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Tracking wheat rust on a continental scale

James A Kolmer

The rusts of wheat are important fungal plant pathogens that can be disseminated thousands of kilometers across continents and oceans by wind. Rusts are obligate parasites that interact with resistance genes in wheat in a gene-for-gene manner. New races of rust develop by mutation and selection for virulence against rust resistance genes in wheat. In recent years, new races of wheat leaf rust, wheat stripe rust, and wheat stem rust have been introduced into wheat production areas in different continents. These introductions have complicated efforts to develop wheat cultivars with durable rust resistance and have reduced the number of effective rust-resistance genes that are available for use. The migration patterns of wheat rusts are characterized by identifying their virulence against important rust resistance genes in wheat and by the use of molecular markers.

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Introduction

The rusts of wheat (*Triticum aestivum*) cause common and widespread wheat diseases that can be found in most areas of the world where wheat is grown. Wheat stem rust is caused by *Puccinia graminis* f. sp. *tritici*, wheat leaf rust by *Puccinia triticina*, and wheat stripe rust by *Puccinia striiformis*. The disease-causing wheat rust fungi are spread in the form of clonally produced dikaryotic urediniospores, which can be wind blown for thousands of kilometers from initial infection sites. Epidemics of wheat rusts can occur on a continental scale because of the widespread dispersal of urediniospores [1]. Wheat rust fungi are highly specific obligate parasites that interact with wheat in a gene-for-gene relationship [2,3]. This high degree of specificity has made durable rust resistance in wheat difficult to achieve because the virulence of wheat rust fungi against wheat resistance genes is highly diverse, resulting in the existence of many different pathogenic races. Rust races that are virulent against resistance genes that are newly deployed in wheat can rapidly increase in frequency over a large geographic area [4], thus rendering the resistance genes ineffective.

The specific interactions between resistance genes in wheat and avirulence genes in the rusts serve as extremely useful markers for characterizing rust populations. Near-isogenic [5] or single-gene lines [6] of wheat, which differ only by the presence of a single rust resistance gene, are used to identify races of rust fungi. Two genes for leaf rust resistance in wheat, *Lr10* [7•] and *Lr21* [8•], have been isolated, cloned, and sequenced. Both genes have sequences that encode nucleotide-binding site (NBS)-leucine-rich repeat (LRR) regions, which are characteristic of disease resistance genes in plants. Incompatible infections occur when the resistance gene interacts with a specific avirulence gene in the rust, and compatible infections occur in the absence of an avirulence gene. Rust populations can be characterized by distribution of races and the frequencies of virulence against specific rust resistance genes on a defined set of wheat differential hosts.

The avirulence genes that are present reflect only a small proportion of the total genetic variation present in rust populations, but this variation is subject to intense selection by the resistance genes in commonly grown wheat cultivars. Selectively neutral markers such as isozymes or more recently developed molecular markers, such as random amplified polymorphic DNA (RAPD), simple sequence repeat (SSR) and amplified fragment length polymorphism (AFLP), can also be used to characterize and compare rust populations. As the wheat rust fungi are spread easily within and between continents, it is essential to document the genetic changes in rust populations over large geographic areas so as to facilitate the development of rational strategies to prolong the effectiveness of rust resistance genes in wheat. This review examines genetic variation and recent changes in world-wide populations of the three wheat rusts.

Wheat leaf rust (*Puccinia triticina*)

Wheat leaf rust is the most common and widely distributed of the three wheat rusts (Figure 1). Extensive annual surveys of wheat leaf rust virulence phenotypes are conducted in both Canada [9•] and the US [10•]. Every year 40–60 races of *P. triticina* are identified in North America using a standardized set of 16 near-isogenic lines of Thatcher wheat. Leaf rust infections become established in the fall on winter wheats that are grown in the southern US, and the urediniospores are wind dispersed to the

Figure 1

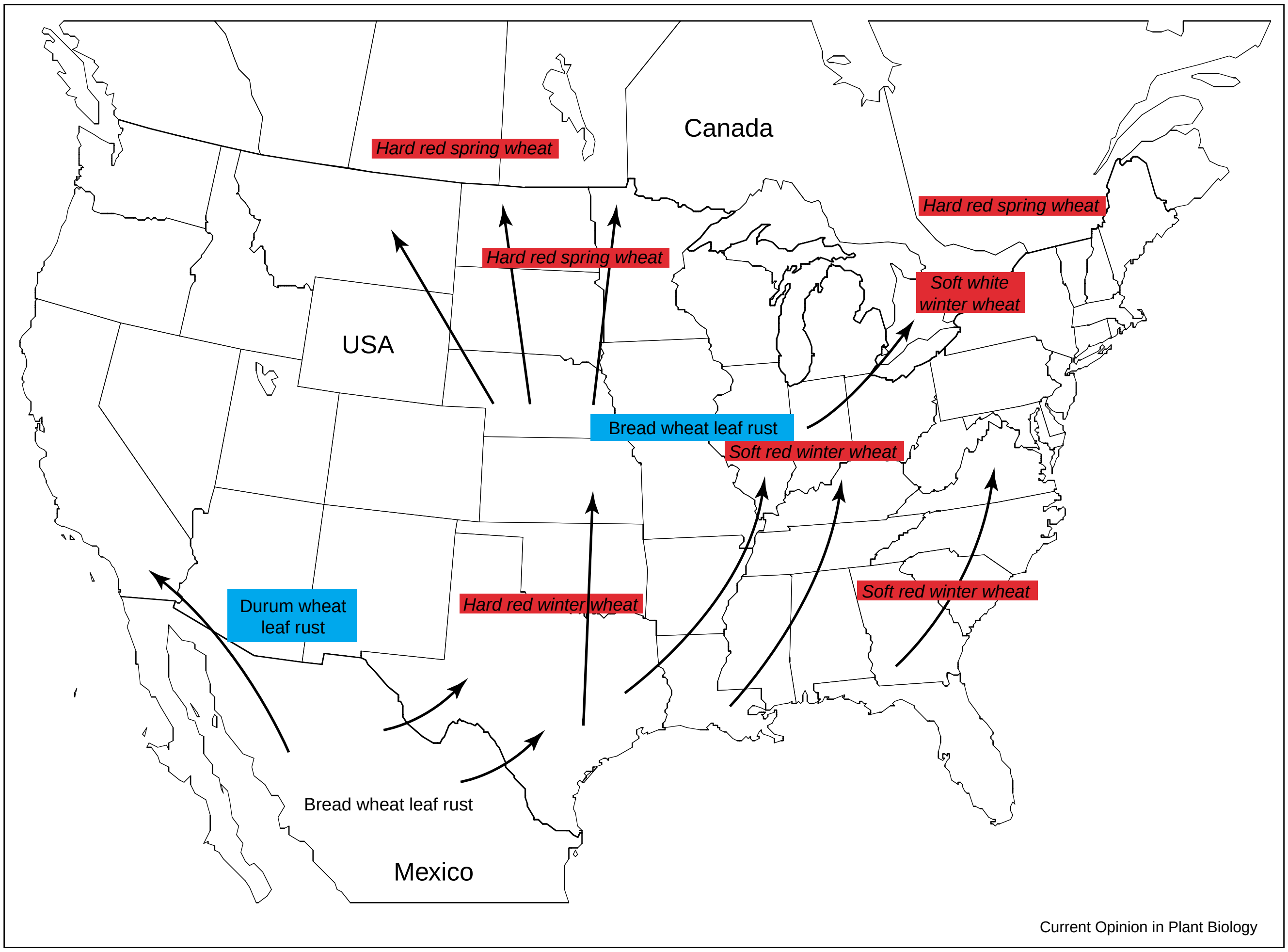
Wheat leaf rust caused by *Puccinia triticina*. (Courtesy of X Zhang, University of Minnesota, St. Paul, Minnesota, USA.)

northern US and Canada in the following spring and summer by the southerly winds (Figure 2). Distinct populations of *P. triticina* races can be found in the US because of the use of wheat cultivars that have different leaf rust resistance genes. Soft red winter wheats that are grown in the southeastern US have the leaf rust resistance genes *Lr9*, *Lr11*, *Lr18*, *Lr24*, and *Lr26*, and are infected by selected races that have virulence against these genes [11]. Similarly, races with virulence against *Lr3ka*, *Lr11*, *Lr24*, and *Lr26* have been selected by the hard red winter wheat cultivars that have been grown in the southern Great Plains region of Texas, Oklahoma, and Kansas since the late 1980s [12,13]. Races with virulence against *Lr2a* and *Lr16* are found in the northern Great Plains, where hard red spring wheats that have these genes are grown. Although the frequency of predominant races might differ in different wheat production regions, certain races have become widespread throughout the US and Canada because of the wind dispersal of infectious urediniospores. In 1996, races of *P. triticina* with virulence against *Lr17* were first detected in significant frequencies in the Great Plains of the US and in the prairie provinces of Canada. These races had been selected by a wheat cultivar grown in Kansas that had the resistance gene *Lr17*. By 2002, races with virulence against *Lr17* had spread to every wheat-growing area of the US, and were the most common races, accounting for over 33% of the *P. triticina* population [10•]. Distinct populations of *P. triticina* races in North America were also distinguished by use of RAPD [14] and AFLP [15] markers. The races with virulence against *Lr17* were determined to have AFLP phenotypes that were very distinct compared to those of the other groups of *P triticina* races in North America. A combination of the molecular data and the virulence data supported the hypothesis that this group of *P. triticina* isolates had been recently introduced to the US, most probably from Mexico.

In 2001, a new race of leaf rust with high virulence to durum wheat was detected in northwestern Mexico [16••]. This leaf rust race is unique as it is avirulent against many of the resistance genes present in common hexaploid wheat; yet the new race is virulent to most durum cultivars that were tested from a world-wide collection. This race might have been introduced to Mexico from other durum-growing areas of the world or might have evolved by mutation and selection in the durum fields of Mexico. Collections of leaf rust from fields of durum wheat in South America, western Europe, and the Middle East predominantly contained races that were identical or very similar to those from Mexico, as determined on the set of 16 standard Thatcher near-isogenic differential wheat lines (ME Ordoñez, JA Kolmer, unpublished). A single group of *P. triticina* races that are virulent to durum wheats has rapidly spread and become established in Europe and the Americas.

Australia and New Zealand are geographically separated from other major wheat-producing areas of the world, yet new rust races from other continents have been introduced into these countries numerous times. In 1981, a leaf rust race was detected in New Zealand that was virulent to the cultivar Karamu. This race differed from all other leaf rust races in New Zealand and Australia in terms of its

Figure 2



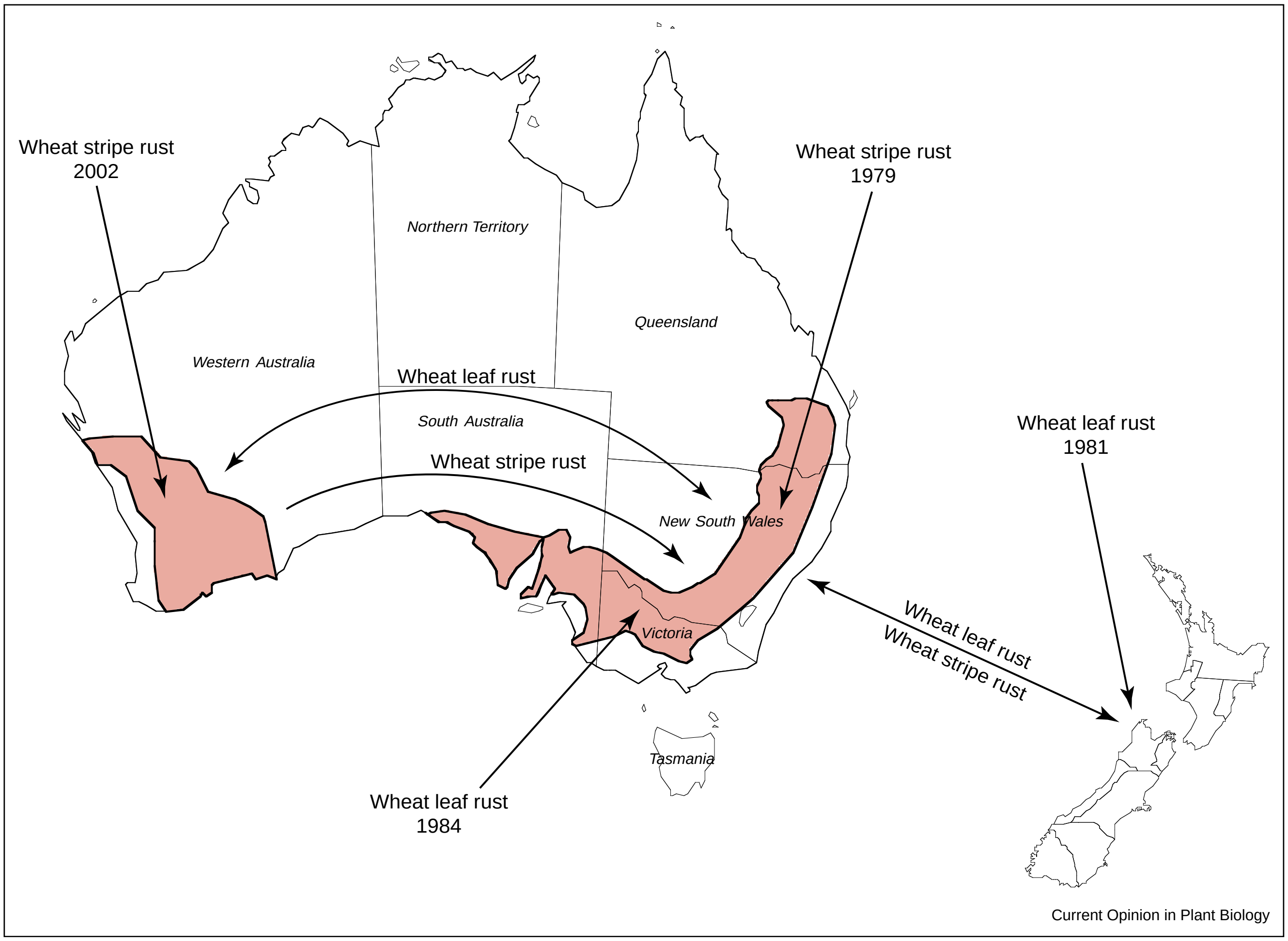
Current Opinion in Plant Biology

Migration of wheat leaf rust, *Puccinia triticina*, in North America. Leaf rust overwinters in Mexico and throughout much of the southern US. In the spring, urediniospores are carried in the southerly winds to wheat-production areas in the US Great Plains, the Canadian prairies, the Ohio Valley region, and the north-eastern US states. Leaf rust can also overwinter in northern areas of wheat production, such as southern Ontario in Canada, with adequate snow cover and moderate temperatures. The hard red winter wheats, soft red winter wheats, soft white winter wheats, and hard red spring wheats have different genes for resistance to leaf rust. Different leaf rust races occur in areas with different wheat classes because of host selection for virulent races. In recent years, durum leaf rust from Mexico has migrated to durum fields in southern California.

virulence to host differentials and isozyme variation at the glucosephosphate isomerase locus (*GOT*) (Figure 3; [17]). The virulence and isozyme differences could not be explained by single-step mutation of a race from the Australasian *P. triticina* population and subsequent selection. Within a few years, the introduced race was the most common leaf rust race in New Zealand. In 1984, a new leaf rust race that was distinct for virulence to wheats containing the *Lr16*, *Lr27* and *Lr31* genes and for isozyme variation at the phosphoglucosmutase locus (*Pgm2*) was detected in Australia [18]. On the basis of the virulence and isozyme data, it was hypothesized that this race was also introduced into Australasia. Additional races that were derived by mutation from the original introduction increased rapidly in frequency throughout eastern and western Australia and New Zealand, displacing races that had been common before 1984. Of the six leaf rust races identified in 2003 or 2004 in Australia, all were derived by mutation from the race introduced in 1984 (RF Park, pers. comm.).

In 2000, a leaf rust race with virulence against *Lr24* was detected in Australia for the first time [19••] although wheat cultivars that have this resistance gene had been grown since 1983. The emergence of the *Lr24*-virulent race could be explained by the mutation of and selection from a previously existing race. It is remarkable that *Lr24*-virulent races were not detected previously in Australia. In the US, virulence against *Lr24* appeared within a few years of the introduction of winter wheat cultivars with *Lr24* on the southern Great Plains [20]. In Australia, almost all released wheat cultivars are highly resistant to leaf rust, and the effective population size of *P. triticina* in which

Figure 3



Eastern and western wheat-production areas of Australia and New Zealand. A new race of wheat leaf rust was introduced into New Zealand in 1981. An additional new leaf rust race was introduced into eastern Australia in 1984. This new Australian leaf rust race, and derivatives of this race that have arisen by mutation, have spread throughout Australia. Wheat stripe rust was introduced for the first time to Australia in 1979 in the eastern wheat region. In 2002, stripe rust was found for the first time in western Australia having originated from a foreign source. This new stripe rust race had spread to eastern Australia by 2003.

mutation might occur is greatly reduced. The delay of the emergence of *Lr24*-virulent leaf rust races in Australia might reflect a reduced probability of mutation to *Lr24* virulence in the Australian population. Many of the winter wheats grown in the southern US are susceptible to leaf rust, which allows a very large leaf rust population to survive, creating a reservoir for mutation and selection.

Long-distance dissemination of *P. triticina* also occurs in Europe, since four races accounted for 64% of all isolates in collections from western Europe [21]. Multiple isolates of the same race from different countries have been found to have identical RAPD banding patterns [22], providing further evidence of the long-distance transport of *P. triticina* isolates in Europe. Isolates from central Europe were found to have high frequencies of virulence against the *Lr3*, *Lr3ka*, *Lr3bg*, and *Lr26* genes, whereas most isolates from western Europe were avirulent to these genes [23]. Cluster analysis of RAPD markers indicated that isolates from Great Britain, Italy, and Spain were more closely related to each other than to isolates from Hungary and Slovakia, which indicated the existence of at least two major groups of *P. triticina* in Europe. Isolates from South America had RAPD phenotypes that were nearly identical to those of isolates from western Canada, yet the two populations had different patterns of virulence [23]. Similarity of RAPD phenotypes suggest that the western Canadian and South American isolates of *P. triticina* might have derived from introductions from the same source, presumably from Europe, but the subsequent selection of these introductions by wheats that had different resistance genes might account for the dissimilarities in the virulence of *P. triticina* isolates from western Canada and South America.

Wheat stripe rust (*Puccinia striiformis* f. sp. *tritici*)

Stripe rust (Figures 3 and 4) in Australia provides a classic example of a pathogen being introduced to a new continent followed by mutation and selection for virulence [24]. Stripe rust was accidentally transported by man from Europe to the wheat-growing region of eastern Australia and was first detected in 1979. The initial introduction was virulent against the resistance gene *Yr2*. Within ten years, 15 different races that were virulent against resistance genes *YrA*, *Yr5*, *Yr6*, *Yr7* and *Yr8*, which were derived by single-step changes in virulence from the original introduction, were detected in Australia and New Zealand. Isolates of different *P. striiformis* races collected from 1979 to 1991 in Australia showed no polymorphism for RAPD or AFLP markers [25], supporting the hypothesis that they were derived from a single introduction followed by mutation and selection for virulence. Another introduction of stripe rust, apparently from a foreign source, occurred in western Australia, where a new race with virulence against *Yr6*, *Yr7*, *Yr8*, *Yr9* and *YrA* was observed in 2002 [26•]. By 2003, the new race had also spread into the wheat-producing area of eastern Australia, and by 2004, accounted for the majority of stripe collections in the entire country. Stripe rust was also recently introduced to South Africa, being detected first in 1996 [27••]. Initially, a single race with virulence against *Yr2*, *Yr6*, *Yr7*, *Yr8*, *Yr11*, *Yr14*, *Yr17* and *Yr19* was present. By 1998, a second race with additional virulence against *Yr25* was detected.

Figure 4

Wheat stripe rust caused by *Puccinia striiformis*. (JA Kolmer, USDA-ARS, Cereal Disease Laboratory, St. Paul, Minnesota, USA.)

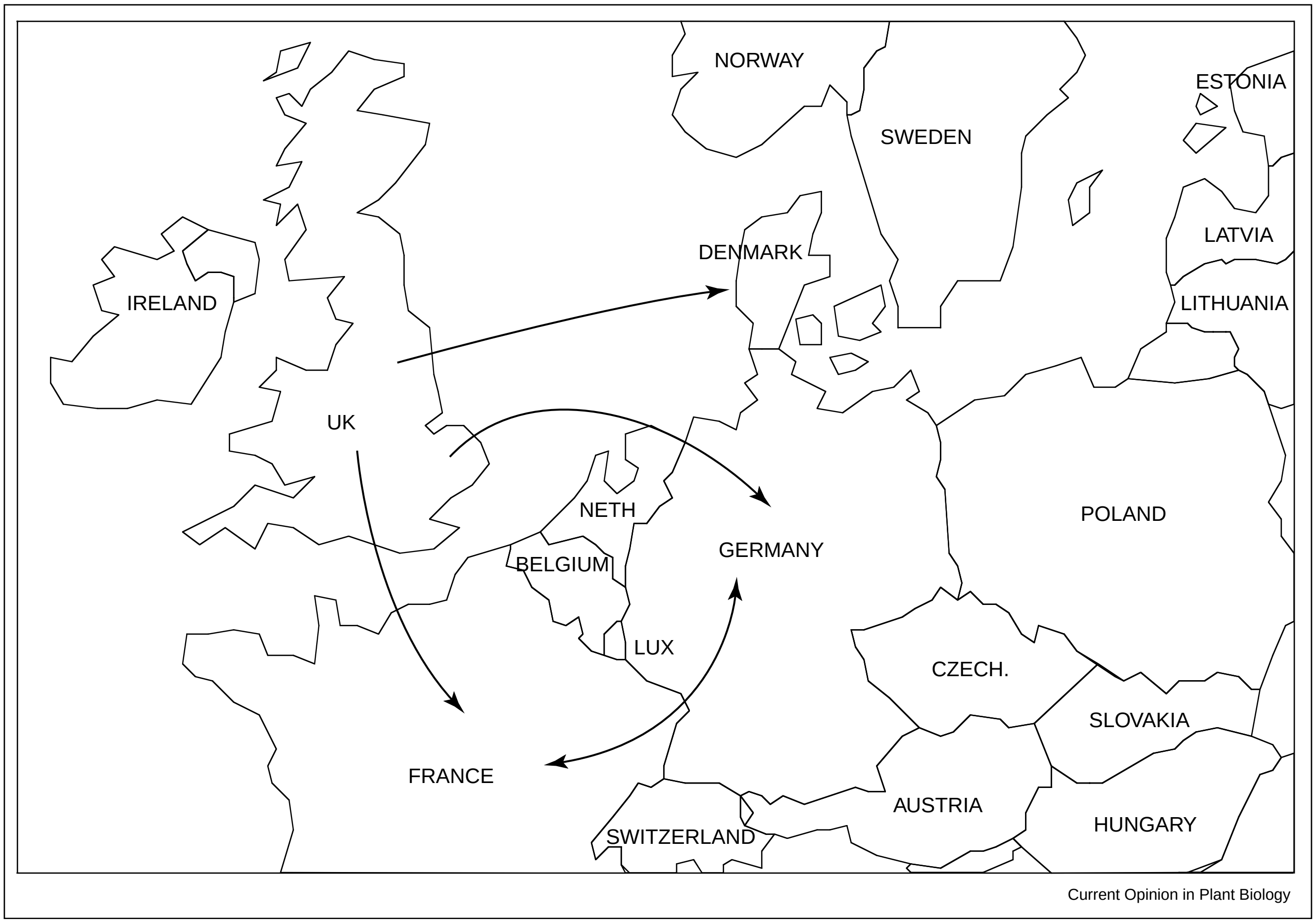
Stripe rust is a long-established disease of wheat in northwestern Europe, although local extinction events within individual countries occur periodically. For example, in 1996, stripe rust was not found at any location in Denmark [28••]. The following year the disease was observed at ten locations, and in 1998, the disease had spread throughout Denmark. The isolates collected in 1997 and 1998 were of two races with virulence against *Yr17* that had not been found previously in Denmark. Isolates of the two races had three AFLP phenotypes, which were found in both 1997 and 1998. The diversity of AFLP phenotypes was greater in collections of isolates that were obtained before 1996. On the basis of the virulence and AFLP data, it was hypothesized that stripe rust was re-introduced into Denmark in 1997 from other countries to the south or west. Further research with virulence patterns and AFLP markers determined that a single stripe rust population with regular west to east migration is found in the UK, France, Germany, and Denmark (Figure 5; [29••]). Isolates of five *P. striiformis* clones that had identical virulence patterns and AFLP phenotypes were wind-disseminated from the UK to Denmark. The AFLP and virulence data also indicated the migration of stripe rust from the UK to Germany and France.

In North America, stripe rust has long been a common disease of wheat in the Pacific Northwest region of the US and Canada. In 2000, wheat stripe rust was severe in California and in the south central states of Texas, Oklahoma, Arkansas, but only low levels of infection occurred throughout the Great Plains region of the US [30•]. New races with virulence against the *Yr8* and *Yr9* genes and to the cultivars Clement and Compair were found in the isolates collected in 2000. Virulence against *Yr8* and *Yr9* had not been previously found in North America. Mexico or California might have been the inoculum source for the stripe rust epidemic in the south central states. The stripe rust races introduced to western Australia have virulence characteristics similar to those of the new US races.

Wheat stem rust (*Puccinia graminis* f. sp. *tritici*)

Wheat stem rust epidemics occurred regularly from 1900–1955 in North America [31]. Heavy epidemics in 1916, 1935, 1937, 1950–1954 and other years caused massive yield losses in wheat. Since then, wheat stem rust has successfully been controlled in North America and most parts of the world by the use of highly resistant wheat

Figure 5



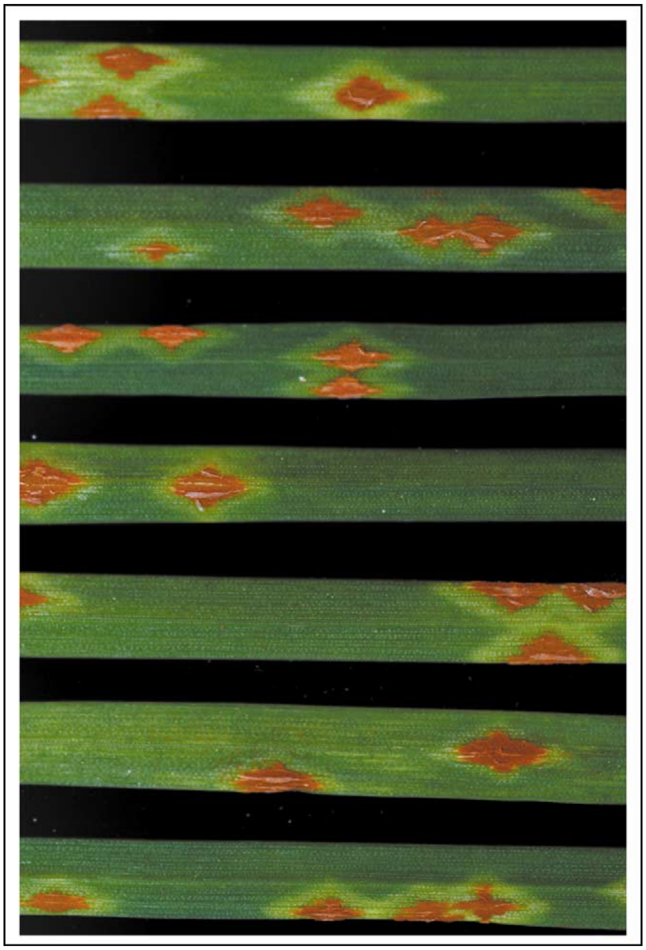
Migration of wheat stripe rust, *Puccinia striiformis,* in northwestern Europe. A single migratory wheat stripe rust population was characterized in Denmark, France, Germany, and the UK. Wheat leaf rust also migrates between countries in northwestern Europe. However, wheat leaf rust isolates from Hungary, Slovakia, and the Czech Republic can be distinguished from those found in northwestern Europe on the bases of virulence and molecular markers.

cultivars, and by eradication of the barberry (*Berberis vulgaris*) alternate host. This has greatly reduced the number of stem rust races and the effective size of the *P. graminis* population, therefore directly reducing the chances of a virulence mutation occurring in an adapted *P. graminis* genotype. In recent years, relatively few races of wheat stem rust have been found in North America [32•]. In the US, six highly discrete virulence groups of *P. graminis* have been described in the asexual population on the Great Plains using 16 host differential single gene lines [33]. These virulence groups correlated very highly with patterns of isozyme variation at 13 loci [34]. In 1989, a stem rust race that had high virulence against the stem rust resistance gene *Rpg1* in barley appeared in the Great Plains region of the US and Canada [35]. This race was avirulent to nearly all of the spring and winter wheats grown in North America, yet was virulent to nearly all of the barley cultivars that were tested. The new race differed for virulence against the resistance genes *Sr6*, *Sr12*, *Sr13*, and *Sr33* when compared to the previously characterized virulence groups, and was virulent to the cultivar McNair. The stem rust isolates that had virulence against *Rpg1* also differed from other stem rust races in their isozyme genotypes at two loci. It is likely that the stem rust race with virulence to *Rpg1* was wind disseminated to the Great Plains region from a sexual population of *P. graminis* in the Pacific Northwest. The stem rust populations in North and South America are speculated to have originated by introductions from the same or similar sources in Europe because there is no clear difference between isolates from these regions for virulence patterns or RADP markers [36].

Many wheat cultivars throughout the world have the stem rust resistance gene *Sr31*, which was introgressed into wheat on a translocated chromosomal fragment from rye. This gene has provided highly effective resistance for many years. Stem rust races with virulence against

Sr31 were not detected despite the extensive cultivation of wheat with this gene. In 1999, high levels of stem rust infections were found in Uganda on wheat genotypes that had the rye translocation and *Sr31* [37•]. Subsequent tests confirmed that a new race of *P. graminis* with virulence against *Sr31* had been found in central Africa. The new phenotypes were also virulent against *Sr38*, for which virulence had never been previously detected. In 2004, heavy stem rust infections were observed on International Center for Wheat and Maize Improvement (CIMMYT)-derived lines wheat lines in Kenya (RP Singh, pers. comm.). The stem rust infections in Kenya were caused by the same race that had virulence against *Sr31* and *Sr38* originally detected in Uganda (Y Jin, pers. comm.). This new race of wheat stem rust might soon migrate further and threaten wheat production in the Middle East and Central Asia. The stem rust race from central Africa is also virulent to many of the wheats currently grown in Canada and the US (Figure 6). It will be essential to develop germplasm that has effective resistance to this dangerous new race in any potentially affected regions.

Figure 6



Virulent infection types of wheat stem rust, *Puccinia graminis*, from Kenya on North American spring wheats. (Courtesy of Y Jin, USDA-ARS, Cereal Disease Laboratory, St. Paul, Minnesota, USA.)

Conclusions

New races of wheat leaf rust, wheat stripe rust, and wheat stem rust continue to emerge and can be transported for thousands of kilometers across continents and oceans to wheat growing areas that are far removed from the sites of their original detection. Introductions of foreign rust races can have dire consequences for wheat production and wheat improvement programs. New races of rust can have virulence against resistance genes that are not currently present in wheat cultivars or breeding lines, rendering these genes ineffective even before the genes are selected for use in a breeding program. Introduced races might also be virulent to genes that are currently used to provide resistance in wheat cultivars, thus shortening the effective life-span of a cultivar. In the future, it will be essential to continue the monitoring of wheat rust populations world-wide to allow wheat pathologists and breeders to anticipate and prepare for the occurrence of new races that might potentially threaten wheat production. The genotyping of rust populations using recently developed SSR markers [38•,39•], combined with testing for virulence against important rust resistance genes, will further refine analyses of the migration of these globally important plant pathogens.

Acknowledgements

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Microsatellite loci were characterized for the wheat leaf rust fungus. Thirty-six primer pairs amplified leaf rust DNA. Twelve primer pairs were polymorphic among 15 leaf rust isolates from Europe. Twelve of the primers also cross-amplified the DNA of wheat leaf rust. The development of markers such as microsatellites will allow the determination of leaf rust molecular genotypes. Previous molecular markers, such as RAPDs and AFLPs, could only distinguish phenotypes because they were unable to differentiate heterozygotes and homozygotes.

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Microsatellite primers were characterized for the wheat stripe rust fungus. Twenty-four primer pairs amplified stripe rust DNA, and 12 primers were polymorphic among 64 French isolates and 32 Chinese isolates. Twelve of the primers also cross-amplified the DNA of wheat leaf rust. The development of markers such as microsatellites will allow the determination of stripe rust molecular genotypes. Previous molecular markers, such as RAPDs and AFLPs, could only distinguish phenotypes because they were unable to differentiate heterozygotes and homozygotes.