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Studies on the origin, spread, and evolution of an important group of *Puccinia recondita* f. sp. *tritici* pathotypes in Australasia

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Abstract

Wheat brown rust pathotype (pt) 104-2,3,(6),(7),11 was first detected in Australasia in Victoria during 1984. Although it appeared similar to a pre-existing pathotype, 104-2,3,6,(7), detailed greenhouse tests revealed nine pathogenic differences between the two rusts. Six differences involved contrasting virulence/avirulence for the resistance genes/specificities *Lr12*, *Lr27* + *Lr31* and *Lr16*, and three uncharacterised genes, present in the wheat cultivars Gaza and Harrier, and in triticale cultivar Lasko. Differences in partial virulence between the pathotypes were found for the genes *Lr2a*, *Lr13* and *Lr26*. A comparison of the phenotypes for 13 isozyme systems in the two pathotypes revealed two differences, including a *Pgm2* allele in pt 104-2,3,(6),(7),11 not found in other contemporary Australasian *Puccinia recondita* f. sp. *tritici* pathotypes. On the basis of these differences, it was concluded that pt 104-2,3,(6),(7),11 was introduced into the Australasian region before or during 1984.

Seven variants of pt 104-2,3,(6),(7),11, that differed by single virulences, were detected during 1984–1992. Pt 104-2,3,(6),(7),11 and a derivative pathotype with virulence for *Lr20* underwent rapid increases in frequency, largely displacing pathotypes which predominated before 1984. Although first detected in eastern Australia, both pathotypes spread to New Zealand, and the derivative pathotype appeared in Western Australia. The rapid spread and increase of these pathotypes could not be explained by host selection. Pt 104-2,3,(6),(7),11 and derivatives may therefore be more aggressive than other contemporary Australasian pathotypes.

Abbreviations: NSW = New South Wales; *Prt* = *Puccinia recondita* f. sp. *tritici*; Qld = Queensland; SA = South Australia; WA = Western Australia.

Introduction

Annual pathotype surveys for the wheat brown (leaf) rust organism (*Puccinia recondita* Rob. ex Desm. f. sp. *tritici* Eriks. & Henn.) and the wheat black (stem) rust organism (*P. graminis* Pers. f. sp. *tritici* Eriks. & Henn.) have been conducted in Australia and New Zealand (Australasia) since the 1920s. Pathogenicity studies on *P. graminis* f. sp. *tritici* at the University of Sydney have indicated that in the absence of sexual recombination, new pathotypes were derived primarily from existing pathotypes by single (or rarely double) step mutations at loci governing pathogenicity [Luig, 1977]. Occasionally, a pathotype which differed from existing pathotypes at many pathogenicity loci was detected, and in such cases it was concluded that the new pathotype had originated from a region outside Australasia [Luig, 1977; Watson, 1981]. Such introductions were postulated for the origin of wheat stem rust standard races 126 (first detected in 1925), 21 (1954), and 194 and 326 (1969) [Luig, 1977]. A study which compared the isozyme phenotypes of Australian pathotypes of *P. graminis* f. sp. *tritici* supported this hypothesis [Burdon *et al.*, 1982].

A similar situation is thought to exist for the Australasian wheat brown rust population. Luig *et al.* [1985] concluded that an isolate of *Prt*, first detected in New Zealand in 1981 [now designated

53-1,(6),(7),10,11; Park and Wellings, 1992], was introduced into the Australasian region, most probably sometime before the 1981 wheat growing season, when it was first detected. It differed from existing pathotypes in five pathogenic attributes and was also found to have the *Got b* allele which had not previously been recorded in Australasian isolates of *Prt*. It is now known that this pathotype also differed from pre-1981 Australasian pathotypes of *Prt* in possessing avirulence for the adult plant resistance gene *Lr22b* [McIntosh and Gordon-Werner, 1989; Park and McIntosh, 1994].

Pathotype 104-2,3,(6),(7),11 was first detected in Victoria in 1984, and was considered at the time to be related to pt 104-2,3,6,(7), first found in 1976. This paper presents results of comparative tests of the pathogenicity and enzyme phenotypes of these two pathotypes. The evolution of pathotypes putatively derived from pt 104-2,3,(6),(7),11, the increase and spread of these pathotypes in the Australasian region, and their significance in relation to wheat breeding and cultivation in the region are also discussed.

Materials and methods

Pathotype nomenclature

Pathotypes (races or strains) of *Prt* were identified following the system originally described by Watson and Luig [1961], and expanded by the addition of further differentials [Park and Wellings, 1992]. The differential set comprising four genotypes permitted a standard race designation [Johnston and Browder, 1966] and an additional 11 wheats numbered from 1 to 11, were used as supplementary differentials (Table 1). The numbers assigned to differentials were used to indicate virulence for the resistance gene present in the corresponding differential. Thus, a pathotype designated as 104-2,3,6 was standard race 104, and was also virulent on seedlings of the supplementary differentials Gaza (2), Spica (3), and Gatcher (6). Where infection types were intermediate between established virulent and avirulent responses, the number corresponding to the differential was enclosed in parentheses.

Seedling and adult plant tests

The accession numbers of the cultures used in comparisons of pathotypes 104-2,3,6,(7) and 104-2,3,(6),(7),11 were 76694 and 84045, respectively. For seedling tests, seed of host materials were sown in 9 cm plastic pots, and seedlings were inoculated 7–10 days later by spraying a suspension of uredospores in light mineral oil. Plants were then incubated for 18–24 h at ambient greenhouse temperatures in a growth room in which mist was generated by an ultrasonic humidifier. Infected plants were kept at 15–25 °C with natural lighting, and disease responses were recorded after 11–14 days using the infection type scale of Stakman *et al.* [1962]. Adult plant tests involved raising two plants of each of two lines per 25 cm plastic pot in a temperature-controlled room (*c.* 20 °C). Plants were inoculated at anthesis, following the previously described procedure, and responses were recorded after 21 days.

Survey procedures

Australia and New Zealand were divided into five regions on the basis of differences in climate and wheat cultivars grown [Zwer *et al.*, 1992] (Fig. 1). Samples of rusted wheat collected from these regions were forwarded by mail to the University of Sydney, Plant Breeding Institute by researchers, farmers, and other interested persons. Supplementary collections were made by Institute staff during survey tours which involved random crop inspections every 20–30 km along predetermined routes.

Leaf samples which lacked sufficient inoculum were increased initially by inoculating susceptible Sonora W195* seedlings. Often, mixed infection types were encountered on differential lines, indicating the presence of more than one pathotype in the original sample. In these cases, single pustule isolates were established from selected differentials, which were then individually applied to differential sets. Samples with sufficient inoculum were cut into short pieces, agitated in glass tubes containing light mineral oil, and the resulting suspension applied to seedlings as described above. Temperatures during the postinoculation phase were maintained at 20–25 °C.

Isozyme studies

The identity and purity of cultures used in isozyme studies were confirmed by infecting differential sets with each. Approximately 50–100 mg of eight isolates of *Prt* were individually germinated and prepared for enzyme extraction following the procedures of Burdon *et al.* [1982]. Crude enzyme extracts were absorbed onto paper wicks and subjected to horizontal starch-

* W numbers refer to the Plant Breeding Institute wheat collection.

Table 1. Infection types produced by pathotypes 104-2,3,6,(7) [76694] and 104-2,3,(6),(7),11 [84045] of *Puccinia recondita* f. sp. *tritici* on seedlings of the Australasian wheat brown rust differential sets and selected cereal lines at 17 ± 1 °C

Cultivar	Accession number[a]	Relevant host resistance gene	Pathotype	
			104-2,3,6,(7)	104-2,3,(6),(7),11
International series				
Malakof	W970	*Lr1*	3+	3+
Carina	W971	*Lr2b*	2+C	;12−
Brevit	W972	*Lr2c*	3+	3+
Webster	W973	*Lr2a*	;12−	;−
Loros	W974	*Lr2c*	3+	3+
Mediterranean	W1728	*Lr2a, Lr3a*[b]	;12−	;
Hussar	W976	*Lr11*	;12−	;12−
Democrat	W977	*Lr3a*	3+	3+
Australasian series				
1. Thew	W203	*Lr20*	;N	;1−N
2. Gaza	W277	*Lr23*	3+	22−
3. Spica	W2341	*Lr14a*	3+	3+
4. Kenya	W1483	*Lr15*	;	;
5. Klein Titan	W1633	*Lr3ka*	;	;
6. Gatcher[c]	W3201	*Lr27 + Lr31*	3+	XX+
7. Timson[c]	W3689	*Lr17*	X+X++	X++
8. CS 2A/2M	C77.1	*Lr28*	0;	0;
9. Mildress	W3662	*Lr26*	;1−	0;=
10. Egret	W3606	*Lr13*	XX+	X++3
11. Exchange[c]	W2554	*Lr16*	2+C	3+
Miscellaneous lines				
Thatcher	W1201	−	3+	3 +
Thatcher + *Lr23*	W3555	*Lr23*	3+	3+
Harrier	−	?	3+	;12=
Lasko	−	?	33−	;

[a] W numbers refer to the PBI wheat collection. C numbers refer to the PBI Cytogenetics register.
[b] Not all accessions of Mediterranean possess *Lr2a* [Singh and McIntosh, 1985]
[c] Gatcher and Exchange also carry *Lr10*, and Timson is heterogeneous for *Lr10*. *Lr10* is largely ineffective in Australia.

gel electrophoresis using one continuous (A: histidine) and three discontinuous (B: triscitrate-borate; C: lithium; D: morpholine citrate) buffer systems. Details of these systems were given by Clayton and Tretiar [1972], Brown *et al.* [1978] and Moran and Marshall [1978]. Gels were assayed for the following enzymes: Glucose-1-phosphate uridylyl transferase (GPUT; EC 2.7.7.9), malate dehydrogenase (MDH; EC 1.1.1.37), Triose phosphate isomerase (TPI; EC 5.3.1.1), phosphoglucose mutase (PGM; EC 2.7.5.1) on system A; esterase (EST; EC 3.1.1.2), leucine aminopeptidase (LAP; EC 3.4.11.1) and methylumbelliferyl esterase (UMB; EC 3.1.?.?) on system B; β-glucosidase (βGLU; EC 3.2.1.21), dipeptidase (DIP; EC 3.4.?.?), glucosephosphate iosmerase (PGI; EC 5.3.1.9), glutamate oxalate transaminase (GOT; EC 2.6.1.1) and menadione reductase (MDR; EC 1.6.99.2) on system C; and aconitase (ACO; EC 4.2.1.3) on system D.

Following electrophoresis, the anodal portion of all gels were assayed for enzyme activity. In the case of GOT, the cathodal portion was also assessed. Enzyme assays were similar to those described in Mitton *et al.* [1979], Collins *et al.* [1984] and Wendel and Weeden [1989]. All isozyme patterns were confirmed by repeat runs.

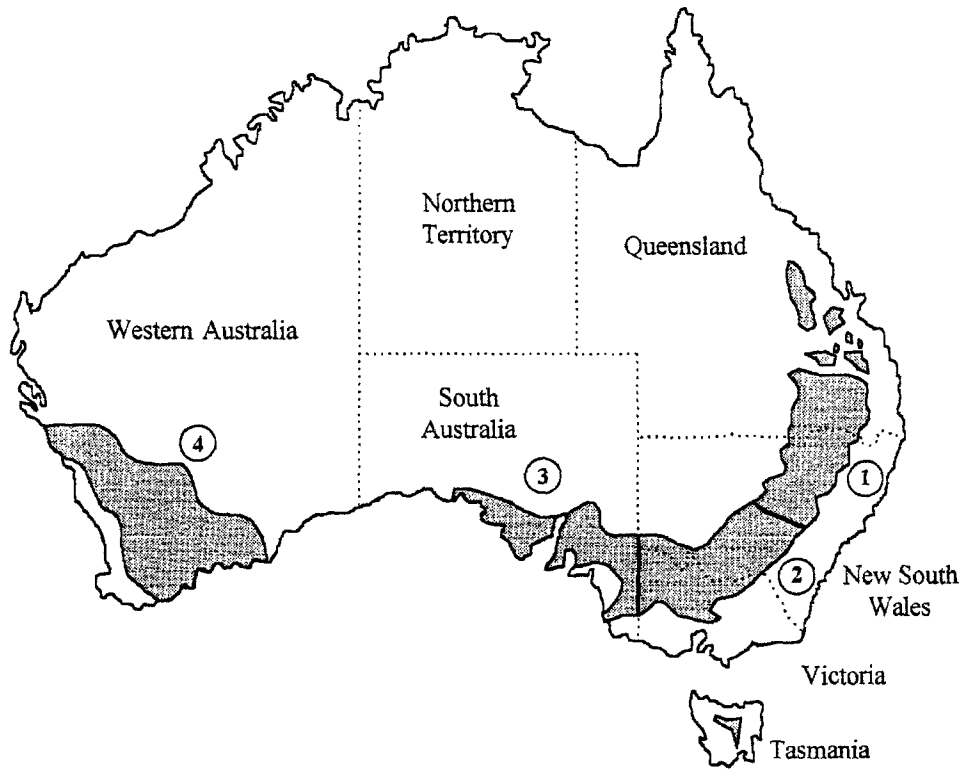

Fig. 1. The wheat producing area of Australia, and the four epidemiologically diverse regions.

Results

Seedling and adult plant tests

Initial tests indicated that whereas isolate 76694 was avirulent on seedlings with gene *Lr16*, 84045 was virulent. A differential line for *Lr16* (cv Exchange) was therefore included in the Australian differential set as the eleventh tester, and culture 84045 was designated pt 104-2,3,(6),(7),11 to indicate virulence on this line. More detailed seedling tests revealed five further differences between the two pathotypes (Table 1). The two pathotypes differed in pathogenicity for the *Lr27 + Lr31* complementary gene specificity in Gatcher, and for uncharacterised resistance in the wheat cultivar Harrier and the triticale cultivar Lasko (Table 1). Multipathotype tests of Harrier and Lasko showed the resistance in these cultivars to be different (R. F. Park, unpubl.). The response of Lasko was found to be temperature sensitive, with resistance being expressed only at low temperatures (Fig. 2). The durum differential Gaza also differed in response to the two pathotypes, despite the fact that both were virulent on the Thatcher + *Lr23* line, indicating the pathotypes differ for virulence for an additional gene in Gaza. Although both pathotypes were avirulent for *Lr2a*, *Lr13* and *Lr26*, subtle, but consistent, differences between the pathotypes were observed on the differentials with these genes (Table 1). These differences were also apparent on seedlings of near-isogenic lines possessing *Lr2a, Lr13* and *Lr26* (R. F. Park, unpubl.).

Comparative tests of the two pathotypes on lines possessing the complementary genes *Lr27 + Lr31* confirmed results of the earlier experiment indicating that pt 104-2,3,6,(7) was fully virulent and pt 104-2,3,(6),(7),11 was avirulent for this resistance under both cool and warm conditions (Table 2). These tests also indicated that the latter was partially virulent for *Lr27 + Lr31* when compared to an older, avirulent pathotype (122-2,3, culture 73003) under warm conditions. These results were confirmed in tests of adult plants of the breeding line SUN 6B, which possesses *Lr27 + Lr31* (Table 3).

Controlled tests on adult plants revealed that whereas pt 104-2,3,6,(7), was virulent for the adult plant resistance (APR) gene *Lr12* (infection type 3+), pt 104-2,3,(6),(7),11 was avirulent (infection type ;1).

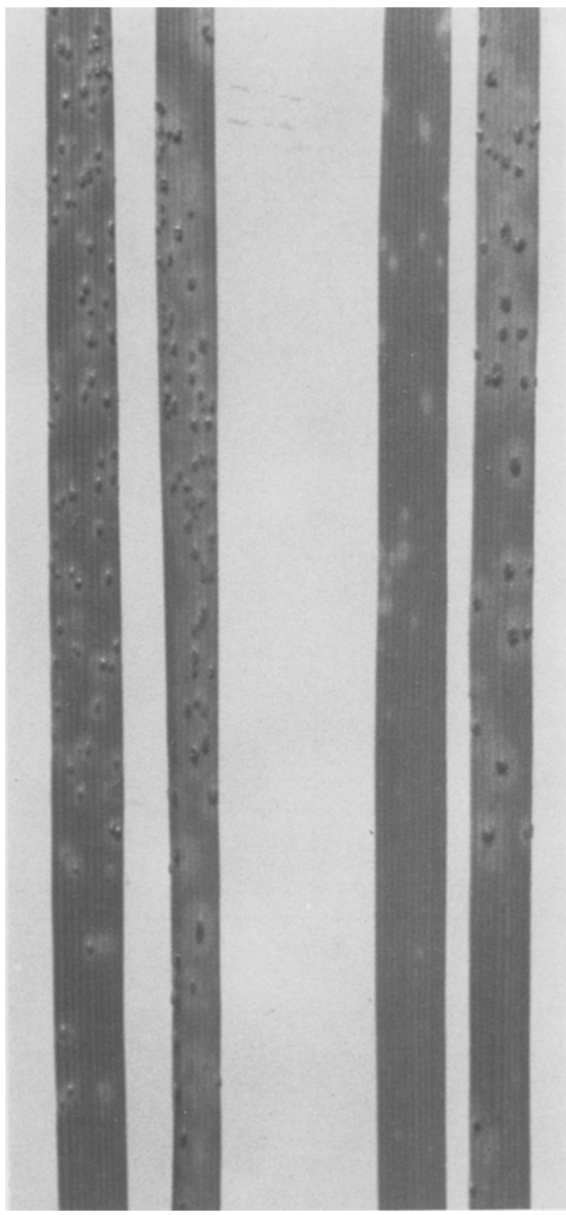

Fig. 2. Seedling leaves of triticale cultivar Lasko infected with *Puccinia recondita* f. sp. *tritici* pathotypes 104-2,3,6,(7) [76694], left hand pair, and 104-1,2,3,(6),(7),11 [89172], right hand pair. After inoculation, leaves on the left hand side of each pair were kept at 17 ± 1 °C, and on the right hand side, 24 °C.

Both pathotypes were avirulent for the genes *Lr13, Lr22a, Lr30, Lr35,* and *Lr37.*

Survey results

Although pt 104-2,3,(6),(7),11 was first detected during the 1984 cropping season, no further isolation was made until three years later (Table 4). The 1985–1986 survey periods coincided with a period of transition in staff, and in view of the initially perceived similarity of this pathotype with pt 104-2,3,6,(7) it is likely that isolates of pt 104-2,3,(6),(7),11 were mistakenly identified as pt 104-2,3,6,(7) during this time. By 1988, pt 104-2,3,(6),(7),11 had spread to northern NSW and Qld (Region 1), and in the following year it was found in all three eastern Australian wheat growing regions plus New Zealand (Table 4).

In 1988, a derivative pathotype, 104-1,2,3,(6),(7),11, with added virulence for *Lr20* (Fig. 3), was detected for the first time in demonstration plots of wheat at Camden, NSW, and soon after at Terang, Victoria (both in Region 2) (Table 5). In the following year, this pathotype was detected in Regions 1, 2, and 3, and in 1990 it was detected in Region 4 (Table 5). The frequency of this pathotype increased in all three regions during 1989–1992, and by 1992 it was most common in samples originating from Regions 2 and 3 (Table 5). Further mutants which presumably developed directly or indirectly from pt 104-2,3,(6),(7),11 were detected in 1990, 1991 and 1992 (Fig. 3). These included two isolates which involved partial increases in virulence. Isolate 91026, identified as pt 104-2,3,(6),(7),11 from a sample collected near Canberra (Region 2) during 1991, gave distinctly higher infection types on lines with *Lr26*. The response of lines with *Lr26* to this isolate was shown to be modified by temperature, whereas that of the putative parental pathotype was not (data not presented). One isolate of pt 104-2,3,(6),(7),11 (92316) collected near Geraldton, WA possessed greater virulence for *Lr17* in comparison with the standard isolate of this pathotype (84045). Unlike pt 104-2,3,6,7 (791023), isolate 92316 could not be considered to be fully virulent on *Lr17* since it produced an X++3+ infection type on seedlings of Songlen at warm temperatures (Table 2) and an X infection type on adult plants of Thatcher + *Lr17* (Table 3). Isolate 92316 was therefore concluded to possess a level of partial virulence for *Lr17* which was intermediate between that of pt 104-2,3,6,(7) and 104-2,3,6,7.

For the four year period 1989–1992, the most commonly isolated pathotype of *Prt* in all mainland states was either pt 104-2,3,(6),(7),11 or the derivative pathotype 104-2,3,6,(7),11, and in New Zealand (Region 5), these two pathotypes comprised between 26% (1990) and 44% (1992) of isolates identified (Table 4).

Table 2. Infection types produced by various pathotypes of *Puccinia recondita* f. sp. *tritici* on seedling leaves of selected wheats at two temperatures

Host line	Resistance gene	Pathotype [culture]				
		122-2,3 [73003]	104-2,3,6,(7) [76694]	104-2,3,6,7 [791021]	104-2,3,(6),(7),11 [84045]	104-1,2,3,(6),(7),11 [92316]
Cool[a]						
Gatcher	*Lr27 + Lr31*	X–	3+	3+	X–X=	X–
Timgalen[b]	*Lr27 + Lr31*	;1–	3+	3+	X+X++	X+X++
SUN 6B	*Lr1, Lr3a, Lr27 + Lr31*[c]	;1–	3+	3+	X+	X
Timson	*Lr17*	X+	X++3	3+	X++3	3+
Songlen	*Lr17*	X++3	3+	3+	3+	3+
Warm[d]						
Gatcher	*Lr27 + Lr31*	;1–	3+	3+	X=	X–
Timgalen	*Lr27 + Lr31*	X=	3+	3+	XX–	XX–
SUN 6B	*Lr1, Lr3a, Lr27 + Lr31*	X=X≡X	3+	3+	X–X=	X–
Timson	*Lr17*	X–X=	X++	3+	X++3	3+
Songlen	*Lr17*	;	X+X++	3+	X+	X++3+

[a] 12–18 °C.
[b] The selection of Timgalen used lacked *Lr3a*.
[c] Genes *Lr1* and *Lr3a* were ineffective against these pathotypes.
[d] 20 °C.

Table 3. Infection types produced by various pathotypes of *Puccinia recondita* f. sp. *tritici* on the flag leaves of selected wheats

Host line	Resistance gene	Pathotype [culture]				
		122-2,3 [73003]	104-2,3,6,(7) [76694]	104-2,3,6,7 [791021]	104-2,3,(6),(7),11 [84045]	104-1,2,3,(6),(7),11 [92316]
SUN 6B	*Lr1, Lr3a, Lr27 + Lr31*	;1–	3+	3+	X–X=	X–X=
Timson	*Lr17*	;;1–	;	33+C	;;+	33+C
Thatcher + *Lr17*	*Lr17*	;	;1–1	3+	;1–	X
Thatcher		3+	3+	3+	3+	3+

Isozyme studies

Initial screening of the *Prt* isolates detected single zones of enzyme activity for all but EST, PEP and PGM where 2, 2 and 3 zones were detected, respectively. In all cases these zones were putatively identified as separate loci. Allelic variation was found at the EST2, GOT and PGM2 loci where bands were well resolved and reproducible. All other enzyme systems were either monomorphic showing single uniform bands consistent with the hypothesis of a single invariant locus (βGLU, LAP, MDH, PGI and TPI); or were insufficiently resolved for accurate assessment (ACO, GPUT, MDR, PEP and UMB).

The multilocus isozyme phenotype obtained for pt 104-2,3,6,(7) was the same as those obtained for all pathotypes tested, with the exception of pts 104-2,3,(6),(7),11, the derivative 104-1,2,3,(6),(7),11 and 53-1,(6),(7),10,11 (Table 6). Comparison of pts 104-2,3,6,(7) and 104-2,3,(6),(7),11 revealed two isozyme differences, with the former possessing a distinctive allele at the *Est* locus (*Est2 b*), whereas the latter possed a different allele at the *Pgm2* locus (*Pgm2 c*). The latter allele was only detected in pt 104-2,3,(6),(7),11, but was also present in the derivative pts 104-1,2,3,(6),(7),11 and 122-1,2,3,(6),(7),11 (Table 6).

As reported previously by Luig *et al.* [1985], pt 53-1,(6),(7),10,11 possessed an allele at the *Got* locus (*Got*

Table 4. Frequency (%) of pathotypes and number of isolates related to an including 104-2,3,6,(7) and 104-2,3,(6),(7),11 of *Puccinia recondita* f. sp. *tritici* identified from regions of Australasia, 1984–1992

Region	Year								
	1984	1985	1986	1987	1988	1989	1990	1991	1992
104-2,3,6,(7) group									
1	91	90	86	78	38	26	7	4	0
2	64	61	62	56	23	4	0	0	0
3	0	20	–[a]	50	0	0	0	2	0
4	0	–	–	–	–	–	0	0	0
5	0	0	0	0	0	0	0	0	0
No. isolates	195	54	52	37	62	16	9	2	0
104-2,3,(6),(7),11 group									
1	0	0	0	0	23	65	71	87	85
2	2	0	0	22	63	91	90	96	97
3	0	0	–	0	0	73	92	86	100
4	0	–	–	–	–	–	100	100	100
5	0	0	0	0	0	36	26	33	44
No. isolates	1	0	0	4	56	142	232	125	523

[a] No samples received.

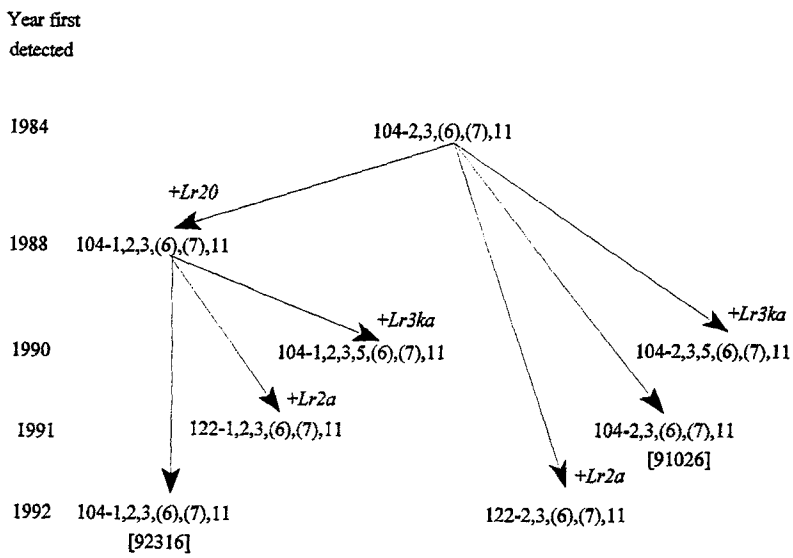

Fig. 3. Putative origins of seven pathotypes of *Puccinia recondita* f. sp. *tritici* considered to be derived directly or indirectly from pathotype 104-2,3,(6),(7),11 in Australasia during 1984–1992. Isolates 91026 and 92316 differ from their putative parents by partial increases in virulence for *Lr26* and *Lr17*, respectively. These changes are not reflected in pathotype designations.

b) which was not present in the other pathotypes (Table 6). This pathotype was also found to be heterozygous at the *Pgm2* locus for a first migrating allele (*Pgm2 a*), which was not present in any other pathotype. *Pgm2* was not examined in the study of Luig *et al.* [1985].

Discussion

The pathogenic and enzymic differences found between isolates 76694 and 84045 of pts 104-2,3,6,(7) and 104-2,3,(6),(7),11 respectively indicate that these two cultures are not closely related. These two cultures

Table 5. Frequency (%) and number of isolates of pathotypes 104-2,3,(6),(7),11 and 104-1,2,3,(6),(7),11 of *Puccinia recondita* f. sp. *tritici* identified in regions of Australasia, 1984–1992

Region	Year								
	1984	1985	1986	1987	1988	1989	1990	1991	1992
Pt 104-2,3,6,(7),11									
1	0	0	0	0	23	47	47	61	43
2	2	0	0	22	58	60	54	48	30
3	0	0	–[a]	0	0	55	28	35	34
4	0	–	–	–	–	–	0	0	0
5	0	0	0	0	0	31	14	33	3
No. isolates	1	0	0	4	54	98	110	60	92
Pt 104-1,2,3,(6),(7),11									
1	0	0	0	0	0	18	23	26	40
2	0	0	0	0	5	31	36	48	63
3	0	0	–	0	0	18	64	49	62
4	0	–	–	–	–	–	98	100	100
5	0	0	0	0	0	5	12	0	30
No. isolates	0	0	0	0	2	44	120	64	420

[a] No samples received.

Table 6. Multilocus isozyme phenotypes for pathotypes of *Puccinia recondita* f. sp. *tritici* collected in Australasia

Pathotype	Isolate accession number	Isozyme phenotype		
		Pgm2	*Est 2*	*Got*
10-1,2,3,4	72469	bb[a]	ab	aa
26-1,3	67028	bb	ab	aa
76-2,3,6,(7)	76583	bb	ab	aa
122-2,3	73003	bb	ab	aa
104-2,3,6,(7)	76694	bb	ab	aa
104-2,3,(6),(7),11	88024	bc	aa	aa
104-1,2,3,(6),(7),11	89172	bc	aa	aa
122-1,2,3,(6),(7),11	91067	bc	aa	aa
53-1,(6),(7),10,11	81043	ab	ab	ab

[a] Alleles are designated 'a', 'b', and 'c' in order of decreasing electrophoretic mobility.

are assumed to be representative of pathotypes 104-2,3,6,(7) and 104-2,3,(6),(7),11 in Australasia, since all isolates of these pathotypes identified in routine pathogenicity surveys gave identical infection types on the seedling differentials used. This is further supported by the results of a previous study, in which more than 50 isolates of *P. recondita* f. sp. *tritici* considered to belong to the 104-2,3,6,(7) group were all found to have the same isozyme phenotype [Burdon *et al.*, 1983]. In addition, pathotypes 104-1,2,3,(6),(7),11 and 122-1,2,3,(6),(7),11, both considered to be related to 104-2,3,(6),(7),11, gave identical results in both adult plant (R. F. Park, unpubl.) and isozyme (Table 6) studies.

A comparison of the multilocus isozyme phenotype for pt 104-2,3,(6),(7),11 with those of other pathotypes present in Australasia when this pathotype was first detected does not support the hypothesis that this pathotype has a hybrid origin. It is therefore most likely that pt 104-2,3,(6),(7),11 was introduced to the Australasian region before or during the 1984 wheat growing season. An exotic origin is further supported by the presence of the unique *Pgm2 c* allele.

The exact origin and the means by which pt 104-2,3,(6),(7),11 was transported to Australia are not clear. Previous studies indicated that two pathotypes of *P. graminis* f. sp. *tritici* were introduced into the Australasian region from southern Africa by wind [Watson and de Sousa, 1982], and that *P. striiformis* f. sp. *tritici* was introduced into the region from Europe by man [Wellings *et al.*, 1987]. No data on the *Pgm2* locus were collected during a survey of North American isolates of *Prt* occurring during or before 1983 [Burdon and Roelfs, 1985]. However, virulence for *Lr16* was rare or absent in North America during the early 1980s [Long *et al.*, 1985; Samborski, 1985], making it unlikely that pt 104-2,3,(6),(7),11 originated from this region. Surveys of pathogenic variation in South

Africa also revealed that virulence for *Lr16* was rare during 1983–1985 [Pretorius *et al.*, 1987]. Comparison of the pathogenicity profile of pt 104-2,3,(6),(7),11 with those given for countries in which virulence for *Lr16* was reported as high during this period is difficult because of differences in the differential sets used to type isolates of *Prt*. Studies of isozymic variability in *Prt* may be a useful approach in trying to determine the origin of this pathotype.

The reason(s) for the rapid increase in frequency of pt 104-2,3,(6),(7),11 and the concurrent decline in frequency of pt 104-2,3,6,(7) and its derivatives are not clear. The only gene for which pt 104-2,3,(6),(7),11 is virulent and for which pt 104-2,3,6,(7) is avirulent is *Lr16*, a gene not present in any currently grown Australian wheat cultivar. The introduction of *P. graminis* f. sp. *tritici* pt 21-0 in 1954 was similarly followed by a rapid increase in the frequency of this pathotype, which within several seasons had largely replaced pathotypes predominating before 1954 [Luig, 1985]. As with pt 104-2,3,(6),(7),11, pt 21-0 did not possess virulence attributes giving it an advantage over existing pathotypes [Luig, 1985]. Presumably in both of these cases, the exotic pathotypes were more aggressive, but as stated by Luig [1985], it is remarkable that a change in frequency of pathotypes can occur so rapidly in the absence of a pathogenic advantage.

The higher frequency of pt 104-1,2,3,(6),(7),11 in SA during 1990–1992 may be related to the cultivation of wheat cultivars with *Lr20* in this region (e.g. Aroona, Lance, Schomburgk and Tatiara). The rapid spread of pt 104-1,2,3,(6),(7),11 into Regions 1 and 3 following its initial detection in Region 2 in 1988 could also indicate that independent mutations to virulence for *Lr20* occurred in pt 104-2,3,(6),(7),11. All other pathotypes which developed directly or indirectly from pt 104-2,3,(6),(7),11 (Fig. 3) have remained at relatively low levels. The only pathotypes within this group which may be significant in future are 122-2,3,(6),(7),11 and 122-1,2,3,(6),(7),11, which possess virulence for *Lr2a*, and perhaps culture 920316 of pt 104-1,2,3,(6),(7),11, which has increased virulence on *Lr17*. Currently, however, the area sown to cultivars with these genes is very small. The detection of seven step-wise mutants of pt 104-2,3,(6),(7),11 during the period 1984–1992 demonstrates the propensity of *Prt* to change, despite the absence of sexual recombination in Australasia.

Pathogenicity surveys in the Australasian region over the past 75 years have indicated that exchange of inoculum occasionally occurs between the eastern and Western Australian wheatbelts, and more commonly between eastern Australia and New Zealand, and that this generally occurs in a west to east direction [Luig, 1985]. The data obtained for pts 104-2,3,(6),(7),11 and 104-1,2,3,(6),(7),11 are consistent with these observations. Pt 104-2,3,(6),(7),11 was first detected at Mt Derimut, Victoria, in February 1984. By 1989, it and the derivative pt 104-1,2,3,(6),(7),11 had spread into NSW, Qld and SA, and both were recorded in New Zealand. Twelve isolates of pt 104-2,3,(6),(7),11 were identified from New Zealand in 1989, originating from both islands. Only one isolate of pt 104-1,2,3,(6),(7),11 was identified. This originated from the south island and was collected about two months after the initial collection of pt 104-2,3,(6),(7),11. It is therefore possible that pt 104-1,2,3,(6),(7),11 arose in New Zealand through a mutational event independent of that/those occurring in Australia. Pt 104-1,2,3,(6),(7),11 was detected at Salmon Gums, WA, in September 1990, providing another example of east to west movement of rust inoculum within Australia, and pointing to the likely future occurrence of wheat stripe rust in that state. At their nearest points, Regions 3 and 4 are separated by 1300 k of arid land. Given the predominance of pt 104-1,2,3,(6),(7),11 in Region 3 during 1990 (Table 5), it is possible that the isolate that reached Region 4 originated from Region 3. The ability of *Prt* to spread throughout the Australasian region so readily necessitates a national approach to controlling this disease.

Watson and Luig [1968] observed up to six different phenotypes in the interaction of *Sr15* and different isolates of *P. graminis* f. sp. *tritici*, and described this phenomenon as progressive increase in virulence. It is unlikely that the differences found in pathogenicity for *Lr2a, Lr13,* and *Lr26* would be significant in an agricultural context, but they are useful markers in studies of variability in *Prt*. The partial virulence of pt 104-2,3,(6),(7),11 on *Lr27 + Lr31* may result in moderate levels of rusting on adults plants, however, field tests are needed to confirm this.

The gene present in Harrier for which pt 104-2,3,(6),(7),11 was avirulent is not present in any other Australian wheat variety, and its origin is currently unknown. Greenhouse studies comparing the response of Australasian *Prt* pathotypes present prior to 1985 showed that all except pts 53-1,(6),(7),10,11 and 104-2,3,(6),(7),11 are virulent on Harrier, again supporting an exotic origin for these rusts. Variation in the response of Lasko triticale was also observed using pathotypes isolated in the United Kingdom [Jones *et al.*, 1991]. A final difference between the two patho-

types is in pathogenicity for the adult plant resistance gene *Lr12*. The frequency of virulence for this gene was between 80–100% in isolates originating from Region 1 during the period 1975–1985, and this declined to 0% by 1992 because of the displacement of older *Lr12* virulent pathotypes with the *Lr12* avirulent pt 104-2,3,(6),(7),11 [Park and McIntosh, 1994].

The introductions of pts 53-1,(6),(7),10,11 and 104-2,3,(6),(7),11 into the Australasian region during the late 1970s early 1980s were very significant for wheat growing in this region. The former pathotype is virulent for *Lr13*, a feature not previously detected in Australasia and resulting in several cultivars being rendered susceptible. An epidemic of this pathotype occurred in central Qld during 1988. *Lr13*-virulent mutants of resident *Prt* pathotypes within Australasia were not identified despite the relatively widespread use of resistant wheats with *Lr13*. The occurrence of pt 104-1,2,3,(6),(7),11 in WA in 1990 was followed by an epidemic year in 1991 when between 100,000 and 120,000 ha of wheat were sprayed with fungicide, primarily for the control of rust. In unprotected crops, yield losses of up to 37% were incurred [R. Loughman, pers. comm.]. The appearance of such pathotypes therefore have important implications for resistance breeding and plant quarantine in the Australasian region. Further studies using isozymic or molecular markers (for example, RFLPs or RAPDs) may help determine the origin of exotic pathotypes and in so doing reduce the possibility of similar introductions.

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