

## Epidemiology and control of stripe rust [*Puccinia striiformis* f. sp. *tritici*] on wheat

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**Abstract:** Stripe rust of wheat, caused by *Puccinia striiformis* f. sp. *tritici*, is one of the most important diseases of wheat worldwide. This review presents basic and recent information on the epidemiology of stripe rust, changes in pathogen virulence and population structure, and movement of the pathogen in the United States and around the world. The impact and causes of recent epidemics in the United States and other countries are discussed. Research on plant resistance to disease, including types of resistance, genes, and molecular markers, and on the use of fungicides are summarized, and strategies for more effective control of the disease are discussed.

**Key words:** disease control, epidemiology, formae speciales, races, *Puccinia striiformis*, resistance, stripe rust, wheat, yellow rust.

**Résumé :** Mondialement, la rouille jaune du blé, causée par le *Puccinia striiformis* f. sp. *tritici*, est une des plus importantes maladies du blé. La présente synthèse contient des informations générales et récentes sur l'épidémiologie de la rouille jaune, sur les changements dans la virulence de l'agent pathogène et dans la structure de la population et sur les déplacements de l'agent pathogène aux États-Unis et autour de la planète. L'impact et les causes des dernières épidémies qui ont sévi aux États-Unis et ailleurs sont examinés. La synthèse contient un résumé de la recherche sur la résistance des plantes à la maladie, y compris les types de résistance, les gènes et les marqueurs moléculaires, et sur l'emploi des fongicides, et un examen des stratégies pour une lutte plus efficace contre la maladie.

**Mots clés :** lutte contre les maladies, épidémiologie, formae speciales, races, *Puccinia striiformis*, résistance, rouille jaune, blé.

### Introduction

Although stripe rust probably occurred long before wheat was grown for food, the disease was first described by Gadd in Europe in 1777 (Eriksson and Henning 1896). The first reports of stripe rust and its distribution around the world were given by Hassebrauk (1965), Stubbs (1985), Line (2002), and Li and Zeng (2003). Stripe rust of wheat has been reported in more than 60 countries and on all continents except Antarctica.

Research on the epidemiology and control of stripe rust has been conducted for more than a century and reviewed in several excellent articles. Hassebrauk wrote a treatise in Germany that was published in four parts (Hassebrauk 1965, 1970; Hassebrauk and Röbbelen 1974, 1975). The section on the genetics of host-pathogen relationships and resistance breeding was revised and translated into English by Röbbelen and Sharp (1978). Later, various aspects of stripe rust were reviewed by Rapilly (1979), Stubbs (1985), Line and Qayoum (1992), and Line (2002). This review

provides important basic and recent information on epidemiology and control of stripe rust, including research conducted by the author.

### The disease

#### Symptoms, disease development, and signs of the pathogen

The pathogen causing stripe rust infects the green tissues of plants of cereal crops and grasses. Infection can occur anytime from the one-leaf stage to plant maturity provided plants are still green. Symptoms appear about 1 week after infection, and sporulation starts about 2 weeks after infection, under optimum temperature conditions. The fungus forms tiny, yellow- to orange-colored rust pustules, called uredia. Each uredium contains thousands of urediniospores. A single urediniospore is too small to be seen with the naked eye, but spores on mass are yellow- to orange-colored and powdery. Stripes of uredia or necrosis are not formed on the leaves of seedlings, but as the plants age, generally after stem elongation. Depending on the level of plant resistance and the temperature, various amounts of chlorosis or necrosis appear (hypersensitive response), with or without sporulation. The necrotic stripes or elongated spots that form on leaves of adult plants are distinguishable from spots caused by necrotrophic pathogens. The pathogen of

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stripe rust utilizes water and nutrients from the host plants, which weakens the plants.

### Environmental factors affecting stripe rust

As with other diseases, the three factors in the disease triangle are all essential for disease development. However, the development of stripe rust, compared with many other diseases, depends even more on the very specific weather conditions when pathogen inoculum (urediniospores) and susceptible host plants are present. The three most important weather factors affecting epidemics of stripe rust are moisture, temperature, and wind.

Moisture affects spore germination, infection, and survival. Urediniospores require at least 3 h of continuous moisture (dew formation) on the plant surfaces to germinate and infect plants (Rapilly 1979). Moist regions with frequent dew formation during the growing season provide conditions conducive for stripe rust. Western Washington and western Oregon in the Pacific Northwest (PNW) of the United States typically have cool and moist-weather patterns, and therefore, stripe rust occurs there every year. Because high moisture promotes infection, irrigated cereal crops are vulnerable to stripe rust. In rain-fed areas, light rains create the ideal conditions for infection. After rains, high moisture in the air and soil often creates dew for several nights, providing favorable conditions for infection. Rain can also disperse spores to spread the disease because raindrops release urediniospores either by direct impact or by splashing (Rapilly 1979). Stripe rust can be predicted by monitoring precipitation in a region and dew formation in the fields. High moisture promotes disease by favoring spore germination, but also adversely affects spore survival. Because urediniospores do not exhibit fungistasis, they can germinate immediately after they are produced if dew is present and the temperature is in the correct range. Since urediniospores kept under high-moisture conditions lose viability more quickly than those kept dry, spores should be kept dry to maintain viability during sample shipment and storage. With regard to epidemics of stripe rust, dry spores survive longer than moist spores, and therefore, are more likely to survive to infect next crops and be disseminated over longer distances. The dry weather in late summer and the various stages of wheat crops in the PNW allow urediniospores produced on late-harvested spring wheat to survive the summer and be able to infect seedlings of winter wheat planted in the fall. This is one of the reasons why stripe rust occurs every year in this region. Moisture also affects spore dispersal. Depending on the relative humidity, urediniospores can spread individually or in clusters (Rapilly 1979). Clusters increase in size as humidity increases. High humidity leads to stronger adhesion of urediniospores to the leaves (Rapilly 1979). Nevertheless, high humidity generally increases disease by increasing infection frequency.

Temperature affects spore germination and infection, latent period, sporulation, spore survival, and host resistance. *Puccinia striiformis* Westend. prefers cool climates, so stripe rust mainly occurs in temperate regions and areas of high elevation in tropical regions. The disease can start very early in the crop season and, therefore, can cause more severe damage in some areas than leaf rust [*Puccinia triticina* Eriks.] and stem rust [*Puccinia graminis* Pers.:Pers. f. sp.

*tritici* Eriks. & E. Henn.], which have higher temperature optima for development. Effects of temperature on stripe rust were well reviewed by Rapilly (1979) and Line (2002). Variation exists among isolates of *P. striiformis* for response to temperature. In a recent study, Milus and Seyran (2004) determined the spore germination rate and latent period of 6 isolates collected before 2000 to represent "old races" and of 14 isolates collected after 2000 to represent "new races". They found significant temperature  $\times$  isolate interactions for latent period and spore germination rate. Eight of the 14 new isolates, and only 1 of the 6 old isolates, germinated significantly faster at 18 °C than at 12 °C. Two of the six old isolates germinated significantly faster at 12 °C than at 18 °C. All new isolates had significantly shorter latent periods at 18 °C than at 12 °C. Of the six old isolates, four had similar latent periods at both temperatures, one had a shorter latent period at 18 °C, and one had a shorter latent period at 12 °C. These results indicate that stripe rust caused by the new isolates tends to develop faster than the old isolates at relatively high temperatures.

With regard to the effects of temperature on development of stripe rust, night temperatures play a more critical role than daytime temperatures (Stubbs 1985). Both dew formation and low temperatures occur together most frequently at night, and therefore, infections are more likely to occur at night. Hot weather, especially hot nights, limits disease development and survival of the pathogen. The use of average and maximum daily temperatures to determine if stripe rust occurs in a region or in a season is misleading. For example, if an area has a daily maximum temperature of 28 °C and minimum temperature of 22 °C, while another area has a daily maximum temperature of 34 °C and minimum temperature of 16 °C, then both areas have an average daily temperature of 25 °C. If we use both the daily average and maximum temperatures, we would conclude that the first area is more likely to have stripe rust. In reality, the second area is more likely to have stripe rust because it has a period of time with temperatures that allow infection, while the lowest temperature in the first area would be marginal for infection to occur. The temperature range in the second area is typical of most areas in the PNW in the late spring and summer. The cool weather at night allows stripe rust to develop and the pathogen to survive.

Temperature is the major factor effecting the winter survival of the pathogen of stripe rust. Rapilly (1979) considered that temperatures below -10 °C might halt the pathogen development. Cold-weather conditions reduce the pathogen winter survival by winter killing the pathogen in the infected leaves. Therefore, temperature is one of the major weather factors used to predict occurrence of stripe rust. In the late 1970s and early 1980s, Coakley and Line (reviewed by Line 2002) developed models for forecasting stripe rust in the PNW based on accumulated negative degree days (daily temperatures lower than 7 °C, the optimum temperature for infection) from 1 December to 31 January and on accumulated positive degree days (daily temperatures higher than 7 °C) from 1 April to 30 June. The models explained why stripe rust was not a major problem in the 20-year period before 1960, but was a major problem from 1960 to 1979 in the PNW. The models have also been used in the United States and Canada for predicting stripe rust.

Chen et al. (2003a) analyzed the weather data in 2001 and 2002 for Washington State and identified weather factors favorable for the epidemic of 2002. The temperatures from 1 November 2001 to 31 January 2002 were significantly higher than normal and the temperatures in May 2002 significantly lower; more rain occurred in 2002 than in 2001. Similarly, X.M. Chen (unpublished data<sup>1</sup>) analyzed weather data in Manhattan, Kansas (USA), from 1957 to 2003 and explained the epidemics of 1957, 1958, 2001, and 2003. In general, temperatures in late May and June for those years were significantly lower than normal (the average over 100 years). For example, the monthly mean temperature in May 2003 was 17.4 °C, which is 0.9 °C lower than normal (18.3 °C), and in June 2003, it was 21.4 °C, which is 2.3 °C higher than normal (23.7 °C). Unlike the PNW, the central Great Plains of United States do not have normal temperatures favorable for stripe rust in May and June. However, temperatures below normal are favorable for the disease. Temperature data, coupled with precipitation in May and June and incidence of stripe rust in the spring in Texas (USA), should provide accurate forecasting of stripe rust for the central Great Plains.

Wind can affect *P. striiformis* by drying urediniospores, which reduces on-site germination and infection, but also increases the duration of spore viability. More importantly, wind plays a major role in the spread of stripe rust. Wind is generally not a limiting factor within a small area, especially in an area with local inoculum. For example, in the region of eastern Washington, northern Idaho (USA), and northeastern Oregon (about 300 km from the West to the East and 400 km from the South to the North), *P. striiformis* can overwinter and oversummer. The region has its own local inoculum, but is also under the influence of inoculum from outside of the region. Stripe rust usually starts in the south of the region (northern Oregon and south central Washington) because of its warmer weather and early planted crops, then is quickly spread by wind throughout the region. Wind can be a limiting factor for spreading rust spores over long distances. The central (such as Kansas and Nebraska) and northern (South Dakota and North Dakota) Great Plains of United States usually receive inoculum of stripe rust from the southern Great Plains (Texas and Louisiana). The timing, type, and direction of winds determine the earliness, scale, and development rate of epidemics of stripe rust. For the large area east of the Rocky Mountains, initial infections occur in the south (Texas) in the fall, and the rust continues to increase even in the winter. This area provides spores for the eastern and the northern portion of the region. Depending on the wind direction when spores are produced in southern Texas, we can predict whether the disease will spread eastward and (or) northward.

The spread patterns of stripe rust in the Great Plains and the southeastern United States from 2000 to 2004 (Fig. 1) based on annual surveys by collaborators can be explained by the storms that occurred in the spring of each year. Inoculum was available in the southwest of the region (Texas) in all 4 years, but the central region had severe stripe rust only in 2001 and 2003. In 2000 and 2002, storms

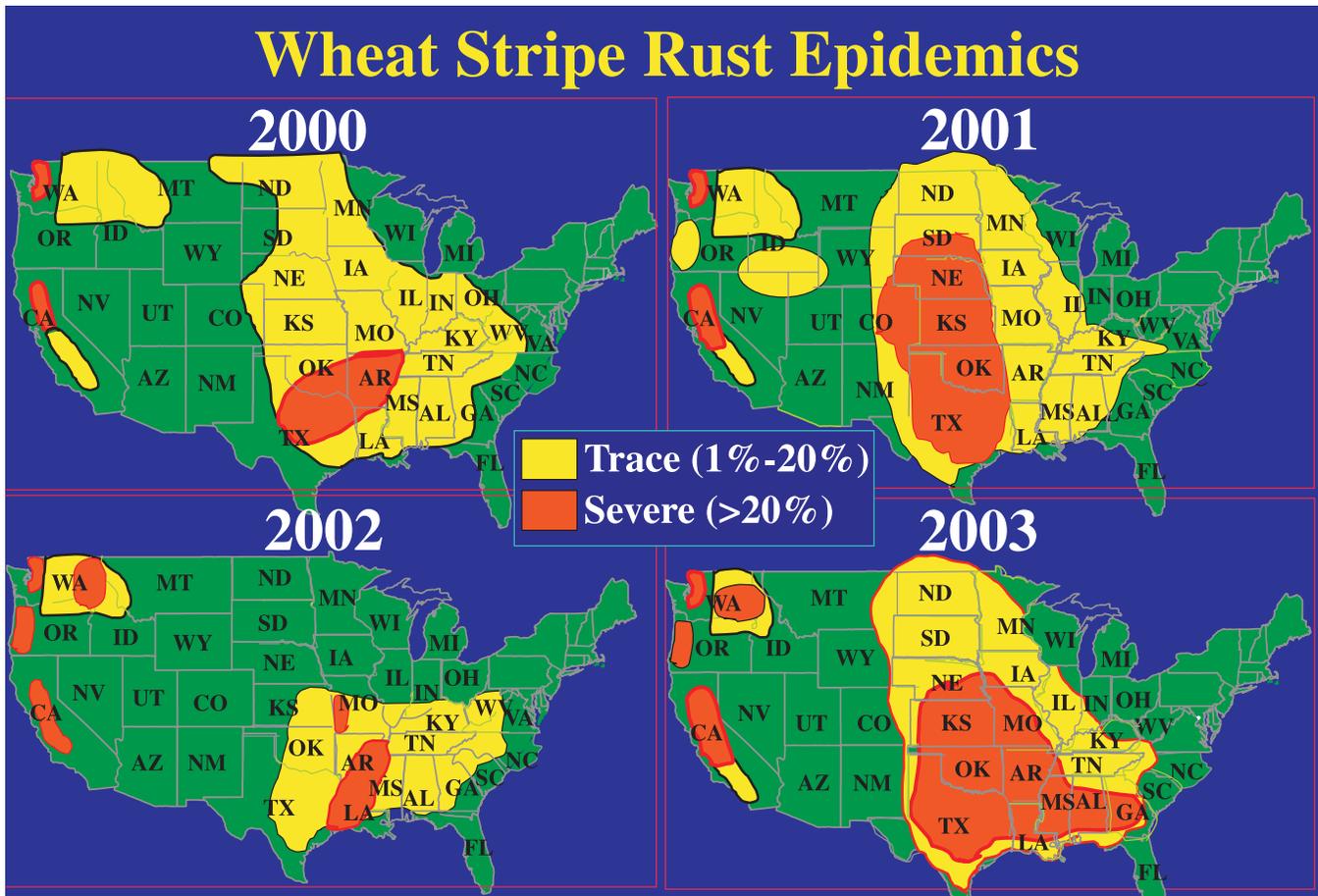
spread spores mostly eastward. In 2003, the disease started developing earlier than in the previous years; the first sample of stripe rust was collected as early as 18 January from southern Texas. The author predicted an epidemic of stripe rust for the region when he saw the major storm moving from South to North in April 2003 (X.M. Chen 2004, unpublished data).

## Impact of stripe rust

Stripe rust reduces the yield and quality of grain and forage. Seed produced from crops damaged by stripe rust have low vigor and thus poor emergence after germination. Stripe rust can cause 100% yield losses if infection occurs very early and the disease continues developing during the growing season. We have often observed 100% yield losses of highly susceptible wheat lines such as PS 279 in our experimental fields near Mount Vernon, Washington, and occasionally observed complete yield losses near Pullman, Washington. In most wheat-producing areas, yield losses caused by stripe rust have ranged from 10% to 70% depending on susceptibility of the cultivar, earliness of the initial infection, rate of disease development, and duration of disease.

Estimates of yield losses of cereals caused by rusts since 1918 are maintained by the United States Department of Agriculture, Agricultural Research Service, Cereal Disease Laboratory in St. Paul, Minnesota, at the Web site <http://www.cdl.umn.edu/loss.html>. However, wheat yield losses caused by stripe rust were not recorded until 1958 when a loss of 2.9 million bushels (about 78 996 t and 4% of production) was recorded for the State of Washington. The most severe yield losses recorded for Washington State were 25% (591 108 t) in 1960 and 17% (787 236 t) in 1976. In the past 46 years in the Washington State (from 1958 to 2003), stripe rust occurred almost every year. Severe damage (a statewide yield loss of 5% or more) occurred in 12 of the 46 years, all of which occurred before 1990 and most of which before 1984. Yield losses were generally low after the early 1980s because of the use of resistant cultivars and the application of effective fungicides. In 2002, weather conditions were extremely favorable for stripe rust in Washington State and affected 440 000 acres (70% of the acreage; 1 acre = 0.404 685 6 ha) of the spring-wheat crop and 45 000 acres (2.5% of the acreage) of the winter-wheat crop, 20% of the total wheat acreage. About 170 000 acres were sprayed with fungicides at a cost of more than US\$2.5 × 10<sup>6</sup>. Without the fungicide application, stripe rust could have caused a yield loss of 20%–25% (155 268 – 193 404 t), valued at about US\$26 × 10<sup>6</sup> to US\$33 × 10<sup>6</sup>. Similar frequent epidemics have occurred in Oregon and Idaho because of their geographic proximity to Washington State and similar weather conditions and cropping systems. Significant damage caused by stripe rust was not recorded in California (USA) until 1974, when a yield loss of 8% was estimated. From 1975 to 1997, yield losses in California were below 1%. Beginning in 1998, stripe rust became an increasingly severe problem. In 2003, the disease caused

<sup>1</sup>Chen, X.M. 2004. Changing stripe rust epidemiology and prospects for control. Invited oral presentation on 24 February, section Fungal Pathology presided by B. Bowden, at the National Wheat Worker's Workshop, 22–25 February 2004, Kansas City, Missouri, USA.

**Fig. 1.** Distribution of stripe rust [*Puccinia striiformis* f. sp. *tritici*] in the United States from 2000 to 2003.

a 25% yield loss (Table 1) and the epidemic was so severe and widespread that fungicides were used for the first time in California. In addition to the favorable weather conditions in 2003, the major reason for the severity of the epidemic was that the major wheat cultivars with race-specific resistance were overcome by new races of *P. striiformis* f. sp. *tritici* Eriks.

In the United States, stripe rust was primarily a problem in the PNW (Washington, Oregon, and Idaho) and California prior to 2000. Since 2000, the disease has become increasingly important in the south central states (Texas, Louisiana, Oklahoma, Arkansas, and Mississippi) and the central Great Plains (Kansas, Colorado, Missouri, and Nebraska). In Arkansas, the only significant epidemic that was recorded before 2000 occurred in 1987 with a statewide yield loss of 2.5%. The disease caused yield losses of 7%, 5%, and 3% in 2000, 2002, and 2003, respectively, plus the millions of dollars that were spent on fungicide applications each year. Stripe rust seldom caused significant damage in Kansas before 2000, but the State suffered yield losses of 7.3% (706 606 t) in 2001 and 10.6% (1 573 110 t) in 2003. Similarly, Colorado had an 8% (156 358 t) yield loss in 2001, and Nebraska had a 10% (256,328 metric tons) yield loss in 2003. Because of the large acreage of wheat grown in the central Great Plains, especially Kansas, yield losses to stripe rust in 2003 ( $2.42 \times 10^6$  t) were a record for the United States (Chen et al. 2004). Including the cost of fun-

gicide applications, losses due to stripe rust amounted to more than  $\text{US}\$300 \times 10^6$  in the United States in 2003. Yield losses in the United States from 2000 to 2003 are listed in Table 1.

In recent years, stripe rust caused severe damage to wheat in several other countries. Although the disease was observed for the first time in South Africa in 1996, it caused a widespread epidemic in spring wheat that year because of cultivar susceptibility and favorable weather conditions (Pretorius 2004). Epidemics occurred again in the central and western Free State in 1997, the eastern Free State in 1998, and all wheat-growing areas characterized by a weather pattern of summer rainfall in 2002. The 1998 epidemic on about 42 000 ha of winter wheat in the eastern Free State resulted in losses of  $\text{ZAR} 12 \times 10^6$  ( $\sim\text{US}\$2.25 \times 10^6$ ) (Pretorius 2004). An epidemic of stripe rust occurred in China in 2002 and affected about  $6.6 \times 10^6$  ha of wheat in 11 provinces and caused a yield loss of  $1.3 \times 10^6$  t (Wan et al. 2004). Stripe rust was a dominant disease in Central Asia (Kazakhstan, Kyrgyzstan, Tajikistan, Turkmenistan, and Uzbekistan) in the late 1990s and early 2000s, accounting for yield losses of 20%–40% in 1999 and 2000 (Morgounov et al. 2004). A severe epidemic in Australia in 1983 cost wheat growers  $\text{AU}\$8 \times 10^6$  for fungicide application to control stripe rust while an epidemic in 2003 cost  $\text{AU}\$40 \times 10^6$  for fungicide application (Wellings and Luig 1984; Wellings and Kandel 2004).

**Table 1.** Wheat yield losses caused by stripe rust [*Puccinia striiformis* f. sp. *tritici*] in the United States from 2000 to 2003.

Location <sup>a</sup>	Yield loss in metric tons (t) and as percentage (%) of production							
	2000		2001		2002		2003	
	(t)	(%)	(t)	(%)	(t)	(%)	(t)	(%)
Arkansas	597 304	7.00	—	<0.01	268 771	5.00	116 485	3.00
California	100 137	3.00	74 009	2.00	196 057	6.00	1 021 520	25.00
Colorado	—	<0.01	758 471	8.00	—	0.00	102 793	1.00
Idaho	—	<0.01	0	0.00	58 361	0.50	58 282	0.50
Kansas	23 683	0.05	3 428 392	7.30	—	<0.01	7 632 502	10.60
Louisiana	6 542	0.50	5 339	0.50	36 159	3.00	40 348	5.00
Nebraska	—	<0.01	39 317	0.50	—	0.00	1 243 176	10.00
Oklahoma	194 564	1.00	499 071	3.00	—	<0.01	239 485	1.00
Oregon	11 656	1.50	1 401	0.10	19 388	0.50	1 857 777	0.10
South Dakota	—	<0.01	16 137	1.00	—	0.00	163 388	2.00
Texas	44 273	0.50	218 974	1.50	106 678	1.00	398 960	3.00
Washington	220 322	1.00	212 604	1.00	363 568	2.30	342 925	2.00
United States total <sup>a</sup>	1 198 480	0.40	5 240 498	2.80	1 056 687	0.5	11 746 401	3.40

**Note:** The data for yield losses were extracted from the database maintained by the United States Department of Agriculture, Agricultural Research Service, Cereal Disease Laboratory in St. Paul, Minnesota, at the Web site <http://www.cdl.umn.edu/loss/loss.html>. —, occurrence of stripe rust, but no significant yield loss.

<sup>a</sup>Not all states included in the United States total are listed among the locations, but only the 12 states that had the most important yield losses.

## The pathogen

### Taxonomy and biology

Stripe rusts of cereal crops and grasses are caused by different formae speciales of *P. striiformis*, a fungus in the order Uredinales of Basidiomycetes. The species name of the pathogen had undergone several changes, *Uredo glumarum* (Schmidt 1827), *Puccinia striaeformis* (Westendorp 1854), *Puccinia straminis* (Fuckel 1860), *Puccinia glumarum* (Eriksson and Henning 1894), before its current name was revived in 1953 (Hylander et al. 1953; Stubbs 1985). Its life cycle consists of dikaryotic uredial and telial stages. Teliospores can germinate to form haploid basidiospores, but unlike the pathogens causing stem rust and leaf rust, the pathogen of stripe rust does not have any known alternate hosts for the basidiospores to infect, and thus, it does not have any known pycnial and aecial stages.

### Formae speciales

*Puccinia striiformis* is subdivided into formae speciales based on specialization on different genera and species of host plants. Eriksson (1894) was the first to separate *P. striiformis* into formae speciales and reported five formae speciales: *P. striiformis* f. sp. *tritici* on wheat, *P. striiformis* f. sp. *hordei* on barley, *P. striiformis* f. sp. *secalis* on rye, *P. striiformis* f. sp. *elymi* on *Elymus* spp., and *P. striiformis* f. sp. *agropyron* on *Agropyron* spp. Later, three more formae speciales were reported: *P. striiformis* f. sp. *dactylidis* on orchard grass (*Dactylis glomerata* L.) (Manners 1960; Tollenaar 1967), *P. striiformis* f. sp. *poae* on Kentucky blue grass (*Poa pratensis* L.) (Britton and Cummins 1956; Tollenaar 1967), and *P. striiformis* f. sp. *leymii* on *Leymus secalinus* (Georgi) Tzvel. (Niu et al. 1991). More recently, Wellings et al. (2004a) considered *P. striiformis* on grass *Hordeum* spp. in Australia to be a new forma specialis, which differs from both *P. striiformis*

f. sp. *tritici* and *P. striiformis* f. sp. *hordei*. The subdivision of *P. striiformis* into formae speciales, especially *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei*, has been questioned because of their overlapping host ranges (Sydow and Sydow 1904; Straib 1935; Newton and Johnson 1936). However, Zadoks (1961) and Stubbs (1985) considered them to be different based on greenhouse and field studies. Newton et al. (1985) showed that *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei* were different based on isozyme and double-stranded RNA analyses. In the United States, stripe rust occasionally occurred in barley fields, but had never caused significant damage before 1991 when *P. striiformis* f. sp. *hordei* first appeared in this country (Line and Qayoum 1992). Chen et al. (1995a) clarified the relationships of *P. striiformis* f. sp. *hordei*, *P. striiformis* f. sp. *tritici*, and *P. striiformis* f. sp. *poae* (from blue grass), using virulence and random-amplified polymorphic DNA (RAPD) analyses. They reported that isolates of *P. striiformis* f. sp. *hordei* were avirulent on most wheat cultivars, and that isolates of *P. striiformis* f. sp. *tritici* were avirulent on most barley cultivars. Isolates of *P. striiformis* f. sp. *hordei* and *P. striiformis* f. sp. *tritici* did not infect blue grass, and isolates of *P. striiformis* f. sp. *poae* did not infect barley or wheat. Random-amplified polymorphic DNA analyses separated the isolates of *P. striiformis* f. sp. *hordei*, *P. striiformis* f. sp. *tritici*, and *P. striiformis* f. sp. *poae* from each other. *Puccinia striiformis* f. sp. *hordei* and *P. striiformis* f. sp. *tritici* were more closely related to each other than they were to *P. striiformis* f. sp. *poae*. Among the formae speciales, *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei* are far more important economically than the others. Because the epidemiology of stripe rust on barley was recently reviewed (Brown et al. 2001) and because races of the pathogen in the United States were summarized (Chen 2004), this review will focus on stripe rust of wheat.

## Races of the pathogen of stripe rust on wheat

The formae speciales *P. striiformis* f. sp. *tritici*, the pathogen causing stripe rust on wheat, is further separated into races based on avirulence or virulence to cultivars or genotypes of wheat. Races are differentiated by infection types produced on a set of selected plant genotypes or single-gene lines that are referred to as differentials. Hungerford and Owens (1923) were the first to report that “specialized varieties” occurred in *P. striiformis* based on specificity on genera of wheat and grasses. However, Allison and Isenbeck (1930) were the first to establish the existence of races in *P. striiformis* f. sp. *tritici* based on specificity on wheat cultivars.

### Race studies in Europe

In Europe, extensive studies on races of *P. striiformis* f. sp. *tritici* were conducted by Gassner and Straib (1932) in Germany in the 1930s. They introduced a set of differential genotypes of wheat, barley, and rye. The set was used by Fuchs (1960) in cooperation with Zadoks (1961) for race surveys in Europe, and later for international race surveys by Stubbs (1985). Johnson et al. (1972) introduced the binary notation for races of *P. striiformis* f. sp. *tritici* and eliminated the use of barley and rye cultivars as differentials. The original set of differential wheat cultivars in this race differentiation system included two groups, the “world differentials” and the “European differentials”. The world differential genotypes included ‘Chinese 166’, ‘Lee’, ‘Heines Kolben’, ‘Vilmorin 23’, ‘Moro’, ‘Strubes Dickkopf’, and ‘Suwon 92 × Omar’. The European differential genotypes included Hybrid 46, ‘Riechersberg 42’, ‘Heines Peko’, ‘Nord Desprez’, ‘Compair’, ‘Carstens V’, ‘Spaldings Prolific’, and ‘Heines VII’. ‘Clement’ was added to the world set by Johnson and Taylor (1976), and *Triticum aestivum* subsp. *spelta* (L.) Thell. ‘Album’ was added to the world set by Wellings and McIntosh (1990). This system is still used by countries in Europe and some countries on other continents (Singh 1992; Boshoff et al. 2002; Calonnec et al. 2002; Wellings and Kandel 2004). In addition to the standard world and European set of differential genotypes, other cultivars and (or) single *Yr* gene lines have often been used as supplemental differentials to describe virulence or avirulence patterns of isolates of *P. striiformis* f. sp. *tritici* (Boshoff et al. 2002; Hovmøller et al. 2002; Wellings and Kandel 2004).

### Race studies in China

The race identification system for *P. striiformis* f. sp. *tritici* in China evolved using a different set of wheat differentials. Fang (1944) conducted the first study, using seven wheat genotypes (‘Carstens V’, ‘Heines Kolben’, ‘Vilmorin 23’, 9 H 77, ‘Hybrid 128’, ‘Carina’, and ‘Michigan Amber’) and a barley genotype, ‘Heil’s Franken’, and identified nine races (designated C1 to C9) in southwestern China. Lu et al. (1956) used Gassner and Straib’s (1932) 14 differential genotypes and identified 16 races from 50 samples. Later, Chinese scientists found that the differential genotypes used in Europe were not suitable for differentiating

races in China, and selected seven wheat genotypes as differentials, using CYR (for Chinese yellow rust) and numbers to designate races of *P. striiformis* f. sp. *tritici* (Wang et al. 1963). Of these seven differential genotypes, only ‘Strubes Dickkopf’ was also used in Germany and later included in the world differential set. Although changes among differentials have been made several times since the early 1960s, the race nomenclature system has been stable. The current Chinese differential set consists of 17 wheat genotypes (‘Trigo Eureka’, ‘Fulhard’, ‘Letescens 128’, ‘Mentana’, ‘Virgilio’, ‘Abbondanza’, ‘Early Premium’, ‘Funo’, ‘Danish 1’, ‘Jubilejina 2’, ‘Fengchan 3’, ‘Lovrin 13’, ‘Kangyin 655’, ‘Shuiyuan 11’, ‘Zhong 4’, ‘Lovrin 10’, and Hybrid 46) (Wan et al. 2004). Among the 17 differentials, only Hybrid 46 is in common with the current European differential set. Traditionally, the major virulence patterns were named as races with CYR while similar virulence patterns, especially virulence to key differential genotypes such as ‘Lovrin 10’, ‘Lovrin 13’, Hybrid 46, and ‘Shuiyuan 11’, were regarded as “pathotypes”. For example, the first race virulent on Hybrid 46 detected in 1991 was designated CYR30 (virulent on ‘Trigo Eureka’, ‘Fulhard’, ‘Lutescens 128’, ‘Mentana’, ‘Virgilio’, ‘Abbondanza’, ‘Early Premium’, ‘Funo’, ‘Danish 1’, ‘Fengchan 3’, ‘Lovrin 13’, ‘Lovrin 10’, and Hybrid 46, but avirulent on ‘Jubilejina 2’, ‘Kangyin 655’, ‘Shuiyuan 11’, and ‘Zhong 4’), while an isolate detected in 1996 that had all CYR30 virulence factors plus virulence on ‘Jubilejina 2’ was designated as pathotype H46-9 (Wan et al. 2004). Some previous pathotypes were late redesigned as CYR races. These “pathotypes” should be treated as races, and thus, a total of 67 races (32 CYR races and 35 “pathotypes”) have been identified based on their virulence and avirulence patterns on the 17 differential genotypes (Wan et al. 2004).

### Race studies in North America

Early indications of *P. striiformis* f. sp. *tritici* races in North America were obtained from studies of host range and cultivar resistance (Hungerford and Owens 1923; Allison and Isenbeck 1930). Line (2002) reviewed the research on race identification in North America beginning in the 1920s. The current system for race identification in the United States was first established by Line and his colleagues in 1968 (Line et al. 1970). The first set of differential genotypes consisted of five wheat genotypes (‘Lemhi’, ‘Chinese 166’, ‘Heines VII’, ‘Moro’, and ‘Suwon 92 × Omar’). Over the last three decades, the number of wheat genotypes used for differentiating races of *P. striiformis* f. sp. *tritici* has grown to 20, as listed in Table 2 (Chen et al. 2002). For race identification, plants were incubated in the dark for 16–24 h at a constant temperature of 10 °C in dew chambers during inoculation, then submitted to a diurnal cycle of temperatures, gradually changing from 4 °C at 0200 to 20 °C at 1400, under a daily photoperiod of 16 h (light), after inoculation. The infection types of plants are recorded 18–22 days after inoculation, using a 0 to 9 scale (Line et al. 1970). Races of *P. striiformis* f. sp. *tritici* in North America were sequentially designated by a CDL number before 2000 (Line and Qayoum 1992). CDL stands for Cereal Disease Laboratory of the United States Depart-

**Table 2.** Wheat genotypes used to differentiate races of *Puccinia striiformis* f. sp. *tritici* in the United States.

Differential No.	Cultivar or line	Genotype identification No. <sup>a</sup>	Type of wheat	Yr gene <sup>b</sup>	Year of incorporation to differential set
1	'Lemhi'	CI 011415	Spring	<i>Yr21</i>	1968
2	'Chinese 166'	CI 011765	Winter	<i>Yr1</i>	1968
3	'Heines VII'	PI 201195	Winter	<i>Yr2, YrHVII</i>	1968
4	'Moro'	CI 013740	Winter	<i>Yr10, YrMor</i>	1968
5	'Paha'	CI 014485	Winter	<i>YrPa1, YrPa2, YrPa3</i>	1974
6	'Druchamp'	CI 013723	Winter	<i>Yr3a, YrD, YrDru</i>	1969
7	'Riebesel 47-51' or Yr5 <sup>c,d</sup>	YR 00004	Spring	<i>Yr5</i>	2004
8	'Pro dura'	CI 017460	Spring	<i>YrPr1, YrPr2</i>	1974
9	'Yamhill'	CI 014563	Winter	<i>Yr2, Yr4a, YrYam</i>	1974
10	'Stephens'	CI 017596	Winter	<i>Yr3a, YrS, YrSte</i>	1976
11	'Lee'	CI 012488	Spring	<i>Yr7, Yr22, Yr23</i>	1977
12	'Fielder'	CI 017268	Spring	<i>Yr6, Yr20</i>	1980
13	'Tyee'	CI 017773	Winter	<i>YrTye</i>	1983
14	'Tres'	CI 017917	Winter	<i>YrTr1, YrTr2</i>	1989
15	'Hyak'	PI 511674	Winter	<i>Yr17</i>	1990
16	'Express'	DA 984034	Spring	Unknown	1998
17	Yr8 <sup>b</sup>	YR 000008	Spring	<i>Yr8</i>	2000
18	Yr9 <sup>b</sup>	YR 000009	Spring	<i>Yr9</i>	2000
19	'Clement'	PI 518799	Winter	<i>Yr9, YrCle</i>	2000
20	'Compair'	PI 325842	Spring	<i>Yr8, Yr19</i>	2000

<sup>a</sup>CI, crop index number; PI, plant identification number; YR, line number for resistance gene to yellow rust.

<sup>b</sup>Refer to Chen and Line (1992a, 1992b, 1993), Chen et al. (1995b, 1998a), and McIntosh et al. (1998) for the *Yr* genes.

<sup>c</sup>'Riebesel 47-51' (Yr9) was used as differential No. 7 before 2004, but was replaced by the Yr5 near-isogenic line. 'Riebesel 47-51' was resistant (infection type (IT) 0) to all races detected before 2000, but it had ITs 0, 2, 3, 4, 5, 6, and 7 when tested with races detected after 2000. Urediniospores collected from 'Riebesel 47-51' with ITs 4 to 7 produced lower ITs and eventually were unable to produce spores. This change of differential No. 7 did not change the description of avirulence and virulence for all the described races.

<sup>d</sup>Yr5, Yr8, and Yr9, developed by the Plant Breeding Institute, University of Sydney, Australia, are near-isogenic lines in the 'Avocet Susceptible' background (Wellings et al. 2004b).

ment of Agriculture, Agricultural Research Service located at Pullman, Washington. The race prefix was changed to PST for *P. striiformis* f. sp. *tritici* because the laboratory name had been changed and because PSH had been used as the prefix for races of *P. striiformis* f. sp. *hordei* (Chen et al. 1995a, 2002).

A total of 109 races of *P. striiformis* f. sp. *tritici* have been identified in the United States (Table 3), of which 59 were identified before, and 50 since, the year 2000. Nearly as many races were identified in the past 4 years as in the previous 40 years before 2000 because the disease was more widespread in those years and several differentials were added. Detailed descriptions for races PST-1 to PST-39, which were detected before 1987, were given by Line and Qayoum (1992). A summary of races PST-40 to PST-80 were presented by Line and Chen (1996) and Chen et al. (2002).

Line and Qayoum (1992) described seven epidemic regions (Fig. 2) based on geographic barriers, prevailing winds, crop cycles, rust occurrence, and virulence of the wheat pathogen of stripe rust. Races of *P. striiformis* f. sp. *tritici* differed more among the regions before 2000 than after 2000. Region 1 (eastern Washington, northeastern Oregon, and northern Idaho, USA, and southeastern British Columbia, Canada) is an important wheat-growing region with an environment that is favorable for *P. striiformis* f. sp. *tritici* survival in the winter and for disease development in the spring. In addition, growing both winter wheat and

spring wheat in the region provides continuous green host plants for pathogen survival and development of stripe rust. Prior to 2002, the greatest number of races and races with the widest range of virulence occurred in region 1. In 2002, race PST-78 and similar races caused severe stripe rust on spring wheat in the PNW (Chen et al. 2003a). These races were first identified in California (region 6) and the south central states (region 7) in 2000 and were predominant from Texas to Manitoba, Canada, in 2001 (Chen and Moore 2002; Chen et al. 2002). Regions 2 (western Montana, USA, and southern Alberta, Canada) and 3 (southeastern Oregon, northern Nevada (USA), northern Utah (USA), southern Idaho, and western Colorado) are somewhat isolated from other regions in the west, and generally have winters that are too cold for the survival of the pathogen. Consequently, severe epidemics are less frequent in those regions. All races of *P. striiformis* f. sp. *tritici* identified in regions 2 and 3 were first detected in region 1. When stripe rust is severe in region 1, epidemics often occur later in the season in region 2. Because of the late development of epidemics, spring-wheat cultivars are the most vulnerable to stripe rust, especially in Alberta (Line and Qayoum 1992). Races of *P. striiformis* f. sp. *tritici* in region 4 (northern California and western Oregon) were thought to originate locally, with the introduction of inoculum from other regions seldom occurring (Line and Qayoum 1992). However, more recent virulence data for both *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei* showed that predomi-

**Table 3.** Races of *Puccinia striiformis* f. sp. *tritici* (PST), virulence descriptions, and year of first detection in the United States.

PST race	Differentials subject to virulence <sup>d</sup>	Year of first detection
1	1, 2	1963
2	1, 2, 5	1964
3	1, 3	1964
4	1, 3	1968
5	1, 3, 4	1972
6	1, 6, 8, 12	1974
7	1, 3, 5	1974
8	1, 3, 9	1975
9	1, 3, 6, 8, 12	1976
10	1, 2, 3, 9	1976
11	1	1976
12	1, 5, 6, 12	1976
13	1, 5, 6, 8, 12	1976
14	1, 8, 12	1976
15	1, 3, 6, 8, 10	1977
16	1, 3, 9, 11	1977
17	1, 2, 3, 9, 11	1977
18	1, 3, 4, 9	1977
19	1, 3, 6, 8, 10, 12	1977
20	1, 6, 8, 10, 12	1978
21	2	1980
22	1, 3, 12	1981
23	1, 3, 6, 9, 10	1981
24	1, 3, 5, 12	1981
25	1, 3, 6, 8, 9, 10, 12	1982
26	1, 3, 9, 12	1983
27	1, 3, 12, 13	1983
28	1, 3, 4, 12	1983
29	1, 3, 4, 5	1983
30	1, 4, 6, 8, 12	1983
31	1, 3, 5, 11	1983
32	1, 4	1984
33	1, 3, 9, 12, 13	1984
34	1, 3, 4, 5, 12	1985
35	1, 10	1985
36	1, 3, 4, 9, 12	1987
37	1, 3, 6, 8, 9, 10, 11, 12	1987
38	1, 3, 11	1987
39	1, 2, 4	1989
40	1, 4, 14	1989
41	1, 3, 4, 14	1989
42	1, 3, 11, 12	1990
43	1, 3, 4, 5, 12, 14	1990
44	1, 4, 5	1990
45	1, 3, 12, 13, 15	1990
46	1, 3, 6, 9, 10, 11	1991
47	1, 6, 8, 12, 13	1992
48	1, 6, 8, 12, 13, 14	1992
49	1, 3, 11, 14	1992
50	1, 3, 4, 5, 14	1992
51	1, 3, 4, 12, 14	1992
52	1, 4, 8, 12, 14	1993
53	1, 6, 10	1994
54	1, 3, 4, 6, 8, 9, 10, 12	1994

**Table 3 (continued).**

PST race	Differentials subject to virulence <sup>d</sup>	Year of first detection
55	1, 6, 10, 11	1994
56	1, 4, 6, 8, 12, 14	1995
57	1, 3, 4, 6, 8, 10, 12	1996
58	1, 11, 12, 16	1998
59	1, 3, 11, 12, 16	1998
60	1, 12, 16	2000
61	1, 4, 10, 12	2000
62	1, 2, 12, 16	2000
63	1, 8, 12, 16	2000
64	1, 2, 11, 12, 16	2000
65	1, 8, 10, 12, 16	2000
66	1, 2, 10, 11, 12, 16	2000
67	1, 2, 3, 11, 12, 16	2000
68	1, 3, 12, 16, 17	2000
69	1, 2, 11, 12, 16, 18	2000
70	1, 3, 11, 12, 16, 18	2000
71	1, 8, 10, 12, 18, 19	2000
72	1, 6, 8, 10, 12, 18, 19	2000
73	1, 2, 3, 11, 12, 16, 18, 19	2000
74	1, 8, 10, 12, 17, 18, 19	2000
75	1, 4, 8, 10, 12, 17, 18, 19	2000
76	1, 2, 12, 16, 17, 18, 20	2000
77	1, 11, 12, 16, 17, 18, 19, 20	2000
78	1, 3, 11, 12, 16, 17, 18, 19, 20	2000
79	1, 8, 11, 12, 16, 17, 18, 19, 20	2000
80	1, 3, 8, 11, 12, 16, 17, 18, 19, 20	2000
81	1, 14	2001
82	1, 11, 17	2001
83	1, 3, 6, 11	2001
84	1, 8, 10, 12, 18	2001
85	1, 8, 10, 12, 17, 18	2001
86	1, 8, 10, 12, 16, 18, 19	2001
87	1, 2, 11, 12, 16, 17	2001
88	1, 11, 12, 16, 17, 20	2001
89	1, 12, 16, 17, 18, 19, 20	2001
90	1, 3, 11, 12, 14, 16, 17, 18, 19, 20	2001
91	1, 9, 10	2002
92	1, 10, 12	2002
93	1, 6, 10, 12	2002
94	1, 9, 10, 12	2002
95	1, 4, 8, 10, 12, 14	2002
96	1, 4, 6, 8, 10, 12, 14	2002
97	1, 3, 10, 11, 12, 16, 17, 18, 19, 20	2002
98	1, 3, 8, 10, 11, 12, 16, 17, 18, 19, 20	2002
99	1, 3, 9, 10, 11, 12, 16, 17, 18, 19, 20	2002
100	1, 3, 8, 9, 10, 11, 12, 16, 17, 18, 19, 20	2003
101	1, 2, 3, 8, 9, 10, 11, 12, 16, 17, 18, 19, 20	2003
102	1, 3, 8, 9, 10, 11, 12, 14, 16, 17, 18, 19, 20	2003
103	1, 9, 10, 11, 12, 16, 17, 18, 19, 20	2003
104	1, 2, 3, 9, 10, 11, 12, 16, 17	2003
105	1, 8, 10, 11, 12, 16, 17, 20	2003
106	1, 8, 10, 11, 12, 16, 17	2003

**Table 3** (concluded).

PST race	Differentials subject to virulence <sup>a</sup>	Year of first detection
107	1, 3, 4, 5, 9, 10, 14	2003
108	1, 3, 4, 6, 9, 10, 12	2003
109	1, 4, 8, 10, 12	2003

<sup>a</sup>See Table 2 for identity of differential genotypes.

nant races in region 4 were similar to those in other regions (Chen et al. 2002, 2003a, 2004). Stripe rust was once considered to be indigenous to region 5 (western Washington and southwestern British Columbia) and not influenced by outside inoculum. Because of mild winter and cool spring and summer temperatures, stripe rust is severe in region 5 every year. Therefore, one of our major field sites for testing wheat germplasm is in region 5. Region 5 is a major center of diversity where *P. striiformis* f. sp. *tritici* has existed for more than 100 years (Humphrey et al. 1924). Stripe rust also occurs on several species of wild grasses and on cultivated rye, and these hosts may contribute to the diversity of races. The recent spread of stripe rust of barley to this region indicates that region 5 is not totally isolated from other regions, especially regions 4 and 6 (central California) (Chen et al. 1995a). In recent years, races first detected in California and in the south central states also occurred in region 5. Region 6 (central California) has a favorable environment for stripe rust during the early part of the growing season (October to May), but a less favorable environment during summer, when wheat and triticale are not grown as crops. Virulence data from recent years suggest that this region may not be as isolated as previously thought (Chen et al. 2002). The population in region 6 may influence other regions, especially region 7 and Mexico. Conversely, the population in region 6 may be influenced by *P. striiformis* f. sp. *tritici* from other regions, especially Mexico (Chen et al. 2002). Region 7 includes all wheat-producing areas east of the Rocky Mountains from Texas, United States, to Ontario, Canada, and from the eastern slopes of the Rocky Mountains in Colorado to Virginia (USA). The winter weather in the southern portion of the region aids in the survival of the pathogen and in the increase of the disease, but hot-summer weather limits the development of severe epidemics in the northern states of the region. Stripe rust of wheat emerged as a devastating disease in the central United States after 2000. Isolates collected from region 7 and northern Mexico before 1985 were all identified as PST-3 (previously CDL-3). Later, a closely related race, PST-8, was found in region 7. In recent years, the race composition has been more complex and similar to that of other regions (Chen et al. 2002, 2004). From 2000 to 2003, races of *P. striiformis* f. sp. *tritici* that were predominant in California were also predominant in region 7 (Chen et al. 2002, 2004).

Much of the early information on separation of races among the epidemic regions was based upon studies on stripe rust of wheat. The spread of stripe rust of barley in North America does not support these separate epidemic regions. Stripe rust of barley was first reported in Mexico in 1987, then in southern Texas (region 7) in 1991, and has spread against prevailing wind and crossed geographic bar-

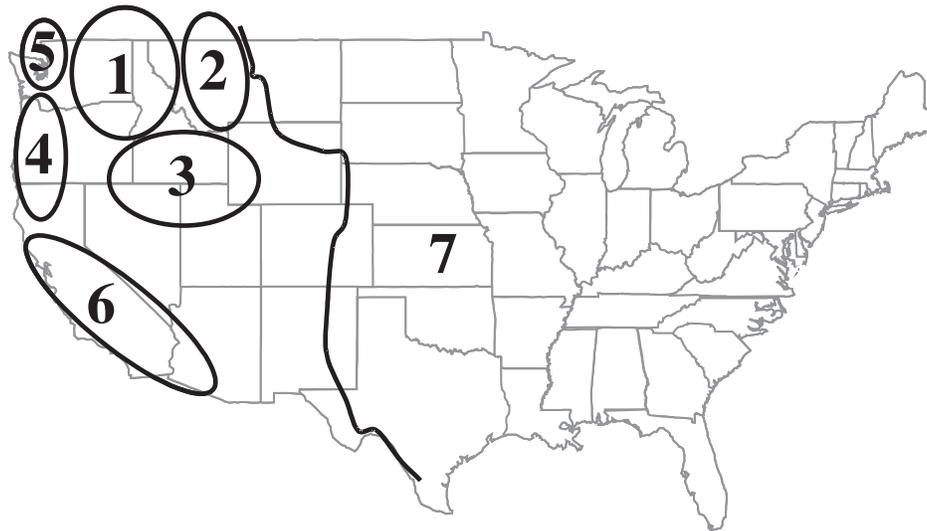
riers to become established in all western barley-production areas (Fig. 2). Stripe rust of barley spread to region 2 (Montana) and region 3 (southern Idaho) by 1993, region 6 (California) by 1994, and region 4 (northern California and western Oregon), region 5 (western Washington), and region 1 (eastern Washington, northeastern Oregon, and northern Idaho) by 1995 (Chen et al. 1995a). The prevalent races that were detected in Texas and California also were detected in the PNW (Chen 2004). The regions where stripe rust of barley has been reported are apparently in a single epidemic system.

The detection of a new group of races of *P. striiformis* f. sp. *tritici* represented by PST-78 (virulent to differentials 1, 3, 11, 12, 16, 17, 18, 19, and 20) in 2000 in California and south central states, and the widespread epidemics that they have caused in the United States, triggered interest into their origin. Races in this new group share the virulence of PST-77 to differentials 1, 11, 12, 16, 17, 18, 19, and 20 (Tables 2 and 3). The most closely related races to PST-77 and PST-78 that were detected before 2000 are PST-58 (virulent to differentials 1, 11, 12, and 16) and PST-59 (virulent to differentials 1, 3, 11, 12, and 16). PST-58 and PST-59 were first detected in 1998 in California (Chen et al. 2002). Races such as PST-68 (virulent to differentials 1, 3, 12, 16, and 17) and PST-70 (virulent to differentials 1, 3, 11, 12, 16, and 18), detected in 2000, and PST-88 (virulent to differentials 1, 11, 12, 16, 17, and 20), detected in 2001, might be intermediates between PST-58 and PST-77 or PST-59 and PST-79. These intermediates occurred at much lower frequencies than PST-77, PST-78, and subsequently detected more virulent races PST-98 (virulent to differentials 1, 3, 8, 10, 11, 12, 16, 17, 18, 19, and 20) and PST-100 (virulent to differentials 1, 3, 8, 9, 10, 11, 12, 16, 17, 18, 19, and 20).

Races of *P. striiformis* f. sp. *tritici* with wide virulence tended to be predominant in recent years (Table 4). This phenomenon does not support the general concept that isolates with fewer virulence genes are more aggressive and have better fitness than isolates with more virulence genes (Vanderplank 1963; Line and Qayoum 1992). In contrast, races of *P. striiformis* f. sp. *hordei* with a narrow spectrum of virulence tended to be more predominant than races with a wide spectrum of virulence (Chen 2004). In stripe rust of wheat, races with the narrowest spectrum of virulence, such as PST-11 and PST-21 that are virulent only to 'Lemhi' and 'Chinese 166', respectively, among the 20 differentials, have never occurred at a high frequency.

Race frequency is determined by two opposing forces. The first force is virulence. Virulence is essential for obligate parasites, such as *P. striiformis*, to infect host plant, grow, and reproduce. The more virulence genes a race has, the more cultivars it is capable of infecting, increasing its frequency in the pathogen population. The second force is the cost of unneeded virulence. A wide virulence spectrum may result in reduced fitness and aggressiveness (Vanderplank 1963; Line and Qayoum 1992). Selection pressure from host plants has a major effect on the first force. If the host population contains relatively few resistance genes, races with only the virulence genes to match these resistance genes should be favored by selection and, therefore, should tend to be predominant. If the host popu-

**Fig. 2.** Epidemic regions of stripe rust [*Puccinia striiformis* f. sp. *tritici*] in the United States and Canada as determined by Line and Qayoum (1992) and Line (2002). The seven regions (separated by the solid lines or circles) were determined based on virulence distribution of *P. striiformis* f. sp. *tritici* and disease patterns.



lation contains many resistance genes, races capable of overcoming more of these genes should become predominant. These forces can account for the current situations for both the *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei* populations in the United States as discussed above. Some of the races of *P. striiformis* f. sp. *tritici* detected after 2000 have both the advantages of virulence to more wheat cultivars and high aggressiveness (Milus and Seyran 2004).

Stripe rust in Canada is influenced by epidemics in the United States. Western Canada is part of regions 1, 2, and 5, based on wind direction, geographic proximity, and cropping systems (Line and Qayoum 1992). Eastern Canada is included in region 7. Before 2000, stripe rust was mainly a problem in western Canada (British Columbia, Alberta, and Saskatchewan). In recent years, stripe rust also has appeared in Manitoba and Ontario (eastern Canada), as it has in the northern Great Plains of the United States (Chen and Moore 2002). Weather conditions and cropping systems allow the pathogen to overwinter and overwinter in far western Canada (British Columbia). Unique races may develop in this area. Inoculum for most of Canada is airborne from either far western Canada or the PNW and Great Plains of the United States. Su et al. (2003) identified 36 races from 57 isolates collected from 1984 to 2002 in British Columbia, Alberta, Saskatchewan, and Manitoba, using 24 wheat genotypes, including the set of world or European differentials and some of the United States differentials. They also found that some of the races could not be differentiated by using only the world or European differentials. Most of the races that they identified had similar virulence genes to those of the United States races. The unique races identified in their study were race 11 (collected from Bow Island, Alberta, in 1990), race 12 (collected from Vauxhall, Alberta, in 1990), race 29 (collected from Foremost, Alberta, in 1995), race 16 (collected from Creston, British Columbia, in 1991), and race 18 (collected from Bow Island in 1993). Races 11, 12, and 29 were virulent on the *Yr15* donor, *Triticum turgidum* L. var. *dicoccoides* selection G-25. Virulence to *Yr15* has

not been detected in the United States. Races 16 and 18, virulent on 'Compair' (*Yr8*, *Yr19*), were collected before 2000. In contrast, races virulent on 'Compair' were not detected in the United States until 2000 (Chen et al. 2002). In the study by Su et al. (2003), the wheat 'Fielder' (*Yr6*, *Yr20*) was used to propagate urediniospores of isolates of *P. striiformis* f. sp. *tritici*. Thus isolates avirulent to 'Fielder' would have been eliminated from the study. About 31% of United States races are avirulent on 'Fielder' (Table 3). Those races could not have been identified in their study. We tested one isolate from Manitoba in 2001 and five isolates from Ontario in 2003. The 2001 isolate was race PST-78, the predominant race in the Great Plains of United States. The 2003 isolates were races PST-73, PST-97, PST-98, and PST-100, all of which were present and the latter two, predominant, in the Great Plains of United States.

Many of the wheat genotypes that have been used to differentiate races of *P. striiformis* f. sp. *tritici* have more than one gene for resistance. The presence of two or more genes in a single differential genotype makes it difficult to quickly detect new races that have virulence to a single resistance gene. Wellings et al. (2004b) at the Plant Breeding Institute, University of Sydney, Cobbity, NSW, Australia, developed near-isogenic lines in the 'Avocet Susceptible' background for 13 *Yr* genes including *Yr1*, *Yr5*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr10*, *Yr15*, *Yr17*, *Yr18*, *Yr24*, *Yr26*, and *Yr32*, which have been used throughout the world for monitoring virulence genes of *P. striiformis* f. sp. *tritici*. More effort is needed to develop near-isogenic lines for the other *Yr* genes. The near-isogenic lines with *Yr* gene should eventually replace the current differential genotypes used for identifying races.

#### Race studies in Australasia

The situation of stripe rust of wheat in Australia is a good example of how the pathogen can change in virulence. *Puccinia striiformis* f. sp. *tritici* was introduced into Australia in 1979 and spread to New Zealand in 1980 as a single race. It evolved into 15 races within 10 years (Wellings and

**Table 4.** Frequencies and distributions of *Puccinia striiformis* f. sp. *tritici* (PST) races in the United States and Canada from 2000 to 2003.

PST race	Frequency (%)				Distribution in regions <sup>a</sup>			
	2000	2001	2002	2003	2000	2001	2002	2003
1	0.6				6			
3	0.6	1.6			1	1, 7		
6	0.6				6			
8		1.1				6		
11	1.9			0.3	5			6
12			0.6				5, 6	
14	1.9		0.3		1, 5		5	
18	0.6		0.3		1		5	
19	0.6				5			
20	11.3	1.1	5.3		1, 5	5	1, 5	
21	2.5			0.5	1			1, 6
22			0.6				5	
23	0.6	0.5	0.9		1	5	5	
24			0.3				5	
25	0.6		2.4	1.3	1		5	5
26	0.6		0.9	0.3	1		5	
29	2.5				1			
30	1.9	0.5	0.3		5	5	5	
32	1.9				5			
35	3.1	2.7	1.2		1, 5, 6, 7	1, 5	5	
38		0.5				1		
40			0.3				1	
41			0.3				1	
42		0.5				1		
43				0.3				1
45				0.8				1
49			0.3				6	
51	0.6				1			
52	0.6				1			
53	1.9		0.6		1, 5		1, 5	
54			0.3	0.5			4	5
55			0.3				7	
56			0.3				1	
58	10.7	2.7			1, 6, 7	1, 7		
59	11.2	6.0			1, 6, 7	1, 6, 7		
60	3.1				4, 6, 7			
61	0.6	1.6	0.6		5	1, 5	5	
62	0.6	0.5			6	7		
63	1.3				7			
64	5.7	4.3			6, 7	6		
65	2.5		0.3		1, 5, 6		5	
66	1.3				6, 7			
67	1.9	8.2	0.3		6, 7	1, 5, 6, 7	6	
68	1.3	1.6			6, 7	1, 6		
69	1.3	0.5			5	7		
70	0.6				7			
71	1.9	4.9			5	1, 5		
72	1.3	3.3			1, 6	1, 5, 7		
73	1.3	1.1		0.5	1, 6	5		7
74	3.1	1.6	1.5		5, 7	1, 5	1, 5	
75	0.6				5			
76	0.6				7			
77	0.6	2.7	5.0	2.4	7	1, 7	1, 4, 6, 7	1, 5, 6, 7
78	7.6	23.4	31.1	5.6	6, 7	1, 7	1, 4, 6, 7	1, 6, 7
79	1.3	3.8	0.3	1.6	7	7	5	1, 6, 7

**Table 4** (concluded).

PST race	Frequency (%)				Distribution in regions <sup>a</sup>			
	2000	2001	2002	2003	2000	2001	2002	2003
80	12.0	17.4	8.9	4.8	6, 7	7	1, 4, 6, 7	1, 6, 7
81		0.5				1		
82		1.1				1, 7		
83		0.5				7		
84		1.1	0.3			1	1	
85		0.5	0.9	0.3		5	5	7
86		0.5				1		
87		1.1				6		
88		0.5				7		
89		0.5	0.3			7	7	
90		3.3	2.4			7	1, 6, 7	
91			0.9	0.3			5	5
92			1.5	0.5			1, 5	5
93			0.6	0.5			5	1, 5
94			0.3	0.3			6	6
95			2.4	0.3			1	1
96			1.8				1, 4, 6, 7	
97			11.2	10.6			1, 4, 6, 7	1, 5, 6, 7
98			9.5	29.4			1, 4, 6, 7	1, 2, 5, 6, 7
99			2.1	2.9			1, 6, 7	1, 6, 7
100				33.1				1, 2, 5, 6, 7
101				0.3				6
102				0.5				2, 7
103				0.3				6
104				0.3				7
105				0.5				5, 7
106				0.3				6
107				0.3				1
108				0.3				5
109				0.5				1

<sup>a</sup>Refer to Fig. 2 for mapping of the seven regions (Line and Qayoum 1992; Line 2002): 1, eastern Washington, northeastern Oregon, and northern Idaho (United States) and southeastern British Columbia and southwestern Alberta (Canada); 2, western Montana (United States) and southeastern Alberta (Canada); 3, southern Idaho, southeastern Oregon, northern Nevada, northern Utah, and western Wyoming (United States); 4, western Oregon and northern California (United States); 5, northwestern Washington (United States) and southwestern British Columbia (Canada); 6, central and southern California and western Arizona (United States); 7, the huge area east of the Rocky Mountains (United States) and southern Saskatchewan, Manitoba, and Ontario (Canada).

McIntosh 1990). The first race was virulent to *Yr2*. Later races had various combinations of virulence to *Yr1*, *Yr2*, *YrA*, *Yr5*, *Yr6*, *Yr7*, *Yr8*, and *YrSp*. Although the appearance of some of the virulence genes was not related to the deployment of corresponding resistance genes in wheat production, as was the case for virulence to *Yr5*, predominant races with specific virulence combinations, such as those virulent to *Yr7* and *Yr17*, were selected by widely grown wheat cultivars (Wellings and McIntosh 1990; Wellings and Kandel 2004). The most recently detected race in western Australia appeared in 2002 and spread to eastern Australia in 2003. It was virulent to *Yr6*, *Yr7*, *Yr8*, *Yr9*, and *YrA*, indicating another foreign introduction (Wellings et al. 2003; Wellings and Kandel 2004). These virulence combinations have also occurred in the *P. striiformis* f. sp. *tritici* populations in North America (Chen et al. 2002, 2003a), South America (Madariaga et al. 2004), Europe (Manninger 2004), and China (Wan et al. 2004).

### Population structure of *P. striiformis* f. sp. *tritici* based on DNA polymorphism and genome and functional genomic research, using fungal DNA libraries

Chen et al. (1993) were among the first to use molecular markers to determine the population structure of the pathogen of stripe rust in wheat. They detected DNA polymorphisms among races and among single-spore isolates within races, using 115 single-spore isolates. Cluster analysis based on RAPD data separated isolates virulent to resistance gene *Yr1* of wheat from those avirulent to *Yr1*. They detected a low but significant correlation between the virulence and RAPD data. Virulence groups were highly associated with epidemic regions, but the RAPD groups were generally not associated with geographic regions. The low association between virulence and RAPD patterns indicates that DNA polymorphism is independent of virulence. Selec-

tion for virulence by growing cultivars with race-specific resistance may play a major role in determining race structure of the pathogen.

As advances in biotechnology have been made, other molecular techniques have been used to study *P. striiformis* f. sp. *tritici* populations. Shan et al. (1998) used a moderately repetitive DNA sequence as a probe and detected a high level of genetic variation among 160 isolates of stripe rust from wheat in six provinces in China. They reported a low level of genetic differentiation among regions as well as within regions. Zheng et al. (2001) used amplified fragment length polymorphism (AFLP) to show that several new pathotypes might have evolved independently of the reference strains. Justesen et al. (2002) used disease incidence data and AFLP markers to demonstrate that the recent *P. striiformis* f. sp. *tritici* population in Denmark could be traced back to the aerial dispersal of urediniospores from France and (or) Germany. Recently, Komjáti et al. (2004) used intergenic spacer (IGS) sequences, simple sequence repeats (SSR), and sequence-related amplified polymorphism (SRAP) techniques to reveal polymorphisms in *P. striiformis*. They detected variation between *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei*, but not among isolates of *P. striiformis* f. sp. *tritici*.

Markell et al. (2004) used both SSR and AFLP techniques to clearly distinguish pre-2000 isolates from isolates collected after 2000 in the United States. They also found that the pre-2000 isolates in the United States were more closely related to isolates from Denmark than the after-2000 isolates in the United States. Based on their results, the group of races first identified in 2000 (Chen et al. 2002) may have a different origin from races identified prior to 2000. The early pathogen of stripe rust in the United States was postulated to be more likely from Asia than from Europe (reviewed in Line 2002). Based on virulence patterns of the after-2000 races, Chen et al. (2002) related these races to the pathogen population in Mexico. Research is underway to test the hypothesis. Molecular studies on a global scale are needed to identify the origin of new races detected in various countries and to reveal evolutionary mechanisms of the pathogen.

To study the global genomic structure, Chen and Ling (2004) have constructed a BAC library from urediniospores of race PST-78 of *P. striiformis* f. sp. *tritici*. PST-78 represents the group of races first identified in 2000 that have been responsible for the devastating epidemics in the United States since 2000 (Chen et al. 2002, 2004). Strains with virulences similar to that of PST-78 have also been identified in other countries (Wellings et al. 2003). The BAC library was constructed by cloning *Hind*III-digested high molecular weight DNA extracted from urediniospores into the *pIndigo* BAC-5 cloning vector transformed into *Escherichia coli* competent cell DH10B. The BAC library consists of 22 272 clones with an average insert size of 50 kb and covers about 15 times the *P. striiformis* genome (Chen and Ling 2004). The BAC library will be useful for studying the genomic structure, constructing physical maps, sequencing the genome, and cloning genes of the pathogen of stripe rust.

Understanding the pathogen genome may be useful for developing better techniques to study the disease epidemiol-

ogy, which should lead to more effective control of the disease. Chen and Ling (2004) also generated a high-quality full-length cDNA library consisting of 42 240 clones from race PST-78. Examination in cDNA insert size of random samples from the cDNA library revealed that 99% of the clones reach full length with the average cDNA insert of 1.5 kb. This full-length cDNA library is useful for identifying fungal genes. Of 167 full-length cDNA clones that were sequenced, 126 clones had the complete sequences, and 41 had partial sequences. The results of BLAST (basic local alignment search tool; Altschul et al. 1997) search of the fungal gene database showed that 36 cDNA clones had more than 45% homology (in a sequence length of at least 97 amino acids) to genes with clear functions identified in other fungi. These genes code for an elongation factor, a mitogen-activated protein kinase, deacetylase, calnexin, transaldolase, TATA-binding protein, uridine 5'-diphosphate (UDP)-glucose dehydrogenase, and enolase. Other clones also had various levels of homology with genes in other fungi, including chitinase in *Aspergillus nidulans* (Eidam) Winter, xylanase B in *Neocallimastix patriciarum* Orpin & Munn, and a pectin lyase precursor in *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk (Chen and Ling 2004; P. Ling, M.N. Wang, and X.M. Chen, unpublished data). The evolutionary relationships of *P. striiformis* f. sp. *tritici* to other fungi were determined based on their elongation-factor sequences. *Puccinia striiformis* f. sp. *tritici* was more related to *P. graminis* f. sp. *tritici*, the pathogen of stem rust of wheat and barley, than to other fungal species. As expected, fungal species in Basidiomycetes were more related to each other than to fungi in other groups (P. Ling, M.N. Wang, and X.M. Chen, unpublished data). Polymerase chain reaction (PCR) primers were designed based on DNA sequences of selected genes (P. Ling, M.N. Wang, and X.M. Chen, unpublished data) to establish a genomic map, using BAC clones, to study population structure and migration of *P. striiformis* f. sp. *tritici*, and to identify molecular mechanisms controlling the host-pathogen interaction.

### Migration and introduction of the pathogen causing stripe rust

Long-distance dispersal of rust pathogens of cereals has been reviewed by Nagarajan and Singh (1990), Eversmeyer and Kramer (2000), and Brown and Hovmøller (2002). As observed in many other airborne fungal pathogens, long-distance dispersal in the air and occasionally by human activities enables pathogens of stripe rust to spread to new geographic areas. The pathogen of stripe rust of wheat has been in Asia and Europe for thousands of years and on the American continents for over one hundred years (Stubbs 1985; Line 2002). The pathogen was not in Australia and New Zealand until 1979 when it was first reported in Australia (O'Brien et al. 1980). The pathogen was possibly introduced from Europe through urediniospore-contaminated clothing (Wellings and McIntosh 1987). It probably spread by wind dispersal to New Zealand the following year from eastern Australia (Wellings and McIntosh 1990). Stripe rust was not detected in western Australia until August 2002 (Wellings et al. 2003). According to virulence and molecular analyses (Wellings et al. 2003), the strain responsible for

the widespread epidemic in 2002 was probably a foreign introduction. In central Africa, stripe rust was first reported in northern Zambia in 1958 (Angus 1965), but the disease was not found in South Africa until 1996 when it was detected in the Western Cape (Pretorius et al. 1997). From there it spread to most of South Africa in 1997 (Boshoff et al. 2002).

Seasonal, long-distance dispersal has been documented for several rust pathogens throughout the world (Brown and Hovmøller 2002; Nagarajan and Singh 1990). Stakman (1934) demonstrated the seasonal movement of urediniospores of the pathogen of wheat stem rust from Northern Mexico or from Texas to Canada. The urediniospore dispersal route was subsequently described as the “*Puccinia* path”. Stripe rust showed similar occurrence patterns from Texas to Canada in the later 1950s and early 2000s, so it may follow the same *Puccinia* path (Pady et al. 1957; Chen et al. 2002). Line and Qayoum (1992) documented urediniospore migration from eastern Washington to Montana and Alberta, Canada, and to southern Idaho.

Because the fungus of stripe rust requires relatively cool temperatures and host crops, certain epidemic regions may serve as sources of inoculum for recipient regions. Seasonal migration in both directions of urediniospores between high-elevation mountainous areas and plains has been documented in China (Brown and Hovmøller 2002; Wan et al. 2004). Hovmøller et al. (2002) demonstrated that a single, clonal population exists in the United Kingdom, Germany, France, and Denmark, and that Denmark was the recipient of migrant spores from other countries. Prevailing winds, environments, and crop seasons affect the seasonal migration of urediniospores in North America (Fig. 3). The north central states of the United States, and Canada, may be the recipients of urediniospores from the south central states of the United States and from Mexico. The south central region of the United States may be the recipient of urediniospores from the high-elevation areas of Mexico. Long-distance dispersal of urediniospores from California may spread new races to the central United States and to Mexico. The dispersal of urediniospores against the prevailing wind is also possible, as demonstrated by the westward spread of *P. striiformis* f. sp. *hordei* from southern Texas to California and the PNW of the United States from 1991 to 1995 (Chen et al. 1995a) and by the spread of PST-78 and similar races of *P. striiformis* f. sp. *tritici* in the United States from 2000 to 2002 (Chen and Moore 2002; Chen et al. 2002, 2003a, 2004). Studies on race identification, using samples from various regions, support these findings. However, more studies using molecular haplotypes are needed to confirm these conclusions.

## Control of stripe rust, using plant resistance

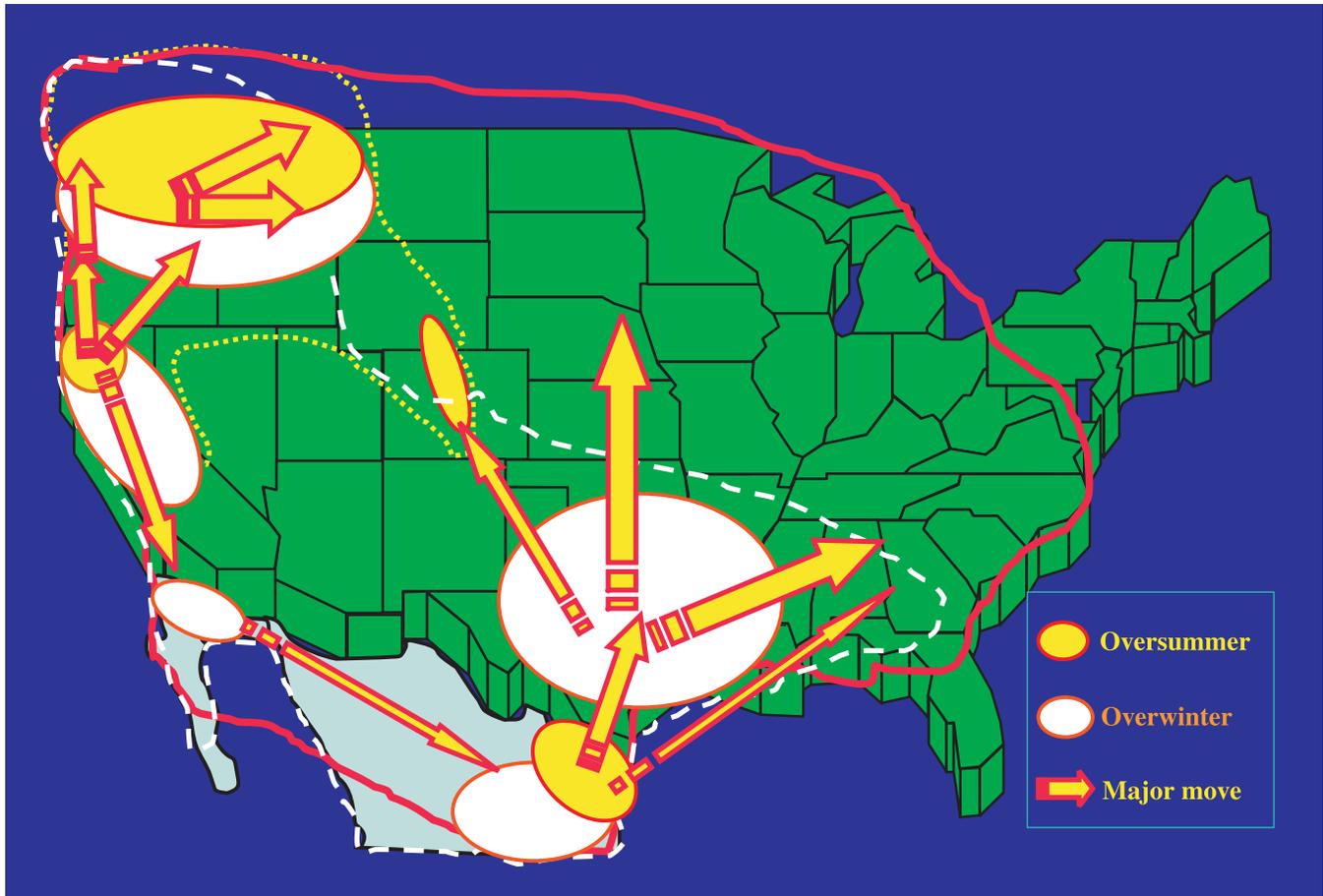
### Types of resistance to stripe rust

Growing resistant cultivars is the most effective, economical, and environmentally friendly method of disease control (Röbbelen and Sharp 1978; Line and Chen 1995). Resistance to stripe rust can be broadly categorized as all-stage resistance (also called seedling resistance), which can be detected at the seedling stage, but is also expressed at all

stages of plant growth, and as adult-plant resistance, which is expressed at later stages of plant growth. The previously used term of seedling resistance can be misinterpreted as resistance that is only effective in the seedling stage, and therefore, should be replaced with all-stage resistance. All-stage resistance is race specific (Chen and Line 1992a, 1992b, 1993; Chen et al. 1995b, 1998a). Cultivars with race-specific, all-stage resistance often become susceptible soon after they are released because of the rapid evolution of new races (Line and Qayoum 1992; Line and Chen 1995, 1996). Many of the races listed in Table 2 caused devastating epidemics during the last 46 years in the United States (Line and Qayoum 1992; Chen et al. 2004). Some types of adult-plant resistance are also race specific (Lupton et al. 1971; Priestley and Dodson 1976; McIntosh et al. 1995). However, high-temperature, adult-plant (HTAP) resistance, which is present in many winter-wheat cultivars and some spring-wheat cultivars currently grown in the PNW, is durable and non race specific (Qayoum and Line 1985; Milus and Line 1986a, 1986b; Chen and Line 1995a, 1995b; Line and Chen 1995; Chen et al. 1998a). The effectiveness of HTAP resistance increases as temperature become higher and plants grow older. Cultivars with only HTAP resistance are susceptible to all races at the seedling stage. However, as the plant ages and temperature increases during the growing season, the plant becomes more resistant, and rust development slows or may even stop. In HTAP resistance, the flag leaf, which makes the largest contribution to grain yield, is more resistant to stripe rust than lower leaves. HTAP resistance protects the crop by lowering the infection type, the number of new infections, and the amount and spread of inoculum. Sporulation is completely inhibited on cultivars with the highest degree of HTAP resistance (Chen and Line 1995a, 1995b).

Although field data of infection types and severities and race composition can indicate the presence of HTAP resistance, greenhouse tests, using isolates virulent to the seedlings of a genotype under controlled temperature conditions are needed for confirmation. Qayoum and Line (1985) conducted detailed studies on the effects of various temperature ranges on wheat cultivars with different levels of HTAP resistance. Two temperature ranges are used routinely for separating all-stage and HTAP resistance in greenhouse tests. Cultivars are first tested at the seedling stage with as many races as possible at the standard low diurnal temperature cycle (gradually changing from 4 °C at 0200 to 20 °C at 1400) to identify races virulent on the seedlings, and then, adult-plants (boot to heading stage) susceptible in the seedling test are tested at the standard high diurnal temperature cycle (gradually changing from 10 °C at 0200 to 35 °C at the 1400) with selected races that are virulent to the cultivars in the seedling tests. Cultivars with HTAP resistance will have lower infection types in the adult-stage test than in the seedling test. The two temperature ranges simulate the normal temperature ranges early and late in the growing season of cereal crops in most wheat-production areas of the PNW. These temperature ranges can be used directly or modified to select cultivars with a useful level of HTAP resistance for other regions. Even though expression of HTAP resistance can be as high as all-stage resistance, it is generally expressed as partial resistance. The level of

**Fig. 3.** Regions of oversummering and overwintering in North America for the pathogen of stripe rust [*Puccinia striiformis* f. sp. *tritici*] and movement of *P. striiformis* f. sp. *tritici* urediniospores among the regions. The major oversummering and overwintering regions are indicated. The pathogen can occur in the area within the red irregular circle, overwinter within the white-dash irregular circle, and oversummer within the yellow-dash irregular circle. The major spore movements by wind between regions are indicated by arrows. Minor movements by wind and human activities between some of the regions are also possible.



HTAP resistance (measured as disease severity) in fields is greatly influenced by inoculum pressure and temperature. Thus, a level of HTAP resistance may be adequate in one region (for example, eastern Oregon, eastern Washington, and Kansas) where stripe rust usually develops late, but inadequate in another region (for example, western Oregon, western Washington, and California) where the disease develops early. Nevertheless, cultivars with any level of HTAP resistance are better than susceptible cultivars for reducing damage due to stripe rust, based on our yield-loss studies with cultivars with various levels of resistance. The epidemics of stripe rust in California and the PNW in recent years mainly occurred on cultivars lacking HTAP resistance. Many of the currently grown winter- and spring-wheat cultivars have HTAP resistance. However, the challenge is to more efficiently incorporate HTAP resistance into susceptible cultivars that carry special quality traits and high-yield potential and to combine HTAP resistance with effective all-stage resistance because HTAP resistance is often controlled by quantitative-trait loci (QTL) (Chen and Line 1995a, 1995b) and masked by effective all-stage resistance. Use of marker-assisted selection is a promising ap-

proach to meet this challenge. Collaborative studies to develop molecular markers for various sources of HTAP resistance are underway. Combining HTAP resistance with effective all-stage resistance is the best approach for developing wheat cultivars with high-level and durable resistance because the all-stage resistance can provide complete control when it is effective, and the HTAP resistance can reduce damage when the all-stage resistance is overcome by new races (X.M. Chen<sup>1</sup>).

#### Genetics of resistance to stripe rust of wheat

Genetics of resistance to stripe rust has been studied for a century. Biffen (1905) first demonstrated that resistance to stripe rust in wheat follows Mendel's laws. Seventy genes with official (*Yr* followed by a number) or provisional (*Yr* followed by letters) symbols have been reported (Table 5). Multiple resistance alleles have been reported for the *Yr3* and *Yr4* loci (Lupton and Macer 1962; Chen and Line 1993; Chen et al. 1996). Most of the 70 genes are unique as indicated by different chromosomal locations, responses to races, and wheat genotypes or wild species. Many reported genes have not been named (Chen et al. 1998a; Chen 2002).

**Table 5.** Genes for resistance to stripe rust [*Puccinia striiformis* f. sp. *tritici*], examples of wheat genotypes containing the genes, their chromosomal locations, types of resistance, and references.

Yr gene	Example of wheat genotype	Chromosomal location	Resistance type <sup>a</sup>	Reference
<i>Yr1</i>	'Chinese 166'	2AL	RS, AS	Lupton and Macer 1962
<i>Yr2</i>	'Heines VII'	7B	RS, AS	Lupton and Macer 1962
<i>Yr3a</i>	'Cappelle Desprez'	1B	RS, AS	Lupton and Macer 1962
<i>Yr3b<sup>b</sup></i>	Hybrid 46		RS, AS	Lupton and Macer 1962
<i>Yr3c</i>	'Minister'	1B	RS, AS	Lupton and Macer 1962
<i>Yr4a</i>	'Cappelle Desprez'	6B	RS, AS	Lupton and Macer 1962
<i>Yr4b</i>	Hybrid 46	6B	RS, AS	Lupton and Macer 1962
<i>Yr5</i>	<i>Triticum aestivum</i> subsp. <i>spelta</i> 'Album'	2BL	RS, AS	Macer 1966
<i>Yr6</i>	'Heines Kolben'	7BS	RS, AS	Macer 1966
<i>Yr7</i>	'Lee'	2BL	RS, AS	Macer 1966
<i>Yr8<sup>c</sup></i>	'Compair'	2D (2A, 3D)	RS, AS	Riley et al. 1968
<i>Yr9<sup>d</sup></i>	'Clement'	1RS/1BL	RS, AS	Macer 1975
<i>Yr10</i>	'Moro'	1BS	RS, AS	Macer 1975
<i>Yr11</i>	'Joss Cambier'		RS, AP	McIntosh 1988
<i>Yr12</i>	'Frontier'		RS, AP	McIntosh 1988
<i>Yr13</i>	'Hustler'		RS, AP	McIntosh 1988
<i>Yr14</i>	'Kador'		RS, AP	McIntosh 1988
<i>Yr15</i>	<i>Triticum turgidum</i> var. <i>dicoccoides</i> G-25	1BS	RS, AS	Gerechter-Amitai et al. 1989
<i>Yr16</i>	'Bersee'	2D	NRS, AP	Worland and Law 1986
<i>Yr17<sup>e</sup></i>	VPM1	2AS	RS, AS	Bariana and McIntosh 1993
<i>Yr18</i>	'Jupateco 73R'	7DS	NRS, HTAP	Singh 1992
<i>Yr19</i>	'Compair'	5B	RS, AS	Chen et al. 1995b
<i>Yr20</i>	'Fielder'	6D	RS, AS	Chen et al. 1995b
<i>Yr21</i>	'Lemhi'	1B	RS, AS	Chen et al. 1995b
<i>Yr22</i>	'Lee'	4D	RS, AS	Chen et al. 1995b
<i>Yr23</i>	'Lee'	6D	RS, AS	Chen et al. 1995b
<i>Yr24</i>	Yr24/6*AVS	1BS	RS, AS	McIntosh et al. 1998
<i>Yr25</i>	'Strubes Dickkopf'	1D	RS, AS	McIntosh et al. 1998
<i>Yr26<sup>f</sup></i>	R55	1BS	RS, AS	McIntosh et al. 1998
<i>Yr27</i>	'Ciano 79'	2BS	RS, AS	McDonald et al. 2004
<i>Yr28</i>	Synthetic	4DS	RS, AS	Singh et al. 2000
<i>Yr29</i>	'Pavon F76'	1BL	NRS, AP	McIntosh et al. 2001
<i>Yr30</i>	'Opata 85'	3BS	NRS, AP	McIntosh et al. 2001
<i>Yr31</i>	'Pastor'	2BS	RS, AS	McIntosh et al. 2003
<i>Yr32</i>	'Carstens V'	2AS	RS, AS	Eriksen et al. 2004
<i>Yr33<sup>g</sup></i>	'Batavia'	7DL	RS, AS	McIntosh et al. 2004
<i>Yr34</i>	WAWHT2046	5AL	AP	McIntosh et al. 2004
<i>Yr35</i>	98M71	6BS	RS, AS	R.A. McIntosh 2004, personal communication
<i>Yr36</i>	'Glupro', RSL No. 65	6BS	NRS, HTAP	J. Dubcovsky 2004, personal communication
<i>Yr37</i>	S14	2DL	RS, AS	R.A. McIntosh 2004, personal communication
<i>YrH52</i>	<i>T. turgidum</i> var. <i>dicoccoides</i> H52	1BS	RS, AS	Peng et al. 2000
<i>Yrns-B1</i>	Lgst. 79-74	3BS	NRS, AP	Börner et al. 2000
<i>YrSP</i>	'Spaldings Prolific'	2BS	RS, AS	McIntosh et al. 1995
<i>YrA</i>	'Anza'		RS, AS	McIntosh et al. 1998
<i>YrCle</i>	'Clement'	4B	RS, AS	Chen et al. 1998a
<i>YrDru</i>	'Druchamp'	5B	RS, AS	Chen et al. 1998a
<i>YrDru2</i>	'Druchamp'	6A	RS, AS	Chen et al. 1998a
<i>YrDa1</i>	'Daws'	1A	RS, AS	Chen et al. 1998a
<i>YrDa2</i>	'Daws'	5D	RS, AS	Chen et al. 1998a
<i>YrH46</i>	Hybrid 46	6A	RS, AS	Chen et al. 1998a
<i>YrHVII</i>	'Heines VII'	4A	RS, AS	Chen et al. 1998a
<i>YrMin</i>	'Minister'	4A	RS, AS	Chen et al. 1998a

**Table 5.** (concluded).

<i>YrMor</i>	'Moro'	4B	RS, AS	Chen et al. 1998a
<i>YrND</i>	'Nord Desprez'	4A	RS, AS	Chen et al. 1998a
<i>YrSte</i>	'Stephens'	2B	RS, AS	Chen et al. 1998a
<i>YrSte2</i>	'Stephens'	3B	RS, AS	Chen et al. 1998a
<i>YrTye</i>	'Tyee'	6D	RS, AS	Chen et al. 1998a
<i>YrTr1</i>	'Tres'	6D	RS, AS	Chen et al. 1998a
<i>YrTr2</i>	'Tres'	3A	RS, AS	Chen et al. 1998a
<i>YrYam</i>	'Yamhill'	4B	RS, AS	Chen et al. 1998a
<i>YrV23</i>	'Vilmorin 23'	2B	RS, AS	Chen et al. 1998a
<i>YrJh1</i>	'Jinghe 8811'	2A	RS, AS	Zhang et al. 2001
<i>YrJh2</i>	'Jinghe 8811'	4D	RS, AS	Zhang et al. 2001
<i>YrGui1</i>	'Guinong 22'		RS, AS	Cao et al. 2004
<i>YrGui2</i>	'Guinong 22'		RS, AS	Cao et al. 2004
<i>YrGui3</i>	'Guinong 22'		RS, AS	Cao et al. 2004
<i>YrJu1</i>	'Jubilejna II'		RS, AS	Zhao et al. 2004
<i>YrJu2</i>	'Jubilejna II'		RS, AS	Zhao et al. 2004
<i>YrJu3</i>	'Jubilejna II'		RS, AS	Zhao et al. 2004
<i>YrJu4</i>	'Jubilejna II'		RS, AS	Zhao et al. 2004
<i>YrA1</i>	'Gaines', 'Nugaines'		NRS, HTAP	Chen et al. 1998a
<i>YrA2</i>	'Nugaines'		NRS, HTAP	Chen et al. 1998a
<i>YrA3</i>	'Luke'		NRS, HTAP	Chen et al. 1998a
<i>YrA4</i>	'Luke'		NRS, HTAP	Chen et al. 1998a
<i>YrA5</i>	'Druchamp'		NRS, HTAP	Chen et al. 1998a
<i>YrA6</i>	'Druchamp'		NRS, HTAP	Chen et al. 1998a
<i>YrA7</i>	'Stephens'	6BS	NRS, HTAP	Chen et al. 1998a
<i>YrA8</i>	'Stephens'		NRS, HTAP	Chen et al. 1998a

<sup>a</sup>AS, All-stage resistance (also called seedling resistance); AP, adult-plant resistance; HTAP, high-temperature, adult-plant resistance; RS, race-specific resistance; NRS, non-race-specific resistance.

<sup>b</sup>*Yr3b* was originally reported in Hybrid 46 by Lupton and Macer (1962). Chen and Line (1993) found that a second gene in Hybrid 46, presumably this gene, was not located at the *Yr3* locus, and provisionally designated it *YrH46*.

<sup>c</sup>*Yr8* was originally from *Triticum comosum* (Sibth. & Sm.) Richter and translocated on chromosome 2D of wheat in 'Compair', 'Hobbit Sib', and 'Maris Widgeon', on chromosome 3D in CS 3D/2M, and on chromosome 2A in CA 2A/2M 4/2 (see McIntosh et al. 1998).

<sup>d</sup>*Yr9* was originally from rye and translocated on chromosome 1B in many wheat genotypes (see McIntosh et al. 1998). Some wheat cultivars carrying the 1A/1R wheat-rye translocation also may carry this gene.

<sup>e</sup>*Yr17* was originally from *Triticum ventricosum* Ces. It was transferred to many wheat cultivars including 'Hyak' and 'Mandsen' in the PNW.

<sup>f</sup>*Yr26* was originally from *Triticum turgidum* L. R55. The gene was previously thought to be on chromosome 6AS (6AL.6VS) (see McIntosh et al. 1998) and later was remapped on 1BS (Ma et al. 2001).

<sup>g</sup>*Yr33* is more readily detected in the seedling stage at elevated temperatures (McIntosh et al. 2004).

Most of the known genes confer all-stage resistance. The genetics of HTAP resistance from several sources has been studied, but the genes or QTL have not been designated.

The genetic control of durable HTAP resistance has been analyzed by biometrical methods. Milus and Line (1986a, 1986b) showed that non-race-specific HTAP in the winter-wheat 'Gaines', 'Nugaines', and 'Luke' is quantitatively inherited. They reported that the HTAP resistance in these three cultivars is partially recessive with no maternal inheritance and that most of the gene action among loci is additive. They detected epistatic gene action in some crosses and estimated that one to two genes condition HTAP resistance in each cultivar. Chen and Line (1995a, 1995b) studied the genetics of durable HTAP resistance and the relationships between HTAP resistance and race-specific seedling resistance in wheat 'Druchamp' and 'Stephens'. HTAP resistance in these two cultivars is partially recessive, and the gene action of resistance is mostly additive. Maternal cytoplasmic effects were observed in crosses involving 'Stephens', 'Druchamp', and the club wheat 'Paha'.

'Druchamp' and 'Stephens' each have two to three different HTAP resistance genes. The genes for HTAP resistance in both 'Stephens' and 'Druchamp' are also different from their race-specific, seedling resistance genes. HTAP resistance in the two cultivars has high heritability. The results suggest that HTAP and all-stage resistance from different sources can be combined to achieve a higher level of durable resistance in a single cultivar (Chen and Line 1995b).

Winter-wheat genotypes are the major sources of HTAP resistance in the PNW. 'Jagger', a hard red winter wheat cultivar that has been widely grown in the Great Plains since 2000, might have inherited HTAP resistance from 'Stephens'. Relatively few sources of HTAP resistance have been identified in spring-wheat genotypes. *Yr18*, a gene for adult-plant resistance present in many spring-wheat cultivars (Singh 1992), confers HTAP resistance (X.M. Chen 2004, unpublished data). Recently, a gene identified in *T. turgidum* var. *dicoccoides* and designated *Yr36* (J. Dubcovsky, personal communication) was shown to confer HTAP resistance (X.M. Chen 2004, unpublished data).

Spring wheat 'Alpowa' and 'Express', although susceptible to many races in the PNW since their release in the early 1990s, are resistant in the field and in adult-plant tests when inoculated with seedling-virulent races under high temperatures in the greenhouse (X.M. Chen 2004, unpublished data). Genetic and molecular-mapping studies are underway to identify genes conferring HTAP resistance in these cultivars. Ultrastructural and molecular studies are also needed to reveal mechanisms of HTAP resistance.

On a global scale, the genes listed in Table 5, for which matching virulence occurs in races of *P. striiformis* f. sp. *tritici*, can still provide effective resistance if they are used in certain combinations in individual cultivars and if these cultivars are deployed wisely among regions and within a region. A multiline approach utilizing a mixture of lines with different resistance genes in a common genetic background has provided durable resistance to stripe rust in club wheat. Two multiline cultivars have been released, 'Crew' in 1983 and 'Rely' in 1993 (Allan et al. 1983, 1993). 'Crew' is no longer grown because later-released cultivars of club wheat have a higher yield potential and better agronomic traits. 'Rely' has been widely grown in the PNW since its release, and its overall resistance remains adequate. Mixtures of cultivars carrying different resistance genes have also enhanced the durability of resistance (Finckh and Mundt 1992). About one third of the total wheat acreage in Washington of the PNW has been planted with mixtures of two or three cultivars.

### Molecular markers for resistance genes

The development of molecular markers for mapping resistance genes to stripe rust and of marker-assisted selection has been among the most active areas of research on stripe rust. Molecular markers have been identified for *Yr5* (Sun et al. 2002; Yan et al. 2003; Chen et al. 2003b), *Yr9* (Shi et al. 2001), *Yr10* (Frick et al. 1998; Shao et al. 2001; Bariana et al. 2002; Smith et al. 2002; Wang et al. 2002), *Yr15* (Chagué et al. 1999; Peng et al. 2000), *Yr17* (Robert et al. 1999; Seah et al. 2001), *Yr18* (Suenaga et al. 2003), *Yr24* (Zakari et al. 2003), *Yr26* (Ma et al. 2001), *Yr28* (Singh et al. 2000), *Yr32* (Eriksen et al. 2004), *Yr33* (McIntosh et al. 2004), *Yr34* (McIntosh et al. 2004), *Yr36* (J. Dubcovsky 2004, personal communication), *YrH52* (Peng et al. 2000), and *Yrns-B1* (Börner et al. 2000) (Table 6). Depending upon the closeness of markers to the genes, these markers should be useful in marker-assisted selection. Most of the markers were identified using RAPD, SSR, and AFLP techniques, while the markers for *Yr17* were identified using the clone of a gene-like sequence for resistance to disease (Seah et al. 2001). They used an approach similar to the resistance gene analog polymorphism (RGAP) technique that we developed (Chen et al. 1998b).

We constructed a linkage group consisting of 10 RGAP markers for the QTL for HTAP resistance to stripe rust in the winter wheat 'Stephens' (Chen and Line 2000). In that study with phenotypic and marker data of 121 F<sub>7</sub> lines derived from the 'Stephens' × 'Michigan Amber' cross, we identified cosegregating RGAP markers for two of the three genes conferring all-stage resistance in 'Stephens' (X.M. Chen 2004, unpublished data). Using RGAP and *Yr* near-

isogenic lines of wheat developed in the Plant Breeding Institute at the University of Sydney, Australia, we identified numerous RGAP markers that were specific for each of the 10 *Yr* near-isogenic lines (*YrA*, *Yr1*, *Yr5*, *Yr7*, *Yr8*, *Yr9*, *Yr10*, *Yr15*, *Yr17*, and *Yr18*) (Chen et al. 1998a), and constructed high resolution maps for *Yr9* (Shi et al. 2001), *Yr5* (Yan et al. 2003), and *Yr15* (Chen and Yan 2002). We identified six, four, and one RGAP markers cosegregating with *Yr5*, *Yr9*, and *Yr15*, respectively, and several other markers closely linked to these genes. We also developed markers for sequence-tagged sites (STSs) and cleaved amplified polymorphic sequences (CAPSs), based on the sequences of RGAP markers that were codominant and cosegregating with *Yr5* (Yan et al. 2003; Chen et al. 2003b). The molecular markers for *Yr5* and *Yr15* are currently being used to combine these two genes, each of which confers resistance to all races of *P. striiformis* f. sp. *tritici* in the United States. Codominant RGAP markers that cosegregate with *Yr5* and have very strong homologies with many clones of genes for plant resistance have provided us with markers to clone *Yr5*, using a gene-landing approach based on resistance gene analogs. We have constructed a BAC library for hexaploid wheat, using the genomic DNA from a *Yr5* near-isogenic line (Chen and Ling 2004). We have identified 12 positive BAC clones with the STS markers for *Yr5* and are currently subcloning them to identify the complete sequence of the resistance gene candidates.

Most wheat cultivars are resistant to stripe rust of barley caused by *P. striiformis* f. sp. *hordei* and most barley cultivars are resistant to stripe rust of wheat caused by *P. striiformis* f. sp. *tritici* (Chen et al. 1995a). The wheat 'Lemhi', which is susceptible to all races of *P. striiformis* f. sp. *tritici* except PST-21, is resistant to all of the PSH races tested. Similarly, the barley 'Stephoe', which is susceptible to all races of *P. striiformis* f. sp. *hordei*, is resistant to all races of *P. striiformis* f. sp. *tritici* that were tested. Recent genetic studies (Chen and Pahalawatta 2004) show that 'Lemhi' has a dominant gene (provisionally designated *RpsLem*) for resistance to two *P. striiformis* f. sp. *hordei* races used in the study. The gene is closely linked to *Yr21*, a gene for resistance to PST-21 (Chen et al. 1995b). Additionally, 'Stephoe' has a dominant gene and a recessive gene (provisionally designated *RpstS1* and *rpstS2*, respectively) for resistance to races PST-41 and PST-45 of *P. striiformis* f. sp. *tritici*. Using the RGAP technique, we constructed a linkage group for the linked genes, *RpsLem* and *Yr21*, in 'Lemhi' with 11 RGAP markers, and a linkage group for the dominant gene, *RpstS1*, in 'Stephoe' with 12 RGAP markers. Using a RGAP marker linked in repulsion to the resistant allele and a set of nulli-tetrasomic 'Chinese Spring' lines, the linkage group for *RpsLem* and *Yr21* was mapped to wheat chromosome 1B, confirming the chromosomal location of *Yr21* (Chen et al. 1995b). Using a chromosome-specific microsatellite marker, *Hvm68*, we mapped *RpstS1* on barley chromosome 4H. The results show that resistances in wheat to stripe rust of barley and in barley to stripe rust of wheat are qualitatively controlled by a single or a few genes. These genes may provide effective resistance when introgressed from wheat into barley and from barley into wheat against stripe rust of barley and

**Table 6.** Molecular markers and resistance genes to other diseases linked to genes for resistance to stripe rust in wheat.

Yr gene	Linked gene or marker	Reference
Yr5	<i>Xwgp-17-2B, Xwgp19-2B, Yr5STS7/8</i>	Chen et al. 2003b
Yr8	<i>Sr34</i>	McIntosh et al. 1998
Yr9	<i>Lr26, Sr31, Xwgp4, Xwgp7, Xwgp8, Xwgp9</i>	Shi et al. 2001
Yr10	<i>RgaYr10a, S26-M47, S13-M63</i>	Smith et al. 2002
Yr15	<i>Nor1, UBC212a, Xgwm413</i>	Peng et al. 2000
Yr17	<i>Lr37, Sr38, Vrgal</i>	Seah et al. 2001
Yr18	<i>Lr34, Xgwm295.1, Xgwm44</i>	Suenaga et al. 2003
Yr24	<i>Xgwm11-1B</i>	Zakari et al. 2003
Yr26	<i>Xgwm11, Xgwm18</i>	Ma et al. 2001
Yr27	<i>Lr13, Lr23</i>	McDonald et al. 2004
Yr28	<i>Xmwig634-4DS</i>	Singh et al. 2000
Yr29	<i>Lr46</i>	McIntosh et al. 1998
Yr30	<i>Sr2, Lr27</i>	McIntosh et al. 1998
Yr31	<i>Yr27, Yr23, Lr23</i>	McIntosh et al. 1998
Yr32	<i>Xwmc198, M62/P19-156, M59/P37-375</i>	Eriksen et al. 2004
Yr33	A 7DL marker	McIntosh et al. 2004
Yr34	<i>Xgwm6-5A, B1</i>	McIntosh et al. 2004
Yr36	<i>Xucw74-6B, Xucw77-6B</i>	J. Dubcovsky 2004, personal communication
YrH52	<i>Xgwm273a, UBC212a</i>	Peng et al. 2000
Yrns-B1	<i>Xgwm493</i>	Börner et al. 2000
YrA7	<i>Xgwm88.2, Xucw71</i>	Santra et al. 2005

stripe rust of wheat, respectively. Before taking this approach, studies should be conducted to determine if *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei* can combine their virulence genes to produce new isolates causing diseases on both wheat and barley crops.

### Control of stripe rust, using fungicides

Much of the early research on use of fungicides to control stripe rust in the United States was done by Hardison (1963, 1975), Powelson and Shaner (1966), and later, by Line and his associates (Line and Rowell 1973; Line 1976; Rakotondradona and Line 1984; reviewed in Line 2002). The first large-scale, successful use of fungicides to control stripe rust in North America occurred in 1981 (Line 2002). By that time, Line and his associates had demonstrated the effectiveness of triadimefon (Bayleton) and developed guidelines for the timely application for economical control. Through their efforts, triadimefon received emergency registration and was widely used in the PNW (Line 2002). Stripe rust was unusually severe throughout the PNW in 1981. When stripe rust was not controlled, highly susceptible cultivars, such as the winter club wheat 'Omar', were totally destroyed, while cultivars with moderate resistance to stripe rust had yield losses of 50%. Fungicide use prevented multimillion dollar losses (Line 2002). New foliar fungicides highly effective for control of stripe rust subsequently have been registered. In the 1990s, Cu and Line (1994) developed an expert system called MoreCrop (Managerial option for reasonable economical control of rusts and other pathogens) that combined information on management practices and use of fungicides and resistant cultivars into an integrated disease management program. Use of MoreCrop continues today.

Annual tests to determine efficacy, rates, and timing of fungicide application for control of stripe rust continue (Chen and Wood 2002, 2003, 2004), and new and more effective fungicides have been registered. Five fungicides, Tilt® (propiconazole), Quadris® (azoxystrobin), Stratego™ (propiconazole + trifloxystrobin), Headline™ (strobilurin), and Quilt™ (azoxystrobin + propiconazole) are currently registered in United States for use on wheat and barley to control stripe rust. In our laboratory, we conduct annual tests on about 24 winter-wheat and 16 spring-wheat cultivars to determine their responses to fungicide application. Data on yield losses from stripe rust and on yield increases attained with fungicide application for each cultivar are used to guide growers on whether or not to use fungicides. In recent years, use of fungicides has successfully reduced yield damage caused by stripe rust. In 2002, when stripe rust was widespread on susceptible and moderately susceptible spring-wheat cultivars, fungicide application (at a cost of about US\$2.5 × 10<sup>6</sup>) saved Washington State wheat growers about US\$30 × 10<sup>6</sup> (Chen et al. 2003a).

Nevertheless, the use of fungicides adds a huge cost to wheat production, which is a burden for many growers, especially in developing countries. The use of fungicides may also create health problems for users, adversely affect the environment, and result in the selection of fungicide-resistant strains of the pathogen. To avoid these problems, growing cultivars with adequate level of durable resistance is the best strategy to control stripe rust.

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## References

- Allan, R.E., Line, R.F., Peterson, C.J., Rubenthaler, G.L., Morrison, K.L., and Rohde, C.R. 1983. Crew, a multiline wheat cultivar. *Crop Sci.* 23: 1015–1016.
- Allan, R.E., Peterson, C.J., Line, R.F., Rubenthaler, G.L., and Morris, C.F. 1993. Registration of “Rely” wheat multiline. *Crop Sci.* 33: 213–214.
- Allison, C., and Isenbeck, K. 1930. Biologische specialisierung von *Puccinia glumarum tritici* Erikss. und Henn. *Phytopathol. Z.* 2: 87–98.
- Altschul, S.F., Madden, T.L., Schaffer, A.A., Zhang, J.H., Zhang, Z., Miller, W., and Lipman, D.J. 1997. Gapped BLAST and PSI-BLAST: a new generation of protein database search programs. *Nucleic Acids Res.* 25: 3389–3402.
- Angus, A. 1965. Annotated list of plant pests and diseases in Zambia. Parts 1–7 and supplements. Mount Makulu Research Station, Chilanga, Zambia.
- Bariana, H.S., and McIntosh, R.A. 1993. Cytogenetic studies in wheat. XIV. Location of rust resistance genes in VPM1 and their genetic linkage with other disease resistance genes in chromosome 2A. *Genome*, 36: 476–482.
- Bariana, H.S., Brown, G.N., Ahmed, N.U., Khatkar, S., Conner, R.L., Wellings, C.R., Haley, S., Sharp, P.J., and Laroche, A. 2002. Characterization of *Triticum vavilovii*-derived stripe rust resistance using genetic, cytogenetic and molecular analyses and its marker-assisted selection. *Theor. Appl. Genet.* 104: 315–320.
- Biffen, R.H. 1905. Mendel’s law of inheritance and wheat breeding. *J. Agric. Sci.* 1: 4–48.
- Börner, A., Röder, M.S., Unger, O., and Meinel, A. 2000. The detection and molecular mapping of a major gene for non specific adult plant disease resistance against stripe rust (*Puccinia striiformis*) in wheat. *Theor. Appl. Genet.* 100: 1095–1099.
- Boshoff, W.H.P., Pretorius, Z.A., and van Niekerk, B.D. 2002. Establishment, distribution, and pathogenicity of *Puccinia striiformis* f. sp. *tritici* in South Africa. *Plant Dis.* 86: 485–492.
- Britton, M., and Cummins, G.B. 1956. The reaction of species of *Poa* and grasses to *Puccinia striiformis*. *Plant Dis. Rep.* 40: 643–645.
- Brown, J.K.M., and Hovmøller, M.S. 2002. Aerial dispersal of pathogens on the global and continental scales and its impact on plant disease. *Science (Washington, D.C.)*, 297: 537–541.
- Brown, W.M., Jr., Hill, J.P., and Velasco, V.R. 2001. Barley yellow rust in North America. *Annu. Rev. Phytopathol.* 39: 367–384.
- Calonnet, A., Johnson, R., and de Vallavieille-Pope, C. 2002. Genetic analyses of resistance of the wheat differential cultivars Carstens V and Spaldings Prolific to two races of *Puccinia striiformis*. *Plant Pathol. (London)*, 51: 777–786.
- Cao, Z.J., Jing, J.X., Wang, M.N., Xu, Z.B., Shang, H.S., and Li, Z.Q. 2004. Analysis of stripe rust resistance inheritance of wheat cultivar Guinong 22. *Acta Bot. Boreali Occident. Sin.* 24: 991–996.
- Chagué, V., Fahima, T., Dahan, A., Sun, G.L., Korol, A.B., Ronin, Y.I., Grama, A., Röder, M.S., and Nevo, E. 1999. Isolation of microsatellite and RAPD markers flanking the *Yr15* gene of wheat using NILs and bulked segregant analysis. *Genome*, 42: 1050–1056.
- Chen, X.M. 2002. Genetics of wheat resistance to stripe rust. *In* *Wheat rusts in China. Edited by Q. Li and S.M. Zeng.* Chinese Agricultural Press, Beijing, China. pp. 173–184. [In Chinese.]
- Chen, X.M. 2004. Epidemiology of barley stripe rust and races of *Puccinia striiformis* f. sp. *hordei*: the first decade in the United States [online]. *In* *Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK.* European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. 2.8. Available from <http://www.crpmb.org/2004/1029chen> [accessed 8 July 2005].
- Chen, X.M., and Line, R.F. 1992a. Identification of stripe rust resistance genes in wheat cultivars used to differentiate North American races of *Puccinia striiformis*. *Phytopathology*, 82: 1428–1434.
- Chen, X.M., and Line, R.F. 1992b. Inheritance of stripe rust resistance in wheat cultivars used to differentiate races of *Puccinia striiformis* in North America. *Phytopathology*, 82: 633–637.
- Chen, X.M., and Line, R.F. 1993. Inheritance of stripe rust resistance in wheat cultivars postulated to have resistance genes at *Yr3* and *Yr4* loci. *Phytopathology*, 83: 382–388.
- Chen, X.M., and Line, R.F. 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 Line, R.F. 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., and Line, R.F. 2000. Developing molecular markers for quantitative trait loci conferring durable, high-temperature, adult-plant resistance in wheat to stripe rust with resistance gene analog polymorphism [online]. *In* *VIIIth International Plant and Animal Genome Conference. 9–12 January 2000, Town and Country Hotel, San Diego, Calif.* Available from <http://www.intl-pag.org/pag/8/abstracts/pag8874.html> [accessed 8 July 2005]. p. 101. [Abstr.]
- Chen, X.M., and Ling, P. 2004. Towards cloning wheat genes for resistance to stripe rust and functional genomics of *Puccinia striiformis* f. sp. *tritici* [online]. *In* *Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK.* European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A2.10. Available from <http://www.crpmb.org/icrpmb11/abstracts.htm> [accessed 8 July 2005].
- Chen, X.M., and Moore, M.K. 2002. Epidemics and races of *Puccinia striiformis* in North America in 2001. *Phytopathology*, 92: S14–S15.
- Chen, X.M., and Pahalawatta, V. 2004. Genetics and molecular mapping of resistance genes in wheat and barley against inappropriate formae speciales of *Puccinia striiformis* [online]. *In* *Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK.* European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A2.9. Available from <http://www.crpmb.org/icrpmb11/abstracts.htm> [accessed 8 July 2005].
- Chen, X.M., and Wood, D.A. 2002. Control of stripe rust of spring wheat with foliar fungicides, 2001. *Fungicide and*

- Nematicide Tests [serial online], Report 57:CF03. The American Phytopathological Society, St. Paul, Minn. doi: 10.1094/FN57.
- Chen, X.M., and Wood, D.A. 2003. Control of stripe rust of spring wheat with foliar fungicides, 2002. Fungicide and Nematicide Tests [serial online], Report 58:CF004. The American Phytopathological Society, St. Paul, Minn. doi: 10.1094/FN58.
- Chen, X.M., and Wood, D.A. 2004. Control of stripe rust of spring wheat with foliar fungicides, 2003. Fungicide and Nematicide Tests [serial online], Report 59:CF022. The American Phytopathological Society, St. Paul, Minn. doi: 10.1094/FN59.
- Chen, X.M., and Yan, G.P. 2002. Development of RGAP markers for stripe rust resistance gene *Yr15* and use of the markers to detect the gene in breeding lines. *Phytopathology*, 92: S14.
- Chen, X.M., Line, R.F., and Leung, H. 1993. Relationship between virulence variation and DNA polymorphism in *Puccinia striiformis*. *Phytopathology*, 83: 1489–1497.
- Chen, X.M., Line, R.F., and Leung, H. 1995a. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f. sp. *hordei* to other rusts. *Phytopathology*, 85: 1335–1342.
- Chen, X.M., Line, R.F., and Jones, S.S. 1995b. Chromosomal location of genes for stripe rust in spring wheat cultivars Compair, Fielder, Lee, and Lemhi and interactions of aneuploid wheats with races of *Puccinia striiformis*. *Phytopathology*, 85: 375–381.
- Chen, X.M., Jones, S.S., and Line, R.F. 1996. Chromosomal location of genes for resistance to *Puccinia striiformis* in seven wheat cultivars having resistance genes at *Yr3* and *Yr4* loci. *Phytopathology*, 86: 1228–1233.
- Chen, X.M., Line, R.F., Shi, Z.X., and Leung, H. 1998a. Genetics of wheat resistance to stripe rust. *In* Proceedings of the 9th International Wheat Genetics Symposium. 2–7 August 1998, University of Saskatchewan, Saskatoon, Sask. Edited by A.E. Slinkard. University Extension Press, University of Saskatchewan, Saskatoon, Sask. Vol. 3. pp. 237–239.
- Chen, X.M., Line, R.F., and Leung, H. 1998b. Genome scanning for resistance gene analogs in rice, barley, and wheat by high resolution electrophoresis. *Theor. Appl. Genet.* 97: 345–355.
- Chen, X.M., Moore, M.K., Milus, E.A., Long, D.L., Line, R.F., Marshall, D., and Jackson, L. 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., Moore, M.K., and Wood, D.A. 2003a. Epidemics and control of stripe rust on spring wheat in the Pacific Northwest in 2002. *Phytopathology*, 93: S16.
- Chen, X.M., Soria, M.A., Yan, G.P., Sun, J., and Dubcovsky, J. 2003b. Development of sequence tagged site and cleaved amplified polymorphic sequence markers for wheat stripe rust resistance gene *Yr5*. *Crop Sci.* 43: 2058–2064.
- Chen, X.M., Milus, E.A., Long, D.L., and Jackson, L.F. 2004. Impact of wheat stripe rust and races of *Puccinia striiformis* f. sp. *tritici* in the United States. *In* Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A2.11. Available from <http://www.crpmb.org/icrpmb11/abstracts.htm> [accessed 8 July 2005].
- Cu, R.M., and Line, R.F. 1994. An expert advisory system for wheat disease management. *Plant Dis.* 78: 209–215.
- Eriksen, L., Afshari, F., Christiansen, M.J., McIntosh, R.A., Jahoor, A., and Wellings, C.R. 2004. *Yr32* for resistance to stripe (yellow) rust present in the wheat cultivar Carstens V. *Theor. Appl. Genet.* 108: 567–575.
- Eriksson, J. 1894. Über die Spezialisierung des Parasitismus bei den Getreiderostpilzen. *Ber. Dtsch. Bot. Ges.* 12: 292–331.
- Eriksson, J., and Henning, E. 1894. Die Hauptresultate einer neuen Untersuchung über die Getreiderostpilze. *Z. Pflanzenkr.* 4: 197–203.
- Eriksson, J., and Henning, E. 1896. Die Getreideroste. Nortstedt & Söner, Stockholm.
- Eversmeyer, M.G., and Kramer, C.L. 2000. Epidemiology of wheat leaf rust and stem rust in the central great plains of the USA. *Annu. Rev. Phytopathol.* 38: 491–513.
- Fang, C.T. 1944. Physiological specialization of *Puccinia glumarum* Erikss., and Henn. in China. *Phytopathology*, 34: 1020–1024.
- Finckh, M.R., and Mundt, C.C. 1992. Stripe rust, yield, and plant competition in wheat cultivar mixtures. *Phytopathology*, 82: 82–92.
- Frick, M.M., Huel, R., Nykiforuk, C.L., Conner, R.L., Kusiak, A., and Laroche, A. 1998. Molecular characterisation of a wheat stripe rust resistance gene in Moro wheat. *In* Proceedings of the 9th International Wheat Genetics Symposium. 2–7 August 1998, University of Saskatchewan, Saskatoon, Sask. Edited by A.E. Slinkard. University Extension Press, University of Saskatchewan, Saskatoon, Sask. Vol. 3. pp. 181–182.
- Fuchs, E. 1960. Physiologische Rassen bei Gelbrost (*Puccinia glumarum* (Schm.) Erikss. et Henn.) auf weizen. *Nachrbl. Dtsch. Pflanzenschutzd. Braunsch.* 12: 49–63.
- Fuckel, L. 1860. Enumeratio fungorum Nassovia. *Jahrb. Ver. Naturk. Herzogth. Nassau*, 15: 9.
- Gassner, G., and Straib, W. 1932. Die Bestimmung der Biologischen Rassen des Weizengelbrostes [*Puccinia glumarum tritici* (Schmidt) Erikss. u. Henn.]. *Arb. Biol. Reichsanst. Land-Forstwirtschaft.* 20: 141–163.
- Gerechter-Amitai, Z.K., van Silfhout, C.H., Grama, A., and Kleitman, F. 1989. *Yr15* — a new gene for resistance to *Puccinia striiformis* in *Triticum dicoccoides* sel. G-25. *Euphytica*, 43: 187–190.
- Hardison, J.R. 1963. Commercial control of *Puccinia striiformis* and other rusts in seed crops of *Poa pratensis* by nickel fungicides. *Phytopathology*, 53: 209–216.
- Hardison, J.R. 1975. Control of *Puccinia striiformis* by two new systemic fungicides, Bay MEB 6447 and BAS 31702 F. *Plant Dis. Rep.* 59: 652–655.
- Hassebrauk, K. 1965. Nomenklatur, geographische Verbreitung und Wirtsbereich des Gelbrostes, *Puccinia striiformis* West. *Mitt. Biol. Bundesanst. Land-Forstwirtschaft. Berl.–Dahl.* 116: 1–75.
- Hassebrauk, K. 1970. De Gelbrost *Puccinia striiformis* West. 2. Befallsbild. Morphologie und Biologie der Sporen. Infektion und weitere Entwicklung. Wirkungen auf die Wirtspflanze. *Mitt. Biol. Bundesanst. Land-Forstwirtschaft. Berl.–Dahl.* 139: 1–111.
- Hassebrauk, K., and Röbbelen, G. 1974. Der Gelbrost *Puccinia striiformis* West. 3. Die Spezialisierung. *Mitt. Biol. Bundesanst. Land-Forstwirtschaft. Berl.–Dahl.* 156: 1–150.
- Hassebrauk, K., and Röbbelen, G. 1975. Der Gelbrost *Puccinia striiformis* West. 4. Epidemiology — Bekämpfungsmassnahmen. *Mitt. Biol. Bundesanst. Land-Forstwirtschaft. Berl.–Dahl.* 164: 1–183.
- Hovmöller, M.S., Justesen, A.F., and Brown, J.K.M. 2002. Clonality and long-distance migration of *Puccinia striiformis* f. sp. *tritici* in north-west Europe. *Plant Pathol. (London)*, 51: 24–32.

- Humphrey, H.B., Hungerford, C.W., and Johnson, A.G. 1924. Stripe rust (*Puccinia glumarum*) of cereals and grasses in the United States. *J. Agric. Res.* (Washington, D.C.), 29: 209–227.
- Hungerford, C.W., and Owens, C.E. 1923. Specialized varieties of *Puccinia glumarum* and hosts for variety *tritici*. *J. Agric. Res.* (Washington, D.C.), 25: 363–401.
- Hylander, N., Jørstad, I., and Nanfeldt, J.A. 1953. Enumeratio uredinearum Scandinavicarum. *Opera Bot.* 1: 1–102.
- Johnson, R., and Taylor, A.J. 1976. Yellow rust of wheat. Plant Breeding Institute, Cambridge, UK, 1975 Annual Report. pp. 106–109.
- Johnson, R., Stubbs, R.W., Fuchs, E., and Chamberlain, N.H. 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. *Trans. Br. Mycol. Soc.* 58: 475–480.
- Justesen, A.F., Ridout, C.J., and Hovmøller, M.S. 2002. The recent history of *Puccinia striiformis* f. sp. *tritici* in Denmark as revealed by disease incidence and AFLP markers. *Plant Pathol.* (London), 51: 13–23.
- Komjáti, H., Pasquali, M., Hubbard, A., Lee, D., and Bayles, R. 2004. IGS, SSR and SRAP analysis of *Puccinia striiformis* isolates. In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A2.33. Available from <http://www.crpmb.org/icrPMC11/abstracts.htm> [accessed 8 July 2005].
- Li, Z.Q., and Zeng, S.M. 2003. Wheat rusts in China. Chinese Agricultural Press, Beijing, China.
- Line, R.F. 1976. Chemical control of *Puccinia striiformis* and *Puccinia recondita* on wheat in Northwestern United States. In Proc. 4th Eur. Mediterr. Cereal Rusts Conf. 5–10 September 1976, Interlaken, Switzerland. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. pp. 105–108.
- 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 Chen, X.M. 1995. Successes in breeding for and managing durable resistance to wheat rusts. *Plant Dis.* 79: 1254–1255.
- Line, R.F., and Chen, X.M. 1996. Wheat and barley stripe rust in North America. In Proceedings of the 9th European and Mediterranean Cereal Rusts and Powdery Mildews Conference. 2–6 September 1996, Lunteren, Netherlands. Edited by G.H.J. Kema, R.E. Nike, and R.A. Damen. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, 24(Suppl.): 101–104.
- Line, R.F., and Rowell, J.B. 1973. Systemic fungicides for control of rusts and smuts. In Proceedings of the 2nd International Congress of Plant Pathology. 5–12 September 1973, Minneapolis, Minn. International Society for Plant Pathology. American Phytopathological Society, St Paul, Minn. Symposium Paper 549.
- Line, R.F., and Qayoum, A. 1992. Virulence, aggressiveness, evolution, and distribution of races of *Puccinia striiformis* (the cause of stripe rust of wheat) in North America, 1968–87. *US Dep. Agric. Agric. Res. Serv. Tech. Bull.* 1788.
- Line, R.F., Sharp, E.L., and Powelson, R.L. 1970. A system for differentiating races of *Puccinia striiformis* in the United States. *Plant Dis. Rep.* 54: 992–994.
- Lu, S.I., Fan, K.F., Shia, S.M., Mu, W.T., Kong, S.L., Yang, T.M., Wang, K.N., and Lee, S.P. 1956. Studies on stripe rust of wheat. 1. Physiologic specialization of *Puccinia glumarum* (Schmidt) Erikss. & Henn. *Chin. J. Plant Pathol.* 2: 153–166.
- Lupton, F.G.H., and Macer, R.C.F. 1962. Inheritance of resistance to yellow rust (*Puccinia glumarum* Erikss., and Henn.) in seven varieties of wheat. *Trans. Br. Mycol. Soc.* 45: 21–45.
- Lupton, F.G.H., Wilson, F.E., and Bingham, J. 1971. Breeding for non-race specific resistance to yellow rust and to mildew. 1970 Annual Report, Plant Breeding Institute Cambridge, UK.
- Ma, J., Zhou, R., Dong, Y., Wang, L., Wang, X., and Jia, J. 2001. Molecular mapping and detection of the yellow rust resistance gene *Yr26* in wheat transferred from *Triticum turgidum* L. using microsatellite markers. *Euphytica*, 120: 219–226.
- Macer, R.C.F. 1966. The formal and monosomic genetic analysis of stripe rust (*Puccinia striiformis*) resistance in wheat. In Proceedings of the 2nd International Wheat Genetics Symposium. 19–24 August 1963, Lund, Sweden. Edited by J. MacKey. *Hereditas* 2(Suppl.): 127–142.
- Macer, R.C.F. 1975. Plant pathology in a changing world. *Trans. Br. Mycol. Soc.* 65: 351–374.
- Madariaga, R., Mellado, M., and Becerra, V. 2004. Significance of wheat yellow rust (*Yr*) genes in Chile. In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A2.38. Available from <http://www.crpmb.org/icrPMC11/abstracts.htm> [accessed 8 July 2005].
- Manners, J.G. 1960. *Puccinia striiformis* Westend. var. *dactylidis* var. nov. *Trans. Br. Mycol. Soc.* 43: 65–68.
- Manninger, K. 2004. Virulence survey for wheat rusts in Hungary during 2000–2003. In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A2.41. Available from <http://www.crpmb.org/icrPMC11/abstracts.htm> [accessed 8 July 2005].
- Markell, S.G., Milus, E.A., and Chen, X.M. 2004. Genetic diversity of *Puccinia striiformis* f. sp. *tritici* in the United States. In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A2.43. Available from <http://www.crpmb.org/icrPMC11/abstracts.htm> [accessed 8 July 2005].
- McDonald, D.B., McIntosh, R.A., Wellings, C.R., Singh, R.P., and Nelson, J.C. 2004. Cytogenetical studies in wheat. XIX. Location and linkage studies on gene *Yr27* for resistance to stripe (yellow) rust. *Euphytica*, 136: 239–248.
- McIntosh, R.A. 1988. Catalogue of gene symbols for wheat. In Proceedings of the 7th International Wheat Genetics Symposium. 14–19 July 1988, Cambridge, UK. Edited by T.E. Miller and R.M.D. Koebner. Institute of Plant Science Research, Cambridge, UK. Vol. 2. pp. 1225–1323.
- McIntosh, R.A., Wellings, C.R., and Park, R.F. 1995. Wheat rusts: an atlas of resistance genes. Commonwealth Scientific and Industrial Research Organization, Australia, and Kluwer Academic Publishers, Dordrecht, Netherlands.
- McIntosh, R.A., Hart, G.E., Devos, K.M., Gale, M.D., and Rogers, W.J. 1998. Catalogue of gene symbols for wheat. In Proceedings of the 9th International Wheat Genetics Symposium. 2–7 August 1998, University of Saskatchewan, Saskatoon, Sask. Edited by A.E. Slinkard. University Extension Press, University of Saskatchewan, Saskatoon, Sask. Vol. 5. pp. 1–235.
- McIntosh, R.A., Devos, K.M., Dubcovsky, J., and Rogers, W.J.

2001. Catalogue of gene symbols for wheat: 2001 supplement [online]. Available from <http://grain.jouy.inra.fr/ggpages/wgc/2001upd.html> [accessed 31 December 2004].
- McIntosh, R.A., Devos, K.M., Dubcovsky, J., Morris, C.F., and Rogers, W.J. 2003. Catalogue of gene symbols for wheat: 2003 supplement [online]. Available from <http://grain.jouy.inra.fr/ggpages/wgc/2003upd.html> [accessed 31 December 2004].
- McIntosh, R.A., Devos, K.M., Dubcovsky, J., and Rogers, W.J. 2004. Catalogue of gene symbols for wheat: 2004 supplement [online]. Available from <http://grain.jouy.inra.fr/ggpages/wgc/2004upd.html> [accessed 31 December 2004].
- Milus, E.A., and Line, R.F. 1986a. Number of genes controlling high-temperature adult-plant resistance to stripe rust in wheat. *Phytopathology*, 76: 93–96.
- Milus, E.A., and Line, R.F. 1986b. Gene action for inheritance of durable, high-temperature, adult-plant resistance to stripe rust in wheat. *Phytopathology*, 76: 435–441.
- Milus, E.A., and Seyran, E. 2004. New races of *Puccinia striiformis* f. sp. *tritici* more aggressive than older races at 18 °C. In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. Cereal Rusts and Powdery Mildews Bulletin, Abstr. A2.50. Available from <http://www.crpmb.org/icrPMC11/abstracts.htm> [accessed 8 July 2005].
- Morgounov, A., Yessimbekova, M., Rsaliev, S., Baboev, S., Mumindjanov, H., and Djunusova, M. 2004. High-yielding winter wheat varieties resistant to yellow and leaf rust in Central Asia. In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. Cereal Rusts and Powdery Mildews Bulletin, Abstr. A2.52. Available from <http://www.crpmb.org/icrPMC11/abstracts.htm> [accessed 8 July 2005].
- Nagarajan, S., and Singh, D.V. 1990. Long-distance dispersion of rust pathogens. *Annu. Rev. Phytopathol.* 28: 139–153.
- Newton, A.C., Caten, C.E., and Johnson, R. 1985. Variation for isozyme and double-stranded RNA among isolates of *Puccinia striiformis* and two other cereal rusts. *Plant Pathol. (Oxford)*, 34: 235–247.
- Newton, M., and Johnson, T. 1936. Stripe rust, *Puccinia glumarum* in Canada. *Can. J. Res. Sect. C, Bot. Sci.* 14: 89–108.
- Niu, Y.C., Li, Z.Q., and Shang, H.S. 1991. *Puccinia striiformis* West. f. sp. *leymi* and f. sp. *elymi*, two new formae speciales. *Acta Univ. Agric. Boreali Occident.* 19: 58–62.
- O'Brien, L., Brown, J.S., Young, R.M., and Pascoe, I. 1980. Occurrence and distribution of wheat stripe rust in Victoria and susceptibility of commercial wheat cultivars. *Australas. Plant Pathol.* 9: 14.
- Pady, S.M., Johnston, C.O., and Rogerson, C.T. 1957. Stripe rust of wheat in Kansas in 1957. *Plant Dis. Rep.* 41: 959–961.
- Peng, J.H., Fahima, T., Roder, M.S., Huang, Q.Y., Dahan, A., Li, Y.C., Grama, A., and Nevo, E. 2000. High-density molecular map of chromosome region harboring stripe-rust resistance genes *YrH52* and *Yr15* derived from wild emmer wheat, *Triticum dicoccoides*. *Genetica (The Hague)*, 109: 199–210.
- Powelson, R.L., and Shaner, G.E. 1966. An effective chemical seed treatment for systemic control of seedling infection of wheat by stripe rust (*Puccinia striiformis*). *Plant Dis. Rep.* 50: 806–807.
- Pretorius, Z.A. 2004. The impact of wheat stripe rust in South Africa. In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference. 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. Cereal Rusts and Powdery Mildews Bulletin, Abstr. A1.29. Available from <http://www.crpmb.org/icrPMC11/abstracts.htm> [accessed 8 July 2005].
- Pretorius, Z.A., Boshoff, W.H.P., and Kema, G.H.J. 1997. First report of *Puccinia striiformis* f. sp. *tritici* on wheat in South Africa. *Plant Dis.* 81: 424.
- Priestley, R.H., and Dodson, J.K. 1976. Physiological specialization of *Puccinia striiformis* to adult plants of winter wheat cultivars in the United Kingdom. In Proc. 4th Eur. Mediterr. Cereal Rusts Conf. 5–10 September 1976, Interlaken, Switzerland. pp. 87–89.
- Qayoum, A., and Line, R.F. 1985. High-temperature, adult-plant resistance to stripe rust of wheat. *Phytopathology*, 75: 1121–1125.
- Rakotondradona, R., and Line, R.F. 1984. Control of stripe rust and leaf rust of wheat with seed treatments and effects of treatments on the host. *Plant Dis.* 68: 112–117.
- Rapilly, F. 1979. Yellow rust epidemiology. *Annu. Rev. Phytopathol.* 17: 59–73.
- Riley, R., Chapman, V., and Johnson, R. 1968. The incorporation of alien disease resistance in wheat by genetic interference with the regulation of meiotic chromosome synapsis. *Genet. Res.* 12: 713–715.
- Röbbelen, G., and Sharp, E.L. 1978. Mode of inheritance, interaction and application of genes conditioning resistance to yellow rust. *Fortschr. Pflanzenzücht.* 9: 1–88.
- Robert, O., Abelard, C., and Dedryver, F. 1999. Identification of molecular markers for the detection of the yellow rust resistance gene *Yr17* in wheat. *Mol. Breed.* 5: 167–175.
- Santra, D.P., Watt, C., Uauy, C., Kidwell, K.K., Chen, X.M., Dubcovsky, J., and Campbell, K.G. 2005. Mapping QTL for high temperature adult-plant resistance to stripe rust in wheat (*Triticum aestivum* L.) [online]. In XIII Plant and Animal Genome Conference. 15–19 January 2005, Town and Country Convention Centre, San Diego, Calif. Available from [http://www.intl-pag.org/13/abstracts/pag13\\_p331.html](http://www.intl-pag.org/13/abstracts/pag13_p331.html) [accessed 8 July 2005]. p. 155. [Abstr.]
- Schmidt, J.K. 1827. Allgemeine ökonomisch-technische Flora oder Abbildungen und Beschreibungen aller in bezug auf Ökonomie und Technologie, merkwürdigen Gewächse. Jena, Germany. Vol. I. p. 27.
- Seah, S., Bariana, H., Jahier, J., Sivasithamparam, K., Lagudah, E.S. 2001. The introgressed segment carrying rust resistance genes *Yr17*, *Lr37* and *Sr38* in wheat can be assayed by a cloned disease resistance gene-like sequence. *Theor. Appl. Genet.* 102: 600–605.
- Shan, W.X., Chen, S.Y., Kang, Z.S., Wu, L.R., and Li, Z.Q. 1998. Genetic diversity in *Puccinia striiformis* Westend. f. sp. *tritici* revealed by pathogen genome-specific repetitive sequence. *Can. J. Bot.* 76: 587–595.
- Shao, Y.T., Niu, Y.C., Zhu, L.H., Zhai, W.X., Xu, S.C., and Wu, L.R. 2001. Identification of an AFLP marker linked to the stripe rust resistance gene *Yr10* in wheat. *Chin. Sci. Bull.* 46: 1466–1469.
- Shi, Z.X., Chen, X.M., Line, R.F., Leung, H., and Wellings, C.R. 2001. Development of resistance gene analog polymorphism markers for the *Yr9* gene resistance to wheat stripe rust. *Genome*, 44: 509–516.
- Singh, R.P. 1992. 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., Nelson, J.C., and Sorrells, M.E. 2000. Mapping *Yr28* and other genes for resistance to stripe rust in wheat. *Crop Sci.* 40: 1148–1155.
- Smith, P.H., Koebner, R.M.D., and Boyd, L.A. 2002. The development of a STS marker linked to a yellow rust resistance derived from the wheat cultivar Moro. *Theor. Appl. Genet.* 104: 1278–1282.
- Stakman, E.C. 1934. Epidemiology of cereal rusts. *In Proc. Pac. Sci. Congr.* 5th, 1–14 June 1933, Victoria and Vancouver, B.C. University of Toronto Press, Toronto, Ont. Vol. 4. pp. 3177–3184.
- Straib, W. 1935. Infektionsversuche mit biologische Rassen des Gelbrostes (*Puccinia glumarum* (Schm.) Erikss. et Henn.) im Jahre 1934. *Arb. Biol. Reichsanst. Land- Forstwirtschaft. Berl.-Dahl.* 21: 455–466.
- Stubbs, R.W. 1985. Stripe rust. *In Cereal rusts.* Vol. II. Disease, distribution, epidemiology, and control. *Edited by A.P. Roelfs and W.R. Bushnell.* Academic Press, New York. pp. 61–101.
- Su, H., Conner, R.L., Graf, R.J., and Kuzyk, A.D. 2003. Virulence of *Puccinia striiformis* f. sp. *tritici*, cause of stripe rust on wheat, in western Canada from 1984 to 2002. *Can. J. Plant Pathol.* 25: 312–319.
- Suenaga, K., Singh, R.P., Huerta-Espino, J., and William, H.M. 2003. Microsatellite markers for genes *Lr34/Yr18* and other quantitative trait loci for leaf rust and stripe rust resistance in bread wheat. *Phytopathology*, 93: 881–890.
- Sun, Q., Wei, Y., Ni, C., Xie, C., and Yang, T. 2002. Microsatellite marker for yellow rust resistance gene *Yr5* introgressed from spelt wheat. *Plant Breed.* 121: 539–541.
- Sydow, P., and Sydow, H. 1904. *Monographia Uredinearum.* Vol. 1. Leipzig, Germany.
- 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.
- Vanderplank, J.E. 1963. *Plant diseases: epidemics and control.* Academic Press, New York.
- Wan, A.M., Zhao, Z.H., Chen, X.M., He, Z.H., Jin, S.L., Jia, Q.Z., Yao, G., Yang, J.X., Wang, B.T., Li, G.B., Bi, Y.Q., and Yuan, Z.Y. 2004. Wheat stripe rust epidemic and virulence of *Puccinia striiformis* f. sp. *tritici* in China in 2002. *Plant Dis.* 88: 896–904.
- Wang, K.N., Hong, X.W., Si, Q.M., Wang, J.X., and Shen, J.P. 1963. Studies of the physiological specialization of stripe rust of wheat in China. *Zhiwu Baohu Xuebao (J. Plant Prot.)*, 2: 23–35. [In Chinese with English abstract.]
- Wang, L.F., Ma, J.X., Zhou, R.H., Wang, X.M., and Jia, J.Z. 2002. Molecular tagging of the yellow rust resistance gene *Yr10* in common wheat, P.I. 178383 (*Triticum aestivum* L.). *Euphytica*, 124: 71–73.
- Wellings, C.R., and Kandel, K.R. 2004. Pathogen dynamics associated with historic stripe (yellow) rust epidemics in Australia in 2002 and 2003. *In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference.* 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A2.74. Available from <http://www.crpmb.org/icrpmb11/abstracts.htm> [accessed 8 July 2005].
- Wellings, C.R., and Luig, N.H. 1984. Wheat rusts, the old and the new. Australian Institute of Agricultural Science and Technology, Hawthorn, Occasional Publication, 15: 5–15.
- Wellings, C.R., and McIntosh, R.A. 1987. *Puccinia striiformis* f. sp. *tritici* in Eastern Australia — possible means of entry and implications for plant quarantine. *Plant Pathol. (Oxford)*, 36: 239–241.
- Wellings, C.R., and McIntosh, R.A. 1990. *Puccinia striiformis* f. sp. *tritici* in Australasia: pathogenic changes during the first 10 years. *Plant Pathol. (Oxford)*, 39: 316–325.
- Wellings, C.R., Wright, D.G., Keiper, F., and Loughman, R. 2003. First detection of wheat stripe rust in Western Australia: Evidence for a foreign incursion. *Australas. Plant Pathol.* 32: 321–322.
- Wellings, C.R., Burdon, J.J., and Keiper, F.J. 2004a. The biology of *Puccinia striiformis* on *Hordeum* spp. in Australia: the case for a new *forma specialis*. *In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference.* 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A1.50. Available from <http://www.crpmb.org/icrpmb11/abstracts.htm> [accessed 8 July 2005].
- Wellings, C.R., Singh, R.P., McIntosh, R.A., and Pretorius, Z.A. 2004b. The development and application of near isogenic lines for the stripe (yellow) rust pathosystem. *In Proceedings of the 11th International Cereal Rusts and Powdery Mildews Conference.* 22–27 August 2004, John Innes Centre, Norwich, UK. European and Mediterranean Cereal Rust Foundation, Wageningen, Netherlands. *Cereal Rusts and Powdery Mildews Bulletin*, Abstr. A1.39. Available from <http://www.crpmb.org/icrpmb11/abstracts.htm> [accessed 8 July 2005].
- Westendorp, G.D. 1854. Quatrième notice sur quelques Cryptogames récemment découvertes en Belgique. *Bull. Acad. R. Sci. Belg.* 21: 229–246.
- Worland, A.J., and Law, C.N. 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. Pflanzenzucht.* 96: 331–345.
- Yan, G.P., Chen, X.M., Line, R.F., and Wellings, C.R. 2003. Resistance gene analog polymorphism markers co-segregating with the *Yr5* gene for resistance to wheat stripe rust. *Theor. Appl. Genet.* 106: 636–643.
- Zadoks, J.C. 1961. Yellow rust on wheat: studies in epidemiology and physiologic specialization. *Tijdschr. Plantenziekten*, 67: 69–256.
- Zakari, A., McIntosh, R.A., Hovmoller, M.S., Wellings, C.R., Shariflou, M.R., Hayden, M., and Bariana, H.S. 2003. Recombination of *Yr15* and *Yr24* in chromosome 1BS. *In Proceedings of 10th International Wheat Genetics Symposium.* 1–5 September 2003, Rome, Italy. *Edited by N.E. Pogna, N. Romano, E.A. Pogna, and G. Galterio.* Instituto Sperimentale per la Cerealicoltura, Rome, Italy. Vol. 1. pp. 417–420.
- Zhang, J.Y., Xu, S.C., Zhang, S.S., Zhao, W.S., and Zhang, J.X. 2001. Monosomic analysis of resistance to stripe rust for source wheat line Jinghe 8811. *Acta Agronomica Sinica*, 27: 273–277.
- Zhao, W.S., Xu, S.C., Zhang, J.Y., and Wan, A.M. 2004. Inheritance of stripe rust resistance in wheat cultivar Jubilejna. II. *Acta Phytopylacica Sin.* 31: 127–133.
- Zheng, W.M., Liu, F., Kang, Z.S., Chen, S.Y., Li, Z.Q., and Wu, L.R. 2001. AFLP fingerprinting of Chinese epidemic strains of *Puccinia striiformis* f. sp. *tritici*. *Progr. Nat. Sci.* 11: 587–593.