3. Using race survey outputs to protect wheat from rust

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Abstract

Race (pathotype) surveys of cereal rust pathogens have been conducted in many parts of the world since the early 1900s. The only way to identify rust pathotypes remains virulence testing in greenhouse tests using genotypes ("differentials") carrying different resistance genes. Virulence determinations have rarely targeted genes conferring adult plant resistance because of the technical difficulties of working with adult plants under controlled conditions. Where pathotype surveys have been conducted in a robust and relevant way, they have provided both information and pathogen isolates that underpinned rust control efforts, from gene discovery to post-release management of resistance resources. Information generated by pathotype surveys has been used to: devise breeding strategies; indicate the most relevant isolates for use in screening and breeding; define the distribution of virulence and virulence combinations; allow predictions of the effectiveness/ineffectiveness of resistance genes; and issue advance warning to growers by identifying new pathotypes (both locally evolved and introduced) before they reach levels likely to cause significant economic damage. To be most effective, pathotype surveys should also provide fully characterized isolates (defined pathotypes) for use in identifying new sources of resistance and screening breeding material. Although constrained to some extent by a lack of markers, particularly those not subject to natural selection, surveys have also provided considerable insight into the dynamics of rust pathogen populations, including the evolution and maintenance of virulence, and migration pathways, including periodic long-distance migration events.

Keywords

Avena, genetics, *Hordeum*, leaf rust, *Puccinia*, stem rust, stripe rust, *Triticum*, yellow rust

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Introduction

Formal genetic analysis of disease resistance in plants began over 100 years ago when Biffin (1905) demonstrated that resistance to stripe rust in wheat (caused by *Puccinia striiformis* f. sp. *tritici*) was inherited as a single recessive Mendelian trait. At the time of those studies, Biffin was unaware of pathogenic variability in the stripe rust pathogen. The discovery of physiologic races in the wheat stem rust pathogen (*Puccinia graminis* f. sp. *tritici*) by Stakman and Piemeisel (1917), and later in other rust pathogens of cereals, was important in demonstrating the need to monitor pathogenic variability and to use this information and the most relevant rust isolates in genetic research and resistance breeding. Unfortunately, publications that ignore the importance of pathogenic variability in rust pathogens continue to appear in the scientific literature.

While the discovery of heritable resistance to rust led to great optimism that a solution to rust diseases had been found (Watson and Butler 1984), early attempts to develop genetically resistant wheats involved the development of cultivars with single genes for resistance. Almost invariably, matching virulence in the rust pathogen followed with the cultivar being rendered susceptible in what became known as the "boom and bust" cycle. For example, the first stem rust resistant wheat released in Australia was cv. Eureka (1938), protected by the single resistance gene *Sr6*. It increased in popularity and by 1945 occupied about 18% of the wheat area in northern New South Wales and Queensland (Watson and Luig 1963). Virulence for *Sr6* was first detected in 1942, and its frequency in the *P. graminis* f. sp. *tritici* population in this region increased as the area sown to Eureka increased (Watson and Luig 1963). Many examples of such "boom and bust" cycles were documented in the years since. Similarly, the breakdown of genes such as *Sr24*, *Sr27*, *Sr36*, *SrSatu*, *Yr25* and *YrA* has had a significant impact on wheat production in South Africa (Pretorius et al. 2007a; Pretorius ZA unpublished).

Race (pathotype) Surveys

Pathotype surveys of cereal rust pathogens are conducted in many parts of the world, and typically involve identifying pathotypes present in rust samples collected from crops, volunteer (self sown) cereals, rust susceptible grass species, and experimental plots (including breeders' plots and rust trap nurseries). The only way to identify pathotypes is virulence testing in greenhouse tests using genotypes carrying different resistance genes ("differentials").

The sources of rust samples used to inoculate differential sets differ between laboratories. In some cases, single pustule isolates are established from rust samples before inoculating differential sets (e.g. North America), whereas in others, a spore suspension derived from the original field sample is used (e.g. Australia, India). The latter approach often allows detection of pathotypes present in samples at low levels, but can also confound determinations if pathotypic diversity is high and mixtures are encountered. In these instances, it is necessary to establish single pustule sub-cultures from specific differential genotypes. These are then applied individually to differential sets to determine the component pathotypes. In situations where single pustules are sampled for pathotyping, bulked spore samples from residues are used to inoculate key universally resistant genotypes as a means of detecting rare but potentially important variants.

While some studies have examined virulence for genes conferring adult plant resistance (e.g. Park and McIntosh, 1994), this has not been done on a routine basis because of the technical difficulties of working with adult plants under controlled conditions. However, Pretorius et al. (2000; 2007b) successfully tested mini-adult plants for resistance to leaf and stripe rust in controlled environments, showing that analysis of pathogenicity for adult plant resistance genes is possible.

Race surveys and pre-breeding for rust resistance

Genetic studies of host resistance

An important, but inadequately acknowledged contribution to resistance breeding made by pathotype surveys is the provision of characterized pathogen isolates for use in identifying resistance in germplasm. A comprehensive collection of well characterized rust isolates, coupled with a basic understanding of the genetics of host : pathogen interactions, are powerful tools to resolve the identities and relationships between resistance genes, and to assess the potential value of new resistance sources.

Knowing the virulence attributes of rust isolates used in genetic studies, whether they be greenhouse-based studies of seedling resistance, or field-based studies of resistance expressed at adult plant growth stages, is vital if the results obtained are to be interpreted in a meaningful way. A wheat genotype that displays resistance at adult plant growth stages in the field could carry either seedling resistance, adult plant resistance (APR), or both, and the only way to discriminate between the two types of resistance is to have some understanding of the seedling resistance genes present in the genotype and of the virulence(s) of the isolate(s) present in the field nursery. For example, an assessment of APR to stem rust in a wheat genotype carrying the seedling resistance genes *Sr24* and *Sr31* can only be made using a stem rust isolate carrying virulences for both genes (e.g. TTKST or "Ug99 +Sr24") and to which the wheat genotype is seedling-susceptible. If such an isolate is not available, a mapping population could be developed, and lines lacking either resistance gene can be identified and assessed for adult plant rust response. In the absence of such information it is impossible to relate mapping data to known rust resistance genes.

Using characterized rust isolates to identify resistance genes.

Valuable preliminary information on the genetic basis of rust resistance in cereal germplasm can be obtained using multipathotype tests in which an array of rust cultures with known pathogenicity is used for gene postulation (Loegering et al. 1971). Australian and South African pathogenicity surveys of the wheat rust pathogens have identified groups of pathotypes considered to represent closely related clonal lineages comprising step-wise mutants that differ in virulence/ avirulence for single resistance genes. These are the pathogen equivalent of near-isogenic host series carrying individual rust resistance genes in a common genetic background, and are invaluable in multipathotype testing aimed at postulating the identities of resistance genes and in recognizing potentially new resistance genes.

At least 96 loci confer resistance to *Puccinia coronata* f. sp . *avenae* (*P. c. avenae*) in oats (http://www.cdl.umn.edu/res_gene/ocr.html). A lack of single gene reference stocks for many of these genes, plus high levels of genetic diversity in the pathogen, make it very difficult to identify *Pc* genes in germplasm by multipathotype testing. In Australia, the seedling resistances of many Australian oat cultivars, all of which have been overcome by matching virulence in *P. c. avenae*, are unknown. Detailed studies of pathotypes virulent on 10 oat cultivars that were regarded as seedling resistant to *P.c. avenae* when released between 1991 and 2003 (Barcoo, Bettong, Cleanleaf, Culgoa, Graza 68, Gwydir, Moola, Nugene, Taipan and Warrego) demonstrated that they are pathogenically very similar and were likely derived via single-step mutations (Park RF unpublished). The pathotypes were characterized

extensively on host stocks, and in turn were used to resolve the identities of the resistance genes present in the cultivars. Whilst some of the 10 resistant oat cultivars were regarded as having "new" uncharacterized seedling resistances, it is now clear from the detailed comparative multipathotype studies that most possess combinations of previously characterized genes. For example, cv Cleanleaf was previously reported to carry *Pc38*, *Pc39* and an uncharacterized resistance gene (Bonnett 1996) that on the basis of multipathotype testing is now considered likely to be *Pc52* (Park RF unpublished).

Pathotypes virulent on the 10 oat cultivars have also been invaluable in identifying seedling resistance genes present in other oat germplasm. Detailed multipathotype tests of 166 lines from the 1998 and 1999 Quaker oat nurseries indicated a range of resistance genes, and it was clear that some lines carried the resistance genes present in cultivars Bettong (42 entries), Gwydir (six entries), Nugene (one entry) and Warrego (four entries) (Haque S and Park RF unpublished). These studies also permitted the field identification of 12 nursery entries lacking effective seedling resistance genes, but possessing very high levels of APR to crown rust (Haque 2004).

It is hoped that these related pathotypes will also assist in resolving the confusion surrounding many of the *Pc* genes described so far. Recently, seedling tests of known genetic stocks using a pathotype virulent for *Pc94* and a series of isolates that included the putative parent of this pathotype implicated the presence of this resistance gene in *Avena strigosa* accession CI 3815. This line was originally reported to carry *Pc19* and *Pc30* (Simons et al. 1959; Marshall and Myers 1961), and more recently, to carry five tightly linked genes, designated *Pc81–85* (Yu and Wise 2000). The genetic relationships between *Pc19*, *Pc30* and the *Pc81–85* complex are not known; however, the evidence from our tests suggests that one of these genes and *Pc94* are synonymous. Gene *Pc94* was introgressed into hexaploid oats by Aung et al. (1996) from *A. strigosa* accession RL1697. Tests of RL1697 with the *Pc94*-virulent and -avirulent *P. c. avenae* pathotypes would be a simple means of testing this hypothesis further.

Virulence associations can provide insight into the genetic basis of rust resistance. Australian isolates of *P. triticina* virulent for APR gene *Lr12* are also virulent for the complementary seedling resistance genes *Lr27* and *Lr31* (Park and McIntosh 1994). Similar associations were communicated to these authors by colleagues in South Africa, Argentina and Mexico. Because of this virulence association, and the location of both *Lr12* and *Lr31* on chromosome 4B, Park and McIntosh (1994) predicted the genes were either linked or at the same locus. Subsequent genetic analyses established that the two genes were either completely linked or the same (Singh et al. 1999). If the latter is correct, then *Lr27* acts in a complementary manner with *Lr12* in seedlings to confer resistance, but its presence is not necessary for the adult plant expression of resistance conferred by *Lr12*. This interesting genetic model was developed based on an original observation of an association between virulences for the genes *Lr12* and *Lr27*+*Lr31*, and demonstrates clearly the insight that can be gained from detailed knowledge of pathogen virulence. Furthermore, it also established that virulence on the APR gene *Lr12* can most likely be monitored in seedling-based pathogenicity assays using a differential genotype carrying *Lr27*+*Lr31*.

Pre-breeding

A comprehensive set of well characterized rust isolates permits the identification of potentially new sources of resistance (see example in preceding section on crown rust resistance in oat genotype CI 3815), and also allows an assessment of the effectiveness of new rust resistance genes to local pathotypes prior to their use. For example, although the resistance genes *Lr3ka*, *Lr15* and *Lr41* (leaf rust resistance in wheat), *Sr8b* and *Sr35* (stem rust resistance in wheat), *Yr8* (stripe rust resistance in wheat), and *Rph5*, *Rph6*, *Rph10* and *Rph13* (leaf rust resistance in barley), and *Pc92* and *Pc94* (crown rust resistance in oats) have never been deployed in Australia, virulence to all existed in at least one isolate maintained in an historical cereal rust collection compiled over the past 80 years of pathotype surveys. While this suggests that such resistance genes may not be durable if deployed, where the frequency of virulence is low (e.g. *Lr41*, *Sr35*, *Rph13* and *Pc94*), the existence of such pathogen isolates provides a means of selecting lines carrying these genes with other effective resistance genes.

The Australian Cereal Rust Control Program (ACRCP) at the University of Sydney undertakes "parent building", in which key wheat genotypes (selected to represent the current range of maturity and quality classes) are used as recurrent parents into which new rust resistance genes are backcrossed and then distributed to breeding groups as locally adapted donor sources. In these cases, choice of pathotype to select individual backcross plants from the BC_2 generation onwards is crucial in ensuring the target gene is selected.

Race surveys and breeding for rust resistance

Disease resistance breeding strategies

The recognition of mutation as a major source of variability in wheat rust pathogens led to the development of both gene combinations (Watson and Singh 1952; aka "gene stacking" or "gene pyramiding") and pre-emptive or anticipatory breeding (McIntosh and Brown 1997).

The former strategy assumes that mutation events are independent, and therefore that the frequency of simultaneous mutations for virulence to more than one resistance gene will be extremely low. In a dikaryotic organism in which mutation to virulence may have to occur in two nuclei, the probability is even lower. Following a series of "boom and bust" cycles in northern NSW and Queensland, Luig and Watson (1970) stated that "During the past 15 years, it has become abundantly clear that cultivars with single genes for resistance to stem rust are of limited value in Region 1". Whereas good molecular markers add precision to selection and make it easier to combine resistance genes, breeders had success in the past in combining multiple effective rust resistance genes without such markers. For example, the genes *Sr24* and *Sr26* were combined in the Australian wheat cultivar Sunelg, released over 20 years ago, and *Sr24* and *Sr38* were combined to produce cultivars QAL2000 and QALBis. In both cases, the combinations were produced in the absence of cultures virulent for either gene and of linked molecular markers. In the latter example, seedling stem rust tests using a culture avirulent for both genes allowed reliable identification of lines combining the genes because the genes interact to confer an infection type lower than that produced by the two genes individually (Brown GN unpublished). Similarly, many combinations involving the durable but recessive adult plant resistance gene *Sr2* have been assembled by utilising the linked traits pseudo black chaff and seedling chlorosis (Brown 1997; McIntosh et al. 1995). The linkages of *Lr24* with *Sr24*, and of *Lr37* and *Yr17* with *Sr38* (McIntosh et al. 1995) allowed selection of the stem rust resistance component in the presence of other stem rust genes using the completely linked leaf rust or stripe rust genes as "markers".

Anticipatory breeding is based on the premise that future mutations in a pathogen can be predicted (McIntosh and Brown 1997). Monitoring virulence in rust pathogen populations allows predictions of the effectiveness/ ineffectiveness of resistance genes, and in so doing, provides direction for breeders. For example, the resistance gene *Lr24* remained effective in Australia from 1983 until virulence was detected in a single pathotype in South Australia in 2000 (Park et al. 2002). In contrast, *Lr24* was either ineffective or overcome not long after its deployment in the USA (Long et al. 2000), Canada (Kolmer 1998), and South Africa (Pretorius et al. 1987). The rapid development of *Lr24* virulence in these countries was a clear indication of the potential for this to occur in Australia. Pathotype surveys of *P. triticina* during this period monitored virulence for *Lr24* and the frequency of this gene in breeding populations and wheat cultivars. At least 28 cultivars with *Lr24* were released in Australia following the release of Torres in 1983, and by 1993, the area sown to cultivars with *Lr24* was about 45% in Queensland and 35% in New South Wales (Brown 1994). The reason(s) why virulence for *Lr24* was not detected in Australia for 17 years after the gene was first deployed, although not entirely clear, likely relate in part to its initial deployment in northern NSW and Queensland where leaf rust inoculum levels were low, the result of strict adherence to rust resistance standards in this region from the 1970s onwards (Platz and Sheppard 2007; Wallwork 2007). In this region, fewer mutational events would be expected in situations of small pathogen population size. The initial detection of virulence for *Lr24* in southern regions is consistent with this theory, because leaf rust was at relatively high levels in this region from the 1990s (Park et al. 2002). The presence of *Lr34* in addition to *Lr24* in some cultivars deployed in the north (Singh et al. 2007) also likely contributed to its effectiveness in this region.

Rust resistance screening

Developing germplasm with resistance to rust usually involves screening with rust isolates in either field nurseries and/or the greenhouse. This is most effective, and the resistances selected are most useful when isolates of greatest relevance to the target agricultural production system are used. Pathotype surveys play a central role in this process by providing information on the frequencies and distributions of pathotypes, and the isolates for use in germplasm screening. However, the effectiveness of pathotype surveys in achieving this is determined largely by the differential genotypes used to identify pathotypes. Resistance genes deployed in commercial cultivars should be represented in the differential sets used for pathotype determinations to ensure the relevance of information to breeding programs. Because the resistance genes in use differ between geographical regions, the composition of differential sets will also differ if pathotype surveys are to be relevant to local conditions.

The ACRCP provides greenhouse seedling and field adult plant screening services to all cereal breeding groups in Australia. The pathotypes used to screen breeding material are selected based on their

current relevance and their virulence combinations - knowledge gained from pathotype surveys. Field rust nurseries typically use from 1 to 3 key pathotypes of each species, which are selectively encouraged using cereal genotypes specific to each (e.g. a mixture of wheat genotypes Worrakatta (*Lr24*), Sunstar (*Lr13*) and Marombi (*Lr37*) to promote pathotypes with virulence for each gene individually). A similar service is provided in South Africa where the University of the Free State tests commercial varieties and elite breeding lines with stategic pathotypes on an annual basis.

Long term pathotype surveys have shown that the composition of rust pathogen populations vary enormously with time (see section below). This has at times included situations where new or previously rare genes for avirulence have been introduced and increased in frequency. The "Mackellar" *P. triticina* pathotype, first detected in Victoria in 2004, was considered to be of exotic origin because of more than 5 pathogenic differences from local *P. triticina* pathotypes (Park RF unpublished). Of interest was avirulence for the wheat genotype Morocco (since found to carry a resistance gene located on chromosome 2BS) and a range of Australian wheat cultivars including Halberd, Avocet, Tarsa and Tincurrin (Park RF and Singh D unpublished). Knowledge of this avirulence is very important to avoid inadvertent selection for the gene, which is of limited or no use in resistance breeding because of virulence in virtually all other Australian *P. triticina* pathotypes. Similarly, from 1979 to 2002, virtually all pathotypes of the wheat stripe rust pathogen identified in race surveys were virulent for the gene *Yr3* (Wellings 2007). However, the introduction of a new pathotype in 2002, avirulent for *Yr3* was followed by a rapid decline in virulence for these genes (Wellings 2007). Despite this, selection and deployment of *Yr3* would not be advisable, and its presence in breeding populations is now being monitored, based on knowledge and rust isolates generated by pathogenicity surveys.

Post-breeding management of rust resistance

Whereas most emphasis is placed on identifying, characterizing, and incorporating rust resistance into cereal cultivars, the attention given to post-release management of rust resistant cultivars is unfortunately often much less. An understanding of the resistance genes present in commercial cereal cultivars is important in allowing the risk and implications of resistance gene breakdown to be assessed and managed.

Predicting cultivar vulnerability to mutational change

The wheat cultivar Oxley carrying the stem rust resistance genes *Sr5*, *Sr6*, *Sr8a* and *Sr12*, occupied between 7 and 9% of the area in northern New South Wales and Queensland from 1976 to 1985, despite being susceptible to stem rust pathotype 343-1,2,3,5,6 (Zwer et al. 1992). At that time, a significant proportion of the wheat crop in this region comprised the cultivars Cook (*Sr5*, *Sr6*, *Sr8a*, *Sr36*), Songlen (*Sr2*, *Sr5*, *Sr6*, *Sr8a*, *Sr36*) and Timgalen (*Sr5*, *Sr6*, *Sr8a*, *Sr36*). Watson (1981) warned of the vulnerability of the latter cultivars to mutational change in the Oxley-attacking pathotype, which only had to acquire virulence for *Sr36* to render all sufficiently susceptible to suffer yield losses. Pathotype 343-1,2,3,4,5,6, regarded as a single-step mutational derivative of the Oxley attacking pathotype with virulence for *Sr36*, was duly detected in 1984, after which the area sown to these cultivars declined (Zwer et al. 1992).

Monitoring rust pathogen populations

Rust samples used for pathotype surveys can come from a range of sources, including experimental plots and commercial cereal crops. Where resources do not exist to undertake structured sampling from commercial crops over large areas (e.g. transport), important information on virulence and virulence combinations in rust pathogen populations can nonetheless be gained from undertaking pathotype analyses on samples collected from plots at experimental field sites.

Structured race surveys in which samples are collected across a large area from commercial crops or in areas where cereals are grown should include the collection of information on rust incidence (i.e. surveillance). Such information is important in identifying potential build-up of inoculum. In Australia and South Africa, monitoring the incidence of rust diseases between cropping cycles during the summer period is an important component of rust management. During wet summers, volunteer (self sown) cereals can establish and give rise to "green bridges" that provide opportunities for rusts to build up. In such situations, extension is important to notify the agricultural community of the importance of green bridge destruction in minimising opportunities for oversummering of rust pathogens. This knowledge also provides some advance warning of potential rust build-up, which can be used to advise growers of the need to consider seed treatment and/or advance purchase of fungicides. It is also of relevance to chemical suppliers, who may need to make decisions on whether to stockpile chemicals well in advance of a given production cycle.

Surveillance and pathotype information combined provide an understanding of migration pathways and the distribution of rust pathotypes, which in turn allow assessment of the regional effectiveness of resistance genes and the resistance status of cereal cultivars. In Australia, the eastern and western cereal belts are separated by ca. 1,500 km of desert and long-term race surveys have provided clear evidence of inoculum exchange between the two regions. These surveys have however indicated that this exchange occurs mainly west-to-east on prevailing winds; for example, since 1988 there have been seven incidences of inoculum exchange from west to east, but only one instance where this has occurred in the opposite direction. The appearance of new pathotypes of *P. striiformis* f. sp. *tritici* and *P. triticina* in eastern wheat areas over the past 20 to 30 years in particular, via exotic incursion or local mutation, has resulted in the presence of many virulences that were not recorded in the west (e.g. *Lr13*, *Lr24*, *Yr3*, *Yr4*, *Yr17, Yr27*), and these genes therefore remain important in protecting the Western Australian wheat crop from the respective diseases.

Early warning

The early detection of a new pathotype allows advance warnings to be issued to growers, especially when such pathotypes are identified and characterized before they reach levels likely to cause significant economic damage. To be most effective, early warnings should include an assessment of the risk of a new pathotype to all currently grown cultivars. This can be achieved by initial characterization of the virulence attributes of the pathotype, followed by seedling and eventually adult plant field based tests of cereal cultivars with the new pathotype. For example, virulence for the resistance gene *Yr27* in eastern Australia was detected in late 2008, and at that time, it was known that three wheat cultivars carried this resistance gene. Detailed comparative greenhouse seedling tests of all Australian wheat cultivars with the new pathotype provided not only confirmation of the occurrence of *Yr27* in these three cultivars, but also an indication of a lack of additional effective seedling resistance in all three (Wellings CR unpublished). Since then, extension bulletins have been prepared and distributed to the farming community. The next step will be to establish the new pathotype in field rust nurseries in 2009, to assess whether these cultivars carry residual APR and also to determine the impact of this mutational change on breeding populations.

Rust pathogen population dynamics

Although constrained to some extent by a lack of markers, particularly those not subject to natural selection, surveys have also provided considerable insight into the dynamics of rust pathogen populations, including the evolution and maintenance of virulence, and migration pathways, including periodic long-distance migration events. Because of the isolation of the Australian continent from other cereal-growing regions of the world, the long term surveys of pathogenic variability in *P. graminis* f. sp. *tritici* and *P. triticina* in particular have provided rare insights into rust population dynamics and the processes that generate variability in asexually reproducing pathogen populations. Combined, the survey data strongly implicate periodic introduction of exotic pathotypes, single-step mutation, and more rarely, somatic hybridization, as the major determinants of cereal rust population structure in Australia. All three processes were observed in pathogenicity surveys of *P. triticina* between 1980 and 2005 (Park et al. 1995, 1999). Exotic rust incursions are equally important in South Africa as exemplified in recent years by the introduction of *P. striiformis tritici* pathotype 6E16A- in 1996 (Pretorius et al. 1997) and *P. graminis* f. sp. *tritici* pathotype TTKSF in 2000 (Visser et al. 2009).

Rust pathogen populations vary enormously over time with wide shifts in virulence occurring, often for no apparent reason. In Australia, there have been four instances in which an exotic incursion led to a complete change in the local wheat rust pathogen population: viz. *P. graminis* f. sp. *tritici* race 126 in 1925; *P. graminis* f. sp. *tritici* race 21 in 1954; *P.triticina* race 104 in 1984; and *P. striiformis* f. sp. *tritici* race 134 in 2002. Presumably in each case, the new pathogen genotype was more aggressive and better able to compete, survive and build up, features that also appear to characterize "Ug99".

Future directions in pathogenicity surveys

Australian pathotype surveys of wheat rust pathogens over the past 80 years have clearly shown an increase in the frequency of exotic incursions with time, possibly a consequence of increased international movement of people. The origins of most of these incursions are unknown. The advent of remote sensing and of GIS technologies have provided additional tools for rust surveillance, that in conjunction with pathotype analysis, should provide increased understanding of intercontinental long-distance movement of rust pathogens. The application of new DNA-based marker

systems to study rust pathogen variability will allow more critical appraisals of the role of mutation in generating variability, and also provide insight into global variability in these pathogens. Central to all of this work, and fundamental to sustained genetic control of rust pathogens, will be the need for relevant and informed pathogenicity surveys. Any future increase in restrictions on the movement of biological material will further highlight the need to develop in-country pathotype analysis capabilities, both in terms of infrastructure and skilled personnel.

Acknowledgements

The authors thank the Durable Rust Resistance in Wheat project for making funds available for participation in the meeting at which this paper was presented. RFP acknowledges the financial support of the Australian Grains Research and Development Corporation.

References

Aung T, Chong J, Leggett M (1996) The transfer of crown rust resistance gene *Pc94* from a wild diploid to cultivated hexaploid oat. Cereal Rusts and Powdery Mildews Bull 24 (supplement): 167-171

Biffin R.H. (1905) Mendel's laws of inheritance and wheat breeding. J Agric Sci 1:4-48

Bonnett DG (1996) Host : pathogen studies of oat leaf rust in Australia. PhD Thesis, University of Sydney. 240 pp

Brown GN (1994) Diversity of resistance to wheat rusts in Australian wheats in 1993-94. In: Paull J, Dundas IS, Shepherd KJ, Hollamby GJ (eds) Wheat Breeding Society of Australia 7th Conference, 25-30 September 1994, Adelaide) pp. 199-203

Brown GN (1997) The inheritance and expression of leaf chlorosis associated with gene *Sr2* for adult plant resistance to wheat stem rust. Euphytica 95: 67-71

Haque MS (2004) Rust resistance in oats and variability in the oat stem rust pathogen *Puccinia graminis* f. sp. *avenae*. PhD Thesis, The University of Sydney. 269pp

Kolmer JA (1998) Physiologic specialization of *Puccinia recondita* f.sp. *tritici* in Canada in 1996. Can J Plant Pathol 20:176-181

Loegering WQ, McIntosh RA, Burton CH (1971) Computer analysis of disease data to derive hypothetical genotypes for reaction of host varieties to pathogens. Can J Genet Cytol 13:742-748

Long DL, Leonard KJ, Hughes ME (2000) Virulence of *Puccinia triticina* on wheat in the United States from 1996 to 1998. Plant Disease 84:1334-1341

Luig NH, Watson IA (1970) The effect of complex genetic resistance in wheat on the variability of *Puccinia graminis* f. sp. *tritici*. Proc Linn Soc NSW 95:22-45

Marshall HG, Myers WM (1961) A cytogenetic study of certain interspecific *Avena* hybrids and the inheritance of resistance in diploid and tetraploid varieties to races of crown rust. Crop Sci 1:29-34

McIntosh RA, Brown GN (1997) Anticipatory breeding for resistance to rust diseases in wheat. Annu Rev Phytopathol 35:311-326

McIntosh RA, Wellings CR, Park RF (1995) Wheat rusts. An atlas of resistance genes. CSIRO, Melbourne: 200pp

Park RF, McIntosh RA (1994) Adult plant resistances to *Puccinia recondita* f. sp. *tritici* in wheat. NZ J Crop & Hort Sci 22:151-158

Park RF, Bariana HS, Wellings CR, Wallwork H (2002) Detection and occurrence of a new pathotype of *Puccinia triticina* with virulence for *Lr24* in Australia. Aust J Agric Res 53:1-8

Park RF, Burdon JJ, McIntosh RA (1995) Studies on the origin, spread, and evolution of an important group of *Puccinia recondita* f. sp. *tritici* pathotypes in Australasia. Eur J Plant Pathol 101:613-622

Park RF, Burdon JJ, Jahoor A (1999) Evidence for somatic hybridisation in the leaf rust pathogen of wheat (*Puccinia recondita* f. sp. *tritici*). Mycol Res 103:715-723

Platz GJ, Sheppard JA (2007) Sustained genetic control of wheat rust diseases in north-eastern Australia. Aust J Agric Res 58:854-857

Pretorius ZA, Rijkenberg FHJ, Wilcoxson RD (1987) Occurrence and pathogenicity of *Puccinia recondita* f.sp. *tritici* on wheat in South Africa from 1983 through 1985. Plant Disease 71:1133-1137

Pretorius ZA, Park RF, Wellings CR (2000) An accelerated method for evaluating adult-plant resistance to leaf and stripe rust in spring wheat. Acta Phyto Entomol Hungarica 35:359-364

Pretorius ZA, Pakendorf KW, Marais GF, Prins R, Komen JS (2007a) Challenges for sustainable control of cereal rust diseases in South Africa. Aust J Agric Research 58: 593-601

Pretorius ZA, Pienaar L, Prins R (2007b) Greenhouse and field assessment of adult plant resistance to stripe rust of wheat. Aust Plant Pathol 36: 552-559

Simons M, Sadanaga K, Murphy HC (1959) Inheritance of resistance to strains of diploid and tetraploid species of oats to races of the crown rust fungus. Phytopathology 49: 257-259

Singh D, Park RF, McIntosh R A (1999) Genetic relationship between the adult plant resistance gene *Lr12* and the complementary gene *Lr31* for seedling resistance to leaf rust in common wheat. Plant Pathology 48:567-573

Singh D, Park, RF, and McIntosh RA (2007) Characterisation of wheat leaf rust resistance gene *Lr34* in Australian wheats using components of resistance and the linked molecular marker csLV34. Australian Journal of Agricultural Research 58: 1106-1114

Stakman EC, Piemeisel FJ (1917) Biologic forms of *Puccinia graminis* on cereals and grasses. J Agric Res 10:429-495

Visser B, Herselman L, Pretorius ZA (2009) Genetic comparison of Ug99 with selected South African races of *Puccinia graminis* f. sp. *tritici*. Mol Plant Pathol 10:213-222

Wallwork H (2007) The role of minimum disease standards for the control of cereal diseases. Aust J Agric Res 58:588-592

Watson IA (1981) Wheat and its rust parasites in Australia. In: Evans LT, Peacock WJ (eds) Wheat science - today and tomorrow) Cambridge University Press, Cambridge, UK, pp129-147

Watson IA, Butler FC (1984) Wheat rust control in Australia. National conferences and other initiatives and developments. The University of Sydney, Sydney, Australia

Watson IA, Luig NH (1963) The classification of *Puccinia graminis* var. *tritici* in relation to breeding resistant varieties. Proc Linn Soc NSW 88:235-258

Watson IA, Singh D (1952) The future for rust resistant wheat in Australia. J Aust Inst Agric Sci 18:190-197

Wellings CR (2007) *Puccinia striiformis* in Australia: a review of the incursion, evolution, and adaptation of stripe rust in the period 1979–2006. Aust J Agric Res 58:567-575

Yu GX, Wise RP (2000) An anchored AFLP- and retrotransposon-based map of diploid *Avena*. Genome 43:736-749

Zwer PZ, Park RF, McIntosh RA (1992) Wheat stem rust in Australia 1969 - 1985. Aust J Agric Res 43:399-431