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Pathogen profile

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

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SUMMARY

Stripe (yellow) rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is a serious disease of wheat occurring in most wheat areas with cool and moist weather conditions during the growing season. The basidiomycete fungus is an obligate biotrophic parasite that is difficult to culture on artificial media. *Pst* is a macrocyclic, heteroecious fungus that requires both primary (wheat or grasses) and alternate (*Berberis or Mahonia* spp.) host plants to complete its life cycle. Urediniospores have the capacity for wind dispersal over long distances, which may, under high inoculum pressure, extend to thousands of kilometres from the initial infection sites. Stripe rust, which is considered to be the current major rust disease affecting winter cereal production across the world, has been studied intensively for over a century. This review summarizes the current knowledge of the *Pst*–wheat pathosystem, with emphasis on the life cycle, uredinial infection process, population biology of the pathogen, genes for stripe rust resistance in wheat and molecular perspectives of wheat–*Pst* interactions.

Taxonomy: The stripe rust pathogen, *Puccinia striiformis* Westend. (*Ps*), is classified in kingdom Fungi, phylum Basidiomycota, class Urediniomycetes, order Uredinales, family Pucciniaceae, genus *Puccinia*. *Ps* is separated below the species level by host specialization on various grass genera, comprising up to nine formae speciales, of which *P. striiformis* f. sp. *tritici* Erikss. (*Pst*) causes stripe (or yellow) rust on wheat.

Host range: *Uredinial/telial hosts: Pst* mainly infects common wheat (*Triticum aestivum* L.), durum wheat (*T. turgidum* var. *durum* L.), cultivated emmer wheat (*T. dicoccum* Schrank), wild emmer wheat (*T. dicoccoides* Korn) and triticale (*Triticosecale*). *Pst* can infect certain cultivated barleys (*Hordeum vulgare* L.) and rye (*Secale cereale* L.), but generally does not cause severe epidemics. In addition, *Pst* may infect naturalized and improved pasture grass species, such as *Elymus canadensis* L., *Leymus secalinus* Hochst, *Agropyron* spp. Garetn, *Hordeum* spp. L., *Phalaris* spp. L and *Bromus unioloides* Kunth. *Pycnial/aecial (alternative) hosts*: Barberry (*Berberis chinensis*, *B. koreana*, *B. holstii*, *B. vulgaris*, *B. shensiana*, *B. potaninii*, *B. dolichobotrys*, *B. heteropoda*, etc.) and Oregon grape (*Mahonia aquifolium*).

Disease symptoms: Stripe rust appears as a mass of yellow to orange urediniospores erupting from pustules arranged in long, narrow stripes on leaves (usually between veins), leaf sheaths, glumes and awns on susceptible plants. Resistant wheat cultivars are characterized by various infection types from no visual symptoms to small hypersensitive flecks to uredinia surrounded by chlorosis or necrosis with restricted urediniospore production. On seedlings, uredinia produced by the infection of a single urediniospore are not confined by leaf veins, but progressively emerge from the infection site in all directions, potentially covering the entire leaf surface. Individual uredinial pustules are oblong, 0.4–0.7 mm in length and 0.1 mm in width. Urediniospores are broadly ellipsoidal to broadly obovoid, (16–)18–30(–32) × (15–)17–27(–28) μm, with a mean of 24.5 × 21.6 μm, yellow to orange in colour, echinulate, and with 6–18 scattered germ pores. Urediniospores can germinate rapidly when free moisture (rain or dew) occurs on leaf surfaces and when the temperatures range is between 7 and 12 °C. At higher temperatures or during the later growing stages of the host, black telia are often produced, which are pulvinate to oblong, 0.2–0.7 mm in length and 0.1 mm in width. The teliospores are predominantly two-celled, dark brown with thick walls, mostly oblong-clavate, (24–)31–56(–65) × (11–)14–25(–29) μm in length and width, and rounded or flattened at the apex.

Keywords: genes for resistance, infection procedure, life cycle, molecular aspect, population biology.

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INTRODUCTION

Stripe (yellow) rust, caused by *Puccinia striiformis* Westend. (*Ps*), is one of the most widely destructive plant diseases in modern winter cereal production (Wellings, 2011). The specialized form infecting wheat is referred to as *P. striiformis* f. sp. *tritici* (*Pst*). The sexual stage on *Berberis* spp. and *Mahonia* spp. has only been described recently (Jin *et al*., 2010; Wang and Chen, 2013; Zhao *et al*., 2011, 2013). Mutation, somatic recombination, parasexuality, selection and probably sexual recombination are considered to be the mechanisms that drive pathogen variability (Duan *et al*., 2010a; Hovmøller *et al*., 2011; Mboup *et al*., 2009; Stubbs, 1985). The centre of origin for *P. striiformis* was earlier assumed to be Transcaucasia, where grasses were the primary host (Hassebrauk, 1965), and from there the pathogen dispersed in all directions, including eastward to East Asia and North America in the 1910s (Carleton, 1915; Fraser and Conners, 1925; Humphrey *et al*., 1924), and southward into West Asia and East Africa. Geographically isolated wheat-producing regions were invaded by long-distance pathogen migration: Australia from Europe in 1979 (O'Brien *et al*., 1980); Republic of South Africa from the Middle East in 1996 (Boshoff *et al*., 2002); Western Australia from North America in 2002 (Wellings, 2007). Western China and Central Asia are also speculated as a centre of origin based on a high degree of telial production for isolates sampled in those areas and a high genetic diversity consistent with frequent recombination signatures (Ali *et al*., 2010b; Mboup *et al*., 2009).

Stripe rust is considered to be a low-temperature disease and frequently occurs in temperate areas with cool and moist weather conditions. Recent devastating epidemics have occurred in warmer areas where the disease was previously infrequent or absent (Hovmøller *et al*., 2010; Mboup *et al*., 2009). This led to the proposal that populations of *Pst* had developed adaptation to higher temperature, and supporting evidence was published by Milus *et al*. (2008). The minimum, optimum and maximum temperatures for urediniospore germination are 0 °C, 7–12 °C and 20–26 °C, respectively (Schröder and Hassebrauk, 1964). Of the three wheat rusts (stripe rust, leaf rust and stem rust), stripe rust appears to be the most sensitive to environmental factors, such as air pollution and UV light, which reduce the germination of urediniospores (Sharp, 1967). Resistance of the host is also influenced by temperature and light, which, in turn, influences disease assessment of the infected plant (Sharp and Volin, 1970). Increasing day length or light intensity lowers the infection type (Bever, 1934; Stubbs, 1967; Wellings *et al*., 1988).

Yield losses in wheat from *Pst* infections are usually the result of reduced kernel number per spike, low test weight and reduced kernel quality (Prescott *et al*., 1986). Evidence from historical epidemics and crop losses, and a contemporary analysis of the incidence and crop loss magnitude experienced in current major world wheat-growing regions, concluded that *Pst* was the most serious biotic threat to sustainable international wheat production (Wellings, 2011).

In this review, we summarize the current understanding of *Pst* biology, including studies of host–pathogen interactions, to develop strategies for resistance breeding which may form the basis for the development of practical control strategies in this economically significant pathosystem.

LIFE CYCLE

In the asexual urediniospore stage, *Ps* infects a range of grasses within the Pooideae subfamily of the Poaceae. Collections of *Ps* from certain grasses showed evidence of host specialization, and this led Eriksson (1894) to propose five special forms (Latin: 'formae speciales') based on the originating host genus, namely *P. striiformis* f. sp. *tritici* (*Pst*) specialized on wheat, *P. striiformis* f. sp. *hordei* (*Psh*) on barley, *P. striiformis* f. sp. *secalis* on rye, *P. striiformis* f. sp. *elymi* on *Elymus* spp. and *P. striiformis* f. sp. *agropyri* on *Agropyron* spp. Additional formae speciales were reported: *P. striiformis* f. sp. *dactylidis* (*Psd*) on orchard grass (*Dactylis glomerata* L.) (Manners, 1960; Tollenaar, 1967), *P. striiformis* f. sp. *poae* (*Psp*) on Kentucky blue grass (*Poa pratensis* L.) (Britton and Cummins, 1956; Tollenaar, 1967), *P. striiformis* f. sp. *leymi* on *Leymus secalinus* (Georgi) Tzvel. (Niu *et al*., 1991) and *P. striiformis* f. sp. *pseudo-hordei* (*Psp-h*) on *Hordeum* spp. in Australia (Wellings, 2007).

Liu and Hambleton (2010) presented molecular [internal transcribed spacer (ITS) and β-tubulin sequences] and morphological data from a set of 31 isolates to redefine several issues in the biology and taxonomy of *Ps*. Puccinia series *striiformis* was proposed to include *P. striiformis sensu stricto* (host range *Aegilops*, *Elymus*, *Hordeum*, *Triticum*), *P. striiformoides* (formerly *Psd*) infecting *D. glomerata*, *P. pseudostriiformis* (formerly *Psp*) infecting *Poa* spp. and *P. gansensis* in a single isolate from *Achnatherum inebrians* in China. The descriptions of the last three species agree well with the biology of these collections, including very close host affinity and distinct temperature optima for infection. However, the major formae speciales within *Ps sensu stricto* (i.e. *Pst*, *Psh*, *Psp-h*) will remain as important taxa that assist in understanding the host biology and comparative potential threat of these forms in commercial agriculture.

Ps was long regarded as an autoecious microcyclic rust pathogen (urediniospores and teliospores forming on grass hosts) until Jin *et al*. (2010) identified *Berberis* spp. (*B. chinensis*, *B. holstii*, *B. koreana*, *B. vulgaris*) as alternative hosts supporting pycniospores and aeciospores of *Pst* and *P. pseudostriiformis*. Wang and Chen (2013) demonstrated that Oregon grape (*Mahonia aquifolium*) is also a host for the sexual phase of *Pst*.

Ps and *P. pseudostriiformis* are now classified as heteroecious macrocyclic rust pathogens (Fig. 1). Urediniospores are primarily dikaryotic (n + n) and maintain the dominant asexual stage of the

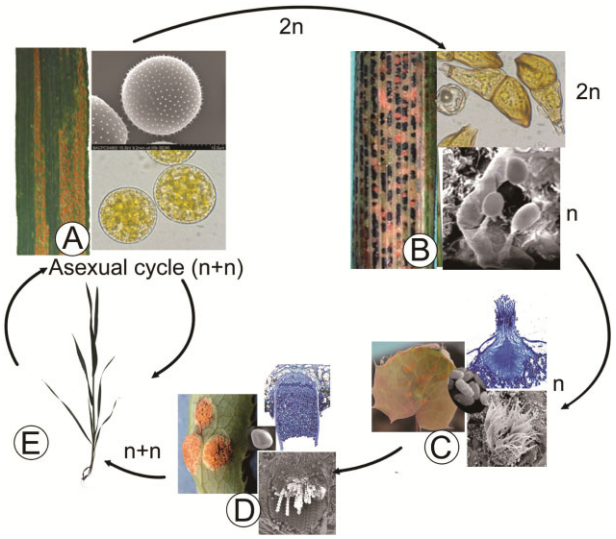

Fig. 1 Life cycle of *Puccinia striiformis*. (A) Uredinia on wheat leaf containing single-celled dikaryotic urediniospores (n + n) originating from aeciospores (n + n) or urediniospores. Top inset: echinulate surface of a urediniospore under a scanning electron microscope (SEM) (×4000). Bottom inset: broadly obovoid urediniospores (×1000). (B) Telia typically form beneath the leaf epidermis near the end of the growing season. Top inset: the two-celled, oblong-clavate teliospores (2n) (×1000). Bottom inset: the elliptoid basidiospores (n) from the germination of teliospores (×2500). (C) Pycnia produced by basidiospore infection on *Berberis chinensis* on upper leaf surfaces via inoculation with germinating teliospores of *P. striiformis*. Top inset: a magnified flask-shaped pycnia (×400). Middle inset: the oblong-shaped pycniospores (×4000). Bottom inset: magnified receptive hyphae (×900). (D) Cluster of sunflower-shaped aecia produced on the lower leaf surface of *Berberis shensiana*. Top inset: a campanulate aecium (×200). Middle inset: flat spherical-shaped aeciospores (×3300). Bottom inset: cluster of aeciospores (×250). (E) A wheat seedling that can be infected by aeciospores produced on barberry plants and can produce urediniospores.

pathogen population on the primary hosts. This phase is responsible for wide-scale stripe rust epidemics reported on cereal crops.

As temperatures rise late in the epidemic phase, *Pst* typically produces thick-walled, predominantly two-celled teliospores. Each cell of a mature teliospore contains a diploid (2n) nucleus formed by karyogamy. *Pst* isolates vary in their ability to produce telia even under similar environmental conditions (Chen XM *et al*., 2012). Unlike those of *P. graminis* f. sp. *tritici*, teliospores of *Pst* do not have dormancy and readily germinate. Wang and Chen (unpublished Washington State University) have determined that, under free water conditions at 12 °C, teliospores germinate (24 h) to produce a promycelium of four cells. Meiosis then produces a single haploid nucleus that forms a basidiospore (48 h) ready to be ejected from the sterigma (60 h). Basidiospores germinate and infect *Berberis* spp. or *Mahonia* spp. (72 h). The authors also determined that a minimum 40-h dew period was required for the infection of barberry leaves from basidiospores, with the highest infection achieved by keeping inoculated barberry plants in a dew chamber at 10 °C for 93 h. The absence of dormancy in teliospores and the consequent short period of available basidiospores led Rapilly (1979) to conclude that a sexual host, should it ever be discovered, would probably have a small role in pathogen survival. In view of this comment and the prolonged dew periods required for infection, it is perhaps unsurprising that there has been limited confirmation of *Pst* on *Berberis* spp. in nature, with just two reports from China (Zhao *et al*., 2011, 2013). It has been concluded that *Berberis* spp. do not play a role in stripe rust epidemics under the natural conditions in the US Pacific Northwest (Chen XM *et al*., 2012).

UREDINIAL INFECTION PROCESS

Almost all biochemical and molecular studies have been based on urediniospores and their derived infection structures (Hahn, 2000; Hahn *et al*., 1997; Ling *et al*., 2007; Mendgen *et al*., 2000; Struck *et al*., 2004; Voegele, 2006; Voegele and Mendgen, 2003; Yin *et al*., 2009; Zhang *et al*., 2008). As an obligate biotrophic plant parasite, *Pst* infects the main hosts (cereal crops, grasses) from urediniospore deposition by wind or raindrops onto the leaf surface. Germination initiates within 3 h of contact with free moisture at a range of temperatures. The cytoplasm of a urediniospore moves into the growing germ tube as it orientates perpendicular to the long axis of epidermal cells during its growth across the leaf surface (Kang, 1996; Kang *et al*., 1997, 2002) until it reaches a stoma (Fig. 2C,D) (Moldenhauer *et al*., 2006; Wang *et al*., 2009). Within 6–8 h post-inoculation (hpi), an appressorium is formed on the stoma, and at 8–12 hpi, a substomatal vesicle is formed within the stomatal cavity, the penetration hyphae are delimited by a septum (Kapooria and Mendgen, 1985) and the primary infection hypha emerges at 12–18 hpi. On contacting the mesophyll cell, a haustorial mother cell containing two to six nuclei develops (Kang *et al*., 1994, 2002) and most of the cytoplasm then moves into the haustorial mother cell, leaving earlier structures to become more or less vacuolated. Haustorial mother cells have a thick, multilayered wall that attaches firmly to the host cell wall. A slender neck forms from the haustorial mother cell, which then invaginates the host cell plasma membrane (Heath and Skalamera, 1997) to form a balloon-shaped feeding structure, known as the haustorium (Fig. 2E; Hovmøller *et al*., 2011; Kang *et al*., 1997, 2003). Haustoria draw nutrients from host cells (Hahn and Mendgen, 2001; Mendgen, 1981; Staples, 2001; Voegele and Mendgen, 2003) and have also been shown to be involved in vitamin synthesis (Sohn *et al*., 2000). Haustoria are typically located in host mesophyll cells and up to 15% of epidermal cells (Hovmøller *et al*., 2011). Unlike stem rust and leaf rust fungi, multinucleate conditions in hyphae, haustorial mother cells and haustoria are very common (Kang *et al*., 1994). From 48 to 120 hpi, the primary infection hypha will give rise to many branched hyphae that develop between host mesophyll cells and produce multiple haustorial mother cells and haustoria, resulting

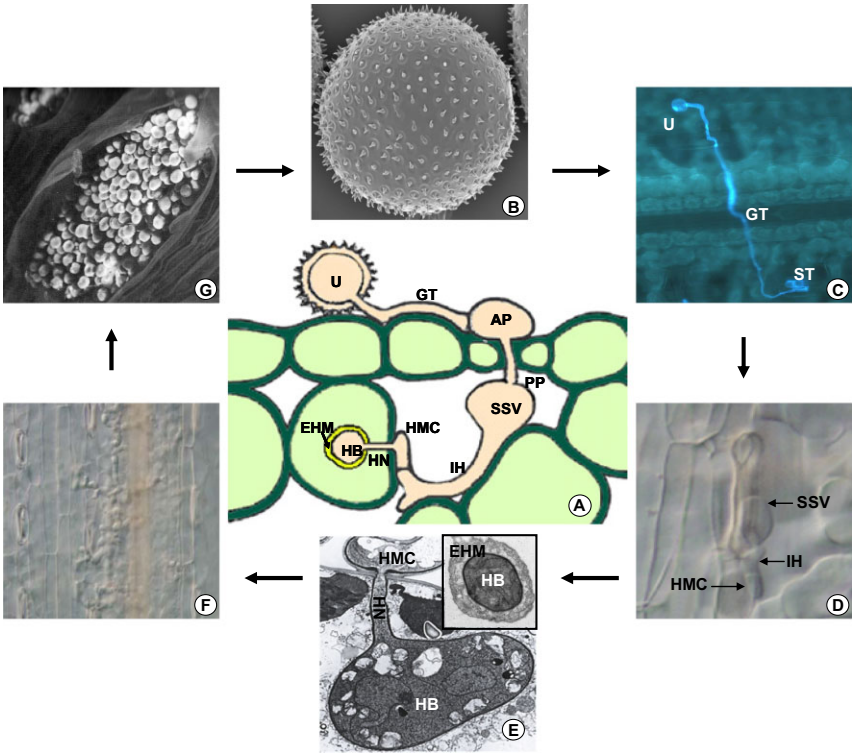

Fig. 2 Uredinial infection process. (A) Schematic representation of early infection structures by *Puccinia striiformis* (*Ps*). (B) Urediniospore under a scanning electron microscope (SEM) (×4000). (C) A urediniospore germ tube (GT) enters the leaf through a stoma (ST). (D) A substomatal vesicle (SSV) is formed, from which an infection hypha (IH) emerges. On contacting with a mesophyll cell, a haustorial mother cell (HMC) is differentiated. (E) A haustorium develops from the haustorial mother cell with a slender neck (Kang, 1996). (F) *Ps* hyphae spread and form colonies in infected leaf tissue. (G) A uredinium under a SEM (Kang *et al*., 1997). AP, appressorium; EHM, extrahaustorial matrix; GT, germ tube; HB, haustorial body; HMC, haustorial mother cell; HN, haustorial neck; IH, infection hypha; PP, penetration peg; SSV, substomatal vesicle; U, urediniospore.

in a branching network of fungal mycelium developing inter- and intracellularly within the host tissue. As the mycelium develops, a pustule bed becomes established from which a uredinium develops. Symptoms of chlorosis can be observed from 6 to 8 days after infection, whereas sporulation (uredinia appearing on the surface of leaves, leaf sheaths, glumes or awns) commences approximately from 12 to 14 days under favourable conditions. Symptoms of wheat stripe rust in the field and a range of seedling infection types in glasshouse tests are shown in Figs 3 and 4, respectively.

Pst is noted for enhanced sensitivity to environmental conditions in comparison with other cereal rust pathogens. The most important factors are temperature, moisture, light and air pollution. Temperature is critical for successful germination and infection. In the presence of adequate moisture, *in vitro* germination studies showed a temperature range of 2–15 °C with an optimum at 7 °C (Sharp, 1965). Similar conclusions were drawn by Rapilly (1979) and Zhang *et al*. (2008). Newton and Johnson (1936) determined that the optimal temperature range for the latent period (time from infection to the beginning of sporulation) was 13–16 °C. These temperatures are, on average, about 10–15 °C lower in each category than those for *P. triticina* and *P. graminis*.

Moisture affects spore deposition, germination, infection and survival. Rapilly and Foucault (1976) observed that the adhesive force between urediniospore and receptive surfaces was significantly greater with high relative humidity, thus increasing the efficiency of spore attachment. Urediniospores require at least 3 h of available moisture on plant surfaces to germinate and infect (Hermansen and Veterinary, 1968; Tu and Hendrix, 1967). Any period of desiccation will irreversibly abort urediniospore germination (Vallavieille-Pope *et al*., 1995). Zadoks (1961) and Rapilly and Fournet (1968) concluded that relative humidity must exceed 50% for sporulation to occur, and that urediniospore production increased exponentially with rising relative humidity.

Light intensity influences the host–pathogen interaction (Stubbs, 1967). Low light intensities in the first 4 days post-infection caused infection type to increase and effectively mask a resistant response (Wellings *et al*., 1988). Seedling tests should always be conducted at light intensities above 10 000 lx in order to ensure consistent infection types (Stubbs, 1985). *Ps* has been shown to be more sensitive to air pollution, relative to other cereal rust pathogens, during urediniospore germination (Sharp, 1967). Later work showed that isolates from various regions varied in sensitivity to air pollutants (Melching *et al*., 1974; Stubbs, 1985).

POPULATION BIOLOGY OF *P. STRIIFORMIS*

Like the other cereal rust fungi, *Pst* has nearly a century of research history investigating detailed aspects of host–pathogen specialization that was fundamental to devising strategies to control the disease in commercial cropping.

Pathogen survey

Pathogenicity surveys for *Pst* have been historically based on the designation of avirulence or virulence responses of isolates inocu-

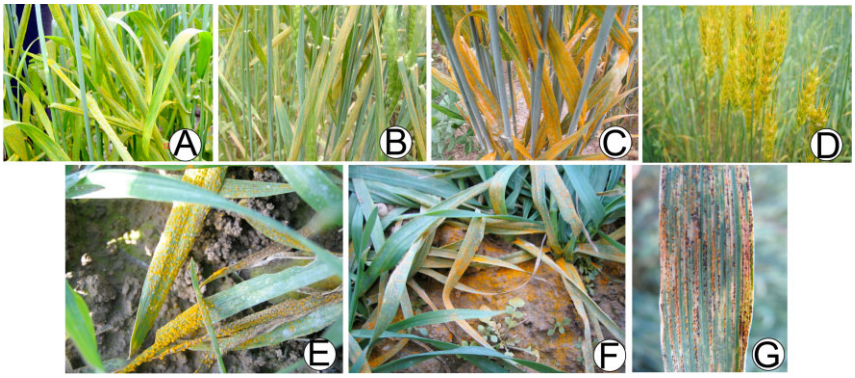

Fig. 3 Wheat stripe rust symptoms in the field. (A–C) Yellow to orange uredinial pustules on susceptible adult plant leaves. (D) Uredinial pustules on the glumes and awns. (E, F) Yellow to orange uredinial pustules on the seedling wheat leaves. (G) Telial and uredinial pustules together on an adult plant leaf.

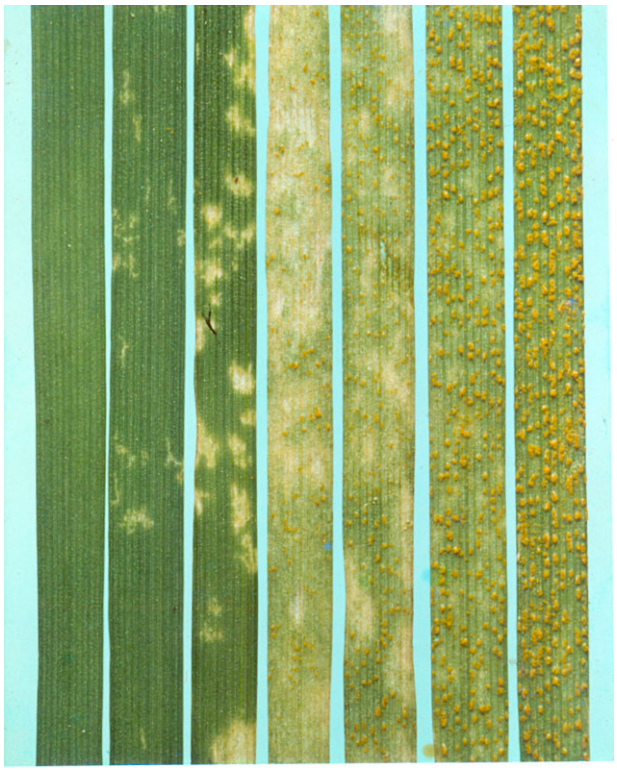

Fig. 4 Range of stripe rust seedling infection types. From the left, leaves show infection types ranging from immune to completely susceptible reaction (McIntosh *et al*., 1995).

lated on host cultivars or genotypes. The assignment of avirulence/virulence in seedling tests has been based on two infection type scales. The 0–9 scale (McNeal *et al*., 1971) is used by scientists in Europe, North America, Syria and Lebanon (Chen and Line, 1993; Hovmøller and Justesen, 2007a; Line *et al*., 1970), whereas the 0–4 scale (Gassner and Straib, 1932a, b) is applied in Australia (Wellings and McIntosh, 1990), China (Chen *et al*., 2009), India (Prashar *et al*., 2007), South Africa (Boshoff *et al*., 2002) and Pakistan (Ali *et al*., 2010a). Designations of avirulence/virulence with respect to specific host resistance genes must be based on the observed deviation from the expected low infection type of that gene, and not on nominal values set within the scale (McIntosh *et al*., 1995).

The differentiation of isolates into distinct races (or pathotypes) requires a set of host materials and prescribed testing conditions, and this was originally proposed for *Ps* by Gassner and Straib (1930, 1932a), and later revised by Fuchs (1965). In this approach, isolates with distinct and unique combinations of virulence and avirulence were described as races and given sequential numbers in order of first detection. Similar approaches continue to be used in North America and China. In North America, several sets of differentials have been developed from the original set of 13 proposed by Line *et al*. (1970) to more than 20 over time (Chen *et al*., 2002; Line and Qayoum, 1991). Races were given sequential CDL (later changed to PST) numbers, and this has been revised to PSTv designations with a new set of 18 wheat *Yr* single-gene lines since 2010 (Wan and Chen, 2012).

In China, pathogenicity surveys of *Ps* have been conducted since the 1940s (Fang, 1944) and in some cases based on the earlier German differentials (Lu *et al*., 1956). In the intervening period, differential sets have changed or been modified, together with changes in commercial wheat cultivars, but the race nomenclature system has remained relatively consistent since the 1960s. The current differential set for the identification of races of *Pst* consists of 20 wheat lines (Chen *et al*., 2009). The major virulence patterns have been formally designated 'CYR' races (Chinese Yellow Rust) with sequential numbers based on their chronological identification. Virulence patterns with low frequencies and limited distribution were temporarily nominated as 'pathotypes' using the abbreviations of specific wheat differential genotypes. There have been 33 CYR races and 35 pathotypes of *Pst* described in China using the current set of wheat differentials (Chen *et al*., 2009; Liu *et al*., 2010; Wan *et al*., 2004).

A substantial revision of *Ps* pathotype nomenclature was proposed by Johnson *et al*. (1972) working in Europe, who applied this system to the international studies of *Ps* conducted by Stubbs and colleagues in the Netherlands. In this approach, pathotypes are designated according to decanary notation using an international and European set of differentials. Supplementary sets with regional significance and international relevance were then devised and/or modified by researchers in India (Prashar *et al*., 2007), southern Africa (Boshoff *et al*., 2002) and Australia (Wellings and McIntosh, 1990).

Dynamics of pathogenic variation

Mutation, somatic recombination, parasexuality, selection and sexual recombination are considered to be mechanisms determining the genetic variability of *Pst* (Duan *et al*., 2010a; Hovmøller *et al*., 2011; Mboup *et al*., 2009; Stubbs, 1985). Single-step mutation of *Pst* from avirulence to virulence, and the reverse, were concluded to be the origin of new avirulence/virulence combinations (Chen *et al*., 2009; Hovmøller and Justesen, 2007b; Wellings, 2007; Wellings and McIntosh, 1990). Gassner and Straib (1932b) were the first to propose the role of mutation in the formation of new races of *Pst*, and estimated a mutation frequency of 0.8×10^{-6} to 1.6×10^{-6}. Similarly, the mutation frequencies in a northwest European population of *Pst* were estimated to range from 1.4×10^{-6} to 4.1×10^{-6} per locus per generation in individual clonal lineages, as determined by amplified fragment length polymorphism (AFLP) analyses (Hovmøller and Justesen, 2007b). Selection of new mutants within *Ps* populations is governed by matching resistance genes in commercial cereal cropping. Despite a relatively large number of mutant derivates detected in eastern Australia over a 25-year period, the *Pst* population was dominated by relatively few pathotypes in particular time periods because of their specific adaptation to widely grown wheat varieties (Wellings, 2007). In addition to mutation and selection, a factor often underestimated in diversity studies in *Ps* is the chance event of pathotype survival in the non-host period. The re-emergence of *Pst* in any wheat growing season is a function of local survival events or long-distance dispersal from regions supporting pathogen populations. The pathotypes surviving in these situations often reflect the dominating pathotypes of the previous season, but the actual pathotypes that survive the non-host period are essentially random and unpredictable.

Although a majority of regional *Pst* populations show evidence of clonality with diversity limited to closely related mutational derivatives, high diversity for both virulence and molecular markers was discovered in *Pst* populations in Gansu, China (Duan, *et al*., 2010a; Mboup *et al*., 2009; Zheng *et al*., 2005) and Pakistan (Bahri *et al*., 2009), suggesting that processes in addition to single-step mutation were involved. Somatic recombination and parasexuality events between isolates may represent an alternative means to generate new pathogenic diversity (Manners, 1988; Park and Wellings, 2012). Experimental evidence for these events has involved pairing isolates contrasting in urediniospore colour and virulence, combined with cytological observations demonstrating anastomosis events (Kang *et al*., 1993; Little and Manners, 1969). Hyphal fusion and nuclear re-assortment may be plausible explanations for the emergence of recombinant isolates in the absence of sexual reproduction. However, somatic recombination has not been demonstrated in *Ps* populations under natural conditions. The discovery of *Berberis* spp. as the alternative host for *Pst* infecting wheat and *P. pseudostriiformis* infecting bluegrass (Jin *et al*., 2010) opened up new possibilities to explain the existence of diverse pathogen populations in certain regions. Some evidence to support this hypothesis in China was reported among collections of aecia from *Berberis* spp. that yielded several pathotypes of *Pst* (Zhao *et al*., 2013). However, it is anticipated that sexuality will be localized in *Ps* populations because of the limited distribution of alternative hosts, climatic conditions and the short dormancy of basidiospores, which will become rapidly exhausted at the end of the wheat growing season (Wellings, 2011).

The dominance of certain pathotypes that cannot be explained on the basis of avirulence/virulence adaptation has led to studies investigating the basis for aggressiveness in certain populations of *Pst*. Races CYR32 and CYR33 have become the most predominant races in China in recent years, and this was attributed to greater parasitic fitness of both races on leading commercial cultivars (Tian *et al*., 2008). Two new strains of *Pst* with the same virulence loci and different AFLP fragments spread rapidly in North America, Australia and Europe in less than 3 years, and were concluded to be highly aggressive. Both strains produced up to two to three times more urediniospores per day than isolates found in the USA and Europe before 2000, which could provide a basis for increased aggressiveness and accelerate their global spread into areas that were previously considered to be too warm for stripe rust epidemics (Hovmøller *et al*., 2008; Markell and Milus, 2008; Milus *et al*., 2008). Although temperature adaptation could not be confirmed in Australian studies (Loladze *et al*., 2013), spore production has been shown previously to be a feature of adapted *Pst* pathotypes (Johnson and Taylor, 1976).

Population structure and diversity

Population structure and diversity of *Ps* were traditionally based on avirulence/virulence analyses, and these studies allowed an appreciation of the broad international population characteristics (Stubbs, 1988). With the development of biotechnology, molecular marker techniques based on polymerase chain reaction (PCR) were successfully applied to population structure and diversity studies of *Ps* from the 1990s. AFLP and random amplification of polymorphic DNA (RAPD) markers revealed a low correlation between virulence and DNA polymorphism in *Pst* populations in China, the USA and Australia (Chen *et al*., 1993; Shan *et al*., 1995; Wellings, 2007). Comparing AFLP lineages, Justesen *et al*. (2002) demonstrated that certain *Pst* populations in Denmark were derived from the aerial dispersal of urediniospores from France and/or Germany, and that frequent migration events occurred between the UK, Germany, France and Denmark (Hovmøller *et al*., 2002). Hovmøller and Justesen (2007a) observed AFLP diversity among unusual pathotypes in northwestern Europe sampled within a short time period in a small area on very few host cultivars with limited or no selection on the pathogen population,

and found three to four times higher diversity than that among isolates sampled from a large number of cultivars with different *Yr* genes in four different countries over 25 years. This suggests a more frequent and even more exotic incursion of *Pst* urediniospores than previously anticipated. The population of *Pst* in Yunnan, southwest China, was considered to be a clonal population compared with that in Gansu, northwest China based on AFLP patterns (Liu *et al*., 2011). These results suggest geographical isolation and local adaptation in the genetic evolution of the *Pst* population in this region.

Simple sequence repeat (SSR) markers with advantages of co-dominance and reliability have been developed and applied to *Pst*. Twelve SSRs distinguished Mediterranean pathotypes specific to southern France from all the other European pathotypes, and separated Chinese isolates into two other classes (Enjalbert *et al*., 2002). The northern French population was more closely related to the northwestern European population, and the southern clonal population was most probably related to the Mediterranean population. It was concluded that the two subpopulations were the result of an ancient divergence of two clonal lineages (Enjalbert *et al*., 2005). Multilocus microsatellite genotyping of worldwide *Pst* samples defined six genetic groups corresponding to their geographical origin. Clonal population structure was observed in northwest European, Middle Eastern, Mediterranean, East and South African, North and South American, and Asian populations. High genotypic diversity and a recombinant structure in Asian populations near the Himalayan Mountain Chain suggested that the Himalayas may represent a centre of diversity for *Ps* (Vallavielle-Pope *et al*., 2012).

Long-distance dispersal

Pst is capable of long-distance dispersal by wind movement and human-assisted transport (Brown and Hovmøller, 2002). Urediniospores of *Pst* can be efficiently air dispersed over hundreds and perhaps thousands of kilometres despite their vulnerability to environmental factors, such as ultraviolet light (Zadoks, 1961).

In North America, *Pst* gradually covered a distance of about 2400 km from northern Mexico and southern Texas to North Dakota within 6 months (Chen *et al*., 2010; Line and Qayoum, 1992).

In Europe, urediniospores can be transported 800 km and even more than 1200 km from north France to Algeria by wind (Stubbs, 1985). The wheat stripe rust pathogen entered New Zealand in 1982, probably as the result of 2000 km of airborne transport from Australia (Wellings and McIntosh, 1990).

In China, evidence suggests that urediniospores are annually dispersed hundreds or even thousands of kilometres from western over-summering areas to the main wheat belt in the east and north (Fig. 5; Chen and Xie, 1999; Chen *et al*., 2007; Xie *et al*., 1992, 1993; Zeng and Luo, 2006). Upland areas 1500–1800 m and 1900–2500 m above sea level in northwestern and southwestern China, respectively, provide environments for year-round pathogen survival and development, and hence these regions are major reservoirs of inoculum and pathogenic diversity (Brown and Hovmøller, 2002; Chen *et al*., 2007, 2009). The pathogen must re-establish each autumn in northern and eastern China, the main winter wheat-growing areas, because the pathogen cannot survive the hot dry summers. Ecological management of wheat stripe rust in the areas of inoculum sources has been considered the major strategy for the sustainable control of wheat stripe rust nationwide, resulting in effective containment of wheat yield losses that potentially amount to more than two million tonnes annually (Chen WQ *et al*., 2012, 2013).

In the US Pacific Northwest, where both winter and spring wheat are grown, *Pst* urediniospores spread readily from winter wheat to spring wheat in the summer and from spring wheat to winter wheat in autumn, in addition to survival on volunteer wheat plants and wild grass species (Chen, 2005; Hendrix *et al*., 1965; Line, 2002).

In Australia, *Pst* was first detected in 1979 and the pathogenic features of the initial isolates predicted that it was of European origin. Wellings *et al*. (1987) provided evidence to support the survival of urediniospores on contaminated travellers' clothing, and predicted that this was the most likely means of entry of *Pst* into Australia. The first detection of *Pst* in remote wheat-growing regions previously free of the disease in South Africa in 1996 and in Western Australia in 2002 has similarly implicated long-distance transport assisted by adherent urediniospores on travellers' clothing (Wellings, 2007).

MOLECULAR PERSPECTIVES

Host–pathogen interactions

Ling *et al*. (2007) published a full-length cDNA library, consisting of 42 240 clones with an average cDNA insert of 1.9 kb, constructed from urediniospores of US race PST-78. Initial characterization and gene analysis were conducted by sequencing about 200 randomly selected clones representing various lengths of open reading frames. The first 51 genes with putative functions involving 11 aspects of pathogen cell biology and pathogenicity were identified.

Cantu *et al*. (2011) accessed the genomic sequence of higher virulent US race PST-130 using next generation sequencing (NGS), and obtained nearly 80 million high-quality paired-end reads that were assembled into 29 178 contigs (64.8 Mb); 22 825 putative coding sequences were identified and tentatively annotated. Cantu *et al*. (2013) re-sequenced the genome of four *Pst* isolates from the US and UK to identify effector candidates and to relate them to their distinct virulence profiles. RNAseq analysis highlighted transcripts encoding secreted proteins that were significantly enriched in haustoria compared with infected tissue. The

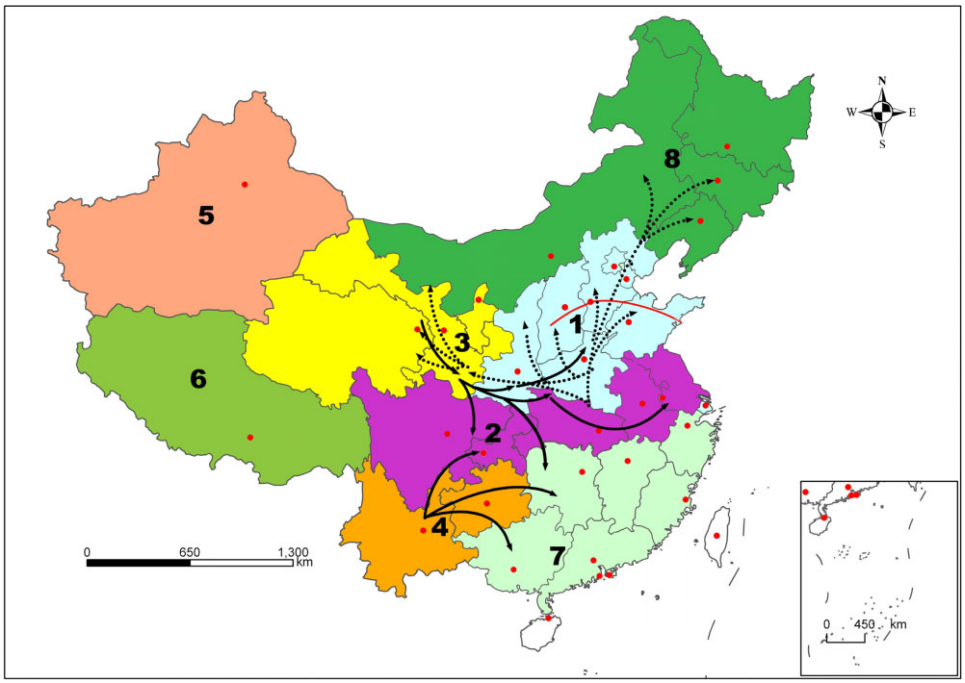

Fig. 5 Ecological zones of wheat stripe rust and aerial dispersal of urediniospores in China. 1, The Guanzhong and Huabei winter wheat region is the main area of winter wheat cultivation and the over-wintering area of *Puccinia striiformis* f. sp. *tritici* (*Pst*) in China. 2, The Chengdu Plain and Jianghan River Basin facultative wheat region is the winter-increasing area and the major spring inoculum source of *Pst*. 3, The Northwest winter and spring wheat region is the most important over-summering area of *Pst* and the major source of inoculum for wheat infection in autumn. 4, The Yunnan and Guizhou wheat region is one of the over-summering areas of *Pst*. 5, The Xinjiang winter and spring wheat region is a relatively independent epidemic zone of *Pst*. 6, The Tibet highland barley and wheat region is a separate epidemic zone of *Pst*. 7, The South late-sowing wheat region does not grow much wheat and stripe rust seldom occurs. 8, The Inner Mongolia and Northeast spring wheat region where wheat stripe rust epidemics occur occasionally. The full black arrows show dispersal of urediniospores from the over-summering areas to the main wheat-growing areas in autumn annually. The broken black arrows indicate the main pathway of urediniospore dispersal in spring. The red line indicates the boundary of *Pst* over-wintering areas and the red circles show provincial capitals.

expression of 22 candidate effector genes was characterized using quantitative reverse transcription-polymerase chain reaction (qRT-PCR), revealing distinct temporal expression patterns during infection in wheat. Five polymorphic effector candidates specifically between two UK isolates, which differ in virulence to two wheat cultivars, were identified among 2999 secreted proteins. These allelic variants are now priority for functional validation as virulence/avirulence effectors in the corresponding wheat cultivars. The comparative sequence analysis of the *Pst* races may provide some candidate genes for the effectors recognized by the stripe rust resistance genes.

A total of 12 282 transcripts of *Pst* race 104E137A$^-$ were assembled by means of NGS platforms to compare the germinated urediniospores and haustoria transcriptomes based on Illumina RNAseq data (Garnica *et al*., 2013). More than 400 genes encoding secreted proteins which constitute candidate effectors were identified from the haustorial transcriptome, with two-thirds of these up-regulated in the tissue of wheat relative to germinated spores. RT-PCR analysis confirmed the expression patterns of 94 effector candidates. The analysis also revealed that urediniospores rely mainly on stored energy reserves for growth and development, whereas haustoria take up host nutrients for massive energy production for the biosynthetic pathway and the ultimate production of spores.

Zhang *et al*. (2008) isolated 4798 expressed sequence tags (ESTs) derived from a germinated urediniospore library and 267 genes with putative functions were identified from Chinese race CYR32. BLASTX searches revealed 13 ESTs homologous to known fungal pathogenicity or virulence factors, and six were shown to have high levels of expression in germinated urediniospores. Potential virulence factors were detected among 15 unique transcripts from a cDNA library constructed from haustoria isolated from *Pst*-infected wheat leaves (Yin *et al*., 2009). Despite some success in transient gene expression using the *β-glucuronidase* (*GUS*) reporter and the antibiotic resistance gene *hygromycin phosphotransferase* (*hpt*) via particle bombardment (Wang *et al*., 2006), Yin *et al*. (2009) were unable to confirm the specific functions of these genes in the infection process. Hypersensitivity-induced reaction genes (*Ta-hir1*, *Ta-hir2*, *Ta-hir3*, *Ta-hir4*) were characterized in wheat–*Pst* interactions (Yu *et al*., 2008; Zhang *et al*., 2009). Studies have revealed an early oxidative burst of reactive oxygen species in the hypersensitive interaction, and have

implicated the role of Ca^{2+} as an intracellular secondary messenger in pathogen defence processes (Long *et al*., 2010).

Several studies have made insights into the molecular basis of compatible interactions between wheat and *Pst*. Yu *et al*. (2008) used suppression subtractive hybridization to identify the genes induced in the fungus during infection, although only two unigenes showed similarity with pathogenic proteins. Ma *et al*. (2009) constructed an EST library from fully susceptible wheat leaves infected with *Pst* to explore the pathogen genes expressed during infection. Among the 2743 unisequences, 446 showed homologies to fungal genes from model fungal species, and 15 genes were homologous to other rust fungal genes. Using a complementary DNA-AFLP, nine transcript-derived fragments from wheat leaves infected by *Pst* were shown to be of pathogenic origin (Wang *et al*., 2009).

Host defence-related genes isolated from interactions between wheat and *Pst* corresponded to different stages in the infection process. Coram *et al*. (2008b) reported that 54 transcripts were induced in both compatible and incompatible interactions, and were considered as basal defence transcripts, whereas 61 transcripts were specific to the incompatible interaction (hypersensitive resistance-specific transcripts) and 19 were specific to the compatible interaction (biotrophic interaction-specific transcripts). A fascinating discovery was the quenching of the divergent expression of *Pst*-regulated genes in both incompatible and compatible interactions in the middle stages of *Pst* infection (Wang *et al*., 2009).

In addition, a number of candidate genes from wheat challenged by *Pst*, such as a transcription factor gene *TabZIP1* (Zhang *et al*., 2009), a novel wheat NAC gene *TaNAC4* (Xia *et al*., 2010), a wheat HSP70 gene *TaHSC70* (Duan *et al*., 2010b), a wheat β-1,3-glucanase gene *TaGlu* (Liu *et al*., 2010), a pathogenesis-related thaumatin-like protein gene *TaPR5* (Wang *et al*., 2010) and three secreted protein genes *PstSP2C7*, *PstSP11L10* and *PstSP11P10* (Dong *et al*., 2011) have been characterized. Meta-analysis of the 28 transcripts confirmed the activity of known resistance (*R*) gene-mediated pathways in the race-specific resistance response, including an oxidative burst that probably contributes to hypersensitive resistance, as well as pathogenesis-related protein expression and activity of the phenylpropanoid pathway (Coram *et al*., 2010).

Resistance gene cloning

Early successes in resistance gene cloning, such as *Yr10*, focused on seedling effective genes which had generally been overcome by new pathotypes (Spielmeyer and Lagudah, 2003). The cloned gene sequences from this, and similar resistances to leaf rust caused by *P. triticina*, showed common molecular motifs that included nucleotide-binding sites which were rich in leucine repeat elements (NBS-LRR). The classical NBS-LRR gene family is considered to have a role in ubiquitous programmed cell death (apoptosis), putative antifungal activities, disease resistance responses, pathogenesis-related responses and unknown functions providing race-specific resistance to stripe rust. In contrast, the reputed durable sources of resistance *Yr18* and *Yr36* showed molecular motifs with distinctly different functions. The former was shown to encode a protein resembling a multidrug ABC transporter (Krattinger *et al*., 2009), whereas the latter includes a kinase and a putative START lipid-binding domain (Fu *et al*., 2009). Functional studies of *Yr39* by Coram *et al*. (2008a) indicated evidence for broad defence responses, including the induction of several R protein homologues, wider induction of the phenylpropanoid pathway and several other putative defence transcripts. These mechanisms are functionally different from those of the NBS-LRR resistance genes, and hence *Yr39* was predicted and proven to be a durable source of resistance to *Pst* (Lin and Chen, 2007; Coram *et al*., 2008a).

Conclusions

The availability of *Pst* sequence data and the integration of genomics, transcriptomics and effector-directed annotation have led to a more comprehensive understanding of the *Pst* pathogenesis system, an important step towards the development of more effective surveillance and management strategies. The cloning of durable resistance genes that demonstrate molecular motifs distinct from those of failed race-specific genes indicates hope for forging substantial advances in determining the molecular nature of durable resistance, and so to provide possibilities for their effective deployment in commercial wheat cultivars.

GENES FOR STRIPE RUST RESISTANCE IN WHEAT

The genetic characterization of resistance has been an active focus of research since the seminal work of Biffen (1905), who first described the Mendelian nature of resistance to stripe rust in wheat cultivar Rivet. The major types of resistance to *Pst* in wheat are seedling (or all-stage) resistance and adult plant resistance (APR). The former is conveniently detected in seedling tests and remains effective throughout all growth stages provided that the same *Pst* pathotype is used in all assessments. APR, which expresses susceptible infection types in seedling tests, develops varying levels of resistance in post-seedling stages in either field or glasshouse studies. Major reviews of the genetic basis for resistance to *Pst* can be found in Chen (2005, 2013), Röbbelen and Sharp (1978) and Wellings *et al*. (2012).

Seedling resistances are frequently conferred by single genes, or simple combinations of single genes, and have generally become vulnerable to single-gene changes to increased virulence in *Pst* populations. APR has been considered to be more robust in terms of resistance in that it has not been as readily overcome by

changing *Pst* populations, although there have been notable severe epidemics arising from the failure of single-gene APR, for example, the sudden susceptibility of Joss Cambier in the UK in 1969 (Johnson and Taylor, 1972). A major conceptual development in searching for strategies to contain stripe rust epidemics was the description of durable resistance, which was defined as wheat cultivars that remained resistant when cultivated over large areas, for many years and in environments conducive to *Pst* epidemics (Johnson and Law, 1975).

Seedling resistance

Lupton and Macer (1962) studied the seedling-expressed resistance in seven wheat cultivars and first assigned *Yr* symbols to designate stripe rust resistance genes (*Yr* genes) in wheat. New designations of *Yr* genes are reviewed by an international consultation group, led by Professor R. A. McIntosh, who publishes annual updates (McIntosh *et al*., 2007) and complete revisions every 5 years to coincide with the International Wheat Genetics Symposium (McIntosh *et al*., 2003). To date, more than 50 officially designated and many temporarily designated *Yr* genes for resistance to stripe rust have been described in wheat, and some have been widely used in different areas of the world (McIntosh *et al*., 1995, 2003, 2007; Wellings *et al*., 2012; Xu *et al*., 2013). Among a total of 55 catalogued *Yr* genes, 41 confer seedling resistance, and 14 are APR genes. A majority of *Yr* genes originate from *T. aestivum*, but several are derived from related genera or species, including *Secale cereale* (*Yr9*), *Aegilops* spp. (*Yr8*, *Yr17*, *Yr37*, *Yr38*, *Yr40* and *Yr42*), *T. spelta* (*Yr5*), *T. dicoccoides* (*Yr15*, *Yr35* and *Yr36*), *T. turgidum* (*Yr24/Yr26*, *Yr53*), *T. tauschii* (*Yr28*) and *Thinopyrum intermedium* (*Yr50*).

Adult plant resistance

APR becomes effective at various stages of post-seedling development according to genotype (Boyd, 2006). Several resistance genes, including *Yr16*, *Yr18*, *Yr29*, *Yr30*, *Yr39* and *Yr52*, confer APR, and some, for example *Yr18*, are regarded as durable (Imtiaz, 2004; Morgounov *et al*., 2012).

APR that begins early in the growth cycle will be expected to offer greater yield protection than that operating from flag leaf emergence. Other factors governing the expression of APR are crop nutritional status, where high nitrogen leads to more severe disease expression, and temperature. High-temperature APR (HTAP) is described as quantitatively inherited and pathotype nonspecific, and therefore is concluded to be durable resistance (Chen, 2005, 2013). HTAP, which is triggered in the late stages of plant development when average daily temperatures are typically above 21 °C, causes the initial infection types and severity to decrease, preventing the occurrence of secondary infection events. Genes for HTAP resistance have been genetically characterized, including *Yr36* from *T. turgidum* var. *dicoccoides* located on chromosome 6BS (Uauy *et al*., 2005), *Yr39* in spring wheat cultivar 'Alpowa' located on 7BL (Lin and Chen, 2007), and *Yr52* on 7BL in spring wheat line 'PI 183527' (Ren *et al*., 2012).

Quantitative resistance

Quantitative trait loci (QTLs) conferring resistance, which may have individually small effects on reducing disease expression, can contribute collectively in an additive manner to confer high levels of resistance to *Pst* and segregate according to Mendel's laws (Singh *et al*., 2000; Singh and Rajaram, 1994; Tanksley, 1993). A large number of QTLs for resistance to *Pst* and their associated molecular markers have been reviewed by Boyd (2005), Chen (2005, 2013), Singh *et al*. (2004) and Wellings *et al*. (2012). QTLs for resistance to *Pst* have been mapped to all wheat chromosomes except 1D and 3A (Chen, 2005, 2013; Christopher *et al*., 2013; Imtiaz *et al*., 2004; Lu *et al*., 2009; Suenaga *et al*., 2003; Vazquez *et al*., 2012).

Linkage relationships

All designated *Yr* genes, with the exception of pathotype-specific APRs *Yr11* to *Yr14*, have been mapped to chromosomal positions across the *Triticum* genome, although chromosome 7A remains currently free of described *Yr* genes. Gene location provides the basis for linkage relationships that are important to the development of breeding strategies and in determining the potential value or limitation in deploying certain *Yr* genes. *Yr9* on chromosome 1B is linked to leaf and stem rust resistance genes (*Lr26* and *Sr31*, respectively) and to a quality defect in flour used for bread making (McIntosh *et al*., 1995). *Yr18*, which locates to chromosome 7DS, is the same gene as *Lr34* for resistance to leaf rust and resistance to stem rust and powdery mildew (Krattinger *et al*., 2009; Spielmeyer *et al*., 2005; Suenaga *et al*., 2003). The slow rusting APR gene *Yr29* on 1BL is completely linked to gene *Lr46*, which confers moderate resistance to leaf rust (William *et al*., 2003). *Yr30* on 3BS, involved in APR among several cultivars developed by the International Maize and Wheat Improvement Center, is associated with the durable stem rust resistance gene *Sr2* (Singh *et al*., 2000).

CONCLUSIONS

The wide distribution and frequent occurrence of *Pst* epidemics in world wheat production zones will continue to yield focused effort in the development and deployment of economic control strategies. The increasing use of fungicide management for *Pst* control over the past decade has been driven by the availability of off-patent generic products that have reduced chemical costs and thereby increased commercial economic benefit, even in production systems that have relatively low yield expectations. In addition, the emergence of new *Pst* pathotype lineages across large production regions, either through exotic incursion events or through local evolution to increased virulence, has evidently

thwarted the capacity for breeding programmes to quickly respond with varieties that meet grower's demands for resistance, high yield and market quality. Although resistant varieties remain the goal, commercial industry imperatives will mean that alternative approaches, such as integrating fungicides and cultivation measures (including the regulation of plant date, crop nutrition, interplanting of crops and eradication of volunteer seedlings), may assume greater importance in the short to medium term. Specific management strategies in areas which function as pathogen inoculum sources and center of pathogenic diversity are considered to be the highest priority for sustainable stripe rust control.

In order to advance the development and distribution of resistant wheat cultivars to provide cost-effective and environmentally acceptable disease control, research on *Pst* will continue to build on the large body of information already assembled for this important cereal pathogen. Molecular studies have assisted the identification of certain resistance genes that are likely to provide durable resistance. Efficient marker systems are available for these genes and should enable breeders to incorporate and select several durable genes in populations to build genetic foundations that stabilize and secure their germplasm against the uncertainties of *Pst* dynamics. Although gains in durable resistance are being made, it is essential that genetic diversity is encouraged through the identification and characterization of further durable sources for use in practical plant breeding. Seedling resistances will continue to be described and catalogued, but the application of these genes will centre around debates of stewardship that arise from concerns about the potential evolution and selection of new races and the means to minimize these effects using strategies ranging from the development of gene combinations to regional resistance gene deployment. The annotation and functional analysis of genes involved in the biology and pathogenicity of *Pst* by means of bioinformatics will provide new opportunities to understand the virulence variation in the pathogen and the mechanism of resistance in the host.

In parallel to host resistance, population studies in *Pst* will continue to focus on mechanisms underpinning pathogenic variability within and between hosts. An important area will be to evaluate the significance of apparent restricted sexual populations of *Pst* in certain regions and the role that the alternative *Berberis* hosts may play in generating variability and providing the opportunity for pathogen survival between cereal cropping seasons. Features of pathogen epidemiology, including mechanisms of survival between seasons, and interactions of disease onset and development in relation to variety and environment, remain relevant issues to disease control.

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