006, stripe rust was observed at St. Paul, Minnesota, on April 26, a month earlier than the first observation of the disease at Pullman, Washington, and also much earlier than reports of the disease in Missouri, Illinois, and Indiana, which indicated that the pathogen survived the 2005–2006 winter in Minnesota. The cold winters and hot summers in the northern Great Plains region usually limits stripe rust development in this region.

In California stripe rust regularly overwinters and survives the summer on wheat crops and grasses at high elevations along the coast (Tollenaar and Houston 1966), and on wheat grown as forage or cover crops. Survival of stripe rust on volunteer wheat plants in irrigated fields resulted in a widespread epidemic in California in 1974 (Line 1976). Northeastern California, where stripe rust oversummers, may be a source of inoculum for southern Oregon, southern Idaho, and northern Utah, as well as for central California. Stripe rust inoculum is exchanged among California, Arizona, New Mexico, and northwestern Mexico.

The stripe rust pathogen generally does not survive the summers in the Great Plains, Mississippi Valley, and the southeastern states due to extended hot and humid conditions and the long period between harvest and planting of wheat crops. The late-planted wheat crops at high elevations in Mexico may provide initial inoculum for infection in the fall in the southern Great Plains (Line 2002). Wheat crops in the Rocky Mountain areas from Colorado to western Texas may contribute stripe rust inoculum to the Great Plains. Stripe rust infections occur soon after emergence of wheat in the late fall and early winter in Texas, Louisiana, Arkansas, and Mississippi. If stripe rust occurs, it usually develops slowly in the winter and at a faster rate in the early spring, providing inoculum for areas further north and east. The scope and severity of epidemics depend on inoculum in the south, on wind directions, and on temperature and moisture conditions in the region east of the Rocky Mountains. Stripe rust has been reported in New York, but has never caused significant damage north of Ohio and Virginia.

Origin and historical importance

Stripe rust of wheat is a long established disease in Asia and Europe (Stubbs 1985). Stripe rust was first recognized in the US in 1915 in Arizona (Carleton 1915). However, examination of herbarium specimens has indicated that the disease was present in the western US before 1892 (Humphrey et al., 1924) and possibly occurred in California in the 1700s (Smith 1961). The Caucasus region is the presumed origin of stripe rust (Stubbs 1985; Line 2002).

From 1957 to 2005, the US experienced four waves of regional epidemics of stripe rust. The

first was from 1958 to 1961, and the epidemics were concentrated in the Pacific Northwest and California. Severe epidemics of wheat stripe rust occurred in 1960 and 1961 in Washington (Hendrix 1994) and in 1961 in Oregon (Shaner and Powelson 1971). Stripe rust also was severe in Idaho and Montana (Pope et al., 1963) in those years. In California, the yield losses were estimated at 28%–56% in the Sutter Basin north of Sacramento (Tollenaar and Houston 1966).

The second period of stripe rust epidemics occurred from 1974 to 1978. In 1974, California had an 8% yield loss in wheat production. In 1976, yield losses were 17% in Washington, 13% in Oregon, and 11% in Idaho. The third wave of epidemics occurred from 1980 to 1984. In 1980 and 1981, stripe rust epidemics were widespread in the Pacific Northwest, and yield losses in Washington were estimated to be 13% in 1980 and 11% in 1981. Oregon and Idaho had 5%–9% yield losses during the same period. Yield losses were reduced in 1980–1981 because of the widespread use of fungicides. From the mid-1980s to the late 1990s, yield losses caused by stripe rust were reduced due to widely grown resistant cultivars in the Pacific Northwest and California and the use of fungicides. Yield losses remained below 5% in the western US.

The most recent stripe rust epidemics occurred from 1999 to 2005. In 1999, stripe rust caused a 7% yield loss in California but was not severe in other states. In 2000, stripe rust was widespread and caused severe damage in the south central states with a 7% loss in Arkansas. In 2001 losses in Kansas and Colorado were estimated to be 7% and 8%, respectively. In 2003 states with major yield losses were Kansas (11%), Nebraska (10%), and California (21%). In 2005 stripe rust was very widespread and occurred in more than 30 states, with significant yield losses in Kansas (8%), Texas (15%), Oklahoma (5%), Nebraska (4%), California (5%), Arkansas (5%), and Louisiana (5%).

Taxonomy, life cycle, and host range

Stripe rust on cereal crops and grasses is caused by different *formae speciales* of *P. striiformis*, a basidiomycete rust fungus. The disease was first described by Gadd in 1777 (Eriksson and Henning 1896). Schmidt (1827) named the stripe rust fungus as *Uredo glumarum*. Westendorp (1854) used *P. striaeformis* for stripe rust collected from rye. Fuckel (1860) described stripe rust as *P. straminis*. *Puccinia glumerum*, as described by Eriksson and Henning (1894), was used as the name for the stripe rust fungus until Hylander et al. (1953) revived the name *P. striiformis* Westend.

Since stripe rust pathogens on different cereal crops and grasses are separated into different *formae speciales* (Eriksson 1894), *P. striiformis* Westend. f. sp. *tritici* Eriks. is considered the valid name for the stripe rust pathogen infecting wheat. In addition to wheat stripe rust, Eriksson described stripe rusts on barley as f. sp. *hordei*, on rye as f. sp. *secalis*, on *Elymus* spp. as f. sp. *elymi*, and on *Agropyron* spp. as f. sp. *agropyron*. Later, three more *formae speciales* were proposed: *P. striiformis* f. sp. *poae* on Kentucky bluegrass (*Poa pratensis* L.) (Britton and Cummins 1956; Tollenaar 1967), f. sp. *dactylidis* on orchardgrass (*Dactylis glomerata* L.) (Manners 1960; Tollenaar 1967), and f. sp. *leymi* on *Leymus secalinus* (Georgi) Tzvel (Niu et al., 1991). More recently, Wellings et al. (2004) considered stripe rust on *Hordeum* spp. in Australia to be a new *formae specialis*, different from both *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei*. Not all these *formae speciales* are equally and clearly separated by host specialization. Wheat stripe rust mostly infects wheat but can infect some barley cultivars, while barley stripe rust can infect some wheat cultivars. However, stripe rust of barley does not infect bluegrass, and bluegrass stripe rust does not infect wheat or barley (Chen et al., 1995).

Puccinia striiformis has a hemicyclic life cycle of urediniospores, teliospores, and basidiospores. Teliospores are formed along the sides of uredinia and are the same size and shape as urediniospores, but have black cell walls. Teliospores form more rapidly and more abundantly under hot and humid conditions than under cool and dry conditions. The barley stripe rust fungus (*P. striiformis* f. sp. *hordei*) is more likely to produce telia than *P. striiformis* f. sp. *tritici*. Mature teliospores are two-celled and become diploid with one nucleus

in each cell. Teliospores germinate readily to produce haploid single-celled basidiospores that are unable to infect cereals and grasses. Despite early intensive studies, an alternate host for *P. striiformis* was not found (Eriksson and Henning 1894; Mains 1933; Tranzschel 1934; Straib 1937; Hart and Becker 1939). Alternate host plants for *P. striiformis* may not exist (Hassebrauk 1970), or with the short dormancy of teliospores and readily produced basidiospores, the alternate host may escape infection (Wright and Lennard 1978; Rapilly 1979). In the absence of an alternate host, the teliospores and basidiospores are not functional in the *P. striiformis* life cycle.

Puccinia striiformis f. sp. *tritici* is able to infect a broader range of grass species than stem rust or leaf rust. Hassebrauk (1965) listed about 320 grass species of 50 genera in the Gramineae family that were naturally or artificially infected by wheat stripe rust. The most susceptible genera are *Aegilops*, *Agropyron*, *Bromus*, *Elymus*, *Hordeum*, *Secales*, and *Triticum*. Some *Aegilops* spp., especially *Ae. cylindrica* (common goatgrass), are highly susceptible to stripe rust. These grasses near wheat fields can contribute to stripe rust development as sources of early inoculum, but have limited roles in rust survival because they mature earlier than wheat crops. The importance of grasses as hosts in wheat stripe rust epidemics may vary from region to region. Generally, wild grasses play a less important role than wheat crops and volunteer wheat in the initiation, development, spread, and survival of stripe rust. However, grasses can serve as reservoirs in maintaining diversity of stripe rust races.

Genetic variation in *Puccinia striiformis* f. sp. *tritici*

Virulence variation

The wheat stripe rust pathogen is highly variable for virulence to stripe rust resistance genes in wheat. In 2000, 42 races of wheat stripe rust were found in the US (Chen et al., 2002). Early studies of physiological specialization in *P. striiformis* f. sp. *tritici* used several sets of differential cultivars, different inoculation methods, and varying environmental conditions, which compromised continuity from year to year or among investigators. The differential set used in Europe did not identify important races in the US (Line 2002). Line et al. (1970) first developed a uniform system to identify and describe races of the wheat stripe rust pathogen in the US. The current differential set includes 20 wheat cultivars and lines with various combinations of genes *Yr1*, *Yr2*, *Yr3a*, *Yr4a*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr10*, *Yr17*, *Yr19*, *Yr20*, *Yr21*, and other genes currently undesignated (Chen et al., 2002). A total of 126 races of *P. striiformis* f. sp. *tritici* has been identified since the establishment of this US differential set (Line and Qayoum 1992; Chen et al., 2002, 2007; Chen 2005, 2007).

The emergence of the majority of *P. striiformis* f. sp. *tritici* races in the US can be related to selection by wheat cultivars with race-specific resistance. Line and Qayoum (1992) discussed races selected by widely grown wheat cultivars in the US before 1987. The appearance and rapid development of races with virulence to seedlings of the cultivar Express caused yield losses in California in 1999. The widely grown cultivars RSI 5, Bonus, and Summit in California apparently selected races with virulence to *Yr1*, *Yr9* (on the 1RS.1BL wheat–rye translocation), and the seedling resistance in Express. When these cultivars were released they were highly resistant to the previously detected races but became susceptible within a few years under commercial production. The average length of time of a cultivar with race-specific *Yr* genes retaining effective stripe rust resistance is 3.5 years (Chen 2005).

The soft winter wheat cultivar Stephens has been very popular in the Pacific Northwest since its release in 1978. Wheat stripe rust races with virulence to seedlings of Stephens were first identified in 1977 (Line and Qayoum 1992) and since then have been predominant in this region. Since 2004, races with virulence to Stephens and virulence to *Yr9* have been predominant throughout the US (Chen 2007). The widely grown cultivars Jagger and Jagalene, which likely have stripe rust resistance from Stephens, may have contributed to the widespread occurrence of these races in the

Great Plains region. Because Stephens, Jagger, and Jagalene also have high-temperature adult-plant (HTAP) resistance, these cultivars have not been severely infected. However, races with *Yr9* virulence caused severe epidemics from 2000 to 2005 on susceptible cultivars that lacked any effective resistance.

Other epidemics of stripe rust caused by the introduction of new races are well documented. The best example includes the introduction of the wheat stripe rust pathogen to eastern Australia in the late 1970s (Wellings and McIntosh 1987; Wellings 2007), western Australian in 2002 (Wellings et al., 2003), and South Africa in the mid-1990s (Pretorius et al., 1997). These long-distance introductions of the wheat stripe rust pathogen from one continent to another were thought to be caused by inadvertent human activities. Once present in a new continent, stripe rust spreads quickly to neighboring countries. This was seen in the spread of the barley stripe rust pathogen from Colombia to other South American countries and to Mexico and the US from 1975 to 1991 (Chen et al., 1995). The races with virulence to *Yr9* which have rendered many cultivars with the 1RS.1BL wheat–rye translocation highly susceptible to stripe rust were likely an introduction from outside the US.

Since the group of races with *Yr9* virulence appeared in the US in 2000, numerous new races with additional virulence have subsequently been found. In 2000, the most common races were virulent to the differential lines 'Lemhi', 'Heines VII', 'Lee', 'Fielder', 'Express', 'AVS/6*Yr8', 'AVS/6*Yr9', 'Clement', 'Compair', and 'Produra'. Since then a large number of races with additional virulence to the differential cultivars Tres, Stephens, Yamhill, and Chinese 166 have been detected (Fig.5.2). Races with virulence to Stephens, Yamhill, *Yr8*, and *Yr9* have been the most common races in the US since 2003. In the Pacific Northwest, races with virulence to the cultivars Moro (*Yr10* and *YrMor*) and Paha were detected in 2005. These new races have caused several previously resistant cultivars to become susceptible, or they have reduced the resistance level in cultivars with race-specific resistance and non-race-specific HTAP resistance (Chen 2005, 2007).

Molecular variation

Chen et al. (1993) used RAPD markers to examine molecular variation in *P. striiformis* f. sp. *tritici*. DNA polymorphism was detected among races and among single-spore isolates within races.

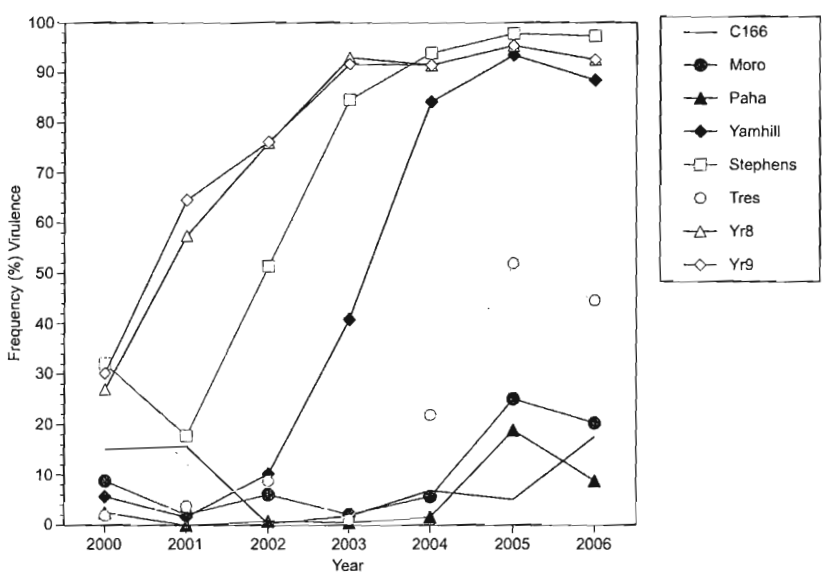

Fig. 5.2 Frequency (%) of isolates of *Puccinia striiformis* f. sp. *tritici* with virulences to selected wheat genotypes in the US from 2000 to 2006. 'Chinese 166' (C166) has resistance gene *Yr1*; 'Moro', *Yr10* and *YrMor*; 'Paha', *YrPa1*, *YrPa2*, and *YrPa3*; 'Yamhill', *Yr2*, *Yr4a*, and *YrYam*; 'Stephens', *Yr3a*, *YrS*, and *YrSte*; and 'Tres', *YrTr1*, and *YrTr2*. The *Yr8* and *Yr9* near-isogenic lines are in the Avocet-susceptible (AVS) background.

Races with virulence to *Yr1* differed for RAPD markers from races avirulent to *Yr1*, indicating different origins of the two groups of races. More recently, Markell et al. (2004) used AFLP markers to study the new wheat stripe rust races in the south central states of the US. The new group of races that was collected after 2000 was virulent to *Yr8* and Yr9 and was clearly different for AFLP genotype compared with races collected before 2000. The results, together with virulence data (Chen et al., 2002; Chen 2005, 2007), indicate that the new group of races was introduced to the US.

In Europe, wheat stripe rust isolates with identical virulences and AFLP phenotypes have been found in the UK and Denmark (Hovmoller et al., 2002). A single population of *P. striiformis* f. sp. *tritici* is present in northwest Europe with regular migration of the rust from the UK to Germany and France. In France two distinct populations of *P. striiformis* f. sp. *tritici* in the north and south of the country were described based on AFLP and virulence variation (Enjalbert et al., 2005).

Stripe rust resistance in wheat

Working with stripe rust of wheat, Biffen (1905) was the first to show that disease resistance in plants was inherited according to Mendel's laws. His research provided the scientific basis for breeding resistant cultivars to control stripe rust and other plant diseases. Much of the early research on resistance and genetics of stripe rust resistance in wheat was conducted in Europe, which was reviewed by Röbbelen and Sharp (1978). Lupton and Macer (1962) determined the genetics of stripe rust resistance in seven wheat genotypes and used the *Yr* symbols to designate resistance genes in wheat.

Breeding for stripe rust resistance in wheat in the US had little incentive from insignificant yield losses during the 40-year period after stripe rust was discovered in 1915 (Line 2002). After the widespread stripe rust epidemics in the early 1960s, wheat breeders and geneticists started work on developing stripe rust resistant cultivars. The first cultivar, Gaines, was released with moderate stripe rust resistance in 1961 by Dr. O. Vogel, a USDA-ARS scientist at Pullman, Washington. This cultivar, and the sister-line cultivar Nugaines, laid the foundation for non-race-specific HTAP resistance that is widely distributed among wheat cultivars grown in the Pacific Northwest and other regions. Since the early 1960s, US scientists have conducted studies to identify, characterize, and map stripe rust resistance genes and to develop cultivars with stripe rust resistance (Röbbelen and Sharp 1978; Line 2002; Chen 2005).

Race-specific resistance

Stripe rust resistance genes up to *Yr40* have been designated (McIntosh et al., 2007) and more than 30 genes with provisional designations have been listed. Races of stripe rust with virulence to most of the race-specific Yr genes have been found (Chen 2005). Currently, only a few race-specific *Yr* genes that are expressed in seedling plants are effective against all identified races in the US. These genes include *Yr5*, *Yr15*, *Yr26*, and *Yr40* (Chen 2005; Kuraparthy et al., 2007).

Although *Yr* genes that are race-specific are generally not durable, the deployment of multiple gene combinations either in homogeneous pure-line cultivars, multiline cultivars, or cultivar mixtures can prolong the effective life span of the *Yr* genes. The most successful example of using a multiline cultivar to control stripe rust is Rely, a club wheat cultivar widely grown in the US Pacific Northwest since its release in 1991 (Allan et al., 1993). Cultivar mixtures combine two or more cultivars with different resistance genes in the same field to reduce disease damage (Finckh and Mundt 1992). Recently more than 30% of the wheat acreage in Washington was planted to mixtures of two or three cultivars. This approach reduces yield losses caused by stripe rust; it also minimizes abiotic stresses such as winter and drought damage. Many wheat breeding programs in the US are currently combining resistance genes, such as *Yr5* and *Yr15*, into elite lines to develop new cultivars with a high level of resistance. In Australia, cultivars with combinations of genes *Yr6*, *Yr7*, *Yr17*, *Yr18*, and *Yr33* are common (Bariana et al., 2007).

High-temperature adult-plant resistance

In contrast to race-specific resistance, HTAP resistance cannot be detected in seedling plants. Cultivars with only HTAP resistance are highly susceptible in the seedling stage. At higher temperatures in older plants resistance is most apparent, with more resistance expressed in flag leaves than in lower leaves. The typical HTAP resistance in the spring wheat cultivar Alpowa is shown in Color Plate 10b. The HTAP resistance was first characterized by Line (1972) and further described in later studies (Qayoum and Line 1985; Milus and Line 1986a,b; Chen and Line 1995a,b; Line and Chen 1995).

The HTAP resistance in Gaines and Nugaines has remained effective for more than 40 years under frequent stripe rust epidemic conditions. Gaines and Nugaines were widely grown in the Pacific Northwest until 1981 (Line 2002), when new cultivars with higher levels of resistance were developed. The cultivar Luke with a much higher level of HTAP resistance than Gaines or Nugaines was released in 1970. Currently, wheat cultivars widely grown in the Pacific Northwest, such as Stephens, 'Madsen', 'Eltan', and 'Rod' soft white winter wheat, 'Bauermeister' hard red winter wheat, and Alpowa, Express, and 'Louise' spring wheat have HTAP resistance.

In the Great Plains, the widely grown cultivar Jagger with HTAP resistance has reduced losses to stripe rust in this region since 2000. Cultivars with HTAP resistance have also reduced yield losses compared with susceptible cultivars in California and the south central states where stripe rust develops in early growth stages (Uauy et al., 2005). Cultivars with HTAP resistance can affect the epidemiology of stripe rust, as stripe rust infections on these cultivars produce fewer urediniospores, thus reducing the overall level of stripe rust inoculum. Increased use of cultivars with HTAP resistance will decrease inoculum levels and thus reduce yield losses over a wide geographic area.

To identify wheat lines with HTAP resistance, adult plants should be tested at defined high (10–35 °C) and low (4–20 °C) temperatures with stripe rust races that are virulent to seedlings of the same genotype. Data from field plots sometimes indicate the presence of HTAP resistance, especially in areas where stripe rust infection occurs in the seedling stage. However, a mixture of avirulent and virulent races to specific *Yr* genes can confound these results. Expression of HTAP resistance can be influenced by temperature, by growth stage at which infections first occur, and by the amount of inoculum. Infection type and disease severity are most commonly used to measure HTAP resistance. Infection types are relatively stable, but can be affected by temperature and plant growth stage. Severity levels of cultivars with HTAP resistance tend to vary across regions and years due to differing levels of rust inoculum and temperatures.

The genetics of HTAP resistance has been studied in numerous wheat cultivars. Segregation for stripe rust resistance in adult plants differed from segregation in seedling plants, which indicated that different genes control resistance in different growth stages (Allan et al., 1966). Milus and Line (1986a) determined that Gaines had one gene and Nugaines and Luke had two genes for HTAP resistance. Gaines and Nugaines had a gene in common, but the HTAP genes in Luke were different. Chen and Line (1995a,b) determined two to three genes or QTLs conferred HTAP resistance in 'Druchamp' and Stephens. The cultivars differed for HTAP resistance genes and for race-specific resistance genes. In these studies, HTAP resistance genes in all cultivars were partially recessive and there was an additive effect when two or more genes were present in a genotype.

Several genes that condition adult-plant or HTAP resistance have been reported (with their known cultivar source): *Yr11* ('Joss Cambier'), *Yr12* ('Frontier'), *Yr13* ('Hustler'), *Yr14* ('Kador'), *Yr16* ('Bersee'), *Yr18* ('Jupateco 73R'), *Yr29* ('Pavon F76'), *Yr30* ('Opata 85'), *Yr34* ('WAWHT2046'), *Yr36* ('Glupro'), and *Yr39* (Alpowa). In addition a large number of QTLs that condition HTAP or adult plant stripe rust resistance have been characterized (Worland and Law 1986; Chen et al., 1998; Boukhatem et al., 2002; Chen 2005; Santra et al., 2006; William et al., 2006; Chen and Lin 2007; Chen and Zhao

2007; Chhuneja et al., 2007; Lin and Chen 2007). The relative abundance of HTAP resistance genes should facilitate incorporation of durable resistance into adapted wheat germplasm.

In 1983, Roy Johnson defined durable resistance as "resistance that remains effective in a cultivar that is widely grown for a long period of time in an environment favorable to the disease" (Johnson 1983). Based on this definition, HTAP resistance has been durable and therefore should be widely used in breeding programs. Although race-specific resistance genes are generally not durable, these genes can be used in combination to prolong their life span. Wheat cultivars with more effective and durable stripe rust resistance can be attained by combining HTAP resistance with effective race-specific resistance.

Slow-rusting resistance

Although there are several definitions given for slow-rusting resistance in the literature, the definition originally given by Caldwell (1968) and followed by Parlevliet (1979) and Singh and Rajaram (1992) is still descriptive. Slow-rusting resistance was characterized as slow disease development in the field despite a susceptible infection type and by one or more resistance components, such as longer latent period, smaller uredinium size, low receptivity, and reduced sporulation. Slow-rusting and HTAP resistances are similar in that both are expressed mostly in adult plants and are characterized by low disease severity in the field. However, cultivars with slow-rusting resistance have a susceptible infection type, while cultivars with HTAP resistance have lower infection types than susceptible cultivars. Slow-rusting resistance usually does not include expression of a hypersensitive response, while HTAP resistance involves some degree of hypersensitivity.

Slow-rusting resistance to stripe rust is present in wheat cultivars. Stripe rust development is much slower on the cultivars Heines VII and Yamhill compared with susceptible cultivars. This resistance is likely due to a longer latent period for stripe rust sporulation. In field plots these two cultivars had lower stripe rust severity compared with susceptible cultivars, even though the predominant races were virulent on the race-specific resistance genes in both cultivars. In field plots at Pullman, Washington, many European wheat cultivars such as 'Cappelle Desprez', 'Vilmorin 23', and 'Hybrid 46' have exhibited slow-rusting resistance. Several spring wheat cultivars, such as 'Eden', 'Macon', and 'Scarlet', have consistently had susceptible infection types but have rust severities lower than highly susceptible cultivars. However, the slow-rusting resistance in these cultivars is not as effective as the HTAP resistance in the cultivars Alpowa, Express, and Louise.

WHEAT STEM RUST

Distribution and epidemiology

Although not the most widespread or common among the wheat rusts, stem rust of wheat can potentially be the most damaging. In the US epidemics have been most frequent and severe in spring wheat and durum wheat in the northern states Minnesota, South Dakota, North Dakota, and to a lesser degree in winter wheat in the southern and central Great Plains states and the Ohio Valley. Stem rust also occurs less frequently in the Pacific Northwest region of Washington and Idaho. Worldwide, stem rust is mostly found in regions with a continental climate where summer temperatures regularly exceed 25 °C. Stem rust has caused losses in wheat in Canada (Kolmer 2001b), the southern Cone of South America (German et al., 2007), continental Europe, the Indian Subcontinent, Australia (Park 2007), eastern Africa (Wanyera et al., 2006), and China (Roelfs et al., 1992).

Stem rust appears as elongated blisterlike pustules, or uredinia, most frequently on the leaf sheaths of a wheat plant, but also on true stem tissues, leaves, glumes, and awns (Color Plate 11). On the leaf sheath and glumes, uredinia rupture the epidermis and give a ragged appearance. Masses of brownish-red urediniospores, up to 10,000 per day, are produced in the uredinia and are easily shaken off plants. The uredinial stage is

the most visible and is the disease stage on green plants. Urediniospores are elongated, 10–15 µm long, and echinulate. The urediniospores are disseminated to newly emerged tissues of the same plant or adjacent plants, where these spores are the source of new infections, or the spores can be windblown over long distances. In the case of long-distance dispersal, spore depositions on crops in a new area are often associated with rain showers. Stem rust pustules develop mostly on the underside of leaves, but may penetrate and sporulate on the upper side. In general, leaves of adult plants are not as receptive as stem tissue for stem rust infection. As infected plants mature, uredinia are replaced by telia, changing color from red to dark brown to black; thus the disease is also called black stem rust. Teliospores are firmly attached to plant tissue. The telial stage is not important in the epidemiology of the disease unless the alternate host common barberry (*Berberis vulgaris*) is present.

The infection process and specialized infection structures for *P. graminis* f. sp. *tritici* are the same as previously described for *P. triticina*, except that a 3-hour period of light is required following 6–8 hours of dew to complete the development of appresoria and penetration peg. Spore germination is optimal at 15–24 °C, and can occur up to 30 °C. The optimal temperature for sporulation is 30 °C and can occur up to 40 °C. A dew period of 6 hours is optimal for spore germination and the subsequent infection process (Roelfs et al., 1992).

In the US, stem rust is rarely observed on winter wheat in the fall. Infections on susceptible cultivars are generally not obvious until the spring after the wheat crop has reached heading. The disease is most obvious when a crop is approaching maturity. Infections of stem rust can severely damage crops that are within 2 weeks of harvest. Infections in winter wheat along the Gulf Coast and Texas in early spring are likely due to infections that survived the winter on fall-planted winter wheat or volunteer plants (Roelfs 1989), though field observations of overwintering events are rare. Stem rust infections on wheat are usually first observed in Texas and Louisiana in the last two weeks of April or the first two weeks of May and reach maximum severity by the middle of May.

Stem rust epidemics during and after the 1930s were due to the presence of a large pathogen population on winter wheat in the southern states that was windblown to the spring wheat region in the northern states and Canada. In years following the 1950s epidemics, however, there has been an increase of resistant cultivars in the southern US. This has reduced the opportunity for the pathogen to infect and overwinter in the south, resulting in very small population size (Kolmer et al., 2007a). There is only a small likelihood that stem rust overwinters on fall-planted winter wheat in the central Great Plains and the Midwest. In the northern spring wheat regions, stem rust occurs most frequently on susceptible wheat lines, and the initial inoculum for the region is almost exclusively from infected wheat in the southern and central Great Plains. In Minnesota and North Dakota, stem rust on wheat is usually first observed in mid-to-late June, with maximum severity in the last week of July or the first week of August. In the Pacific Northwest where plants of the alternate host common barberry are present, both overwintering urediniospores and aeciospores can contribute to the initial infections, but stem rust does not normally develop to epidemic status in this region. Comparisons of area of distribution, optimal temperatures for infection and growth, and alternate hosts for *P. triticina*, *P. graminis* f. sp. *tritici* and *P. striiformis* f. sp. *tritici* are summarized in Table 5.2.

Origin and historical importance

Although the origin of *P. graminis* f. sp. *tritici*, one of the specialized forms in the *P. graminis* species complex, is not clear, one or more of the susceptible alternate hosts of *Berberis* spp. was the likely source of the fungus (Leppik 1967). The rust most likely evolved in a region where the aecial and telial hosts overlapped (Wahl et al., 1984). However, it is difficult to restrict the center of origin to a specific region because of the large number and broad distribution of susceptible species of both the pycnial and telial hosts. Likely, *P. graminis* f. sp. *tritici* originated on a close wild

Table 5.2 Optimal temperatures for infection and growth, areas of distribution, and alternate hosts of *Puccinia triticina* (wheat leaf rust), *P. striiformis* f. sp. *tritici* (wheat stripe rust), and *P. graminis* f. sp. *tritici* (wheat stem rust) in the US.

	Infection temperature	Growth temperature	Distribution in US	Alternate hosts
Puccinia triticina	15–25 °C	20–30 °C	Common and widespread in Great Plains, southeastern US, Ohio Valley; Lower incidence in northeast and Pacific Northwest	*Thalictrum speciosissimum*—present in southern Europe—not native to North America
Puccina striiformis f. sp. *tritici*	10–12 °C	13–16 °C	Common and widespread in Pacific Northwest, California, and Gulf Coast; can be common and severe in southern and mid-Great Plains; lower incidence in northern Great Plains and northeastern US	No alternate host found
Puccinia graminis f. sp. *tritici*	15–24 °C	25–35 °C	Overwinters in south Texas and Gulf Coast; infections found on susceptible wheat in Great Plains and southeastern US; historically most destructive in spring wheat area of Great Plains	*Berberis vulgare*—once common and widespread—currently present in low numbers in Great Plains and Pacific Northwest

relative of common wheat, such as emmer wheat or a grass in the tribe Triticeae. It is not known when stem rust was first established in wheat in North America. Both wheat and susceptible *Berberis* spp. were introduced by early settlers, and the pathogen could have been introduced through imported *Berberis* spp. that had stem rust infections, or urediniospores and teliospores on wheat or barley straw, or urediniospores attached to clothing and implements. The capability of stem rust urediniospores to survive in transcontinental air currents indicates that the introduction could also have been a natural event.

In the early 1900s stem rust epidemics were frequent in the north central US and Manitoba and Saskatchewan in Canada, since all bread wheat cultivars were susceptible and millions of barberry plants were present in the Great Plains region. Only durum cultivars had some resistance to stem rust. Severe epidemics occurred in 1904, 1916, 1919, 1923, and 1927. The 1916 epidemic is especially notable since the large yield losses spurred the national barberry eradication program in the US (Campbell and Long 2001). In 1919 losses of 20% in spring wheat occurred in North Dakota and Minnesota, and 10% losses occurred in the winter wheat grown in Nebraska and Kansas. Even after the release of stem rust resistant spring wheat cultivars, severe epidemics with losses over 50% occurred from 1935 to 1937 and 1950 to 1954 due to the emergence of virulent stem rust races (Kolmer 2001b; Leonard 2001). A sustainable wheat industry could be maintained in the Great Plains of North America only if stem rust resistant cultivars were widely grown in both the winter wheat and spring wheat regions. Since the 1950s epidemics, the incidence of stem rust has been greatly reduced, a cumulative result due to the effect of barberry eradication in reducing the number of stem rust races (Leonard 2001) and the widespread planting of stem rust resistant spring wheat and winter wheat cultivars throughout the Great Plains region. Stem rust is virtually nonexistent today in production fields, and is seen almost exclusively in plots of susceptible winter and spring wheats.

The importance of stem rust in causing grain yield losses in wheat is readily apparent since telia of the fungus can be seen on the stems as the crop matures. Stem rust infections rupture the host plant epidermal tissue, causing an increased loss of water. Nutrients and water diverted by the fungus in the production of urediniospores also contribute to added stress of wheat that can contribute to premature death (Roelfs 1985). Stem rust infected plants are more susceptible to winterkill, produce fewer tillers, and have small heads. Lodging of plants caused by broken straw can occur due to severe stem infections. Severe infections in the last few weeks before harvest can greatly reduce grain yield due to the loss of water during the critical period of grain filling. Grain from stem rust infected wheat is often shriveled, which may reduce the market grade.

Taxonomy, life cycle, and host range

Wheat stem rust belongs to one of several *formae speciales* in *P. graminis*. The fungus is heteroecious, alternating between a telial host in the Poaceae and an aecial host in the Berberidaceae, and macrocyclic, with five spore states that are distinct in morphology and function. Illustrations of the life cycle of *P. graminis* f. sp. *tritici* can be readily found in various monographs and textbooks (Roelfs 1985; Agrios 1997).

The dikaryotic ($n+n$) teliospore is the dormant spore stage and germinates after breaking dormancy, usually after overwintering. The fungus has a brief diploid state when two nuclei fuse in a germinating teliospore. Meiosis follows, producing single-celled, hyaline haploid (n) basidiospores. Basidiospores are windborne to infect the alternate host of susceptible species in *Berberis* spp. and *Mahonia* spp. There are a large number of species in *Berberis* and *Mahonia* listed as susceptible to *P. graminis* (Roelfs 1985), but the common barberry, *B. vulgare*, is considered to be the most important. After infection on the alternate host, flask-shaped pycnia develop on the upper leaf surface, producing single-celled pycniospores (n) and receptive hyphae (n) that serve as gametes. Fertilization occurs when a pycniospore fuses with a receptive hypha of the opposite mating type, resulting in dikaryotic hyphae ($n+n$) that develop into a cluster of tubular or cuplike aecia on the underside of the leaf surface. Aeciospores ($n+n$) are produced in chains in aecia and are windborne to infect the telial hosts (grasses), but do not infect the aecial host. After an infection is established on a telial host, blisterlike uredinia develop at the infection site. Urediniospores ($n+n$) are produced as soon as 7 days after initial infection. Urediniospores are continuously produced in a sporulating uredinium for a sustained period of up to several weeks as long as the host tissue remains viable. The urediniospores are clonally produced for a number of generations and initiate new infections on the telial hosts. Under favorable conditions, urediniospores can reproduce rapidly in wheat crops, generating large quantities of inoculum. Teliospores are produced as the wheat plants approach maturity.

In most areas where wheat stem rust is important, the pathogen survives through the noncrop seasons as mycelia in tissues of dormant crop plants, volunteer plants, or alternative telial hosts (Roelfs 1985). Urediniospores could also be transported from one region to another following the succession of crops within an epidemiological zone. Thus the disease cycle can be completed without the presence of the alternate host. In North America, *P. graminis* f. sp. *tritici* is found primarily on common and durum wheat, barley, foxtail barley (*Hordeum jubatum*), and jointed goatgrass, although artificial inoculation can induce infection on many other grasses.

Genetic variation in *Puccinia graminis* f. sp. *tritici*

Race surveys of *P. graminis* f. sp. *tritici* have been conducted in the US since 1919 (Stakman et al., 1919). The initial set of differentials used to identify wheat stem rust races were the common wheat cultivars Little Club, Marquis, Kanred, and Kota; the durum cultivars Arnautka, Speltz Marz, Mindum, Kubanka, and Acme; the emmers 'White Spring', 'Khapli'; and diploid einkorn wheat (*T. monococcum* ssp. *monococcum*). These various wheats had multiple genes for stem rust resistance and were gradually replaced by differ-

entials with single genes in Chinese Spring or in other backgrounds (McIntosh et al., 1995). The stem rust resistance genes present in the current differential set used in the US and Canada include *Sr5*, *Sr6*, *Sr7b*, *Sr8a*, *Sr9a*, *Sr9b*, *Sr9d*, *Sr9e*, *Sr9g*, *Sr10*, *Sr11*, *Sr17*, *Sr21*, *Sr30*, *Sr36*, and *SrTmp* (Roelfs and Martens 1988). Wheat and *P. graminis* f. sp. *tritici* interact in a gene-for-gene manner (Loegering and Powers 1962; Green 1964); thus the frequency of races with virulence to a resistance gene is related to the frequency of virulence alleles in the rust pathogen.

The alternate host of stem rust, common barberry, was prevalent throughout the north central US in the late 1890s and early 1900s. Pycnial–aecial infections on the barberry plants contributed to the initial inoculum and also to the race diversity of stem rust. The barberry eradication program in the 1920s removed millions of barberry from this region, thus delaying the initial onset of epidemics in the spring wheat region via reduction of initial inoculum and also reducing the race diversity of stem rust (Campbell and Long 2001). From 1919 to the 1950s, 10–38 stem rust races were detected annually in the US, whereas from the 1960s to the present time, fewer than 10 races were usually detected (Groth and Roelfs 1987) (Fig. 5.3). The total amount of wheat stem rust inoculum has been reduced since the 1960s due to the increased use of highly resistant winter and spring wheat cultivars and of winter wheat cultivars with shorter maturity, thus reducing late onsets of stem rust in the southern Great Plains.

In 2006 a single race designated as QFCS accounted for 25 of the 27 total collections, along with single collections of race MCCD (= race 56) and race TTTT (Long et al., 2007). Race QFCS is widely avirulent on many of the *Sr* genes commonly present in spring and winter wheat in the US. The combination of smaller population size and the lack of sexual recombination has stabilized selection of races in the wheat stem rust population and thus has made the resistance of the *Sr* genes much more effective and durable. The use of highly resistant wheat cultivars in Australia (Park 2007) has reduced the number of stem rust races and the overall levels of inoculum.

Roelfs and Groth (1980) determined in 1975 that there were six distinct groups of *P. graminis* f. sp. *tritici* races in North America. Races in each

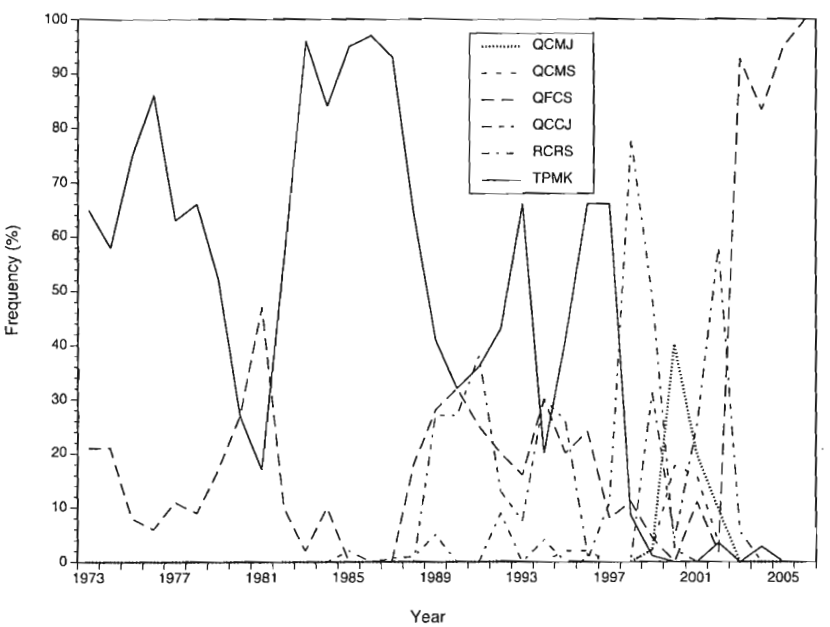

Fig. 5.3 Frequency (%) of races of *Puccinia graminis* f. sp. *tritici* collected from wheat in the US Great Plains. The four letter code race nomenclature is based on infection types to single-gene differentials in Roelfs and Martens (1988).

group were highly related for virulence yet were very distinct from other clusters. Burdon and Roelfs (1985) showed that isozyme variation in wheat stem rust populations was completely correlated with the race groupings. The distinct groups of wheat stem rust races that were described in 1975 in North America were likely ancestral groups of races that existed before barberry eradication. Removal of the sexual cycle would have prevented any further genetic exchange between the different groups of races.

In 1999 a new stem rust race initially designated as Ug-99 was found in Uganda (Pretorius et al., 2000). This race was notable since it was the first to have virulence to gene *Sr31*. Race Ug-99 (designated as TTKS on wheat stem rust differentials) was also virulent to gene *Sr38*. Gene *Sr31* is found in about 30% of CIMMYT germplasm, and *Sr38* is present in European, Australian, and a few CIMMYT wheats (Singh et al., 2006). In addition to these virulences, Ug-99 is virulent to a number of other *Sr* genes present in US spring and winter wheat (Jin and Singh 2006). As of 2007, Ug-99 was found in Uganda, Kenya, Sudan, Ethiopia, Yemen, and Iran. This stem rust race has the potential to spread through the Middle East and then to India and Pakistan, where it could cause devastating grain yield losses in wheat. Since it is widely virulent to many wheat cultivars in this region, it will be imperative to find sources of resistance and incorporate these into current wheat breeding programs.

Stem rust resistance in wheat

In the early part of the 20th century, wheat breeding in the spring wheat region of the northern Great Plains was largely a struggle against stem rust, the most important disease of wheat at the time. Marquis was the most widely grown spring wheat cultivar in the north central US and in Canada. Marquis, developed by the Canada Department of Agriculture in Ottawa and released in 1907, had early maturity and was popular with the milling industry since it was the first high-quality bread wheat developed in North America. However, Marquis and the other bread wheat cultivars were very susceptible to both stem rust and leaf rust. The stem rust epidemics of the early 1900s caused such losses that durum wheat, which was generally resistant to stem rust, had replaced bread wheat in Minnesota. Efforts were thus made to develop bread wheat and durum wheat cultivars that were resistant to stem rust.

The first hard red spring wheat bred for stem rust resistance in North America was 'Ceres', released by the North Dakota Agricultural Experiment Station in 1926 and grown in both the US and Canada. Ceres was developed by crossing the stem rust resistant wheat cultivar Kota with Marquis, and selecting stem rust resistant progeny. However, stem rust race 56, with virulence to the resistance in Kota and Ceres, increased throughout the Great Plains region after the release of Ceres. Widespread epidemics of stem rust on Ceres occurred from 1935 to 1947 due to the increased presence of race 56.

Ceres was largely replaced by Thatcher wheat, developed by the Minnesota Agricultural Experiment Station and released in 1935. Thatcher was developed from a cross between two lines, derived from Kanred/Marquis and from Iumillo durum/Marquis, which combined the good quality characteristics of Marquis with stem rust resistance in Kanred (*Sr5, Sr16*) and Iumillo durum (*Sr9g* and *Sr12*) (Kolmer 2001b). The Thatcher resistance of *Sr5*, *Sr9g*, *Sr12*, and *Sr16* was highly effective against race 56 and the other stem rust races. Many cultivars subsequently released in Canada and the US were derived from Thatcher (Kolmer et al., 1991). None of the known race-specific genes in Thatcher could account for the adult-plant field resistance in Thatcher, which became apparent with the increase of stem rust races with virulence to the seedling genes in Thatcher. The adult-plant resistance in Thatcher appeared to be nonspecific and durable (Kolmer et al., 1991). Genetic analyses indicated that stem rust resistance in Thatcher was complex. At least two genes that condition resistance in adult plants were likely derived from Iumillo durum and were independent of the race-specific genes (Nazareno and Roelfs 1981; Gavin-Vanegas et al., 2007).

Other undesignated race-specific seedling resistance genes were also detected in Thatcher (Nazareno and Roelfs 1981; Knott 2000).

Another source of stem rust resistance in spring wheat during that period was the adult-plant resistance gene *Sr2*, derived from Yaroslav emmer (McIntosh et al., 1995). The stem rust resistant cultivar Hope and the breeding line H-44 were developed by selecting progeny from a cross between Marquis and Yaroslav emmer. Breeding line H-44 was subsequently used as a parent in breeding programs in Canada. The resistance conditioned by *Sr2* is characterized by fewer uredinia and lower overall severity of stem rust at maturity. The cultivar Renown released in 1937 was the first stem rust resistant cultivar with *Sr2* to be released in Canada. Cultivars with *Sr2* were resistant to race 56 and the other stem rust races then present. Gene *Sr2* is present in wheat cultivars in Australia and the CIMMYT program (McIntosh et al., 1995) and in Canadian cultivars (Liu and Kolmer 1998a). Gene *Sr2* is present in some hard red winter and soft red winter cultivars in the US (Roelfs 1988) since Hope was used as a parent in these programs.

From 1950 to 1954 race 15B of wheat stem rust was widespread in the Great Plains region. This race caused high stem rust severities on spring wheats with *Sr2* and the Thatcher stem rust resistance. In 1954 yield losses in spring wheat were over 40% in North Dakota and reached 18% in Minnesota. In response to race 15B, the cultivar Selkirk with genes *Sr2*, *Sr6*, *Sr7b*, *Sr9d*, *Sr17*, and *Sr23* was highly resistant to race 15B and was released in 1954 by the Canada Department of Agriculture. Selkirk was widely grown throughout the spring wheat region of the US and Canada through the 1960s. The *Sr6* gene was particularly effective against race 15B and was actively selected in spring and winter wheat breeding programs in the central and northern Great Plains. The gene is also present with high frequency in CIMMYT germplasm. The widespread use of *Sr6* likely contributed to the rapid decline of race 15B in North America in the late 1950s. Wheat cultivars from Kenya also had *Sr6*. The cultivar Chris was released by the Minnesota Agricultural Experiment Station in 1966 and was the first US cultivar to have *Sr6*.

The resistance derived from Hope, Thatcher, and *Sr6* was the main component of stem rust resistance in the spring wheat region from the 1930s through the 1970s. However, the frequency of *Sr2* and of the Thatcher adult plant resistance has apparently declined in current cultivars given the following observations. Thatcher was moderately resistant to race TTKS (Ug-99) (Pretorius et al., 2000) in field tests in Kenya where race TTKS has been predominant. The Thatcher stem rust resistance is also expressed in seedling plants, but seedling tests with race TTKS failed to detect the Thatcher type of resistance in spring wheat cultivars from the northern Great Plains released between 1996 and 2005 (Jin and Singh 2006). Stem rust race TPMK, which became common after the decline of race 15B (Fig. 5.3), produces moderate to high infection types on seedling and adult plants of Thatcher. Selection of stem rust resistant germplasm in breeding programs based on resistance to TPMK could have placed higher selection pressure on the low infection types conditioned by race-specific genes such as *Sr6* and *Sr9b* than on the nonspecific resistance derived from Thatcher. Resistance due to *Sr2* also appeared to be absent in these cultivars. The absence of *Sr2* in spring wheat was likely associated with intense breeding for Fusarium head blight (caused by *Fusarium graminearum*) resistance, for which a major QTL is located on chromosome 3BS (Anderson et al., 2001) in close repulsion linkage with *Sr2* (Spielmeyer et al., 2003).

High levels of stem rust resistance in hard red winter wheat, in combination with early maturity, have had an impact in reducing the *P. graminis* population size in North America. The stem rust resistance gene *SrTmp*, derived from 'Triumph 64', was likely present in some of the initial hard red winter wheat germplasm (Roelfs and McVey 1979). This gene was effective against the majority of stem rust races in North America, but race 56 and race 15B were virulent to *SrTmp*. The resistance conditioned by *SrTmp*, combined with the early maturity of the Triumph background,

may have contributed to reducing the amount of stem rust present in winter wheat. The *SrTmp* gene is present in several current cultivars (Jin and Singh 2006) and in nearly 10% of breeding lines in the hard red winter wheat region. Other major components of stem rust resistance in hard red winter wheat are *Sr24* and resistance derived from the 1RS.1AL wheat–rye translocation. Originally derived from *Thinopyrum ponticum*, *Sr24* has been common in hard red winter wheat cultivars since the release of Agent in 1967, and to a lesser degree in soft red winter and spring wheat cultivars. Stem rust races with virulence on *Sr24* have not been detected in North America, and the gene is currently present in nearly 50% of the current hard red winter wheat breeding lines and cultivars. Because *Sr24* is tightly linked with *Lr24* (McIntosh et al., 1995), selection for leaf rust resistance produced lines with stem rust resistance. The 1RS.1AL translocation in 'Amigo', with the rye chromosome introduced from 'Insave F.A.' rye via a triticale with greenbug resistance (Sebesta et al., 1994), gives effective resistance to stem rust races in North America as well as to race TTKS.

Derived from Petkus rye, *Sr31* is on the 1RS.1BL wheat–rye translocation with *Lr26* and *Yr9* (McIntosh et al., 1995). Gene *Sr31* is present in some current hard red winter wheat cultivars and in a few soft red winter wheat cultivars in the US, and is present in CIMMYT-derived wheat cultivars that are grown worldwide. This gene provided a very high level of resistance to all known stem rust races prior to the emergence of TTKS in Uganda in 1999. Derived from *T. timopheevi*, *Sr36* is the most common stem rust resistance gene in soft red winter wheat in the US and has been an important source of resistance in Australia (McIntosh et al., 1995). In addition, *Sr36* conditions resistance to the current stem rust races in the US as well as race TTKS (Jin and Singh 2006).

Genes *Sr9b*, *Sr11*, and *Sr17* are also present in spring and winter wheat in North America. Gene *Sr9b* is closely linked with *Lr13* and is present in several spring wheat lines that were selected for leaf rust resistance from Frontana. Virulence to *Sr9b* is common in stem rust races in North America. Virulence to *Sr11* is present in North America and Australia. The origin of *Sr17* is assumed to be Yaroslav emmer; thus it may also be present in wheats with *Sr2*, though many stem rust races are virulent to this gene.

In Australia wheat cultivars with *Sr24*, *Sr26*, *Sr30*, *Sr36*, and *Sr38* have been released in the past 40 years (Park 2007). Gene *Sr26* is derived from *Th. ponticum*, is present only in Australian cultivars, and stem rust races with virulence to this gene have not been detected. Gene *Sr30 is* present in a number of Australian cultivars and also CIMMYT germplasm (McIntosh et al., 1995). Gene *Sr38*, derived from *T. ventricosa*, is linked with *Lr37* and *Yr17* and was selected in Australian germplasm based on resistance to all three rusts. Currently gene designations up to *Sr46* have been given for stem rust resistance genes in wheat (McIntosh et al., 2007).

FUTURE PERSPECTIVES

The emergence of new races of wheat leaf rust, wheat stripe rust, and wheat stem rust continually challenge wheat breeders and plant pathologists to develop effective sources of durable resistance to these pathogens. The eventual cloning and sequencing of genes such as *Lr34/Yr18* and *Sr2* will provide greater insight into how these non-specific resistance genes function, thus offering the potential of designing new resistance genes that will offer greater resistance durability. These genes will likely differ for functional domains and specificity in comparison with the race-specific NBS-LRR resistance genes.

The discovery that the herbicide glyphosate (Anderson and Kolmer 2005) greatly reduces rust infections in glyphosate-resistant wheat is an exciting development that could be utilized immediately to reduce losses in wheat due to rusts, and it may lead to other transgenic strategies to design rust resistance genes or genes for tolerance or resistance to chemical applications in wheat. The characterization of avirulence genes in flax rust (*Melampsora lini*) and the corresponding resistance genes in flax (*Linum usitatissimum*), and an understanding of how their coded func-

tional proteins interact (Dodds et al., 2007), will lead to greater understanding and potential exploitation of gene-for-gene specificity in the wheat rusts. Of course any of these strategies will depend on the eventual governmental, regulatory, and consumer acceptance of transgenic wheat.

In the meantime the frequency and severity of rust epidemics on wheat can be reduced by greater utilization of resources that are already available. Diagnostic markers for genes such as *Lr34/Yr18* should greatly simplify selection of these genes, potentially allowing genes with durable rust resistance to become fixed in wheat germplasm. Increased planting of wheat cultivars with high levels of leaf rust resistance in the US, and removal of susceptible cultivars, will reduce the size of the *P. triticina* population that regularly overwinters, thus reducing the chance of mutation for increased virulence to newly deployed resistance genes. Australia has adopted minimum disease standards for release of wheat cultivars (Wallwork 2007), with the specific goal to reduce the amount of rust inoculum and thus prolong the effective life span of a rust resistance gene by reducing the chances of new virulent races emerging. The effectiveness of this approach can be seen in comparing the effective life span of *Lr24* in the US versus Australia. In the US, virulence to *Lr24* appeared almost immediately after the release of cultivars with this gene, while in Australia races of *P. triticina* with virulence to *Lr24* did not appear until 17 years after cultivars with this gene were first grown (Park et al., 2002). Genes *Lr1* and *Lr13* have also been much more effective in Australia compared with the US due to the greater use of highly resistant cultivars and the reduced size of the *P. triticina* population.

For the stem rust race Ug-99, the challenge will be to incorporate *Sr2* into wheat germplasm in regions where this race poses an immediate threat. This gene was common in older CIMMYT wheat cultivars such as Pavon 76, but more recent germplasm releases appear to lack *Sr2*. Although Ug-99 is virulent to many genes that were originally derived from hexaploid wheat, several genes derived are from wild wheat relatives that condition resistance to this race. It should be feasible to add these genes into wheat germplasm by using tightly linked molecular markers and testing with avirulent stem rust races.

The reproductive capacity, long-distance aerial dispersal, and high degree of genetic variation in the wheat rust fungi almost certainly ensure that new races with virulence to important and widely used resistance genes will continue to arise and pose a threat to wheat production. Ultimately losses due to these pathogens can be avoided only by continued vigilance by wheat breeders and plant pathologists.

REFERENCES

Agrios, G.N. 1997. Plant pathology. 4th ed. Academic Press, Orlando, FL.

Allan, R.E., C.J. Peterson, R.F. Line, G.L. Rubenthaler, and C.F. Morris. 1993. Registration of 'Rely' wheat multiline. Crop Sci. 33:213–214.

Allan, R.E., L.H. Purdy, and O.A. Vogel. 1966. Inheritance of seedling and adult reaction of wheat to stripe rust. Crop Sci. 6:242–245.

Allen, R.E. 1928. A cytological study of *Puccinia glumarum* on *Bromus marginatus* and *Triticum vulgare*. J. Agric. Res. 36:487–513.

Anderson, J.A., and J.A. Kolmer. 2005. Rust control in glyphosate tolerant wheat following application of the herbicide glyphosate. Plant Dis. 89:1136–1142.

Anderson, J.A., R.W. Stack, S. Liu, B.L. Waldron, A.D. Field, C. Coyne, B. Moreno-Sevilla, J.M. Fetch, Q.J. Song, P.B. Cregan, and R.C. Frohberg. 2001. DNA markers for Fusarium head blight resistance QTLs in two wheat populations. Theor. Appl. Genet. 102:1164–1168.

Anikster, Y., W.R. Bushnell, T. Eilam, J. Manisterski, and A.P. Roelfs. 1997. *Puccinia recondita* causing leaf rust on cultivated wheats, wild wheats, and rye. Can. J. Bot. 75:2082–2095.

Barcellos, A.L., A.P. Roelfs, and M.I.B. de Moraes-Fernandes. 2000. Inheritance of adult plant leaf rust resistance in the Brazilian wheat cultivar Toropi. Plant Dis. 84:90–93.

Bariana, H.S., G.N. Brown, U.K. Bansal, H. Miah, G.E. Standen, and M. Lu. 2007. Breeding triple rust resistance for Australia using conventional and marker-assisted selection technologies. Aust. J. Agric. Res. 58:576–587.

Biffen, R.H. 1905. Mendel's law of inheritance of disease resistance. J. Agric. Sci. 4:421–429.

Bossolini, E., S.G. Krattinger, and B. Keller. 2006. Development of simple sequence repeat markers specific for the *Lr34* resistance region of wheat using sequence information from rice and *Aegilops tauschii*. Theor. Appl. Genet. 113:1049–1062.

Boukhatem, N., P.V. Baret, D. Mingeot, and J.M. Jacquemin. 2002. Quantitative trait loci for resistance against yellow rust in two wheat-derived recombinant inbred line populations. Theor. Appl. Genet. 104:111–118.

Britton, M., and G.B. Cummins. 1956. The reaction of species of *Poa* and grasses to *Puccinia striiformis*. Plant Dis. Rep. 40:643–645.

Burdon, J.J., and A.P. Roelfs. 1985. Isozyme and virulence variation in asexually reproducing populations of *Puccinia graminis* and *P. recondita* on wheat. Phytopathology 75:907–913.

Caldwell, R.M. 1968. Breeding for general and/or specific plant disease resistance. p. 263–272. *In* K.W. Finlay and K.W. Shepard (ed.) Proc. Int. Wheat Genet. Symp., 3rd, Canberra, Australia. 5–9 Aug. 1968. Australian Academy of Science, Canberra, Australia.

Caldwell, R.M., H.R. Kraybill, J.T. Sullivan, and L.E. Compton. 1934. Effect of leaf rust (*Puccinia triticina*) on yield, physical characters, and composition of winter wheats. J. Agric. Res. 48:1049–1071.

Campbell, C.L., and D.L. Long. 2001. The campaign to eradicate the common barberry in the United States. p. 16–50. *In* P.D. Peterson (ed.) Stem rust of wheat, from ancient enemy to modern foe. APS Press, St. Paul, MN.

Carleton, M.A. 1915. A serious new wheat rust in this country. Science 42:58–59.

Chen, X.M. 2005. Epidemiology and control of stripe rust on wheat. Can. J. Plant Pathol. 27:314–337.

Chen, X.M. 2007. Challenges and solutions for stripe rust control in the United States. Aust. J. Agric. Res. 58: 648–655.

Chen, X.M., and F. Lin. 2007. Identification and molecular mapping of genes for all-stage and high-temperature adult-plant resistance to stripe rust in 'Express' wheat. Phytopathology 97:S21.

Chen, X.M., and R.F. Line. 1995a. Gene action in wheat cultivars for durable high-temperature adult-plant resistance and interactions with race-specific, seedling resistance to stripe rust caused by *Puccinia striiformis*. Phytopathology 85:567–572.

Chen, X.M., and R.F. Line. 1995b. Gene number and heritability of wheat cultivars with durable, high-temperature, adult-plant resistance and race-specific resistance to *Puccinia striiformis*. Phytopathology 85:573–578.

Chen, X.M., R.F. Line, and H. Leung. 1993. Relationship between virulence variation and DNA polymorphism in *Puccinia striiformis*. Phytopathology 83:1489–1497.

Chen, X.M., R.F. Line, and H. Leung. 1995. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f. sp. *hordei* to other rusts. Phytopathology 85:1335–1342.

Chen, X.M., R.F. Line, Z.X. Shi, and H. Leung. 1998. Genetics of wheat resistance to stripe rust. p. 237–239. *In* R.A. McIntosh, G.E. Hart, K.M. Devos, M.D. Gale, and W.J. Rogers (ed.) Proc. Int. Wheat Genet. Symp., 9th, Vol. 3, Saskatoon, Saskatchewan. 2–7 Aug. 1998. Univ. Saskatchewan, Saskatoon, Canada.

Chen, X.M., M.K. Moore, E.A. Milus, D.L. Long, R.F. Line, D. Marshall, and L. Jackson. 2002. Wheat stripe rust epidemics and races of *Puccinia striiformis* f. sp. *tritici* in the United States in 2000. Plant Dis. 86:39–46.

Chen, X.M., L.N. Penman, and K.L. Richardson. 2007. Races of *Puccinia striiformis*, the stripe rust pathogen in the United States in 2006. Phytopathology 97:S21.

Chen, X.M., and J. Zhao. 2007. Identification of molecular markers for *Yr8* and a gene for high-temperature, adult-plant resistance against stripe rust in the AVS/6*Yr8 wheat line. Phytopathology 97:S21.

Chester, K.S. 1939. The 1938 wheat leaf-rust epiphytotic in Oklahoma. Plant Dis. Rep. 112:1–19.

Chester, K.S. 1946. The nature and prevention of the cereal rusts as exemplified in the leaf rust of wheat. Chronica Botanica, Waltham, MA.

Chhuneja, P., S. Kaur, T. Garg, M. Ghai, S. Kaur, M. Prashar, R.K. Goel, B. Keller, H.S. Dhaliwal, and K. Singh. 2007. Mapping of adult-plant stripe rust resistance genes in diploid A genome wheat species and their transfer to bread wheat. Theor. Appl. Genet. 116:313–324.

Cloutier, S., B.D. McCallum, C. Loutre, T.W. Banks, T. Wicker, C. Feulliet, B. Keller, and M.C. Jordan. 2007. Leaf rust resistance gene *Lr1*, isolated from bread wheat (*Triticum aestivum* L.) is a member of the large psr567 family. Plant Mol. Biol. 65:93–106.

Coakley, S.M., W.S. Boyd, and R.F. Line. 1982. Statistical models for predicting stripe rust on winter wheat in the Pacific Northwest. Phytopathology 72:1539–1542.

Coakley, S.M., W.S. Boyd, and R.F. Line. 1984. Development of regional models that use meteorological variables for predicting stripe rust disease in winter wheat. J. Clim. Appl. Meteorol. 23:1234–1240.

Cummins, G.B., and R.M. Caldwell. 1956. The validity of binomials in the leaf rust complex of cereals and grasses. Phytopathology 46:81–82.

Dodds, P.N., A.M. Catanzariti, G. Lawrence, and J.G. Ellis. 2007. Avirulence proteins of rust fungi: Penetrating the host–haustorium barrier. Aust. J. Agric. Res. 58:521–517.

d'Oliveira, B.D., and D.J. Samborski. 1966. Aecial stage of *Puccinia recondita* on Ranunculaceae and Boraginaceae in Portugal. p. 133–154. *In* R.C.F. Macer and M.S. Wolfe (ed.) Proc. Eur. Brown Rust Conf., 1st, Cereal Rust Conferences, Cambridge, UK. 29 June–4 July, 1964. Plant Breeding Inst., Cambridge, UK.

Duan, X., J. Enjalbert, D. Vautrin, C. Solignac, and T. Giraud. 2003. Isolation of 12 microsatellite loci, using an enrichment protocol, in the phytopathogenic fungus *Puccinia triticina*. Mol. Ecol. Notes 3:65–67.

Dyck, P.L. 1987. The association of a gene for leaf rust resistance with the chromosome 7D suppressor of stem rust resistance in common wheat. Genome 29:467–469.

Dyck, P.L. 1993. Inheritance of leaf rust and stem rust resistance in 'Roblin' wheat. Genome 36:289–293.

Dyck, P.L. 1994. The transfer of leaf rust resistance from *Triticum turgidum* ssp. *dicoccoides* to hexaploid wheat. Can. J. Plant Sci. 74:671–673.

Dyck, P.L., and R. Johnson. 1983. Temperature sensitivity of genes for resistance in wheat to *Puccinia recondita*. Can. J. Plant Pathol. 5:229–234.

Dyck, P.L., and O.M. Lukow. 1988. The genetic analysis of two interspecific sources of leaf rust resistance and their effect on the quality of common wheat. Can. J. Plant Sci. 68:633–639.

Dyck, P.L., and D.J. Samborski. 1982. The inheritance of resistance to *Puccinia recondita* in a group of common wheat cultivars. Can. J. Genet. Cytol. 24:273–283.

Dyck, P.L., D.J. Samborski, and R.G. Anderson. 1966. Inheritance of adult-plant leaf rust resistance derived from the common wheat varieties Exchange and Frontana. Can. J. Genet. Cytol. 8:665–671.

Enjalbert, J., X. Duan, M. Leconte, M.S. Hovmoller, and C. de Vallavielle-Pope. 2005. Genetic evidence of local adaptation of wheat yellow rust (*Puccinia striiformis* f. sp. *tritici*) within France. Mol. Ecol. 14:2065–2073.

Eriksson, J. 1894. Über die Spezialisierung des Parasitismus bei den Getreiderostpilzen. Ber. Dtsch. Bot. Ges. 12:292–331.

Eriksson, J. 1899. Nouvelles études sur la rouille brune des céréales. Ann. Sci. Nat. Bot. Eighth Ser. 9:241–288.

Eriksson, J., and E. Henning. 1894. Die Hauptresultate einer neuen Untersuchung über die Getreiderostpilze. Z. Pflanzenkr. 4:197–203.

Eriksson, J., and E. Henning. 1896. Die Getreideroste. Norstedt & Söner, Stockholm, Sweden.

Everts, K.L., S. Leath, and P.L. Finney. 2001. Impact of powdery mildew and leaf rust on milling and baking quality of soft red winter wheat. Plant Dis. 85:423–429.

Ezzahiri, B., S. Diouri, and A.P. Roelfs. 1992. *Anchusa italica* as an alternate host for wheat leaf rust in Morocco. Plant Dis. 76:1185.

Ezzahiri, B., and A.P. Roelfs. 1989. Inheritance and expression of adult plant resistance to leaf rust in Era wheat. Plant Dis. 73:549–551.

Feuillet, C., S. Travella, N. Stein, L. Albar, L. Nublat, and B. Keller. 2003. Map-based isolation of the leaf rust disease resistance gene *Lr10* from the hexaploid wheat (*Triticum aestivum* L.) genome. Proc. Natl. Acad. Sci. USA 100:15253–15258.

Finckh, M.R., and C.C. Mundt. 1992. Stripe rust, yield, and plant competition in wheat cultivar mixtures. Phytopathology 82:82–92.

Fuckel, L. 1860. Enumeratio fungorum nassoviae. Jahrb. Ver. Naturkd. Herzogthum Nassau 15:9.

Gavin-Vanegas, C.D., D.F. Garvin, and J.A. Kolmer. 2007. Genetics of stem rust resistance in the spring wheat cultivar Thatcher and the enhancement of stem rust resistance by *Lr34*. Euphytica 159:391–401.

German, S.E., A. Barcellos, M. Chaves, M. Kohli, P. Campos, and L. de Viedma. 2007. The situation of common wheat rusts in the Southern Cone of America and perspectives for control. Aust. J. Agric. Res. 58:620–630.

German, S.E., and J.A. Kolmer. 1992. Effect of gene *Lr34* in the enhancement of resistance to leaf rust of wheat. Theor. Appl. Genet. 84:97–105.

Goyeau, H., F. Halkett, M.F. Zapater, J. Carlier, and C. Lannou. 2007. Clonality and host selection in the wheat pathogenic fungus *Puccinia triticina*. Fungal Genet. Biol. 44:474–483.

Goyeau, H., R. Park, B. Schaeffer, and C. Lannou. 2006. Distribution of pathotypes with regard to host cultivars and French wheat leaf rust populations. Phytopathology 96:264–273.

Green, G.J. 1964. A color mutation, its inheritance and the inheritance of pathogenicity in *Puccinia graminis* Pers. Can. J. Bot. 42:1643–1664.

Groth, J.V., and A.P. Roelfs. 1987. The concept and measurement of phenotypic diversity in *Puccinia graminis* on wheat. Phytopathology 77:1395–1399.

Hardeř, D.E. 1984. Developmental ultrastructure of hyphae and spores. p. 333–374. *In* W.R. Bushnell and A.P. Roelfs (ed.) The cereal rusts. Vol. I. Academic Press, Orlando, FL.

Hart, H., and H. Becker. 1939. Beiträge zur Frage des Zwischenwirtes für *Puccinia glumarum*. Z. Pflanzenkr. (Pflanzenpathol.) Pflanzenschutz 49:559–566.

Hassebrauk, K. 1965. Nomenklatur, geographische Verbreitung und Wirtsbereich des Gelbrostes, *Puccinia striiformis* West. Mitt. Biol. Bundesanst. Land-Forstwirtsch. Berlin-Dahlem 116:1–75.

Hassebrauk, K. 1970. Der Gelbrost *Puccinia striiformis* West. 2 Befallsbid. Morphologie und Biologie der Sporen. Infektion und weitere Entwicklung. Wirkungen auf die Wirtsflanze. Mitt. Biol. Bundesanst. Land-Forstwirtsch., Berlin-Dahlem 139:1–110.

Hayes, H.K., E.R. Ausemus, E.C. Stakman, C.H. Bailey, H.K. Wilson, R.H. Bamberg, M.C. Markley, R.R. Crim, and M.N. Levine. 1936. Thatcher wheat. Bull. 325. Minn. Agric. Res. Stn., St. Paul, MN.

Hendrix, J.W. 1994. Stripe rust, what it is and what to do about it. Wash. State Univ. Stn. Circ. 424:1–6.

Herrera-Foessel, S.A., R.P. Singh, J. Huerta-Espino, J. Crossa, J. Yuen, and A. Djurle. 2006. Effect of leaf rust on grain yield and yield traits of durum wheats with race-specific and slow-rusting resistance to leaf rust. Plant Dis. 90:1065–1072.

Herrera-Foessel, S.A., R.P. Singh, J. Huerta-Espino, J. William, G.M. Rosewarne, A. Djurle, and J. Yuen. 2007. Identification and mapping of *Lr3* and a linked leaf rust resistance gene in durum wheat. Crop Sci. 47:1459–1466.

Herrera-Foessel, S.A., R.P. Singh, J. Huerta-Espino, J. Yuen, and A. Djurle. 2005. New genes for leaf rust resistance in CIMMYT durum wheats. Plant Dis. 89:809–814.

Hovmoller, M.S., A.F. Justesen, and J.K.M. Brown. 2002. Clonality and long-distance migration of *Puccinia striiformis* f. sp. *tritici* in north-west Europe. Plant Pathol. 51:24–32.

Huang, L., S.A. Brooks, W. Li, J.P. Fellers, H.N. Trick, and B.S. Gill. 2003. Map-based cloning of leaf rust resistance gene *Lr21* from the large and polyploid genome of wheat. Genetics 164:655–664.

Humphrey, H.B., C.W. Hungerford, and A.G. Johnson. 1924. Stripe rust (*Puccinia glumarum*) of cereals and grasses in the United States. J. Agric. Res. 29:209–227.

Hungerford, C.W. 1923. Studies on the life history of stripe rust, *Puccinia glumarum*. J. Agric. Res. 24:607–620.

Hylander, N., I. Jørstad, and J.A. Nannfeldt. 1953. Enumeratio uredinearum Scandinavicarum. Opera Bot. 1:1–102.

Jackson, H.S., and E.B. Mains. 1921. Aecial stage of the orange leaf rust of wheat, *Puccinia triticina* Eriks. J. Agric. Res. 22:151–172.

Jin, Y., and R.P. Singh. 2006. Resistance in U.S. wheat to recent eastern African isolates of *Puccinia graminis* f. sp. *tritici* with virulence to resistance gene *Sr31*. Plant Dis. 476–480.

Johnson, T. 1956. Physiologic races of leaf rust of wheat in Canada 1931 to 1955. Can. J. Agric. Sci. 36:323–332.

Johnson, R. 1983. Genetic background of durable resistance. p. 5–26. *In* F. Lamberti, J.M. Waller, and N.A. Van der Graaff (ed.) Durable resistance in crops. Plenum Press, New York.

Johnston, C.O. 1931. Effect of leaf rust infection on yield of certain varieties of wheat. J. Am. Soc. Agron. 23: 1–12.

Johnston, C.O., R.M. Caldwell, L.E. Compton, and L.E. Browder. 1968. Physiologic races of *Puccinia recondita* f. sp. *tritici* in the United States from 1926 through 1960. USDA Tech. Bull. 1393.

Kerber, E.R., and T. Aung. 1999. Leaf rust resistance gene *Lr34* associated with nonsuppression of stem rust in the wheat cultivar Canthatch. Phytopathology 89:518–521.

Kerber, E.R. and G.J. Green. 1980. Suppression of stem rust resistance in the hexaploid wheat cv. Canthatch by chromosome 7DL. Can. J. Bot. 12:1347–1350.

Khan, M.A., L.E. Trevathtan, and J. Robbins. 1997. Quantitative relationship between leaf rust and yield in Mississippi. Plant. Dis. 81:769–772.

Knott, D.R. 2000. The inheritance of stem rust resistance in Thatcher wheat. Can. J. Plant Sci. 80:53–63.

Kolmer, J.A. 1992. Gametic phase disequilibria in two populations of *Puccinia recondita* (wheat leaf rust fungus). Heredity 68:505–513.

Kolmer, J.A. 1998. Physiologic specialization of *Puccinia recondita* f. sp. *tritici* in Canada in 1996. Can. J. Plant Pathol. 20:176–181.

Kolmer, J.A. 2001a. Molecular polymorphism and virulence phenotypes of the wheat leaf rust fungus *Puccinia triticina* in Canada. Can. J. Bot. 79:917–926.

Kolmer, J.A. 2001b. Early research on the genetics of *Puccinia graminis* and stem rust resistance in wheat in Canada and the United States. p. 51–82. *In* P.D. Peterson (ed.) Stem rust of wheat: From ancient enemy to modern foe. APS Press, St. Paul, MN.

Kolmer, J.A. 2003. Postulation of leaf rust resistance genes in selected soft red winter wheats. Crop Sci. 43:1266–1274.

Kolmer, J.A., and P.L. Dyck. 1994. Gene expression in the *Triticum aestivum–Puccinia recondita* f. sp. *tritici* gene-for-gene system. Phytopathology 84:437–440.

Kolmer, J.A., P.L. Dyck, and A.P. Roelfs. 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 81:237–239.

Kolmer, J.A., Y. Jin, and D.L. Long. 2007a. Wheat leaf and stem rust in the United States. Aust. J. Agric. Res. 58:631–638.

Kolmer, J.A., and J.Q. Liu. 2000. Virulence and molecular polymorphism in international collections of the wheat leaf rust fungus *Puccinia triticina*. Phytopathology 90:427–436.

Kolmer, J.A., J.Q. Liu, and M. Sies. 1995. Virulence and molecular polymorphism in *Puccinia recondita* f. sp. *tritici* in Canada. Phytopathology 85:276–285.

Kolmer, J.A., D.L. Long, and M.E. Hughes. 2007b. Physiological specialization of *Puccinia triticina* on wheat in the United States in 2005. Plant Dis. 91:979–984.

Kolmer, J.A., D.L. Long, E. Kosman, and M.E. Hughes. 2003. Physiologic specialization of *Puccinia triticina* on wheat in the United States in 2001. Plant Dis. 87:859–866.

Kolmer, J.A., and L.M. Oelke. 2006. Genetics of leaf rust resistance in the spring wheats 'Ivan' and 'Knudson'. Can. J. Plant Pathol. 28:223–229.

Kolmer, J.A., L.M. Oelke, and J.Q. Liu. 2007c. Genetics of leaf rust resistance in three Americano landrace-derived wheat cultivars from Uruguay. Plant Breed. 126:152–157.

Kolmer, J.A., and M.E. Ordoñez. 2007. Genetic differentiation of *Puccinia triticina* populations in Central Asia and the Caucasus. Phytopathology 97:1141–1149.

Kolmer, J.A., R.P. Singh, D.F. Garvin, L. Viccars, H.M. William, J. Huerta-Espino, F.C. Ogbonnaya, H. Raman, S. Orford, H.S. Bariana, E.S. Lagudah. 2008. Analysis of the *Lr34/Yr18* rust resistance region in wheat germplasm. Crop Sci. 48:1841–1852.

Kuraparthy, V., P. Chhuneja, H.S. Dhaliwal, S. Kaur, R.L. Bowden, and B.S. Gill. 2007. Characterization and mapping of cryptic alien introgression from *Aegilops geniculata* with new leaf rust and stripe rust resistance genes *Lr57* and *Yr40* in wheat. Theor. Appl. Genet. 114:1379–1389.

Lagudah, E.S., H. McFadden, R.P. Singh, J. Huerta-Espino, H.S. Bariana, and W. Spielmeyer. 2006. Molecular characterization of the *Lr34/Yr18* slow rusting resistance gene region in wheat. Theor. Appl. Genet. 114:21–30.

Leonard, K.J. 2001. Stem rust—future enemy? p. 119–146. *In* P.D. Peterson (ed.) Stem rust of wheat: From ancient enemy to modern foe. APS Press, St. Paul, MN.

Leppik, E. 1967. Some viewpoints on the phylogeny of rust fungi: IV. Biogenic radiation. Mycologia 59:568–579.

Lin, F., and X.M. Chen. 2007. Genetics and molecular mapping of genes for race-specific all-stage resistance and non–race specific high-temperature adult-plant resistance to stripe rust in spring wheat cultivar Alpowa. Theor. Appl. Genet. 114:1277–1287.

Line, R.F. 1972. Recording and processing data on foliar diseases of cereals. p. 175–178. *In* Proc. Eur. Mediterranean Cereal Rusts Conf. Faculty of Science, Charles University, Prague, Czechoslovakia.

Line, R.F. 1976. Factors contributing to an epidemic of stripe rust on wheat in the Sacramento Valley of California in 1974. Plant Dis. Rep. 60:312–316.

Line, R.F. 2002. Stripe rust of wheat and barley in North America: A retrospective historical review. Annu. Rev. Phytopathol. 40:75–118.

Line, R.F., and X.M. Chen. 1995. Successes in breeding for and managing durable resistance to wheat rusts. Plant Dis. 79:1254–1255.

Line, R.F., and A. Qayoum. 1992. Virulence, aggressiveness, evolution, and distribution of races of *Puccinia striiformis*

(the cause of stripe rust of wheat) in North America, 1968–87. USDA Tech. Bull. 1788.

Line, R.F., F.L. Sharp, and R.L. Powelson. 1970. A system for differentiating races of *Puccinia striiformis* in the United States. Plant Dis. Rep. 54:992–994.

Liu, J.Q., and J.A. Kolmer. 1997. Genetics of leaf rust resistance in Canadian spring wheats AC Domain and AC Taber. Plant Dis. 81:757–760.

Liu, J.Q., and J.A. Kolmer. 1998a. Genetics of stem rust resistance in wheat cvs. Pasqua and AC Taber. Phytopathology 88:171–176.

Liu, J.Q., and J.A. Kolmer. 1998b. Molecular and virulence diversity and linkage disequilibria in asexual and sexual populations of the wheat leaf rust fungus, *Puccinia recondita*. Genome 41:832–840.

Loegering, W.Q., and H.R. Powers. 1962. Inheritance of pathogenicity in a cross of physiological races 111 and 36 of *Puccinia graminis* f. sp. *tritici*. Phytopathology 52:547–554.

Long, D.L., and J.A. Kolmer. 1989. A North American system of nomenclature for *Puccinia recondita* f. sp. *tritici*. Phytopathology 79:525–529.

Long, D.L., J.A. Kolmer, Y. Jin, M.E. Hughes, and L.A. Wanschura. 2007. Wheat rusts in the United States in 2006. Annu. Wheat Newsl. 53:130–141.

Long, D.L., K.J. Leonard, and M.E. Hughes. 2000. Virulence of *Puccinia triticina* on wheat in the United States from 1996 to 1998. Plant Dis. 84:1334–1341.

Lupton, F.G.H., and R.C.F. Macer. 1962. Inheritance of resistance to yellow rust (*Puccinia glumarum* Erikss. & Henn.) in seven varieties of wheat. Trans. Br. Mycol. Soc. 45:21–45.

Mains, E.B. 1930. Effect of leaf rust (*Puccinia triticina* Eriks.) on yield of wheat. J. Agric. Res. 40:417–446.

Mains, E.B. 1932. Host specialization in the leaf rust of grasses, *Puccinia rubigo-vera*. Pap. Michigan Acad. Sci., Arts and Letters 17:289–393.

Mains, E.B. 1933. Studies concerning heteroecious rusts. Mycologia 25:407–417.

Manners, J.G. 1960. *Puccinia striiformis* Westend. var *dactylidis* var. nov. Trans. Br. Mycol. Soc. 43:65–68.

Marasas, C.N., M. Smale, and R.P. Singh. 2004. The economic impact in developing countries of leaf rust resistance in CIMMYT-related spring wheat. Econ. Program Pap. 04-01. CIMMYT, D.F., Mexico.

Markell, S.G., E.A. Milus, and X.M. Chen. 2004. Genetic diversity of *Puccinia striiformis* f. sp. *tritici* in the United States. p. A2.43. *In* Proc. Int. Cereal Rusts & Powdery Mildews Conf., 11th, John Innes Centre, Norwich, UK. 22–27 Aug. 2004.

Marryat, D.C.E. 1907. Notes on the infection and histology of two wheats immune to attacks of *Puccinia glumarum*, yellow rust. J. Agric. Sci. 2:129–138.

Martin, J.N., B.F. Carver, R.M. Hunger, and T.S. Cox. 2003. Contributions of leaf rust resistance and awns to agronomic and grain quality in winter wheat. Crop Sci. 43:1712–1717.

Martinez, F., J.C. Sillero, and D. Rubiales. 2005. Pathogenic specialization of *Puccinia triticina* in Andalusia from 1998 to 2000. J. Phytopathol. 153:344–349.

McCallum, B.D., and P. Seto-Goh. 2006. Physiological specialization of *Puccinia triticina*, the causal agent of wheat leaf rust, in Canada in 2004. Can. J. Plant Pathol. 28:566–576.

McIntosh, R.A. 1992. Close genetic linkage of genes conferring adult-plant resistance to leaf rust and stripe rust of wheat. Plant Pathol. 41:523–527.

McIntosh, R.A., C.R. Wellings, and R.F. Park. 1995. Wheat rusts: An atlas of resistance genes. CSIRO, Sydney, Australia, and Kluwer, Dordrecht, The Netherlands.

McIntosh, R.A., Y. Yamazaki, K.M. Devos, J. Dubcovsky, J. Rogers, and R. Appels. 2007. Catalogue of gene symbols for wheat. 2007 Supplement [Online]. KOMUGI integrated wheat science database. Available at http://www.shigen.nig.ac.jp/wheat/komugi/genes/symbolClassList.jsp (verified 6 Dec. 2007)

Milus, E.A., and R.F. Line. 1986a. Number of genes controlling high-temperature, adult-plant resistance to stripe rust in wheat. Phytopathology 76:93–96.

Milus, E.A., and R.F. Line. 1986b. Gene action for inheritance of durable, high-temperature, adult-plant resistance to stripe rust in wheat. Phytopathology 76:435–441.

Mishra, A.N., K. Kaushal, S.R. Yadav, G.S. Shirsekar, and H.N. Pandey. 2005. A leaf rust resistance gene, different from *Lr34*, associated with leaf tip necrosis in wheat. Plant Breed. 124:517–519.

Navabi, A., R.P. Singh, J. Huerta-Espino, and J.P. Tewari. 2005. Phenotypic association of adult-plant resistance to leaf and stripe rusts in wheat. Can. J. Plant Pathol. 27:396–403.

Navabi, A., R.P. Singh, J.P. Tewari, and K.G. Briggs. 2003. Genetic analysis of adult-plant resistance to leaf rust in five spring wheat genotypes. Plant Dis. 87:1522–1529.

Nazareno, N.R.X, and A.P. Roelfs. 1981. Adult plant resistance of Thatcher wheat to stem rust. Phytopathology 71:181–185.

Newton, M., and T. Johnson. 1936. Stripe rust, *Puccinia glumerum*, in Canada. Can. J. Res. 14:89–108.

Niu, Y.C., Z.Q. Li, and H.S. Shang. 1991. *Puccinia striiformis* West. f. sp. *leymi* and f. sp. *elymi*, two new formae speciales. Acta Univ. Agric. Boreali Occident. 19:58–62.

Oelke, L.M., and J.A. Kolmer. 2004. Characterization of leaf rust resistance in hard red spring wheat cultivars. Plant Dis. 88:1127–1133.

Oelke, L.M., and J.A. Kolmer. 2005. Genetics of leaf rust resistance in spring wheat cultivars Norm and Alsen. Phytopathology 95:773–778.

Ordoñez, M.E., and J.A. Kolmer. 2007a. Simple sequence repeat diversity of a world-wide collection of *Puccinia triticina* from durum wheat. Phytopathology 97:574–583.

Ordoñez, M.E., and J.A. Kolmer. 2007b. Virulence phenotypes of a world-wide collection of *Puccinia triticina* from durum wheat. Phytopathology 97:344–351.

Parlevliet, J.E. 1979. Components of resistance that reduce the rate of epidemic development. Annu. Rev. Phytopathol. 17:203–222.

Park, R.L. 2007. Stem rust of wheat in Australia. Aust. J. Agric. Res. 58:558–566.

Park, R.F., H.S. Bariana, C.R. Wellings, and H. Wallwork. 2002. Detection and occurrence of a new pathotype of

Puccinia triticina with virulence for *Lr24* in Australia. Aust. J. Agric. Res. 53:1069–1076.

Park, R.F., J.J. Burdon, and R.A. McIntosh. 1995. Studies in the origin, spread, and evolution of an important group of *Puccinia recondita* f. sp. *tritici* pathotypes in Australasia. Eur. J. Plant Pathol. 101:613–622.

Park, R.F., A. Jahoor, and F.G. Felsenstein. 2000. Population structure of *Puccinia recondita* in western Europe during 1995, as assessed by variability in pathogenicity and molecular markers. J. Phytopathol. 148:169–179.

Peturson, B., M. Newton, and A.G.O. Whiteside. 1945. The effect of leaf rust on the yield and quality of wheat. Can. J. Res. 23:105–114.

Peturson, B., M. Newton, and A.G.O. Whiteside. 1948. Further studies on the effect of leaf rust on the yield, grade, and quality of wheat. Can. J. Res. 26:65–70.

Pope, W.K., E.L. Sharp, and H.S. Fenwick. 1963. Stripe rust of wheat in the Pacific Northwest in 1962. Plant Dis. Rep. 47:554–555.

Pretorius, Z.A., W.H.P. Boshoff, and G.H.J. Kema. 1997. First report of *Puccinia striiformis* f. sp. *tritici* on wheat in South Africa. Plant Dis. 81:424.

Pretorius, Z.A., F.J. Kloppers, and S.C. Drijepondt. 1994. Effect of inoculum density and temperature on three components of leaf rust resistance controlled by *Lr34* in wheat. Euphytica 74:91–96.

Pretorius, Z.A., R.P. Singh, W.W. Wagoire, and T. Payne. 2000. Detection of virulence to wheat stem rust resistance gene *Sr31* in *Puccinia graminis* f. sp. *tritici* in Uganda. Plant Dis. 84:203.

Qayoum, A., and R.F. Line. 1985. High temperature, adult-plant resistance to stripe rust of wheat. Phytopathology 75:1121–1125.

Rapilly, F. 1979. Yellow rust epidemiology. Annu. Rev. Phytopathol. 17:59–73.

Röbbelen, G., and E.L. Sharp. 1978. Mode of inheritance, interaction and application of genes conditioning resistance to yellow rust. Verlag Paul Parey, Berlin and Hamburg.

Roelfs, A.P. 1985. Wheat and rye stem rust. p. 4–38. *In* A.P. Roelfs and W.R. Bushnell (ed.) The cereal rusts. Vol. II. Academic Press, Orlando, FL.

Roelfs, A.P. 1988. Resistance to leaf and stem rusts in wheat. p. 10–22. *In* N.W. Simmonds and S. Rajaram (ed.) Breeding strategies for resistance to the rusts of wheat. CIMMYT, D.F., Mexico.

Roelfs, A.P. 1989. Epidemiology of the cereal rusts in North America. Can. J. Plant Pathol. 11:86–90.

Roelfs, A.P., and J.V. Groth. 1980. A comparison of virulence phenotypes in wheat stem rust populations reproducing sexually and asexually. Phytopathology 70:855–862.

Roelfs, A.P., and J.W. Martens. 1988. An international system of nomenclature for *Puccinia graminis* f. sp. *tritici*. Phytopathology 78:526–533.

Roelfs, A.P. and D.V. McVey. 1979. Low infection types produced by *Puccinia graminis* f. sp. *tritici* and wheat lines with designated genes for resistance. Phytopathology 69:722–730.

Roelfs, A.P., R.P. Singh, and E.E. Saari. 1992. Rust diseases of wheat: Concepts and methods of disease management. CIMMYT, D.F., Mexico.

Rosewarne, G.M., R.P. Singh, J. Huerta-Espino, J. William, S. Bouchet, S. Cloutier, H. McFadden, and E.S. Lagudah. 2006. Leaf tip necrosis, molecular markers, and β1-proteasome subunits associated with the slow rusting genes *Lr46/Yr29*. Theor. Appl. Genet. 112:500–508.

Saari, E.E., H.C. Young, and M.F. Kernkamp. 1968. Infection of North American *Thalictrum* spp. with *Puccinia recondita* f. sp. *tritici*. Phytopathology 58:939–943.

Samborski, D.J., and P.L. Dyck. 1968. Inheritance of virulence in wheat leaf rust on the standard differential wheat varieties. Can. J. Genet. Cytol. 10:24–32.

Samborski, D.J., and P.L. Dyck. 1982. Enhancement of resistance to *Puccinia recondita* by interactions of resistance genes in wheat. Can. J. Plant Pathol. 4:152–156.

Samborski, D.J., and B. Peturson. 1960. Effect of leaf rust in the yield of resistant wheats. Can. J. Plant Sci. 620–622.

Santra, D.K., M. Santra, C. Uauy, K.G. Campbell, X.M. Chen, J. Dubcovsky, and K.K. Kidwell. 2006. Identifying QTL for high-temperature adult-plant resistance to stripe rust in wheat (*Triticum aestivum* L.). p. 179. *In* Abstracts, Plant and Animal Genome Conf. XIV, San Diego, CA. 14–18 Jan. 2006. Scherago International, New York.

Savile, D.B.O. 1984. Taxonomy of the cereal rust fungi. p. 79–114. *In* W.R. Bushnell and A.P. Roelfs (ed.) The cereal rusts. Vol. I. Academic Press, Orlando, FL.

Schmidt, J.K. 1827. Allgemeine ökonomisch-technische Flora oder Abbildungen und Beschreibungen aller in Bezug auf Ökonomie und Technologie, merkwürdigen Gewächse. Vol. I: 27. Jena, Germany.

Schnurbusch, T., S. Paillard, A. Schori, M. Messmer, G. Schachermayr, H. Winzeler, and B. Keller. 2004. Dissection of quantitative and durable leaf rust resistance in Swiss winter wheat reveals a major resistance QTL in the *Lr34* chromosome region. Theor. Appl. Genet. 108:477–484.

Sebesta, E.E., E.A. Wood, Jr., D.R. Porter, J.A. Webster, and E.L. Smith. 1994. Registration of Gaucho greenbug-resistant triticale germplasm. Crop Sci. 34:1428.

Shaner, G., and R.L. Powelson. 1971. Epidemiology of stripe rust of wheat, 1961–1968. Tech. Bull. 117. Oregon Agric. Exp. Stn., Corvallis, OR.

Sharp, E.L., and E.R. Heln. 1963. Overwintering of stripe rust in winter wheat in Montana. Phytopathology 53:1239–1240.

Singh, R.P. 1991. Pathogenicity variations of *Puccinia recondita* f. sp. *tritici* and *P. graminis* f. sp. *tritici* in wheat-growing areas of Mexico during 1988 and 1989. Plant Dis. 75:790–794.

Singh, R.P. 1992a. Association between gene *Lr34* for leaf rust resistance and leaf tip necrosis in wheat. Crop Sci. 32:874–878.

Singh, R.P. 1992b. Expression of wheat leaf rust resistance gene *Lr34* in seedlings and adult plants. Plant Dis. 76:489–491.

Singh, R.P. 1992c. Genetic association of leaf rust resistance gene *Lr34* with adult plant resistance to stripe rust in bread wheat. Phytopathology 82:835–838.

Singh, R.P. 1993. Genetic association of gene *Bdv1* for tolerance to barley yellow dwarf virus with genes *Lr34* and *Yr18* for adult plant resistance to rusts in bread wheat. Plant Dis. 77:1103–1106.

Singh, R.P., E. Bechere, and O. Abdalla. 1993. Genetic analysis of resistance to leaf rust in nine durum wheats. Plant Dis. 77:460–463.

Singh, R.P., D.P. Hodson, Y. Jin, J. Huerta-Espino, M.G. Kinyua, R. Wanyera, P. Njau, and R.W. Ward. 2006. Current status, likely migration and strategies to mitigate the threat to wheat production from race Ug-99 (TTKS) of stem rust pathogen. *In* CAB reviews: Perspectives in agriculture, veterinary science, nutrition, and natural resources 1, No. 054 [Online]. Available at http://www.cababstractsplus.org/cabreviews/Reviews.asp?action=display&openMenu=relatedItems&ReviewID=25637&Year=2006.

Singh, R.P., J. Huerta-Espino, W. Pfeiffer, and P. Figueroa-Lopez. 2004. Occurrence and impact of a new leaf rust race on durum wheat in northwestern Mexico from 2001 to 2003. Plant Dis. 88:703–708.

Singh, R.P., J. Huerta-Espino, and S. Rajaram. 2000. Achieving near-immunity to leaf rust and stripe rust in wheat by combining slow rusting resistance genes. Acta Phytopathol. Entomol. 35:133–139.

Singh, R.P., A. Mujeebkazi, and J. Huerta-Espino. 1998. *Lr46*—A gene conferring slow rusting resistance to leaf rust in wheat. Phytopathology 88:890–894.

Singh, R.P., and S. Rajaram. 1992. Genetics of adult-plant resistance in Frontana and three CIMMYT wheats. Genome 35:24–31.

Smith, R.E. 1961. Wheat rust as viewed in the early agricultural press in California. Plant Dis. Rep. 45:709–712.

Spielmeyer, W., R.A. McIntosh, J.A. Kolmer, and E.S. Lagudah. 2005. Powdery mildew resistance and *Lr34/Yr18* genes for durable resistance to leaf and stripe rust cosegregate at a locus on the short arm of chromosome 7D of wheat. Theor. Appl. Genet. 111:731–735.

Spielmeyer, W., P.J. Sharp, and E.S. Lagudah. 2003. Identification and validation of markers linked to broad-spectrum stem rust resistance gene *Sr2* in wheat (*Triticum aestivum* L.). Crop Sci. 43:333–336.

Stakman, E.C., M.N. Levine, and J.G. Leach. 1919. New biologic forms of *Puccinia graminis*. J. Agric. Res. 16: 103–105.

Statler, G.D. 1973. Inheritance of leaf rust resistance in Leeds durum wheat. Crop Sci. 13:116–117.

Straib, W. 1937. Über Resistenz bei Gerste gegenüber Zwergrost und Gelbrost. Züchter 9:305–311.

Stubbs, R.W. 1985. Stripe rust. p. 61–102. *In* A.P. Roelfs and W.R. Bushnell (ed.) The cereal rusts. Vol. II. Academic Press, Orlando, FL.

Szabo, L.S., and J.A. Kolmer. 2007. Development of simple sequence repeat markers for the plant pathogenic rust fungus *Puccinia triticina*. Mol. Ecol. Notes 7: 708–710.

Tollenaar, H. 1967. A comparison of *Puccinia striiformis* f. sp. *poae* on bluegrass with *P. striiformis* f. sp. *tritici* and f. sp. *dactylidis*. Phytopathology 57:418–420.

Tollenaar, H., and B.R. Houston. 1966. Effect of temperature during uredospore production and of light on in vitro germination of uredospores from *Puccinia striiformis*. Phytopathology 56:787–789.

Tranzschel, W. 1934. Promežutočnye chozjarva rzavčiny chlebov i ich der USSR. (The alternate hosts of cereal rust fungi and their distribution in the USSR). Bull. Plant Prot. Ser. 2:4–40.

Uauy, C., J.C. Brevis, X.M. Chen, I.A. Khan, L.F. Jackson, O. Chicaiza, A. Distelfeld, T. Fahima, and J. Dubcovsky. 2005. High-temperature adult plant (HTAP) stripe rust resistance gene *Yr36* from *Triticum turgidum* ssp. *dicoccoides* is closely linked to the grain protein content locus *Gpc-B1*. Theor. Appl. Genet. 112:97–105.

Viennot-Bourgin, G. 1941. Diagnose Latine die *Puccinia tritic*-duri nov. sp. Ann. Ed. Natl. Grignon Paris C. Amat Ser. 3. 2:146.

Wahl, I., Y. Anikster, J. Manisterski, and A. Segal. 1984. Evolution at the center of origin. p. 39–78. *In* W.R. Bushnell and A.P. Roelfs (ed.) The cereal rusts. Vol I. Academic Press, Orlando, FL.

Waldron, L.R. 1936. The effect of leaf rust accompanied by heat upon yield, kernel weight, bushel weight, and protein content of hard red spring wheat. J. Agric. Res. 53: 399–414.

Wallwork, H. 2007. The role of minimum disease resistance standards for the control of cereal diseases. Aust. J. Agric. Res. 58:588–592.

Wanyera, R., M.G. Kinyua, Y. Jin, and R.P. Singh. 2006. The spread of stem rust caused by *Puccinia graminis* f. sp. *tritici*, with virulence on *Sr31* in wheat in Eastern Africa. Plant Dis. 90:113.

Webster, J. 1980. Introduction to fungi. Cambridge University Press, Oxford, UK.

Wellings, C.R. 2007. *Puccinia striiformis* in Australia: A review of the incursion, evolution, and adaptation of stripe rust in the period 1979–2006. Aust. J. Agric. Res. 58:567–575.

Wellings, C.R., J.J. Burdon, and F.J. Keiper. 2004. The biology of *Puccinia striiformis* on *Hordeum* spp. in Australia: The case for a new forma specialis. p. A1.50. *In* Proc. Int. Cereal Rusts & Powdery Mildews Conf., 11th, John Innes Centre, Norwich, UK. 22–27 Aug. 2004.

Wellings, C.R., and R.A. McIntosh. 1987. *Puccinia striiformis* f. sp. *tritici* in Eastern Australia—possible means of entry and implications for plant quarantine. Plant Pathol. 36:239–241.

Wellings, C.R., D.G. Wright, F. Keiper, and R. Loughman. 2003. First detection of wheat stripe rust in Western Australia: Evidence for a foreign incursion. Aust. Plant Pathol. 32:321–322.

Westendorp, G.D. 1854. Quatrième notice sur quelques *Cryptogames* récemment découvertes en Belgique. Bull. Acad. R. Sci. Belg. 21:229–246.

William, H.M., R.P. Singh, J. Huerta-Espino, G. Palacios, and K. Suenaga. 2006. Characterization of genetic loci

conferring adult plant resistance to leaf rust and stripe rust in spring wheat. Genome 49:977–990.

Williams, M., R.P. Singh, J. Huerta-Espino, S. Ortiz Islas, and D. Hoisington. 2002. Molecular marker mapping of leaf rust resistance gene *Lr46* and its association with stripe rust resistance gene *Yr29* wheat. Phytopathology 93:153–159.

Winter, G. 1884. Repertorium Rabenhorstii fungi europaei et extraeuraopaei. Centuria XXXI et XXXII. Hedwigia 23:164–175.

Worland, A.J., and C.N. Law. 1986. Genetic analysis of chromosome 2D of wheat: I. The location of genes affecting height, day-length insensitivity, hybrid dwarfism and yellow rust resistance. Z. Pflanzenzücht 96:331–345.

Wright, R.G., and J.H. Lennard. 1978. Mitosis in *Puccinia striiformis*: 1. Light microscopy. Trans. Br. Mycol. Soc. 70:91–98.

Xu, X.Y., G.H. Bai, B. Carver, G.E. Shaner, and R.M. Hunger. 2005a. Mapping of QTLs prolonging the latent period of *Puccina triticina* infection in wheat. Theor Appl. Genet. 110:244–251.

Xu, X.Y., G.H. Bai, B. Carver, G.E. Shaner, and R.M. Hunger. 2005b. Molecular characterization of slow leaf-rusting resistance in wheat. Crop Sci. 45:758–765.

Yehuda, P.B., T. Eilam, J. Manisterski, A. Shimoni, and Y. Anikster. 2004. Leaf rust on *Aegilops speltoides* caused by a new forma specialis of *Puccinia triticina*. Phytopathology 94:94–101.

Zhang, H., and D.R. Knott. 1990. Inheritance of leaf rust resistance in durum wheat. Crop Sci. 30:1218–1222.

Zhang, H., and D.R. Knott. 1993. Inheritance of adult plant resistance to leaf rust in six durum wheat cultivars. Crop Sci. 33:694–697.