

Puccinia striiformis in Australia: a review of the incursion, evolution, and adaptation of stripe rust in the period 1979–2006

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Abstract. The wheat stripe rust pathogen (*Puccinia striiformis* f. sp. *tritici*; *Pst*) was first detected in Australia in 1979. The features of the initial pathotype suggested that it was of European origin, and later work provided evidence that it was most likely transmitted as adherent spores on travellers' clothing. Despite long-held views that this cool temperature pathogen would not adapt to Australian conditions, *Pst* became endemic and progressively adapted to commercial wheat production through step-wise mutation. Several of these mutant pathotypes became frequent in the *Pst* population, causing widespread infection and significant costs to production (yield and quality losses; chemical control expenditure) in certain cultivars and seasons. Pathotype evolution, including adaptation to native barley grass (*Hordeum* spp.) populations, is described.

The occurrence of an exotic pathotype of *Pst* in Western Australia in 2002, and its subsequent spread to eastern Australia, represented a major shift in the pathogen population. This pathotype dominated pathogen populations throughout Australia from 2003, with chemical control expenditure estimated at AU\$40–90 million annually.

Another exotic introduction was detected in 1998. Initial data indicated that certain isolates collected from barley grass were highly avirulent to wheat differentials, with the exception of partial virulence to Chinese 166. Further seedling tests revealed that these isolates, tentatively designated barley grass stripe rust (BGR), were virulent on several Australian barleys, notably those of Skiff parentage. Data, including molecular studies, suggest that BGR is a new *forma specialis* of *P. striiformis*. Field nurseries indicate that BGR is likely to have little impact on commercial barley, although this may change with further pathotype evolution or the release of susceptible cultivars.

Additional keywords: host specialisation, pathotype, epidemiology, pathogen population, mutation, selection.

Introduction

Puccinia striiformis Westend. f. sp. *tritici* (*Pst*) was first detected in eastern Australia in October 1979 (O'Brien *et al.* 1980). Independent collections by J. D. Oates (PBI Castle Hill) and J. Fisher (NSW Agriculture, Wagga Wagga) from southern New South Wales confirmed the first report of the pathogen. Field surveys in the initial period confirmed the epicentre to be near Charlton in the Wimmera region of Victoria (O'Brien *et al.* 1980), and the disease to occur across a 600-km radius extending to Tamworth in northern NSW. Despite earlier predictions that the pathogen would not survive the critical summer phase of its life cycle due to the warm temperatures of Australia (Waterhouse 1936), *Pst* subsequently became adapted and endemic, and has not failed to be recovered in each season since 1979 despite several severe drought seasons.

An early decision was taken by the National Wheat Rust Control Program (NCRCP) at The University of Sydney, Plant Breeding Institute (PBI) Castle Hill, to adopt the common name 'stripe rust'. It was argued that this maintained consistency with the common names for leaf rust (caused by *P. triticina* Eriks.; *Pt*) and stem rust (caused by *P. graminis* Pers. f. sp. *tritici*; *Pgt*) adopted in the US and elsewhere. The alternative system of common names in use throughout Europe, Middle East, and

Central Asia (i.e. yellow, brown, and black rusts) was considered inappropriate. Despite an attempt to adopt 'yellow rust' at a national workshop in 1984, 'stripe rust' has prevailed as the common name in Australia.

The initial collections in 1979 by Oates were dispatched for pathotype (pt) tests to the PBI Rust Laboratory at Castle Hill, whereas the Fisher collection was identified and confirmed by Mr J. Walker (NSW Agriculture) and deposited in Herbarium DAR as a record to confirm the first report (specimen DAR 34145—Voucher specimen deposited in the Plant Pathology Herbarium, NSW Department Primary Industries, Orange Agricultural Institute, Orange, NSW). The collaboration between PBI and NSW Agriculture continued in the early phases of the investigation, with isolated and specialist greenhouse facilities at BCRI Rydalmere made available by Dr K. J. Moore (Director of Biology, NSW Agriculture) to enable Dr R. A. McIntosh (Director of Rust Research, PBI) to undertake pathotype studies. Despite difficulties in establishing viable cultures, McIntosh (pers. comm.) was able to demonstrate that the stripe rust incursion was the result of a single pathotype introduction and he designated pt 104 E137 on the basis of responses on the differential set of Johnson *et al.* (1972).

The occurrence of stripe rust in Australia gave cause for great concern, and a meeting of the NCRCP, established just 4 years earlier in 1975, resulted in an emergency application for funds to construct a specialist greenhouse at PBI Castle Hill. Research was also initiated at the Victorian Department of Agriculture (J. S. Brown), The University of New England (J. F. Brown), the Queensland Wheat Research Institute (R. G. Rees), and later at NSW Agriculture (G. M. Murray) and Latrobe University (P. Keane). These groups focused on epidemiology, crop loss assessment, host range, modelling for decision support in fungicide application, and host-pathogen genetics which provided an early groundwork that defined subsequent approaches to disease control.

This review will seek to document the progressive developments in stripe rust research in Australia, with particular emphasis on the adaptation and evolution of the pathogen. The biology of the disease in Australia will be evaluated from the context of international reports of pathogen behaviour.

Biological forms of the pathogen

Two broad areas of pathogenic variation have been observed in the capacity of pathogens to cause disease. These can be conveniently regarded as specialisation between hosts, and specialisation within hosts. The former refers to variation observed between various host genera in their capacities to support the growth and survival of the pathogen. The International Code of Botanical Nomenclature recognises the taxonomic unit of *forma specialis* (literally 'special form') to describe this variation, which was first reported for *P. striiformis* in Europe by Eriksson and Henning in 1894 (Wellings 1986). Although this concept has been widely adopted, there are exceptions in certain laboratories and in certain eras that must be appreciated in order to interpret published data. For example, the classical work of Gassner and Straib with *P. striiformis* in the 1930s clearly did not recognise the *forma specialis* concept and preferred to describe isolates from various hosts as 'races'. Stubbs (1985) reproduced a table of races described by the earlier German research and later expanded by Dr Eva Fuchs, and here we find Races 24 and 48 specialised to barley as determined by high infection types on the barley tester Heils Franken. These races were evidently the barley form of the stripe rust pathogen. Similarly, early Canadian workers failed to recognise host specialisation and preferred to describe a sequence of races (Line 2002).

In addition to differences in host range, further levels of specialisation within defined collections of certain hosts were observed in the US for the wheat stem rust pathogen (*Pgt*) by E. C. Stakman and colleagues in 1917, and later in *P. striiformis* by Allison and Isenbeck (1930) in Europe. This second level of pathogenic specialisation within hosts has been variously termed biologic form, physiologic race, strain, or pathotype.

Formae speciales in *P. striiformis*

Since the initial work of Eriksson and Henning in 1894 and subsequent contributions in the early 20th Century, a corpus of research has accumulated in an attempt to describe the boundaries of host range variability within *P. striiformis*. The

following represents an attempt to draw conclusions from this work:

P. striiformis f. sp. *tritici* (Eriksson 1894; *Pst*), the pathogen of wheat stripe (yellow) rust, has a host range which is predominantly wheat (*Triticum* spp.), but also includes certain barley, rye, and triticale genotypes. Other hosts include the weedy grasses of the *Pooideae* sub-family within Family *Gramineae*, encompassing species within the *Hordeum*, *Agropyron*, *Phalaris*, *Hystrix*, and *Bromus* genera (Hungerford and Owens 1923; Zadoks 1961; Holmes and Dennis 1985; Wellings 1986).

P. striiformis f. sp. *hordei* (Eriksson 1894; *Psh*), the pathogen of barley stripe rust, principally infects barley (*Hordeum* spp.), although it has also been reported to cause disease on certain wheats (Stubbs 1985). The host range has also been noted to include weedy grasses, in particular *Hordeum murinum* (Zadoks 1961) and *H. jubatum* and *H. leporinum* (Marshall and Sutton 1995). This disease caused major epidemics and serious crop losses when it was first detected in Columbia in 1975, having presumably originated in Europe (Dubin and Stubbs 1986). The disease progressively moved south through the region of the Southern Cone in the period 1975–1982. Later, barley stripe rust moved north to Mexico in 1990 and subsequently to Texas in 1991 (Marshall and Sutton 1995), progressing northward and developing a large array of pathotypes in a short period (Line 2002). Barley stripe rust has not been recorded in Australia and is currently regarded as an exotic plant pest of potential threat to the barley industry.

P. striiformis f. sp. *dactylidis* (Manners 1960; *Psd*), stripe rust infecting cocksfoot (*Dactylis glomerata*), was originally described as morphologically distinctive in urediospore size and therefore ascribed the status of variety within *P. striiformis* (Manners 1960). However, the consensus of opinion leads to the conclusion that this pathogen is best regarded as a distinct *forma specialis* (Tollenaar and Houston 1967; Latch 1976). The host: pathogen association is very close, so that pathogen isolates collected from cocksfoot cannot infect cultivated cereals and grasses, or *vice versa*. The optimum temperature for urediospore germination for *Psd* was noted to be 21–24°C (Manners 1960), in contrast to 6°C for *Pst* (Tollenaar and Houston 1967). Although *Psd* was described in New Zealand in 1975 (Latch 1976), the first report of *Psd* in Australia was not until 1979 and perhaps it was not by chance that it was contemporaneous with the first detection of *Pst*. It is therefore possible that this disease was present but undetected in Australia for some time. It remains a sporadic disease in isolated naturalised communities of *Dactylis glomerata* that occur along roadsides and in pasture situations in the cooler highlands and slopes of eastern Australia; *Psd* has not been detected in Western Australia.

P. striiformis f. sp. *poae* (Tollenaar and Houston 1967) was described as the pathogen causing stripe rust of Kentucky bluegrass (*Poa pratensis*). Temperature optima for urediospore germination (12–18°C) and the close association between pathogen isolates and the host suggest that this is a distinctive *forma specialis*, although the geographic distribution outside the US remains unclear. Stripe rust on bluegrass has not been reported in Australia.

Several other host range variants have been proposed, including f. spp. *agropyri*, *elymi*, *secalis* (Eriksson 1894, in

Zadoks 1961) and varieties *striiformis* and *dactylidis* (Manners 1960). However, an examination of the host range and spore size evidence (Wellings 1986) and more recent data for molecular differentiation (Line 2002) concluded that these designations were not valid.

A potentially new form of *P. striiformis* was detected in Australia in 1998 and described by Wellings *et al.* (2000a). The pathogen, commonly referred to as barley grass stripe rust (BGYR), was observed to cause disease on certain barley cultivars and lines naturally infected in the field (Wellings *et al.* 2000b). BGYR was closely associated with weedy *Hordeum* species, showed broad avirulence on standard wheat differential testers with the exception of Chinese 166 (carrying *Yr1*), and appeared to contrast at one isozyme locus with *Pst*. Further studies demonstrated the unique molecular phenotypes of BGYR isolates compared to a collection of Australian *Pst* pathotypes (Keiper *et al.* 2003), and so provided more evidence for the unique grouping of BGYR as a potentially new *forma specialis* within *P. striiformis*.

Barley cultivars Skiff and its derivatives, including Tantararra, have shown moderate levels of disease in field nurseries inoculated with BGYR. Crop losses have not been reported and the disease is expected to remain of minor importance. However, this situation could rapidly change with the release of BGYR-susceptible cultivars and the opportunities this could provide for the further evolution of more virulent pathotypes adapted to commercial barley cultivars. The release of cv. Maritime in South Australia in 2004 was of concern in this regard with its susceptible response to BGYR observed in several field nurseries.

The detection of BGYR in 1998 represents another incursion event for *P. striiformis*. The origins remain unclear as the international distribution of BGYR remains unresolved. However, R. A. McIntosh (pers. comm.) has observed phenotypically similar stripe rust infections on natural communities of *Hordeum* spp. in California and Chile.

Pathogenic evolution in *Pst*

Early studies in Australia: origin and means of entry

The means of introduction of *Pst* into Australia remained initially unclear. The transport of spores at high altitudes from east Africa had been earlier implicated in the arrival of exotic *Pgt* pathotypes by Watson and DeSousa (1983). This possibility has been more recently supported by studies associating wind connections and floristic similarity among islands in the Antarctic region (Munoz *et al.* 2004). However, the contemporary *Pst* pathotypes from east Africa in the late 1970s were clearly quite different to that detected in Australia (R. W. Stubbs, pers. comm.). The pathotype detected in Australia was also very common in southern Europe in 1979 (Wellings *et al.* 1987). Watson and Butler (1984) suggested that *Pst* spores may have been transported in the undercarriage of international aircraft arriving in Australia from Europe. However during this period, air travel from Europe to Australia required 2 refuelling episodes in Asia and the Subcontinent, and spores, if held in wheel bays, would have been purged. Wellings *et al.* (1987) provided evidence that clearly implicated the retention of *Pst* spores on clothing, and the potential for heavy spore contaminated

clothing to support viable propagules for up to a week at ambient temperatures of approximately 18°C. Subsequent instances of further international transport of *Pst* spores have clearly implicated the role of air travellers (Wellings and Park 2006).

Differential systems for classification

Pathotype variability has traditionally been studied using defined sets of host genotypes inoculated with pathogen isolates in controlled environments. Such studies have largely focused on greenhouse testing of seedling plants and, when most effective, have attempted to relate the results to resistance genes deployed in commercial agriculture. The various sets of differential genotypes employed in the study of *Pst* were reviewed by McIntosh *et al.* (2005). The set of Johnson *et al.* (1972), as modified by Wellings and McIntosh (1990), has been used for continuing studies of pathotype variability in the Cereal Rust Laboratory at PBI Castle Hill and later at Cobbitty from 1991, since the first introduction of *Pst* to Australia in 1979.

The significant additions to the international differential set that were important in Australian studies of *Pst* were:

1. Avocet R (*YrA*). This resistance, which has remained under the designation *YrA* since first described by Wellings *et al.* (1988), was the first to be overcome by the pathogen in 1981. The resistance was common in Australian and international spring wheats (Wellings *et al.* 1988) and several cultivars were severely affected by the new pathotype in the early 1980s (Wellings and McIntosh 1990).
2. Selkirk (*Yr27*). This gene, designated by McDonald *et al.* (2004), was known to be widely distributed in spring wheats (Wellings 1992). Although virulence has not been detected in Australia, certain New Zealand isolates were observed to be virulent for *Yr27* (C. R. Wellings, unpublished data).
3. Trident (*Yr17*). Several Australian breeding groups have used the VPM1 source of rust resistance carrying the linked genes *Yr17*, *Lr37*, *Sr38* that were described by Bariana and McIntosh (1994). The occurrence of virulence for *Yr17* caused noticeably increased levels of rust development on several cultivars that were only protected by this gene.

The above resistances were not present in the international differentials, and were known to be components of resistance in a range of commercial Australian wheats. Their use as supplementary testers subsequently resolved agriculturally important variation in the Australian *Pst* population. Virulence for these genes in *Pst* isolates was designated as a suffix following the international pathotype code.

Pathogenic evolution in Australia

The essential features of the pathogenic evolution in Australia for the 10-year period following the first detection of *Pst* in 1979 were described by Wellings and McIntosh (1990). This period was characterised by several epidemic seasons with a correspondingly large pathogen population size. A range of new pathotypes was detected in this period, and each newly emerging pathotype was observed to be phenotypically closely related to a pre-existing pathotype (Fig. 1). It was concluded that new pathotypes arose as a result of single gene mutations and these were predominantly for increased virulence (Wellings and McIntosh 1990). The exception was an apparent loss of virulence

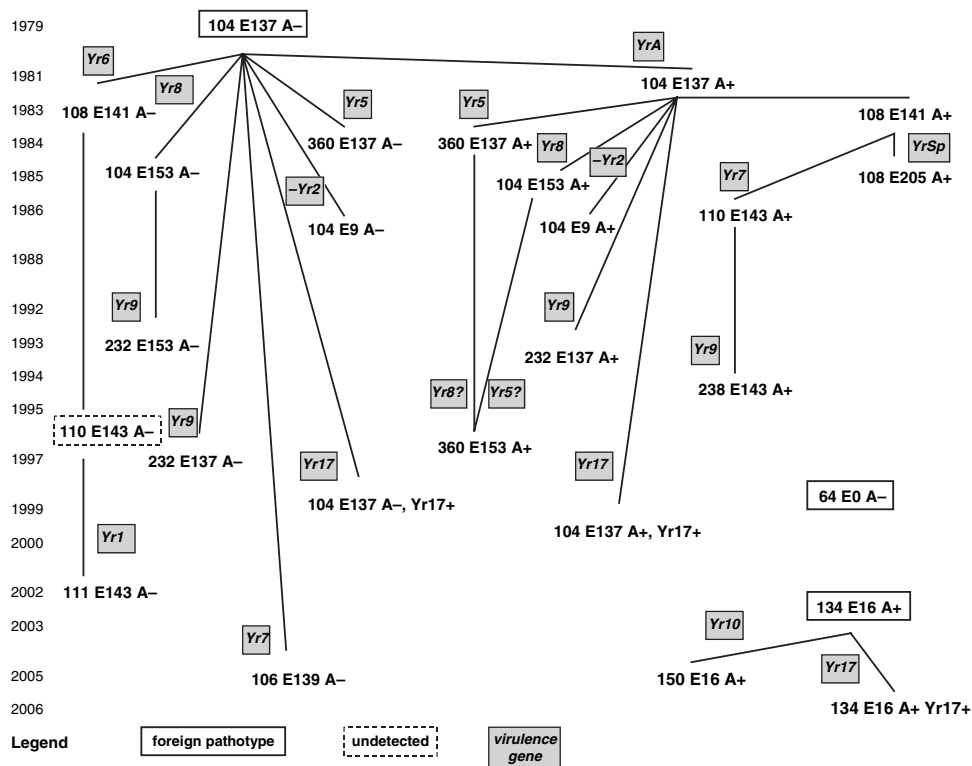


Fig. 1. Progressive emergence of new pathotypes of *P. striiformis* f. sp. *tritici* in relation to presumed mutation events in previously detected pathotypes in Australia, 1979–2006.

for *Yr2* in pts 104 E9 A– and 104 E9 A+. These observations confirmed earlier conclusions from decades of work at PBI with the leaf rust and stem rust pathogens of wheat (Luig 1985). On this basis it was concluded that no new introductions of *Pst* occurred in Australia in this period.

The second decade of surveys revealed similar trends in pathotype evolution (Fig. 1). A presumed exotic introduction was identified in 1999 with the detection of pt 64 E0 A–. This pathotype was recorded at low levels in subsequent seasons, although its relatively high level of avirulence did not provide the capacity for comparative advantage and hence its survival ability was likely impeded. Similarly, most new pathotypes detected in this period were of little significance to commercial agriculture since the respective gene for virulence in the pathogen was rarely (*Yr9*) or never (*Yr1*, *Yr5*, *Yr8*, *YrSP*) deployed in commercial cultivars. Pt 238 E143 A+ arose as a mutant culture with virulence for *Yr9* from pt 110 E143 A+ during routine screening in the greenhouse. Two other cultures with colour variation in pts 104 E137 A– and 134 E16 A+ were also detected in greenhouse screening. These three are the only current examples of pathotype variants recovered during greenhouse studies; the remaining pathotypes were detected in samples collected from field sites.

The origin of several pathotypes remains unclear. Pt 111 E143 A– was identified in 2002 and was closely related to pt 110 E143 A–, although this pathotype has not been detected in Australia to date. Pt 360 E153 A+ detected in 1997 may have arisen as a single gene mutation from either of 2 possible pathotypes, or alternatively may have been the result of a somatic

recombination event between these same putative progenitors. Somatic recombination has only been rarely demonstrated under experimental conditions in *Pst*, and this has usually involved the *Yr1* locus (Little and Manners 1969). Further work is warranted to examine the potential of somatic recombination to yield new pathogenic combinations.

The notable feature of the *Pst* population during the first half of the third decade was the incursion of a new pathotype in Western Australia in 2002 (Fig. 1). Wellings *et al.* (2003) noted that the new pathotype, designated 134 E16 A+, was clearly contrasting from the eastern Australian population, although phenotypically similar to isolates of *Pst* first reported as exotic incursions in the US in 2000 (Chen 2005) and in northern Europe in the same period (Hovmöller and Bayles 2005). It was concluded that this incursion was also likely to be the result of accidental introduction on travellers clothing (Wellings *et al.* 2003). New pathotypes arising from this incursion have been 150 E16 A+, with increased virulence for *Yr10*, and 134 E16 A+ *Yr17*+, with increased virulence for *Yr17* (Fig. 1). The impact of the latter pathotype is currently unknown, but predicted to be significant since a large proportion of wheat cultivars released since the arrival of pt 134 E16 A+ in Australia carry this gene.

Population dynamics in *P. striiformis*

Epidemic variation

Stripe rust epidemics have varied in intensity and duration under Australian conditions over the 28 years from 1979 to 2006. The samples received and processed during annual rust surveys

at the Cereal Rust Laboratory, PBI, provide a measure of this variation (Fig. 2). In general terms, the severe epidemic seasons (1983–1985, 2003–2005) began in early winter (May–July) on cultivars vulnerable to the disease. This seemed to allow inoculum build-up and subsequent rapid disease development in early to mid spring (September–October), especially if moisture and temperature conditions remained favourable for crop growth. In contrast, mild epidemics were frequently associated with dry conditions, late epidemic onset in spring, and relatively poor crop performance (1982, 1991, 1994).

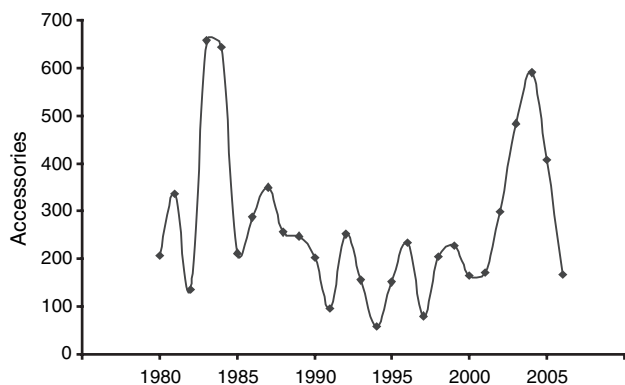


Fig. 2. Annual sample numbers received and accessioned at the PBI Rust Laboratory for pathotype analysis of *Pst* from 1980 to 2006.

Major pathotypes

The original pathotype detected in Australia became the progenitor of a further 20 closely related pathotypes over the 28 years from 1979 to 2006. However, only a subset of these pathotypes became significant or important components of the pathogen population. The infection type (IT) characteristics of these major pathotypes were characterised using the scale described by Wellings *et al.* (1988) and are presented in Table 1. The respective features of these pathotypes are discussed below.

Pt 104 E137 A–

The first pathotype detected in 1979 was concluded to be avirulent on *Yr1*, *Yr5*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr10*, and the resistances in the differential testers Carstens V and Spaldings Prolific. Virulence for Heines VII (*Yr2*, *Yr25*), Vilmorin 23 (*Yr3*), Hybrid 46 (*Yr4*), and the unspecified resistance in Strubes Dickkopf were concluded, although the ITs were not fully compatible. Virulence for Nord Desprez and Suwon92/Omar was indicated by full compatibility. A distinctive feature for this pathotype was the low IT on the tester for *YrA*. The low IT for *YrA* is sensitive to light intensity and hence the second leaf IT can be important in determining the pathotype response to the tester, Avocet R (Wellings *et al.* 1988).

Pt 104 E137 A+

The features of this pathotype were identical to pt 104 E137 A–, with the exception of increased virulence for *YrA* (Table 1).

Table 1. Seedling infection type characteristics of the six major pathotypes of *Pst* recorded in Australia from 1979 to 2006
IT scale 0 (resistant) to 4 (susceptible) as described in Wellings *et al.* (1988); C, chlorotic tissue; N, necrotic; B, brown pustules

Test line	Resistance genotype	Pathotype					
		104 E137 A–	104 E137 A+	108 E141 A–/+	110 E143 A+	104 E137 A– Yr17+	134 E16 A+
Chinese 166	<i>Yr1</i>	0;	0;	0;	0;	0;	0;
Lee	<i>Yr7</i>	1-CN	1-CN	1-CN	33+	1-n	33+
Heines Kolben	<i>Yr2</i> , <i>Yr6</i>	;N1 =	;N	3+	3+	;N1-	3+
Vilmorin 23	<i>Yr3</i>	33 + C	33 + C	33+	3C	33+	;CN1 =
Moro	<i>Yr10</i>	0;	0;	0;	0;	0;	0;
Strubes Dickkopf		33+	33+	33+	3+	33+	1+
Suwon 92/Omar		3 + 4	4	3 + 4	3 + 4	3 + 4	;
Clement	<i>Yr2</i> , <i>Yr9</i>	0;	0;	0;	0;	0;	3-
<i>Triticum spelta</i>	<i>Yr5</i>	0;	0;	0;	0;	0;	0;
Hybrid 46	<i>Yr4</i>	2 + 3	23-	3-	23-	2B	;
Reichersberg 42	<i>Yr7</i>	;1 = N	;N1-C	;CN	33+	;N	1N
Heines Peko	<i>Yr2</i> , <i>Yr6</i>	;N	;N	3+	3+	;N	22 + C
Nord Desprez		3 + 4	3 + 4	3+	3+	3 + 4	1C
Compair	<i>Yr8</i>	0;	0;	0;	0;	0;	3
Carstens V	<i>Yr32</i>	;C	;C	;C	;C	;C	;C
Spaldings Prolific	<i>YrSP</i>	;C	;C	;C1 =	;C	;C	0;
Heines VII	<i>Yr2</i> , <i>Yr25</i>	3	2 + 3	3	33+	3	1C
Avocet R	<i>YrA</i>	2 + C	3+	;C1/3+	3+	3+	3+
Kalyansona	<i>Yr2</i>	3+	3+	3+	3+	3+	3+
Trident	<i>Yr17</i>	;C1	;C1–	;C1+	;C1	3+	2 + C
Yr 15/6* AvS	0;	0;	0;	0;	0;	0;	0;
Hugenoot	<i>Yr25</i>	2 + 3C	2 + 3	2 + 3	3	3-	3
Selkirk	<i>Yr27</i>	1 = NB	1 = NB	1 = B	1 = NN	1-NB	1 = NB
Fed 4*/Kavkaz	<i>Yr9</i>	0;	0;	0;	0;	0;	3 + 4
Federation 1221		3 + 4	4	4	3 + 4	3 + 4	3 + 4
Gregory	<i>Yr33</i>	2 + N	22 + N	2CN	2 + N	22 + N	2 + 3-N

Pt group 108 E141

This group is characterised by virulence for *Yr6* and was presumed to have derived as a single-step mutation from pt 104 E137. This group was aggregated to include A– and A+ types for the purpose of examining frequency distribution over time. The *YrA* avirulent pathotype (108 E141 A–) produced a characteristically lower IT than alternative ‘A–’ pathotypes (Table 1).

Pt 110 E143 A+

This pathotype was detected in 1986 with increased virulence for *Yr7* over pt 108 E141 A+. The combined virulence for *Yr6*, *Yr7*, and *YrA* resulted in the selection of this pathotype for resistance breeding activities at PBI Castle Hill/Cobbitty from 1986 to 2002.

Pt 104 E137 A– *Yr17*+

Increased virulence for *Yr17* in pathotype 104 E137 A– resulted in the detection of this pathotype in 1999. This pathotype would seem to be relatively poor in selective advantage since it is avirulent for *Yr6*, *Yr7*, and *YrA*. These respective resistance genes are present in arrange of commercial Australian cultivars and thus provide protection in the presence of this pathotype.

Pt 134 E16 A+

This pathotype was first detected in Western Australia in 2002 (Wellings *et al.* 2003). The contrasting features for this pathotype, compared to the various pathotypes arising from pt 104 E137 A–, were avirulence for Vilmorin 23, Strubes Dickkopf, Suwon 92/Omar, Nord Desprez, and Heines VII. Virulence for *Yr6*, *Yr7*, *Yr8*, *Yr9*, and *YrA* are combined in this pathotype, although they occurred in various combinations among the pathotypes detected in eastern Australia. The distinctive features of this pathotype compared to previously recorded pathotypes in eastern Australia in 2002 led to the conclusion that it was of foreign origin, possibly from North America (Wellings *et al.* 2003).

Variation in pathotype frequency

Pathotype fluctuations have resulted from adaptation to cultivars, chance events associated with opportunities for over-summer survival, or ability to preferentially survive environmental conditions. Six major pathotypes, or pathotype groups, have dominated the pathogen population at varying levels from 1979 to 2005 (Fig. 3).

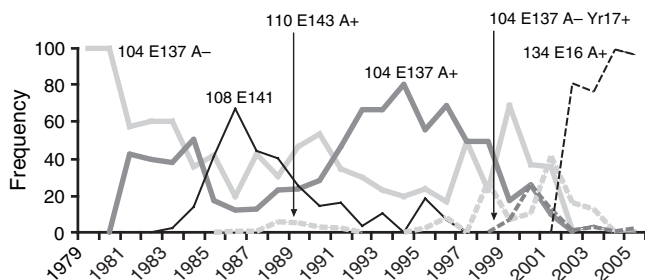


Fig. 3. Relative frequencies of major *Pst* pathotypes over the period 1979–2005.

Pt 104 E137 A–

This pathotype is considered to have formed the founder population of *Pst* in 1979, and has been consistently recovered over the subsequent 24 seasons. It appeared to achieve recovery rates in annual surveys that indicated it remained dominant from 1979 to 1984, and regained this dominance over competing pathotypes in 1990 and 2000.

Pt 104 E137 A+

Virulence for *YrA* provided a selective advantage for this pathotype on wheats carrying this gene (e.g. Avocet, Egret, Banks) in the mid 1980s (Fig. 3). The apparent surge in frequency in the mid 1990s remains unexplained. It is commonly expected that this may have resulted from a selective advantage on wheats carrying the corresponding resistance gene, although it is also likely that this pathotype was simply able to survive the over-summer period and then able to maintain its relatively high frequency during a series of years characterised by a low pathogen population (Figs 2 and 3).

Pt group 108 E141

This pathotype group emerged in 1983 with a comparative advantage of virulence on *Yr6*. The frequency of this pathotype gained momentum over a 2–3-year period, reaching a peak in 1986. During this time period, cv. Millewa was widely grown in southern NSW and the Victorian Wimmera district, and the cultivar proved to be vulnerable to this pathotype. As the wheat industry reduced plantings of Millewa, so the frequency of pt 108 E141 declined over the subsequent 10 years.

Pt 110 E143 A+

The historic high pathogen population in 1986 (Fig. 2) provided a potential opportunity for the emergence of new pathotype variants. Pt 110 E143 A+ was detected as a presumed single-step mutant from pt 108 E141 A+ with increased virulence for *Yr7*. This pathotype represented the most broadly virulent pathotype of *Pst* at the time, and it was used intensively for the purposes of breeding and selection in field nurseries. However, it remained at low frequency in wheat-growing regions for a period of 12 years until the late 1990s when it began to re-emerge and increase in frequency. This coincided with the release and adoption of the hybrid cultivar H45 that carried *Yr7*, and which proved to be moderately susceptible to this pathotype.

Pt 104 E137 A– *Yr17*+

Virulence for *Yr17* arose within pt 104 E137 A– in 1999 giving rise to pt 104 E137 A– *Yr17*+, a variant with virulence for *YrA* was detected in 2000. However, the former has been consistently recovered from 1999 to 2005 at frequencies that have not exceeded 26% in any one season. Although this pathotype has not reached frequency levels approaching that of the other significant pathotypes, it has remained an important component of the pathogen population as it has a distinctive advantage on cultivars that are largely protected only by *Yr17* (e.g. QAL2000, Stylet, Camm).

134 E16 A+

This exotic pathotype was first detected in Western Australia in 2002 and was subsequently reported in eastern Australia in the following year. The epidemics resulting from this pathotype have been widespread and damaging across Australian wheat-growing regions from 2002 to 2005. Drought conditions mitigated the effects of stripe rust in 2006. The pathotype has rapidly become dominant in the pathogen population since the initial introduction, due to its capacity to cause disease on a range of cultivars that were previously resistant or moderately resistant. This dominating feature is similar to that reported for the *Pst* pt PST-78, which was first detected in US in 2000 (Chen 2005). Initial greenhouse tests have provided evidence to suggest that the US pt PST-78 and the Australian 134 E16 A+ are identical (C. R. Wellings and X. M. Chen, unpublished data). The pathogen population in the US that has developed following the introduction of pt PST-78 has given rise to 13 new but related pathotypes over a 5-year period (Chen 2005). In contrast, the Australian pt 134 E16 A+ has given rise to only 2 new pathotypes, viz. 150 E16 A+ (*Yr10* virulent) and 134 E16 A+ *Yr17+* (*Yr17* virulent). Research in the US suggests that the new pathogen population that has emerged since 2000 has become aggressive and more widespread in geographical range due to several factors including an adaptation to warmer temperature environments (Milus *et al.* 2006). As the US and Australia pathogen populations may be closely related, it is possible that this feature may also play a role in the epidemiology of stripe rust in Australia. However, this hypothesis will require further evaluation.

Molecular studies of the Australian isolates of *Pst*

There have been limited studies of the Australian *Pst* population. An examination of the dsRNA phenotypes among a selection of Australia *Pst* isolates revealed a diversity of base pair sizes between pathotypes and between isolates of the same pathotype (Dickinson *et al.* 1990). It was clear that the dsRNA phenotypes were unrelated to pathogenicity, but their biological role remains undetermined. Steele *et al.* (2001) examined a collection of 16 Australian *Pst* isolates comprising 8 pathotypes using RAPD and AFLP methods, and found no evidence of polymorphic variation. Two pathotypes from New Zealand showed identical patterns, confirming the clonal nature of the pathogen and suggesting similar population structures in Australia and New Zealand. Keiper *et al.* (2003) used several molecular methods in an examination of 5 *Puccinia* species and special forms associated with cereals in Australia. They confirmed, among a set of 9 *Pst* isolates, that molecular diversity was low. Recent work by Nazari (2006) provided further support for these conclusions using AFLP assays among an extensive set of pt 104 E137 A– isolates collected over 20 years in Australia. There was some evidence for molecular variation among one of the earliest isolates collected in 1979, suggesting that the founder pathotype may have initially comprised a population of mixed genotype.

Conclusions

The population biology of *Pst* in Australia over a comparatively short period of 27 years from 1979 to 2006 has shown features of

pathogen adaptation that were similar to that described for *Pgt* and *Pt* since detailed studies of pathogenicity were undertaken for these pathogens from the early 20th Century (Luig 1985). The data for *Pst* in Australia suggests that the processes associated with pathogen variation are exotic introduction, mutation, selection, and the chance events associated with pathogen survival during the critical non cropping phase in the warm to hot summer period.

Exotic introduction, including the first occurrence of the pathogen and subsequent new pathogenic variants, has been relatively rare for *Pst* in Australia. The evidence presented indicates an initial introduction of *Pst* pt 104 E137 A– to Australia in 1979, a subsequent introduction of the exotic pt 64 E0 A– in 1999, and a third exotic introduction with pt 134 E16 A+ detected in Western Australia in 2002. A fourth incursion event was implicated in the detection of BGYR in 1998. The relative importance of each of these events to the wheat industry was variable; the pathotypes and their subsequent derivatives originating from the first and third exotic introductions formed the basis of widespread and damaging epidemics. In contrast, the introduction of pt 64 E0 A– appeared to have no impact on crop production.

Mutation within the *Pst* population, as measured by the emergence of new and closely related pathotypes, was a feature of the family of pathotypes derived from pt 104 E137 A–. Although 20 new pathotypes were detected over 28 years, only a relatively small subset of these became important components of the pathogen population. Only 2 mutant derivatives of pt 134 E16 A+ have been detected to date. In contrast, the arrival of a pathotype similar to pt 134 E16 A+ into US from 2000 has resulted in at least 17 pathotypes in 4 years to 2003 (Chen 2005).

Pathotype selection has been an important factor in the selective survival and domination of certain pathotypes. This was evident for pathotype group 108 E141 with adaptation to cultivars protected only by *Yr6*, and pt 110 E143 A+ with a comparative advantage on cultivar H45 carrying *Yr7*. In the former, the first detection of the virulent pathotype was 3 years before the pathotype becoming widely distributed. In the case of pt 110 E143 A+, the period between first detection and economic damage was much greater. These examples serve to indicate the value of a relevant and effective pathotype survey that provides the opportunity to detect the emergence of important pathotypes with sufficient lead time to allow the farming community to respond. Under Australian wheat-growing practices, seed for sowing each season is generally retained on-farm; new cultivars are initially purchased in small quantities and multiplied over several seasons. Hence, the detection of a new and important pathotype is of great value in providing an early warning mechanism that allows the farming community sufficient time to adjust cultivar deployment.

Despite the clear importance of exotic introductions, mutant derivatives, and the selective forces exerted by cultivar resistance genes on the diversity of the *Pst* population in Australia, there is also evidence to suggest that chance events may also be important. For example, pt 104 E137 A+ dominated the *Pst* pathogen population in the mid 1990s during a period of low pathogen inoculum and in the absence of

any apparent comparative advantage on commercial cultivars. It is likely that this pathotype survived the summer period by chance in local habitats and in sufficient quantity in the dry seasons of 1991 and 1994 to re-emerge in commercial fields and maintain its dominant role over other pathotypes. Alternatively, this pathotype may have an adaptation to climatic conditions similar to that suggested for the US *Pst* population after 2000 (Milus *et al.* 2006). This will require further investigation.

Studies of *Pst* in Australia using molecular techniques have supported the clonal nature of pathogen biology. The relative paucity of molecular polymorphism currently available has restricted the capacity to apply unselected genetic markers in population studies. In contrast, the study of pathogenic variation in *Pst* based on modifications of standard differential sets (Johnson *et al.* 1972; Wellings and McIntosh 1990) has demonstrated the progressive occurrence of pathotypes closely related to a pre-existing pathotype. It is presumed that mutation is the basis for the occurrence of new pathogenic variants, although the nature of the mutational change in the genetic code remains unknown. Annual pathogenicity surveys will continue to form the basis for population studies, and provide the farming and wheat-breeding communities with vital information in the quest to maintain effective resistance in commercial agriculture. However, the method of pathotype nomenclature is in need of revision since important events in the *Pst* population in Australia could not be detected using the international system of Johnson *et al.* (1972). The limitations of the latter system and the availability of isogenic lines developed using the semi-dwarf spring wheat Avocet S (Wellings *et al.* 2004) should provide the basis for nomenclature change.

Wheat stripe rust in Australia has caused significant crop loss and resulted in unprecedented costs in chemical control expenditure in epidemic seasons. Unpublished industry estimates suggest that AU\$40–90 million has been expended per annum in 2003–2005 on chemical strategies to contain stripe rust epidemics across all Australian wheat-growing areas. The disease continues to threaten wheat production with potential annual costs in the absence of control measures estimated to be \$180 million (Brennan and Murray 1998). It can be anticipated that control measures will be largely based on the development and release of resistant cultivars, although chemical control may have a place in high input/high yield situations in irrigation areas and high rainfall zones. Breeding for resistance will continue to be based on current awareness of variability in *Pst*, the search for and commercial development of new and effective resistance combinations, and the resolve of industry to adopt best management practices that minimise disease risk.

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