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ANTICIPATORY BREEDING FOR RESISTANCE TO RUST DISEASES IN WHEAT

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ABSTRACT

Anticipatory resistance breeding is the process of predicting future pathotypes and producing resistant germplasm to avert future losses. It is made possible by a national pathotype surveillance program and knowledge that new pathotypes arise predominantly from mutation in existing pathotypes. This is supported by genetic analyses to catalogue the identity and distribution of resistance genes in current cultivars. A national germplasm enhancement program ensuring that both currently effective and potentially new sources of resistance are available in a wide range of adapted genotypes enables rapid cultivar replacement before or soon after the occurrence of new pathotypes. The policy of recommending only rust-resistant cultivars in the more rust-prone areas has resulted in significant reductions in pathogen population size and variability. With increased and more rapid international human travel and transport, there is an increased threat of exotic pathotypes, the effects of which are more difficult to predict. As the frequency and magnitude of epidemics decline, public awareness programs will be required to achieve and maintain the use of rust resistance by the entire wheat industry.

Anticipatory resistance breeding is the process of breeding for resistance to virulent pathotypes before such pathotypes become prevalent and cause significant losses. A program of anticipatory resistance breeding has the following prerequisites:

A knowledge of the epidemiology of the pathogen across the target region. In the case of the airborne cereal rust pathogens this is usually a large area, often covering a number of countries.

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Relevant annual pathogen surveys aimed at detecting new pathotypes with potential to overcome the resistance genes that are deployed. In many cases there is a period of years from pathotype detection to significant crop losses during which alternative cultivars can be promoted, or the final selection, seed increase and recommendation of replacement cultivars can occur.

A knowledge of the main resistance genes that are deployed in current cultivars grown throughout the target region. This requires ongoing genetic research to identify and catalogue resistance genes using the standard methods of cultivar pedigree, low infection type to avirulent pathotypes, responses to pathotype arrays and, where necessary, genetic analysis.

A well co-ordinated system for screening all breeding materials with pathotypes posing the greatest threat, for identifying new sources (genes) of resistance to those pathotypes and, where appropriate, for undertaking the prebreeding often required to have those resistance sources available in locally adapted germplasm.

This paper reviews these prerequisites as they relate to breeding for resistance to the wheat rusts in Australia. The continent of Australia represents a land area approximately the same size as mainland USA and has advantages that simplify an anticipatory breeding strategy. These include physical isolation from other wheat-growing areas, political uniformity allowing pathogen sampling throughout the entire epidemiologic area, and absence of alternate hosts that permit overwinter survival and sexual recombination. In addition, long-term pathogen survey/host genetic analysis programs have provided a substantial basic knowledge of the pathogenic capabilities of the three wheat rust pathogen species and of the main host resistance genes that have been, and are being, deployed in wheat cultivars.

Historic Overview

The Australian wheat crop has been threatened by stem rust virtually since European settlement in the late eighteenth century. Attempts to control stem rust (and leaf rust) by the use of genetically determined resistance were started by William Farrer in the 1880s, but widely accepted resistant cultivars did not become available until the early 1940s. When resistant cultivars were first released to farmers, they were often grown next to susceptible cultivars. In these cases, the resistant cultivars were subjected to the sometimes substantial uredial populations produced by susceptible wheats. Consequently, they acted as screening populations for any mutant uredospore or infrequent pathotype that possessed a virulence gene(s) corresponding to the resistance gene(s) deployed in the resistant cultivar. Rowell & Roelfs (32) estimated that 0.4 ha of susceptible wheat at 10% stem-rust severity could produce 10^{12} uredospores/day. Parlevliet & Zadoks (29) similarly estimated that 1 ha of susceptible wheat with 1% infection of leaf rust could produce 10^{11} uredospores/day. With a mutation frequency of 1×10^{-5} to 1×10^{-6}, and assuming heterozygosity of the dikaryotic pathogen, there was potential for production of many mutant

spores (phenotypes) each day. It is not surprising, therefore, that wheats with single resistance genes gave only ephemeral protection that lasted until the occurrence and selection of a new mutant pathotype, or the increase of a rare virulent pathotype that was already present in the pathogen populations.

After experiencing rapid loss of protection offered by single resistance genes, Watson & Singh (43) proposed that combinations of effective resistance genes would provide longer-lasting protection as this would require pathogen genotypes to undergo multiple simultaneous changes in order to become virulent. The ability of the breeder to construct the desired resistance gene combinations is, however, dependent on a sound knowledge of the incompatible phenotypes (low infection types) produced by each resistance gene and of their interactions, coupled with the availability of pathogen cultures that permit identification of genotypes to obviate time-consuming progeny testing or test crossing. About the same time, it was realized that some resistance genes (and gene combinations) provided longer-lasting resistance than others. To describe this phenomenon, Johnson & Law (7; see also 6) later introduced the concept of durable resistance. A durable resistance was a resistance source that remained effective when used over a wide area for a considerable period of time.

From the 1960s, breeding for stem rust resistance progressed down the pathway of attempting to assemble and deploy multiple gene resistances, a strategy that was combined with the use of now recognized sources of durable resistance, including *Sr26* introgressed to wheat from *Agropyron elongatum* (10, 23) and *Sr2*, the classical Hope/H-44 adult plant resistance transferred from tetraploid Yaraslav emmer to hexaploid common wheat in the 1920s (11, 18, 23, 36).

With the availability of more sources of resistance, and a firm basis for funding basic research on both the pathogen and host, there was a gradual increase in the number of resistant cultivars, and farmers were encouraged to grow only stem rust-resistant wheats. Eventually it became possible for extension agencies to recommend only rust-resistant cultivars for the stem rust-prone summer rainfall areas of northern New South Wales and Queensland where previously the likelihood of significant crop losses was about one year in four.

The last major stem rust epidemic in wheat in Australia, in 1973, was centered on the southeast (Figure 1), where the probability of epidemics was much lower than in the northern areas. A sequence of wetter than average years permitted widespread survival of inoculum that caused an epidemic resulting in massive losses to a crop that was largely unprotected by resistance. By contrast, wheat farmers in the northern areas were unaffected, and in fact have experienced little loss due to stem rust since the early 1960s, because of the widespread use of resistance.

A consequence of the 1973 stem rust epidemic was the establishment of a National Wheat Rust Control Program (NWRCP), which expanded the University of Sydney group to offer screening assistance and advice to all

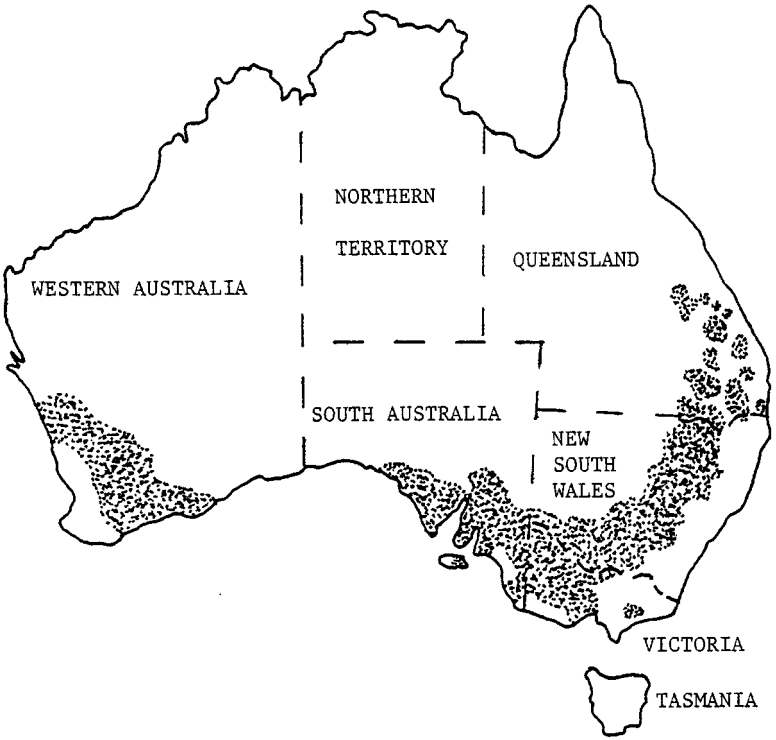

Figure 1 The states and winter cereal-producing areas (*shaded*) of Australia.

wheat-breeding programs in Australia. As a result, the deployment of rust-resistant cultivars in northern areas has strengthened and the use of rust resistance in the southeast and western wheat areas has increased. More importantly, germplasm enhancement based mainly on backcrossing, a responsibility of the NWRCP, has provided a wide range of adapted resistant germplasm covering all quality types, which, although often not released as cultivars, could be mobilized and used in a relatively short time should a major rust threat arise.

The NWRCP also made a major contribution to increased resistance to the other rust diseases. Leaf rust resistance is considered prerequisite to the release of wheat cultivars in the northeast wheat-growing areas, and resistance or moderate resistance is a common attribute of cultivars released in other areas. A major epidemic of leaf rust in Western Australia in 1992 and a moderate resurgence in 1996 suggest a need to improve resistance levels in wheats grown in that state as well as the southeast.

Wheat stripe rust is a relatively recent disease in Australia, having been introduced in 1979 (24). With its rapid spread and establishment on susceptible wheats throughout southeastern Australia (and New Zealand), much of the material in breeding programs and the germplasm enhancement program of the NWRCP had to be discarded. Fortuitously, some material was also resistant or moderately resistant. The facilities available to the NWRCP were upgraded to provide the cooler greenhouse conditions required for stripe rust research, and rapid progress was made in the development of resistant cultivars. All wheats currently recommended for cultivation in eastern Australia are at least moderately resistant to stripe rust as adult plants, and under most conditions do not require additional protection with fungicides.

Finally, considerable progress has been made to develop alternative genotypes to replace certain nonrecommended "niche" cultivars with undesirable levels of susceptibility to one or more of the rusts. For example, rust-resistant wheats for hay and chaff production are now available. The only significantly rusted crop found in the 1996 rust survey of eastern Australia was a stripe-rusted crop of cultivar Ford, which was released to farmers in 1916 (17) but which remains favored as a hay crop. Rust-resistant backcross derivatives of Ford are available to farmers who wish to grow this type of wheat.

The Effects of Rust Resistance on Pathogen Populations

The national wheat rust survey in Australia comprises rust samples provided by farmers, extension workers, breeders, and annual survey trips undertaken by Plant Breeding Institute staff. Trap plots for rust detection were abandoned in the 1960s when it was shown that they contributed no additional information. On regular surveys, random crops are inspected at approximately 20-km intervals.

Where adequate samples are available, uredospore suspensions from collections are inoculated directly to seedlings of differential sets comprising single gene lines, traditional tester genotypes, and currently resistant cultivars to identify pathotypes. Samples with insufficient inoculum for direct use are first increased on susceptible genotypes. Unlike other laboratories, single-pustule isolations are not initially taken for pathotyping. If there is evidence of pathotype mixtures that cannot be identified on the differential sets, single pustules may be sampled from selected host genotypes for increase and further identification on full differential sets.

Until the 1980s, location of stem-rusted or leaf-rusted plants was not difficult and an annual survey covering 2000 to 4000 km was likely to yield several hundred samples originating from crops, stubble regrowth areas, and from wheat plants growing as weeds in other crops or along roadsides. Because of the current widespread use of resistance, stem rust is seldom found in any

of these situations and that found on barley or grasses is usually identified as other forms, e.g. *P. graminis* f. sp. *secalis*, and somatic hybrids of *P. graminis* f. sp. *tritici* and *P. graminis* f. sp. *secalis* that are avirulent on most wheat cultivars (3, 14, 15). Currently, stem rust samples are more likely to come from research stations where susceptible plants may be present and may carry inoculum between seasons.

Simultaneous with the dramatic decline in rust samples, reflecting the much reduced pathogen population, has been an apparent decline in pathogen variability (19).

During 1982 to 1984, there were damaging epidemics caused by the wheat stem rust pathogen on triticale in northern New South Wales and Queensland. The two pathotypes involved sequentially overcame genes *Sr27* and *SrSatu*, which were present singly in triticale cultivars grown at that time (21–23). These pathotypes were incapable of attacking almost all the wheat cultivars grown throughout the region. As a consequence, triticale virtually disappeared from the region, despite the fact that other cultivars with adequate resistance to the "triticale" pathotypes were available. A significant part of these changes was due to new national grain marketing policies and an improved economic outlook for wheat rather than perceived disease vulnerability. These epidemics provided a valuable illustration of the benefits of resistant cultivars to farmers who had not seen a damaging rust epidemic for 20 years or more.

Breeding for leaf rust resistance has been an important secondary objective for the northeastern wheat-producing areas over many years. Losses to leaf rust in Australia were estimated to be at least as high as 30% under experimental conditions (8, 31) but there has not been a perception of significant losses to this disease among farmers, and most commonly, losses would be 10–15% or less (33, 48). As with the stem rust pathogen, there were large and variable *P. recondita tritici* populations until the 1980s. Single genes for resistance were used and were overcome sequentially by the pathogen. Currently, all recommended cultivars in the region have high levels of resistance. This was made possible by the widespread use of *Lr13, Lr24, Lr37,* and probably *Lr34*. The first three of these genes were consciously selected, whereas *Lr34* seems to have been selected passively for its slow rusting attributes or, more recently, through its genetic association with the stripe rust resistance gene *Yr18* (20, 37). Both genes are widely dispersed in CIMMYT-developed, South American, and North American wheat germplasm (12). More recently, D Singh (unpublished) at this laboratory showed that wheat cultivars with *Lr34* are less likely to carry rust through the mild Australian winters and that wheats with *Lr34* have a delayed and reduced rate of leaf rust increase with rising spring temperatures. This attribute of wheats such as Cook, Condor, Oxley, and Sunstar, considered earlier to be susceptible because of relatively high terminal leaf rust levels, has

probably contributed to much reduced carryover of inoculum, reduced rate of rusting and low, if any, yield losses in contrast to wheats such as Gabo and derivatives, and Spica and other Hope derivatives, cultivated in the area in the 1940s, 1950s, and 1960s. These groups possessed genes *Lr23* and *Lr14a*, which became ineffective soon after initial deployment.

Gene *Lr13* has provided stable resistance in Australia for more than 20 years. No mutant pathotype with virulence for *Lr13* was detected in surveys, despite knowledge that this gene was ineffective in South Africa (where it was present, for example, in Inia 66, along with *Lr17* and probably *Lr14a*), in western Europe (e.g. Maris Huntsman), and in Mexico and South America (38). In 1984, pathotype 53-1,(6),(7),10,11 was introduced from New Zealand where it had become prevalent on cultivar Karamu (synonyms WW15, Anza), which carries *Lr34* (37) and is heterogeneous for the presence of *Lr13*. In the Australian northeastern wheat areas, this pathotype was isolated most frequently from cultivar Sunstar. It possesses virulence on seedlings and adult plants with *Lr13*. Despite its wide occurrence on Sunstar, this otherwise widely avirulent pathotype has not mutated to accumulate additional genes for virulence. Consequently, combinations of *Lr13* and any of several earlier "defeated" genes, such as *Lr1*, all *Lr2* alleles, *Lr3*, *Lr14a*, *Lr23*, and adult resistance gene *Lr22b*, provide protection against all Australian *P. recondita* f. sp. *tritici* pathotypes. Of these combinations, *Lr1* and *Lr13* are present in Hartog (= Pavon 'S') and Suneca. Genes *Lr24* and *Lr37* are deployed in a range of other wheats. These genotypes must be considered minimal as a number of cultivars have, in addition, uncatalogued genes for adult plant resistance.

From 1990, a widely avirulent pathotype designated locally as 64-11 was repeatedly isolated from hybrid cultivars that carry *Lr1* and *Lr13* in the heterozygous condition (26). Pt 64-11 is virulent for *Lr1* and has an intermediate level of pathogenicity on wheat seedlings with *Lr13*. Apparently, this intermediate level of pathogenicity is sufficient to cause significantly increased rusting on the heterozygous hybrids compared with other wheats with *Lr13* in the homozygous condition. Recent studies by RF Park and others (unpublished) using both pathogenicity and isozyme analyses provided evidence that this pathotype is a somatic hybrid between pt 53-1,(6),(7),10,11 and 104-2,3,(6),(7),11.

Pathotype 104-2,3,(6),(7),11 was introduced to Australia before or during 1984, after which it and a derivative pathotype with virulence for *Lr20* rapidly increased in frequency, subsequently spreading over the entire continent to become the predominant pathotypes (27). Despite its apparent similarity with the previously predominant pt 104-2,3,6,(7) based on the pathogenicity formula, these two pathotypes differed in several detailed pathogenicity attributes as well as isozymically (27). The mutant derivative, 104-1,2,3,(6),(7),11, has become widely established, perhaps being selected in southern Australia on wheats

with *Lr20*. Pathotype 104-1,2,3,(6),(7),11 was involved in a major leaf rust epidemic in Western Australia in 1992 and again in 1996 was present at high levels following two years at very low incidence. A number of wheats grown in Western Australia are susceptible to leaf rust.

Following its introduction to Australia in 1979, stripe rust became widely established in the Australian southeast and in New Zealand, but has not moved to Western Australia, in contrast to the rapid spread of *P. recondita* f. sp. *tritici* pathotype 104-1,2,3,(6),(7),11. A range of putative stepwise single-locus mutations of the original introduction to Australia was detected in both Australia and New Zealand (28, 44, 46). Steele & Dickinson (39) reported molecular variation among 19 Australian isolates, indicating either local evolution of molecular variation or further introduction events. Selection of some pathotypes, such as those virulent for *YrA*, *Yr6*, and *Yr7* in Australia, and those virulent for *YrA*, *Yr6*, *Yr7*, *Yr9*, and *Yr14* in New Zealand, was possibly enhanced by selection on host genotypes possessing the relevant resistance gene(s). On the other hand, variants virulent for *Yr5*, *Yr8*, *YrSp* (uncatalogued gene present in Spaldings Prolific), and *YrSk* (uncatalogued gene present in Selkirk wheat and various CIMMYT-derived lines) have been detected despite the absence of these genes in commercial cultivars. Two further pathogenic changes are of significance. First, *P. striiformis* f. sp. *tritici* in Australia has acquired virulence for a resistance gene(s) that is widely dispersed in the barley grass (*Hordeum leporinum* complex) population (45). Second, since 1988 there has been a gradual increase in frequency of a variant (104 E9) of the original pathotype 104 E137 with reduced virulence on the differential Heines VII, but not on Kalyansona, which is assumed to carry gene *Yr2* in common with Heines VII (CR Wellings, personal communication). Changes to reduced virulence in clonally propagating populations are uncommon.

Stripe rust control in Australian wheats is predominantly by adult plant resistance, although *Yr17* (derived from VPM1) is present in several wheats and *Yr9* and *Yr10* are present in less widely used cultivars. The likely presence of *Yr18* (linked with *Lr34*), particularly in the eastern states, was described above. Genetic analyses of various local and exotic wheats, classified as adult plant resistant or moderately resistant and considered to be adequately protected without additional chemical control in most circumstances, have shown resistance is based on a low number, usually two to four, of genes with additive effects. These results support repeated reports of transgressive segregation (e.g. 13, 30, 40) of stripe rust response in wheat crosses and further indicate that breeders can rely on the additive nature of resistance as a means of constructing gene combinations conferring high levels of resistance. They also suggest that any loss of resistance is likely to be a stepwise process. However, experience with cultivar Joss Cambier in the United Kingdom provides a

warning on generalizations. This cultivar with an apparently highly effective gene, *Yr11*, for adult plant resistance became highly susceptible following an apparent single mutation event in the pathogen. The routine capture of genetic information on cultivars as they are introduced to agriculture, especially those with very distinctive pedigrees, might enable prediction of the likely magnitude and impact of any pathogenic change.

In attempting to achieve durable resistance to stripe rust, breeders should be aware of the vulnerability of resistance genes effective at the seedling (and adult) stage, and the likelihood of transgressive segregation in crosses among cultivars with intermediate adult plant responses. Attention can also be directed to cultivars such as Oxley and Cook, which represent potential adapted parents with the highest levels of adult plant resistance and which are known to carry at least two genes for resistance.

The Consequences of Low Pathogen Populations

The very significant reductions in rust pathogen populations that result from the recommendation and use of only resistant cultivars, and the public discouragement of susceptible cultivars provide a new challenge to plant breeders. Provided virtually all the area within a large region is sown to resistant cultivars, and provided there are no alternative hosts for survival, the only sources of inoculum will be from off-type plants in current cultivars, from nonrecommended susceptible cultivars grown by nonconformist farmers, or from host sources outside the region. With very high frequencies of adoption of resistant cultivars, these sources of inoculum will be minimal and the likelihood of mutant pathogen variants will also be minimal. Under such circumstances, cultivars with single genes for resistance are likely to contribute to sustained protection. However, the overall strategy would be strengthened by the continued use of gene combinations and genetic diversity. With the modern mass movement of people, an increased threat is posed by introduction of virulent pathotypes from other countries. Travelers, particularly farmers and agricultural workers, need to be made aware of the nature and spread of these diseases and the fact that uredospores can remain viable on personal effects for several weeks (47). Although wheat stem rust is known to survive on the grass *Agropyron scabrum* in northern wheat areas and on barley, experience suggests that these species are not efficient in sustaining significant levels of the pathogen over a period of many years. There is no known alternative host for wheat leaf rust in Australia. Increased virulence of *P. striiformis* f. sp. *tritici* on barley grass provides greater opportunity for survival and increase. However, barley grass is summer dormant and its role relative to wheat in stripe rust epidemiology is still to be established. Current evidence suggests that oversummering of *P. striiformis* occurs at random locations on susceptible wheats. The possibility

that some survival occurs at research stations where susceptible genotypes are often present, needs further examination.

Variability in Wheat Rust Pathogens in Australia

Phenotypic variability in rust fungal populations is determined by the normal evolutionary forces, namely introduction of genotypes from outside the region, mutation, sexual and asexual hybridization, selection, and chance. Various examples of effects of these factors on Australian rust pathogen populations were reviewed earlier (19, 41, 42). The study of rust pathogens in this country from the 1920s has shown sequences of dominant clonal groups as distinctive pathotypes were introduced and became established, often for reasons that were not clear. Once established, components of each group usually underwent sequences of mutations to virulences that may have permitted selection and spread. However, in some cases rapid spread occurred in the absence of obvious selection advantages. For example, Park et al (27) described the probable introduction of *P. recondita* f. sp. *tritici* pathotype 104-2,3,(6),(7),11 and the subsequent spread of this pathotype despite the apparent absence of any selective advantage over locally established pathotypes. By contrast, the occurrence of somatic hybrid pathotype 64-11 was restricted to hybrid wheat hosts with a particular genotype and growing in a relatively limited area (RF Park, personal communication).

Although the predominant direction of airborne movement of uredospores is from west to east, corresponding with the major weather patterns, there are examples indicating spread in the opposite direction. Although the national rust survey may detect a pathotype in one region before another, this may not be a true reflection of actual events as sampling is not exhaustive. Wheat stripe rust, which first appeared in Victoria (24), has not been detected in Western Australia. Similarly, the triticale-attacking *P. graminis* f. sp. *tritici* pathotypes 34-2,12 (virulent on Coorong with *Sr27*) and 34-2,12,13 (virulent on both Coorong and Satu with *SrSatu*) were never sampled from Western Australia even though the putative progenitor pathotype, 34-2, was largely restricted to that state by its pathogenicity attributes relative to contemporary wheat cultivars. It is assumed that the first mutation event leading to 34-2,12 occurred in southeastern Australia. This pathotype then moved northwards where it mutated to virulence on Satu, which was adopted as a replacement cultivar for Coorong.

Watson (42) described a clonal group of *P. graminis* f. sp. *tritici* pathotypes that was considered to represent a somatic hybrid of the clonal group present in Australia prior to 1950 and a post-1950 putative introduction. RF Park and colleagues (personal communication) provided evidence for somatic hybridization between two distinctive exotic *P. recondita* f. sp. *tritici* groups. Although *P. striiformis* has no known alternate host on which sexual reproduction can

occur, it is nevertheless extremely variable (46). Evidence for somatic hybridisation was provided by European workers (5, 50).

The long-term survival of clonal groups is largely dependent on their ability to co-evolve with the introduction of new sources of resistance. If successful mutation or hybrids do not occur, survival ability becomes increasingly difficult, populations decline and are then more subject to chance effects. This is particularly important for oversummer survival. The frequencies of susceptible self-sown or regrowth plants in wheat fields and other out-of-the-way locations such as the vicinity of grain storage facilities and roadsides which are protected from livestock will have an impact on summer survival. This is more likely in summer rainfall areas, or in unusually wet years in other zones. The summer conditions in South Australia in 1993 provided conditions for widespread incidence of stem rust on self-sown wheat and barley. However, many samples taken from barley comprised a somatic hybrid of *P. graminis* f. sp. *tritici* and *P. graminis* f. sp. *secalis* that does not attack wheat (25). An ensuing wheat stem rust epidemic was averted through promotion of resistant cultivars, chemical seed treatment to prevent early seedling infection, and drier than normal conditions for establishment of the 1994 crop.

The Strategy of Anticipatory Resistance Breeding

During the period of widespread concurrent use of both resistant and susceptible cultivars, and relatively large pathogen populations, new virulent mutants of current pathotypes were selected by previously resistant host genotypes. If the effective genes in current cultivars were known, the type of change in the most frequent pathogen genotypes could be predicted, especially where it was known that only a single mutation was required to generate the required virulence.

Given that prediction was possible, an understanding of pathogen variability and host genotypes permitted preemptive crossing and selection for resistance that would be required if the predicted mutation were to occur. Clearly, it is difficult to add resistance genes to host genotypes that are already resistant. The principles of host : pathogen genetics (23) permit the addition of a further resistance gene if the infection type (phenotype) conferred by that gene is lower (less compatible) than the phenotype produced on the recipient parent, or if there are recognizable interaction effects that enable its identification. Usually, interaction results in infection types that are lower than those conferred by the individual genes. Alternatively, the resources of a pathogen culture collection can be exploited. A culture that overcomes part of a current multiple gene resistance may be available for use in the selection process. During the late 1960s and 1970s, IA Watson at this Institute made use of ethyl methanesulphonate (EMS) to produce a laboratory culture of *P. graminis* f. sp. *tritici* that was virulent on seedlings of Timgalen (*Sr5*, *Sr6*, *Sr8a*, *Sr36*) which, at that time,

was a leading prime hard wheat cultivar for the northeastern region. The resultant culture (74-L-1; pathotype 34-1,2,3,4,5,6,7 (for system of nomenclature, see 23) was not only used as a greenhouse test culture to select the next generation of wheat cultivars for the region, but twenty-three years later, remains a key culture for selection in wheat breeding programs. When pathotype 343-1,2,3,4,5,6, virulent on Cook (a Timgalen derivative with the same genotype), appeared in 1983, wheat breeders in the region were able to replace that cultivar within two years with resistant backcross derivatives. The Cook-attacking pathotype did not increase to epidemic levels and no farmer experienced losses caused by it. With the withdrawal and replacement of Cook, there was also a rapid decline and disappearance of the Cook-attacking pathotype (51). The resistant Cook derivatives thereby benefited from the continued effectiveness of *Sr36* along with the various genes that had been added to it (e.g. *Sr9e* in cultivar Diaz, *Sr24* in cultivar Sunco, *Sr26* in cultivar Bass, *Sr38* in cultivar Sunbri).

One to several years may elapse from the time a virulent pathotype is first detected until the occurrence of a loss-causing epidemic. Provided there is a pathogen surveillance program, a knowledge of host genotypes, and a germplasm enhancement program that continually adds resistance genes to current cultivars and advanced breeding lines nominated by breeders, this lag period can be used to select and increase resistant replacements. With adequate isolation, the new pathotype can be used in field screening nurseries to confirm the expected resistant responses. Whereas it may require more than ten years to develop a wheat cultivar, an ongoing germplasm enhancement program provides germplasm that can be exploited much more quickly if required. In some instances, backcross-derived cultivars with added genes for resistance are being released prior to the occurrence of pathotypes virulent on the recurrent parent.

An alternative procedure to the pyramiding of increasingly more effective resistance genes and use of mutant pathotypes or special resource cultures is the exploitation of linked markers. Since the early 1980s, there has been increasing use in Australia of two translocation derivatives of *Agropyron elongatum* (CS 3D-3Ag#3 and CS 3D-3Ag#14 produced by Dr. ER Sears) which possessed *Sr24* and *Lr24*. These were recombined in wheat crosses to produce white seeded wheats preferred by the local grain industry. Selection for *Sr24* to produce cultivars such as Cunningham, Datatine, Goroke, Janz, Sunco, Swift, Tasman, and Vasco was aided by the knowledge that *Sr24* and *Lr24* were inherited in wheat as completely linked genes. A similar situation of complete linkage in an alien segment from *Aegilops ventricosa* permitted the transfer of genes *Sr38*, *Lr37*, and *Yr17* to wheat cultivars including Bowie, Sunbri, Sunstate, Sunvale, and Trident. Finally, the association of the pseudo-black chaff phenotype and a high temperature-induced seedling chlorosis with adult plant resistance gene

Sr2 permits confirmation of the presence of the resistance gene in germplasm where it is otherwise difficult to detect (2). Approximately 45% of the northeast wheat-growing region in 1993 was occupied by wheats possessing *Sr2* in various combinations with one to three additional effective genes. Nine genes, each of which was either comprehensively effective or provided effective resistance in combinations, were present in the remaining cultivars (1).

Even more advantages for selection based on linked characters are provided by molecular markers, especially markers that behave co-dominantly, enabling selection of homozygotes. Whereas a number of alien resistances in wheat can be efficiently marked (e.g. 34, 35, 49), there are few instances where highly desired nonalien wheat genes for rust resistance have been tagged. Closely linked markers for *Lr1* (4), *Lr10* (9) and the linked *Sr15*, *Lr20*, *Pm1* group (16) have been reported, but none of these genes provides sufficiently effective resistance for use in Australian resistance breeding programs. A marker for *Sr2* was identified by S. Johnston (personal communication) at this Institute.

Breeding for Rust Resistance in the Future

With the significant reduction in population size and genetic diversity of the wheat stem rust pathogen throughout Australia and of the wheat leaf rust pathogen in much of eastern Australia, there have been reduced incidences of new pathotypes resulting from mutational changes. Introduction of new pathotypes from outside the region seems to have increased, probably as a result of more frequent and rapid human travel and transport. The major threat in this respect seems to be leaf rust, currently the most widely dispersed and most frequently encountered wheat rust disease. Of greatest threat to Australia is the potential introduction of further pathotypes with virulence for *Lr13*, as these are known to occur very widely, and virulence for *Lr24* which occurs in South Africa and the Americas. However, the probable common occurrence of *Lr34* and additional adult plant resistance genes in a number of wheats grown in eastern Australia should reduce the levels of rusting and magnitude of losses should such events occur.

Stripe rust is likely to be a continuing problem in which the presence of the alternative barley grass host may have a significant role. With the application of improved management practices to wheat production in Australia there are likely to be increased levels of stripe rusting which, in turn, may require higher levels of resistance than present in current recommended cultivars.

For all three wheat rust diseases, there is a need to further discourage the use of nonrecommended susceptible cultivars for specialized uses. For example, the problems associated with the use of older, awnless, nonsemi-dwarf cultivars for hay and chaff production, the use of nonrecommended, noneastern Australian soft wheats in irrigation areas, and the recent trend to long-season, dual purpose

(grazing and grain) winter wheats in high rainfall areas need to be individually addressed. Although these wheats may be successfully grown in "low risk" disease situations, farmers must be aware of the added protection provided by rust control in previously "high risk" areas and should be expected to respond in kind for the benefit of the entire industry. For this to be achieved, breeders must be committed to providing appropriate cultivars to fulfill these developing requirements.

Wheat rusts have largely been controlled in high risk areas in Australia with the use of resistance; further control over the entire country without a downgrading in attitudes and perceptions of the rust threat is a greater challenge in the future as breeders and the industry identify more damaging current problems. The potential cost of reduced efforts in rust control will be the financial burden of crop losses and increased dependency on chemical control strategies.

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CONTENTS