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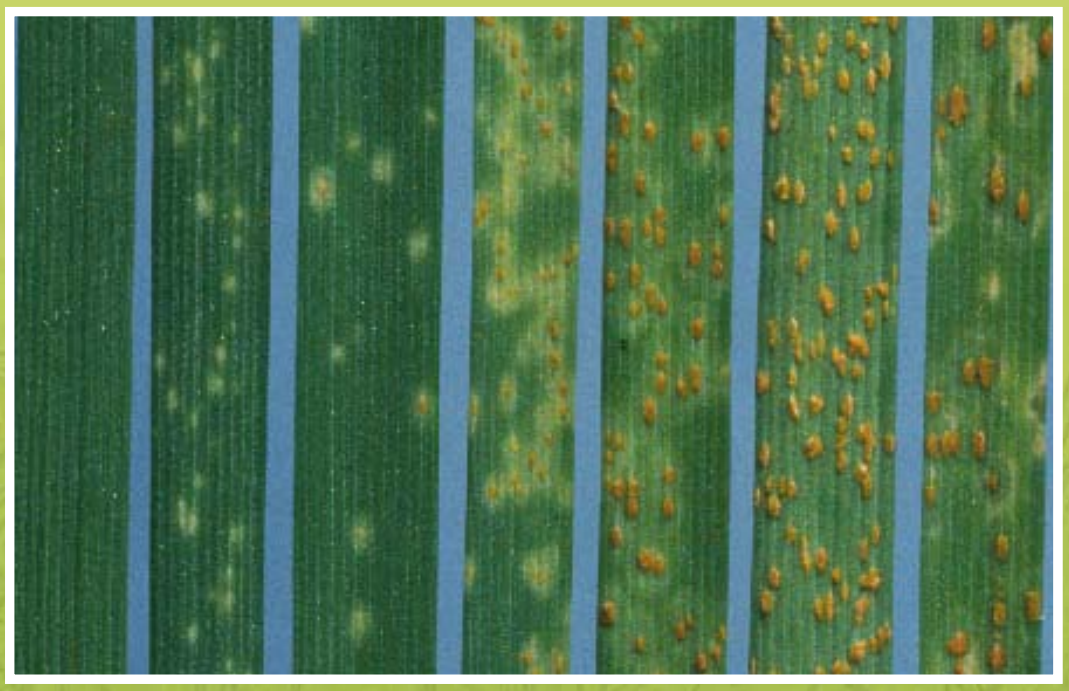
Sustainable wheat rust resistance - Learning from history



About this document:

This review document was prepared by Paul Brennan, PB&B Consulting, PO Box 9055, Rock Valley via Lismore, NSW, 2480, Australia, for the Global Partnership Initiative for Plant Breeding Capacity Building.

Sustainable wheat rust resistance – Learning from history

A review prepared by
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for the
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About GIPB

The Global Partnership Initiative for Plant Breeding Capacity Building (GIPB) is a multi-party initiative of knowledge institutions around the world that have a track record in supporting agricultural research and development, working in partnership with country programmes committed to developing stronger and effective plant breeding capacity (http://km.fao.org/gipb/)

As a partnership of stakeholders from the public, private and civil society sectors, the initiative is aimed at catalyzing and supporting national, regional and global action among relevant international organizations, foundations, universities and research institutes, corporate and business sector, civil society associations, and national and regional bodies.

Mission

The Mission of GIPB is to enhance the capacity of developing countries to improve crops for food security and sustainable development through better plant breeding and delivery systems.

Objectives

A GIPB stakeholder consultation process has defined the following five longer-term specific objectives, aiming at the integrated enhancement of national plant breeding capacity building strategies:

Support for policy development on plant breeding and associated scientific capacity building strategy, to help allocate resources to strengthen and sustain developing countries' capacity to use plant genetic resources for food and agriculture.

Provision of education and training in plant breeding and related scientific capacities relevant to utilization of plant genetic resources.

Facilitate access to technologies in the form of tools, methodologies, know how and facilities for finding genetic solutions to crop constraints.

Facilitate exchange, from public and private breeding programmes, of **plant genetic resources** that can enhance the genetic and adaptability base of improved cultivars in developing countries.

Sharing of information focused on plant breeding capacity building to deliver newly available knowledge to national policy makers and breeders in developing country programmes.

About the author

Dr Paul Brennan is a graduate of the University of Queensland (Australia) and the University of Saskatchewan (Canada) in genetics and plant breeding. He spent thirty three years as a wheat breeder where he released or has been heavily involved in the development of twenty three wheat varieties and is the author of a number of scientific papers and presentations at both national and internal conferences. His wheat varieties commanded over 20% of the Australian wheat deliveries over an extended period. During his time as a wheat breeder Dr Brennan co-supervised a number of graduate students with several university professors.

The needs of a competitive wheat breeding program required the deployment of many advanced plant breeding and genetic skills which were obtained through inservice training at the (then) Plant Breeding Institute (Cambridge), attending national and international conferences and through personal research.

Dr Brennan held appointments as the Director of the Queensland Wheat Research Institute, as a director of the Sugar Research and Development Corporation and to the Northern Panel for the Grains Research and Development Corporation. These appointments facilitated the development of skills in evaluating research proposals, reviewing research programs and negotiating local, national and international research programs. During this time Dr Brennan employed the plant molecular biology skills learned in Cambridge to undertake local, national and international programs in the development of molecular marker technology for wheat breeding.

Dr Brennan also devoted considerable time and energy to the development of skills in the exploitation (commercialisation) of and policy for plant intellectual property.

Dr Brennan has conducted his own consulting business in plant breeding and the application of molecular biology to plant breeding since 1999. In addition to the further development of the skills mentioned above, Dr Brennan has consulted to a wide range of national and international organisations and across more than thirty agricultural and horticultural species. A considerable focus in Dr Brennan's consultancy work has been in the area of change management for plant improvement investments and to ensure that outcomes from investments in plant breeding have a high potential of meeting the expectations of the investor.

Executive Summary

The wheat rusts have a long history of causing considerable loss in productivity and quality of wheat crops. Much work has been undertaken to address this problem and many successes have been achieved. Sustainable rust resistance has been achieved in a number of situations and has provided valuable guidance for future initiatives where this level of protection has not been achieved. The achievements include understanding the impact of the sexual stage in the rust life cycle in facilitating resistance breakdown and providing inoculum in close proximity to the developing wheat crop, resulting in more frequent and intense epiphytotics. Management options to minimize breakdown events have also been identified. The most significant discovery is that the rusts appear to lack the ability to overcome some sources of resistance, at least in the short term. These are termed durable resistances and their effective deployment in varieties where wheat rusts can be damaging to productivity would appear to be a very effective strategy to achieve sustainable global rust resistance.

The recent development of the stem rust pathotype Ug99 poses a global threat to wheat productivity. This pathotype has virulence against many genes for stem rust, and a majority of varieties where it is currently found and on its currently predicted migration path are susceptible to it. Wheat rusts can migrate long distances over land and sea. Human-facilitated incursions have been reported for stem and stripe rust. Malicious human-mediated incursion has been proposed as a significant concern. The optimal growing temperatures for stem rust are relatively higher than for leaf and stem rust. This has restricted the impact of stem rust to countries in lower latitudes. It is considered that climate change will have a warming effect on higher latitudes, which would encourage the growth and development of stem rust in non-traditional areas.

Genetic solutions to stem rust are advocated as they are low cost, environmentally neutral and the genetic resources required appear to be readily available. This option will only be effective if it is widely adopted by wheat growers in all at-risk regions. The incentive for adoption will be predicated on the varieties meeting the expectations of the whole value chain. Predominant among these expectations is that the variety has all the traits necessary to maximize grower and consumer satisfaction. This portfolio of traits has to be documented for all at-risk regions, and the technology for their successful incorporation into varieties may have to be developed along with that for durable stem rust resistance.

The identification of options to ensure long-term investment in wheat breeding and pre-breeding is a priority, and will vary from country to country. Maximizing the probability for private sector investment in wheat breeding should be a priority, as private sector investment may be less subject to the competing pressures experienced by governments and philanthropic organizations. Re-drafting of plant variety intellectual property legislation will probably be required to ensure that there is an equitable return on private sector investment in plant breeding. The nature of the changes required may well be country specific. It is also probable that there will be circumstances where it is not reasonable to expect a return from investment in wheat breeding.

Variety management strategies for the commercial deployment of resistant varieties are important to ensure that opportunities for the rust to acquire the capability to parasitize previously resistant varieties are minimized. The importance of this is predicated on the limitations of current knowledge to predict that a specific resistance source will be durable.

Priority research needs include the location of sources of durable stem rust resistance and the development of marker assisted breeding strategies to breed varieties incorporating this trait, together with all the traits that will ensure wide deployment of the resistant variety by the grower community in at-risk regions. Emphasis should be placed on chip-based marker technologies for marker identification and breeding, as these will have greater utility and cost efficiency in breeding.

Technologies need to be developed that allow the early identification of durable resistances.

Acknowledgements

The author wishes to acknowledge the considerable and unstinting assistance provided by officers of the University of Sydney, Plant Breeding Institute, in the preparation of this report. In particular, the assistance of Professor Robert Park, Dr Collin Wellings and Dr Harbans Bariana is acknowledged. I also wish to express special thanks for information and reports to Dr Kurt Leonard and Dr Alan Roelfs of the United States of America. Dr Ravi Singh (CIMMYT, Mexico) was unstinting in his quick response for many requests for reprints, and Professor Zac Pretorius provided important insight into the evolution of stem rust in South Africa.

Contents

Early History

The rusts of wheat have probably been a production constraint since the domestication of wheat in the Younger Dryas period some twelve millennia before present (Murphy, 2005). Reports from biblical times document the plight of the Hebrews resulting from rust epidemics (Boyd, 2005). There is also evidence that both the Greeks and Romans made offerings to the fertility god, Robigus, in an attempt to prevent production losses due to rust (McIntosh, Wellings and Park, 1995).

The rust fungi of wheat were all named *Puccinia graminis* by Persoon in 1797 (Singh, Huerta-Espino and Roelfs, 2002). It was recognized that there were more than one species of rust infecting wheat when Leaf rust was separated from Stem rust (de Candolle, 1815). It was not until 1896 that the third species of wheat rust, Stripe rust, was differentiated (Eriksson and Henning, 1896).

The German botanist Heinrich Anton de Bary (1831–1888) was an early pioneer in seeking secular solutions to plant diseases. He was particularly active in understanding sexual reproduction in both Late blight of potato (caused by *Phytophthora infestans*) and Stem rust. De Bary noted the prevalence of stem rust infections adjacent to common barberry, *Berberis vulgaris*, and was successful in infecting barberry with teliospores of stem rust. He observed the development of yellow pustules (aecia) of aeciospores on barberry and was able to infect rye with the aeciospores. These infections produced urediniospores (also called urediospores), which were observed on infected wheat plants. De Barry coined the term ‘heteroecism’ for plant pathogens that required more than one plant species to complete their life cycle, as opposed to ‘autoecism’ for those that did not.

A further degree of specialization within wheat stem rust was based on the host range. These are called ‘*formae speciales’* (abbreviated to f.sp.) and are variants of rust that parasitize only certain species of cereals and have varying levels of cross ability (Eriksson, 1894; Eriksson and Henning, 1896). For example, *Puccinia graminis* f.sp. *tritici* is the stem rust of wheat, while *P. g. secalis* is the stem rust of rye, and they cross readily. The oat *formae speciales*, *P. g. avenae*, cannot cross with either, and parasitizes only oats (McIntosh, 2008).

The understanding of the interaction between host and parasite was further developed through the demonstration that the inheritance of resistance in the host and the parasite's ability to cause disease is controlled by pairs of matching genes (Flor, 1942, 1947, 1955, 1971). The host gene was called the resistance (*R*) gene. The parasite’s gene was called the avirulence (*Avr*) gene. Plants that have a specific *R* gene are resistant to a pathogen with the corresponding *Avr* gene. Conversely, plants with a specific *R* gene are not resistant to a pathogen with the corresponding virulence gene. This is known as the ‘gene for gene’ theory.

The understanding of the pathogenicity of the rust was extended further with evidence of variation within *Puccinia graminis* f.sp. *tritici* for the ability to parasitize some wheat varieties but not others (Stakman and Piemeisel, 1917). These clones are now commonly called pathotypes, but were previously known as races or strains. This phenomenon has now been demonstrated for all three wheat rusts and, indeed, for a very large number of plant pathogens.

Wheat Rusts

Four species of rust are now known to parasitize bread (*Triticum aestivum*) and durum (*Triticum dicoccoides*) wheat. These are stem rust (*Puccinia graminis Pers.* f.sp. *tritici*, Erks. and Henn.), leaf rust (*Puccinia triticina,* Erks.), stripe rust (*Puccinia striiformis* f.sp. *tritici*, Erks. and Henn.) and leaf rust duri type (*Puccinia triticiduri*, V. Bourgin).

Wheat stem rust is the most devastating of the wheat rusts and can cause almost total yield loss in susceptible cultivars in extreme instances (Figure 1). It is characterized by elongated dark brown lesions (uredinium [*pl*. uredinia]; sometimes called uredium [*pl.* uredia]) on the stems and leaves (particularly the mid-rib) of susceptible wheat plants during the growing season. The uredinia produce black spores (teliospores) at the end of the growing season or during periods of stress (Singh, Huerta-Espino and Roelfs, 2002). This characteristic has given rise to its alternative common name 'black rust'. Stem rust prefers higher temperatures than other wheat rusts. It also responds to humid conditions and readily develops between 15° and 30°C.

Leaf rust, also known as 'brown rust', commonly develops on leaf surfaces and has circular uredinia that produce light brown urediniospores (sometimes called uredospores) during the growing season (Figure 2). Urediniospore production tends to be less abundant than in stem rust. Teliospores are produced at the end of the growing season on the leaf undersurface. This rust is favoured by humid conditions and has a lower temperature range (10° to 30°C) in comparison with stem rust. It commonly appears earlier in the growing season than stem rust (Singh, Huerta-Espino and Roelfs, 2002).

Figure 1. Stem rust-infected wheat plants in the field (Photo courtesy of CIMMYT).

Figure 2. Leaf rust-infected wheat plants (photograph courtesy of Colin Wellings, Plant Breeding Institute, University of Sydney).

Stripe rust, also known as 'yellow rust', has long thin uredinia (stripes) with yellow urediniospores on the leaves and stems of susceptible wheat plants. Stripe rust prefers cooler climates than stem and leaf rust, as its temperature optimum range is significantly lower (0° to 23°C; Singh, Huerta-Espino and Roelfs, 2002). It is important in temperate climates, maritime regions, at higher elevations and where wheat is grown as a winter crop in countries that have a warm summer (Johnson, 1992). It has traditionally been found in northern Europe, the Near East, east African highlands, west coast USA and the Andean region of South America (Boyd, 2005). More recently it has migrated to eastern Australia, in 1979 (O'Brien *et al*., 1980), New Zealand in 1980 (Beresford, 1982), South Africa in 1996 (Pretorius, Boshoff and Kema, 1997; Boshoff *et al*., 2002) and Western Australia in 2002 (Wellings *et al*., 2002).

Importance

The significance of wheat rust as a constraint to global food production can, as noted above, be traced back two millennia, and possibly goes back even further.

In the United States of America, yield losses for the northern Great Plains due to stem rust have been estimated for all epidemics from 1920 to 2000 (Leonard, 2001a; Figure 4). These losses reached up 105 million bushels in 1935.

Historically, stem rust has had a significant impact on the value of the Australian wheat crop. The epidemic in 1889 resulted in a loss of approximately AU$ 164 million in 2009 values, and the 1903 epidemic cost AU$ 32 million (McAlpine, 1906); the 1916 epidemic AU$ 100 million; the 1947 epidemic AU$ 204 million (Butler, 1948); and the 1973/74 epidemic caused an AU$ 18–27 million loss to Australian wheat growers (Watson and Butler, 1984).

The value to the Australian wheat industry in preventing loss of production and crop processing quality from the investment in breeding for stem rust resistance is AU$ 128 million per annum in 1988 value (Brennan and Murray, 1988). The benefit of breeding for resistance to all species of rust in Australia is estimated at AU$ 289 million per annum (Brennan and Murray, 1988). It is estimated that the Australian wheat industry spent between AU$ 40 and 90 million between 2003 and 2005 on spraying to control stripe rust (Wellings, 2007). Yield losses due to leaf and stripe in Western Australia for susceptible varieties have been estimated as high as 80% (Beard *et al*., 2007). Similar losses have been estimated in the USA (Eversmeyer and Browder, 1974).

Yield loss of 30–50% have been estimated for the leaf rust epidemics in Pakistan in 1948 and 1954, and substantial losses were experienced in 1978 (Fida, 2005).

The wheat rusts have the capacity, if not controlled effectively, to greatly limit global wheat production. The discovery of a new race of stem rust in east Africa in 1999 with the virulence combination to overcome many of the stem rust resistance genes deployed globally has exacerbated this concern. FAO have estimated that this pathotype, Ug99 (TTKS), has the potential to affect wheat production in many countries, and has identified 29 countries for specific action (FAO, 2008). FAO estimated that this pathotype in 2008 threatened the wheat production in the Near East, eastern Africa and central and south Asia, which together account for 37% of global wheat production.

The effect of climate change will probably be to increase the potential for wheat rust, particularly stem rust, to damage wheat crops, as it is anticipated that there will be preferential warming at high latitudes, where much of the world's wheat is grown (Rosenzweig *et al*., 2000). Warmer temperatures will particularly favour the development of stem rust, which is already causing increased global concern due to the development of the Ug99 pathotype.

Figure 3. Stripe rust-infected wheat plants (photograph courtesy of Colin Wellings, Plant Breeding Institute, University of Sydney).

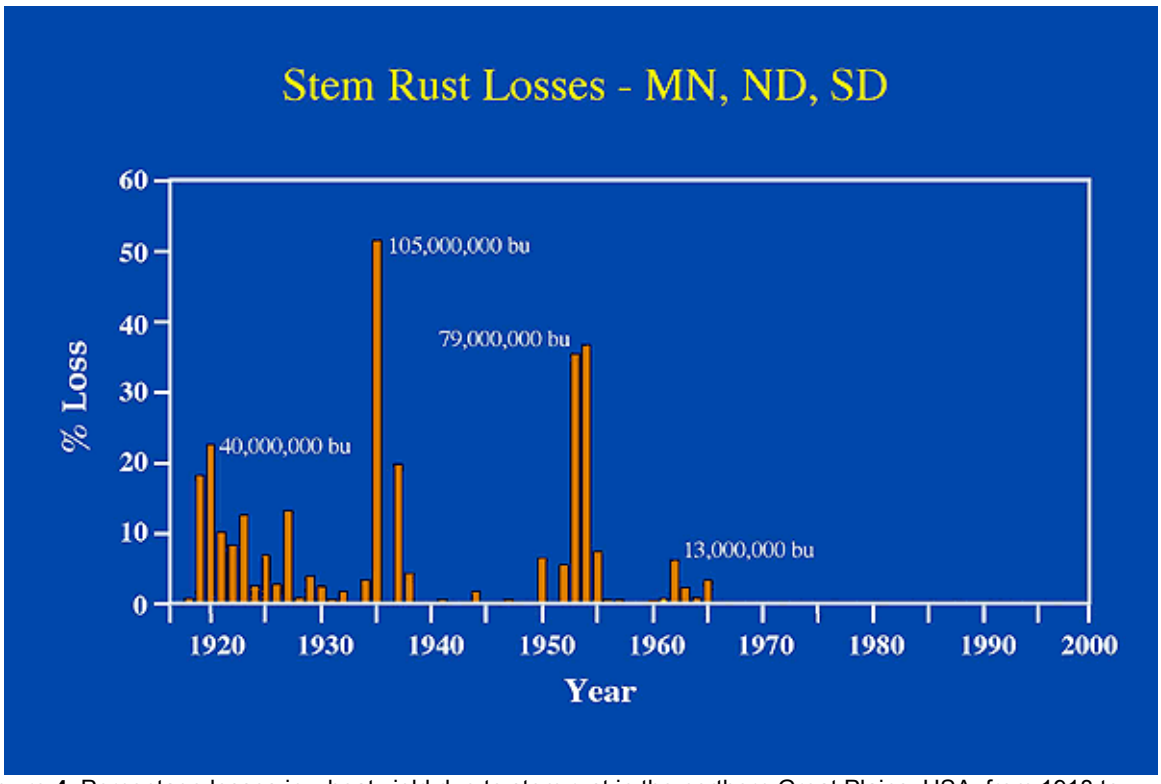

Figure 4. Percentage losses in wheat yield due to stem rust in the northern Great Plains, USA, from 1918 to 2000. Total bushels of wheat lost to stem rust in Minnesota (MN), North Dakota (ND) and South Dakota (SD) are indicated for 1920, 1935, 1953 and 1962. (Reprinted from Leonard, 2001b).

Life Cycle

The life cycle of the wheat rusts are discussed here as knowledge of the life cycle has facilitated the identification of intervention strategies that have mitigated global vulnerability.

The asexual phase of stem rust is most readily observable and it is the rapid multiplication of the urediniospores produced during this stage that is the cause of crop damage (Figure 5). Urediniospore production is replaced by teliospore production towards the end of the growing season for the host, or when the host experiences stress. These spores are thick walled and remain viable and apparently dormant through winter. Like urediniospores, teliospores are dikaryotic as they have two haploid nuclei per cell. During the apparent dormant period the two nuclei in the teliospores fuse to form diploid nuclei, which rapidly begin meiosis to produce four recombinant nuclei. The teliospores germinate in spring and basidia develop on the germinating hyphae (Figure 5). Each basidium produces one basidiospore. Each basidiospore contains one of the recombinant haploid nuclei. They are forcibly ejected from the basidium and carried by air currents. Some land on and successfully parasitize barberry (*Berberis vulgaris*) plants to produce pycnia on the upper side of the leaf. The pycnia produce receptive hyphae (which act as the female organ), spores called pycniospores (which act as the male organ) and a sugary nectar. The pycnia are self-incompatible and the nectar is required to attract insects, which transport the pycniospores to pycnia of the opposite mating type to effect fertilization. The fertilized receptors produce dikaryotic hyphae. These produce a fourth lesion type, the aecia, on the underside of the barberry leaf (Figure 6). The aeciospores produced from the aecia are windblown and can infect receptive wheat plants to produce urediniospores, which can go through many cycles of asexual reproduction during the life of the host (Table 1).

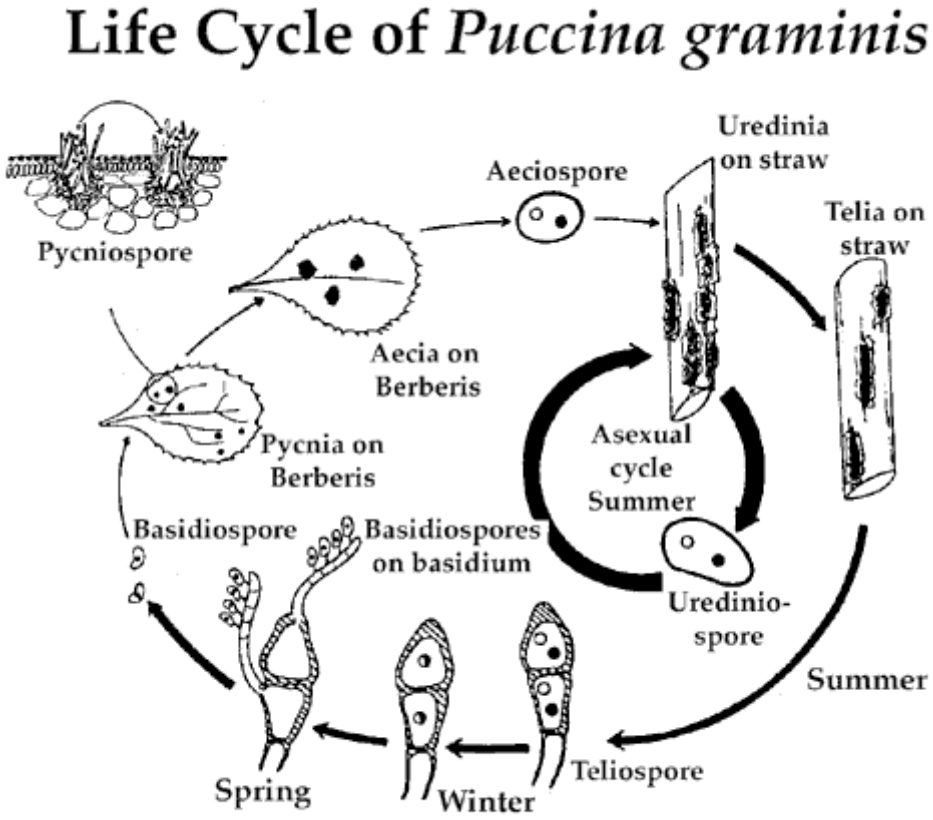

Figure 5. Life cycle of *Puccinia graminis* (courtesy of CIMMYT).

Figure 6. Aecia with aeciospores on the underside of the barberry leaf. (Courtesy of K.J. Leonard, USDA).

Table 1. Life cycles for the wheat rusts[1].

Lesion	Spore and ploidy	Host		
		Stem rust	Leaf rust	Stripe rust
Uredinia	Urediniospore (n + n)	Wheat[2], durum, barley, triticale	Wheat, durum, barley, triticale	Wheat, durum, barley, triticale
Telia	Teliospores (n + n), 2n, (n + n)	Wheat, durum, barley, triticale	Wheat, durum, barley, triticale	Wheat, durum, barley, triticale
Basidium	Basidiospores (n)	Germinating teliospores	Germinating teliospores	

Pycnia	Pycniospores (n)	*Berberis vulgaris* (upper side of leaf)	*Thalictrum speciosissimum*[3] (upper side of leaf)	Not known
Aecia	Aeciospores (n + n) and infect wheat, etc.	*Berberis vulgaris* (lower side of leaf)	*Thalictrum speciosissimum* (lower side of leaf)	

NOTES: (1) *Puccinia triticiduri* has not been included due to its limited economic importance; (2) Bread or hexaploid wheat; (3) Others include *Anchusa* spp., *Isopyrum* spp. and *Clematis* spp.

Leaf rust has a similar life cycle to stem rust, but an alternate host has not been identified for stripe rust (Table 1).

The uredinia/urediniospore stage is the most significant stage in the life cycle of the wheat rusts in creating an epidemic in susceptible varieties that will result in loss of production and processing quality. The extent of these losses will depend on the prevailing environmental conditions. The significant events at this stage include the large number of urediniospores that can be produced from one uredinium; the short generation time from infection to sporulation of uredinia; and the potential for the urediniospores to be transported long distances by wind. These events have been eloquently documented by Stakman (1957):

> "A urediospore may germinate in less than an hour, send out a germ tube that grows along the epidermal surface of wheat, enters through a stoma and is inside of the plant within six hours or even less. The rust tubes or hyphae then branch and grow parasitically between the plant cells, form an extensive network of mycelium over five mm. in extent, which then produces a new crop of 50 thousand to 450 thousand urediospores within a week or ten days. Each of the new spores can then repeat the process, and this can go on and on as long as wheat is green and growing.
>
> "On an acre of moderately rusted wheat there are about 50 thousand billion urediospores, each one capable of surviving a long air journey and starting infection many miles from the place where it was produced.
>
> "The wind has carried spores 600 miles northward, from central Kansas to the Canadian border, over a front more than 400 miles wide. As another example, in early June, 1953, it was calculated that there were 4,000 tons of urediospores, with about 150 billion spores per pound, on four million acres of wheat in northern Oklahoma and south-central Kansas. Winds carried spores northward from this area into the Dakotas and Minnesota, where they were deposited at the rate of 3.5 million an acre in an area comprising 40 thousand square miles."

Stakman's assessment refers specifically to stem rust. Although neither leaf nor stripe rust uredinia have the same capacity to develop urediniospores, they nevertheless, produce very large numbers of spores per uredinia and have similarly short periods between infection and sporulation (under favourable conditions), and have similar capacity for the urediniospores to be transported long distances by wind (Singh, Huerta-Espino and Roelfs, 2002).

The capacity for stem rust to generate genetic variability is derived from two stages in its lifecycle. All cells are dikaryotic (have two nuclei) in the uredinial and aecial stages. The telial stage commences in this way, but the nuclei fuse to form diploid nuclei, which then undergo meiotic division to produce recombinant haploid nuclei. Dikaryotic cells that are, most probably, genetically distinct from their parents are formed during the aecial stage. The new generation of urediniospores produced by aeciospores infecting wheat will be genetically different from their urediniospore progenitors.

Options for intervention

Disruption to the uredinia/urediniospore stage would limit the build up of inoculum, slow the epidemic development and minimize the long-distance transport of the urediniospores. The result would be a minimizing of loss of production and product quality. Options for doing this include spraying with fungicides or developing varieties that are resistant to rust, i.e. varieties that partly or fully suppress infection by, and generation of, urediniospores. Traditionally, breeding resistant varieties has been the option of choice to control the rusts, although fungicide technologies have been developed and used, particularly at times where the breeding option is not available. The varietal option has been selected for cost and environmental reasons. It is considered that the cost of chemical control would be prohibitive in many developing economies (FAO, 2008).

Breeding resistant varieties

The development of plant varieties with a specific trait requires the identification of genetic variability for that trait and the development of effective and efficient breeding strategies to incorporate the target trait into commercially acceptable varieties. The understanding of the genetic basis of resistance to wheat rust commenced in 1908 (Biffin, 1908). Since then a very large global investment has been directed toward the discovery of genetic variability for resistance to stem, leaf and stripe rusts. This endeavour has been facilitated by the discovery that the reaction of the first leaf of a wheat plant to infection by rust is a good predictor of the resistance of the adult plant of that variety to the biotype of the rust used to infect the seedling.

These reactions are assessed on a 0 to 4 scale (Stakman and Levine, 1922, as reported by McIntosh, Wellings and Park, 1995), and are illustrated in Figure 7. Plants are considered to be immune to the biotype used if the seedling leaf showed no reaction to the rust (designated '0') or there is only a slight clearing of the chlorophyll to produce a fleck (designated ';'). The seedling is classified as resistant when the reaction is a small lesion surrounded by a necrotic area or by a halo of chlorosis. These are classified as '1' and '2' type reactions, respectively. The plant is predicted to be susceptible if it produces a '3' (large healthy lesion surrounded by an area of chlorosis) or '4' where the lesion is large and surrounded by little or no chlorosis. A mixed or mesothetic reaction may also be produced.

Similar classificatory systems have been developed for predicting adult-plant resistance to leaf and stripe rust (Figures 8 and 9). It is important to stress that such predictions are not always correct and confirmatory testing of adult plants is required.

It is possible that different genes for resistance may give similar reaction types or that there may be more than one allele for resistance at a particular locus. Both of these have significance for the deployment of the resistance in a breeding programme. The classical approach to determine whether resistances are different loci or different alleles has been to determine the location of the resistance in the wheat genome. Commonly, this has allowed the placement of the gene for resistance on one arm of a specified wheat chromosome and to determine linkage relationships with other known genes (Table 2, adapted from McIntosh *et al.,* 2008). Many genes for resistance to the three wheat rusts have been identified and these are located in all homeologous groups.

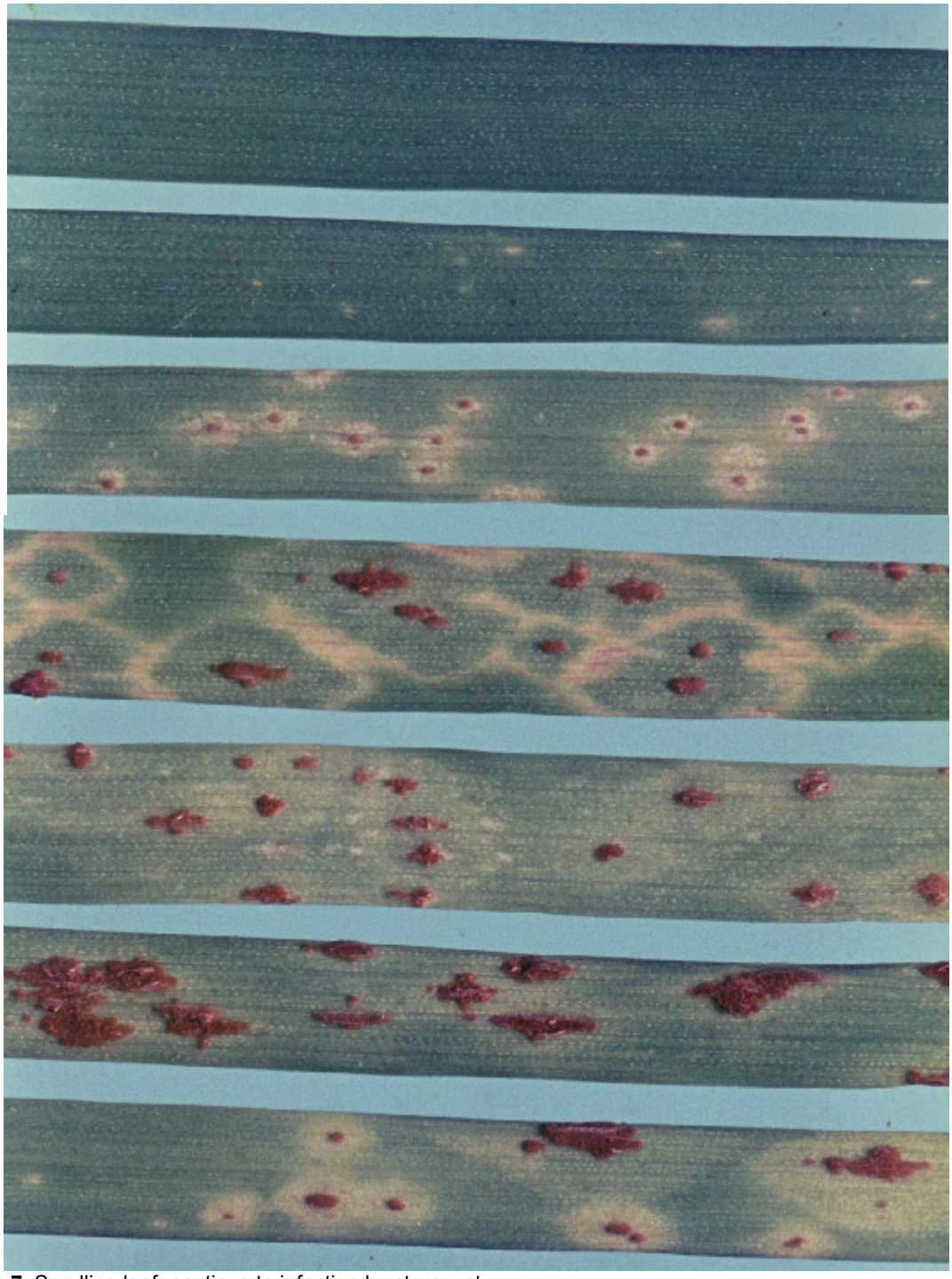

Figure 7. Seedling leaf reactions to infection by stem rust.
KEY: From top to bottom: 0, fleck (;), 1, 2, 3, 4 and mesothetic (from McIntosh, Wellings and Park, 1995).

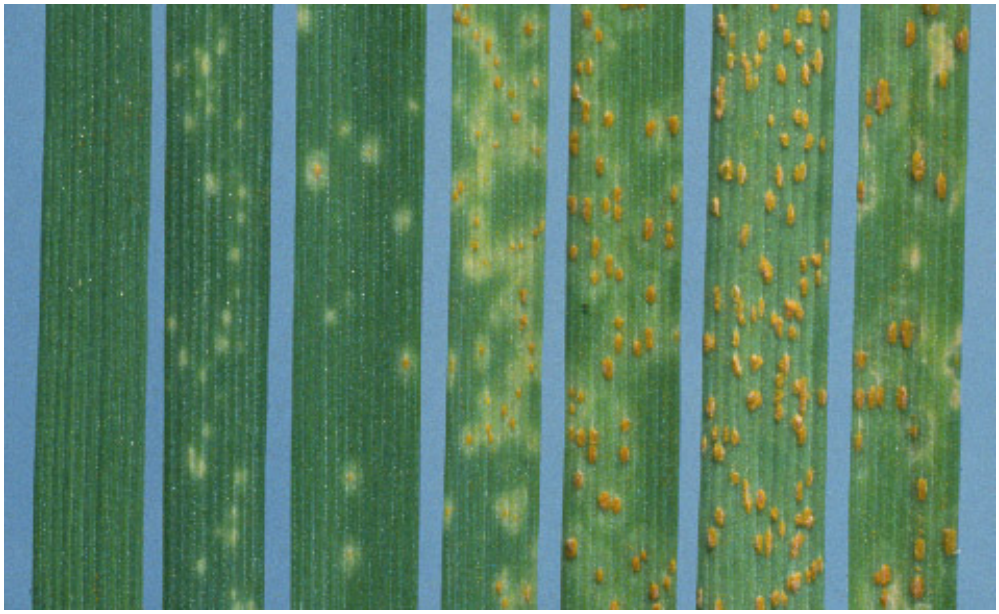

Figure 8. Seedling leaf reactions to infection by stem rust (from McIntosh, Wellings and Park, 1995).

Table 2. Chromosomal location of genes for resistance to wheat rusts (adapted from McIntosh *et al.,* 2008)

Chromosome	Genes	Chromosome	Genes
1AS	Lr_{10}	4BL	Lr_{31}, Lr_{48}, Sr_{37}
1AL	Lr_{59}	4B	Lr_{12}, Lr_{16}, Sr_{23}, Yr_{Cle}, Yr_{Mor}, Yr_{Yam}
1BS	Yr_{10}, Yr_{15} , $Yr_{24/26}$, Yr_{Alp}, Yr_{H52}	4DS	Yr_{28}
1BL	Lr_{33}, Lr_{46}, Lr_{51}, Sr_{14}, Yr_{29}	4D	Sr_{41}, Yr_{22}
1B	Lr_{44}, Lr_{55}, Sr_{31}, Sr_{Zdar}, Yr_{3},Yr_{9}, Yr_{21}	5AL	Yr_{34}
1DS	Lr_{21}, Lr_{60}, Sr_{45}	5AS	Lr_{38}
1DL	Lr_{38}, Sr_{33}	5BS	Lr_{52}
1D	Lr_{42}, Sr_{18}, Yr_{25}	5BL	Lr_{18}
2AS	Lr_{17}, Lr_{37}, Lr_{49}, Sr_{38}, Yr_{17}	5B	Yr_{19}, Yr_{Dru}
2AL	Lr_{38}, Sr_{21}, Yr_{1}, Yr_{32}	5DS	Lr_{57}, Yr_{40}
2A	Lr_{11}, Lr_{45}, Lr_{Tt1}, Sr_{32}, Sr_{34}	5DL	Lr_{1}, Sr_{30}
2BS	Lr_{13}, Lr_{16}, Lr_{23}, Sr_{19}, Sr_{23}, Sr_{36}, Sr_{40}, Yr_{27}, Yr_{31}, Yr_{41},Yr_{Sp},	6AS	Sr_{8}
2BL	Lr_{50}, Lr_{58}, Sr_{9}, Sr_{16}, Sr_{28}, Yr_{5}, Yr_{7}	6AL	Sr_{13}, Sr_{26}
2B	Lr_{35}, Sr_{10}, Sr_{20}, Sr_{32}, Sr_{39}, $Y r_{3}$, Yr_{Ste}, Yr_{V23}.	6A	Yr_{38}, Yr_{D}, Yr_{Dru2}, Yr_{H46}
2DS	Lr_{2}, Lr_{15}, Lr_{22}, Lr_{39}, Sr_{6}, $SuLr_{23}$, Yr_{CK}	6BS	Lr_{36}, Lr_{53}, Yr_{35}, Yr_{36}
2DL	Lr_{54}, Yr_{37}	6BL	Lr_{3}, Lr_{9}, Sr_{11}
2D	Sr_{32}, Sr_{34}, Yr_{8}, Yr_{16}	6B	Yr_{4}, Yr_{Dru}
3AL	Sr_{35}	6DS	Sr_{5}, Sr_{29}, Sr_{42}
3A	Sr_{27}, Yr_{Tr2}	6DL	Lr_{38}, Sr_{29}
3BS	Lr_{27}, Sr_{2}, Sr_{12}, Yr_{30}, Yr_{ns1}	6D	Yr_{20}, Yr_{23}, Yr_{Tye}, Yr_{Tr1}
3B	Yr_{S}, Yr_{Ste}	7AL	Lr_{19}, Lr_{20}, Sr_{15}, Sr_{22}, Sr_{25}
3DS	Lr_{32}, Lr_{38},	7BS	Yr_{6}
3DL	Lr_{24}	More than one chromosome	Sr_{17}, Yr_{39}, Yr_{ZH84}
4AL	Yr_{28}, Lr_{30}, Sr_{7}	7B	Yr_{2}
4A	Yr_{HVII}, Yr_{Min}, Yr_{ND}	7DS	Lr_{29}, Lr_{34}, Sr_{44}, Yr_{18}
4BS	Lr_{25}	7DL	Lr_{19}, Lr_{VPM}, Sr_{25}, S_{r43}

NOTES: *Sr, Lr* and *Yr* indicate genes for resistance to stem, leaf and stripe rust, respectively.

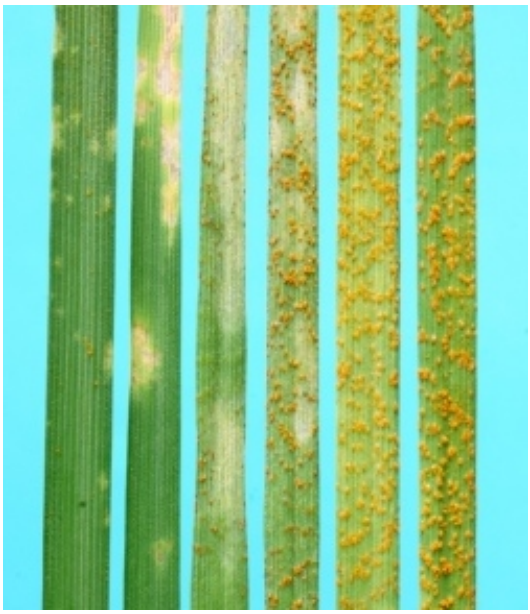

Figure 9. Seedling leaf reactions to infection by stripe rust (from McIntosh, Wellings and Park, 1995).

The breeding methodology for developing wheat rust-resistant varieties varies greatly but usually includes mass screening of seedlings for their reaction to specific pathotypes of the rust(s) early in the breeding cycle, with adult-plant assessments at a later date. This methodology has been chosen as the heritability of rust resistance is quite high and it is much more cost effective for screening the very large populations associated with the early stages of the breeding cycle. Also, seedling screening for rust resistance can readily and effectively be undertaken in the off season.

Breeding outcomes

The development of rust-resistant wheat cultivars using seedling reaction type as a predictor of adult-plant resistance has been conducted globally, with different countries placing emphasis on those rust species of economic concern to them.

Stem rust resistance breeding in Australia

The wheat cultivar Eureka was released for commercial cultivation in 1938 in Australia, with a known seedling-expressed resistance giving adult-plant resistance (Watson and Luig, 1963). Eureka had the Sr_6 gene for stem rust resistance, which conferred resistance to all known pathotypes of rust found in Australia in 1938. In 1942, Eureka became susceptible to stem rust in Australia. "This was the first happening of its kind in Australia and, in the absence of barberry, infection was not readily explained" (Watson and Luig, 1963; Figure 3).

A series of wheat cultivars (including Gabo, Charter, Yalta and Kendee) were released, starting in 1942, to replace Eureka. These varieties had the Sr_{11} gene, which was resistant to all known stem rust pathotypes in Australia at the time of release of Gabo. These varieties became susceptible in 1948. This cyclic phenomenon has continued (Table 3).

The frequencies of biotypes of wheat stem rust in the stem rust-prone areas of north-eastern Australia were examined from before 1929 to 1963 (Watson and Luig, 1963). They demonstrated a very clear relationship between the proportion of the wheat growing area grown to a particular gene for stem rust resistance and the frequency of virulence in the stem rust flora. The trend was for virulence against a particular rust resistance gene to appear some time after the corresponding gene for resistance had been released and rapidly increased in frequency. It would decline rapidly with the decline in the area sown to the varieties it was virulent on once the replacement varieties had become susceptible. The exception to this trend was the variety, Hopps, which remains resistant to stem rust, as have other varieties that carry the Sr_2 gene for stem rust resistance.

Table 3. History of stem rust resistance breeding in Australia (Watson and Luig, 1963; Macindoe and Walkden-Brown, 1968; McIntosh, 1983, 1996; Park, pers. comm.)

Cultivar (first release of the gene for rust resistance)	Gene(s) for stem rust resistance	Year of release	Year resistance became ineffective
Eureka	Sr_6	1938	1942
Gabo	Sr_{11}	1942	1956
Celebration	Sr_3, Sr_4	1945	1954
Festival	Sr_{9b}	1950	1960
Warigo	Sr_{17}	1943	1959
Hopps	Sr_2 Sr_{9d}	1957	Still effective
Mengavi	$Sr3_{36}$	1958	1961
Mendos	Sr_{7a}, Sr_{11}, Sr_{17}, Sr_{36}	1964	1967
Gamut	Sr_6, Sr_{9b}, Sr_{11} +	1965	1973
Timgalen	Sr_5, Sr_6, Sr_8, Sr_{36}	1967	1984
Kite	Sr_{26}	1973	Still effective
Banks	Sr_8, Sr_{9b}, Sr_{12}, Sr_{30}	1979	1989
Hartog	Sr_2, Sr_8, Sr_{9b}, Sr_{12}, Sr_{30}	1982	Still effective
Torres	Sr_{24}	1983	Still effective
Diaz	Sr_{36}, Sr_{9e}	1986	Still effective
Batavia	Sr_2, Sr_{8a}, Sr_{12}, Sr_{30}	1991	Still effective

Watson and Luig (1963) also noted the step-wise changes in virulence in the evolving pathotypes in the stem rust flora. In general, they observed that, based on the range of pathogenicity in a particular pathotype, a single genetic change would occur in a particular pathotype to allow it to parasitize the previously resistant varieties. Watson and Luig (1963) suggested that this might be due to either asexual genetic recombination events or to mutation. These will be considered later in this report, but the addition of virulence without alteration to other quantifiable characteristics of the pathotype would suggest that spontaneous mutation may be implicated in the generation of apparently new virulence in the Australian stem rust flora (Luig and Watson, 1970).

The rate at which the resistance of wheat varieties was overcome after 1963 was much slower (Table 3). This would appear to be of importance for the development of a strategy to breed sustainable wheat rust resistant wheat varieties and will be considered later. Sexual recombination on barberry was not implicated as this species is not found in the parts of Australia where stem rust was most prevalent.

Leaf rust resistance breeding in Australia

A similar study was undertaken of changes in the virulence frequencies in the leaf rust flora of eastern Australia in relation to the leaf rust resistances in the varieties being grown commercially (Watson and Luig, 1961). They described a pattern similar to that for stem rust.

Stem rust resistance breeding in North America

Wheat stem rust has a long history of damaging epidemics in North America (Leonard, 2001a, b). North America has been divided into four zones based on the different imperatives for the development of stem rust epiphytotics. These are: the coastal areas adjacent to the Gulf of Mexico, including the Mississippi Valley (Zone 1); southern Great Plains and southern east coast USA (Zone 2); northern Grain Plains (Zone 3); and northern spring wheat growing areas of the USA and the Canadian Prairies (Zone 4) (Roelfs, 1989).

The temperatures are adequate in Zone 1 for the stem rust fungus to cycle slowly through the urediniospore stage during winter. This provides windborne inoculum for the whole of North America as air movement is from south to north in spring. The prevailing winds in autumn carry urediniospores from the northern reaches of the wheat growing area back to re-infect winter wheat crops sown in Zone 1.

The winter temperatures are generally too low in Zones 2 and 3 for the overwintering of significant amounts of stem rust inoculum. A wheat stem rust infection in these areas early in the season relies on exogenous inoculum from Zone 1.

There is little chance that endogenous inoculum would be available from wheat plants for infection of wheat crops in Zone 4 due to the very cold winters precluding the overwintering of the host and, consequently, of the parasite. However, the alternate host of stem rust, *Berberis vulgaris*, which has been introduced from Europe to use as hedgerows in this and other zones, provides endogenous inoculum. Barberry has also spread from the hedgerows into stands of native vegetation (Leonard, 2001a). The presence of barberry is significant in importance in Zone 4 as teliospores either do not germinate or barberry fails to become infected where winters are mild (Leonard, 2001b). The presence of barberry in this Zone is regarded as a source of the new pathotypes that have been responsible for the loss of effectiveness of the resistance in a considerable number of cultivars (Stakman and Levine, 1922). Pathotypes developed in Zone 4 move south in autumn to re-infect the young wheat crops on the coast of the Gulf of Mexico (Zone 1). Infections of wheat stem rust in Zone 4 may be due to exogenous inoculum from more mature wheat crops further south, or from aeciospores from barberry. The cycling of stem rust from south to north in spring and back south in autumn is known as the 'Puccinia Path' and is not restricted to wheat stem rust (Roelfs, 1982).

Cultural practices and resistant varieties have been used extensively to minimize losses due to the wheat rusts in North America. The use of fungicides has been limited to high-risk situations (Roelfs, 1989). The evolution of the stem rust fungus in North America to overcome the resistances of newly released varieties has been extensively documented over a long period (Stakman and Levin, 1922; Stakman, Stewart and Loegering, 1962).

Stripe rust in Australia

Stripe rust is a recent incursion into Australia, probably from Europe (O'Brien *et al*., 1980). This is based on the similarity of the pathogenicity of the first isolate found in Australia in comparison with a pathotype in Europe.

Since that time, pathotype surveys of stripe rust in Australia have identified 22 virulence changes in the Australian flora, involving the acquisition of pathogenicity against 12 genes for resistances (Wellings, 2007). With few exceptions, these new pathotype were clearly related to pre-existing pathotypes. This conclusion is based on the range of pathogenicity in the new pathotype and on molecular markers (Steele *et al.*, 2001). This suggests that the predominant process of evolution within the Australian flora was through mutation.

The exceptions included an apparent loss of virulence for Yr_2 in two pathotypes, which would impede their survival (Wellings, 2007). A putative exotic incursion was found in 2002 in Western Australia, where stripe rust had not previously been found (Wellings *et al*., 2002). This pathotype was virulent on many Australian varieties and extensive chemical control programmes had to be undertaken. It may have been derived through somatic hybridization, but this is rare in wheat stripe rust (Little and Manners, 1969). This pathotype spread to the eastern states of Australia in 2003.

Conclusions

The deployment of genes for resistance to stem, leaf and stripe rust of wheat has generally failed to deliver sustainable effective resistance in most (all?) countries where this has been attempted. This is due to the ability of these pathogens to acquire the genetic requirements to fully parasitize previously resistant wheat varieties.

There are some significant exceptions to this. They include the Sr_2 and Sr_{26} resistance genes, which have maintained their effectiveness in Australia for a considerable period and through significant epidemics. It is noted that a pathotype virulent against Sr_{26} was identified in glasshouse studies, though nowhere else (Luig and Watson, 1970).

The way forward is to limit the capacity of the wheat rusts to change genetically to acquire the capacity to parasitize previously resistant varieties. This requires an understanding of the processes by which the rust makes these changes.

Generating genetic variability for virulence

The classic processes for generating changes in phenotype frequencies in populations are selection, migration, sexual recombination and mutation. For stem rust, at least, a fifth mechanism, somatic hybridization, has been documented (Watson, 1957).

Selection

The widespread cultivation of a variety carrying an effective gene for resistance to a particular species of wheat rust will ensure that a pathotype having virulence against that variety will have a considerable selective advantage over those pathotypes that do not have the virulence. Consequently, if there is a pathotype with the required virulence in the rust flora it will, most often, rapidly increase in frequency. It is probable that the re-release of the variety Eureka (which has the stem rust resistance gene Sr_6) was such a situation, despite the virulence occurring in a different genetic background (Watson and Luig, 1963).

The ultimate in selection pressure would be to develop varieties that the pathogen cannot develop selection pressure to overcome. This is termed durable resistance and will be considered later in this report.

Migration

Migration is a significant factor in enabling wheat rusts to overcome the effectiveness of host resistance.

It has long been documented that soon after the identification in Australia of a previously unknown pathotype, that pathotype will appear in New Zealand (Hogg *et al.,* 1969; Luig, 1977). This is a distance of about 2000 km across the Tasman Sea and it would appear that urediniospores make this journey on a regular basis.

In early 1969, two stem rust pathotypes with different virulence combinations from the existing stem rust flora were located in different parts of Australia (Watson and de Sousa, 1983). The pathogenicity of these pathotypes was compared with that for the rust flora in Mozambique and found to be similar. Stem rust samples were requested from three east African countries (Angola, Zimbabwe and Mozambique) and compared with the two isolates recovered in Australia. It was concluded that the two exotic isolates found in Australia were identical to two of those from Angola (Watson and de Sousa, 1983). They concluded that, while there was compelling evidence that the two Australian exotic isolates did come from Angola, it is probably a rare event. They also concluded that the crossing of the Indian Ocean and establishment in Australia of rust pathotypes from Angola had only occurred three times in sixty years.

There is now compelling evidence that crossing the Indian Ocean from Africa to Australia is not restricted to wheat stem rust. In 2002, wheat stripe rust was first found in Western Australia (Wellings *et al*., 2002), although it had been in eastern Australia since 1979 (O'Brien *et al*., 1980). The exotic isolate of wheat stripe rust found in Western Australia in 2002 differed in at least five features of pathogenicity from the most common pathotype found in eastern Australia at that time. It also differed from all Australian pathotypes in molecular marker profile (Keiper *et al*., 2003). This isolate is probably not from South Africa, as its pathogenicity is quite different from the current South African pathotypes (Boshoff, Pretorius and van Nierkerk, 2002). It did resemble isolates from Kenya and Ethiopia that were common in the 1990s.

Given the significant hazards to viability of urediniospores in crossing the Indian Ocean and the Tasman Sea, it is not surprising that the new pathotype, Ug_{99}, which developed in Uganda, has quickly migrated to a number of east African and Near Eastern countries. The

most striking feature of this pathotype is that it has virulence against many of the stem rust resistance genes derived from wheat, plus virulence against Sr_{31} from rye and Sr_{38} from *Triticum ventricosum* (Singh *et al*., 2008). Many wheat varieties around the world are susceptible to this pathotype, giving it a significant selective advantage in many wheat growing countries. Ug_{99} spread to Kenya and Ethiopia by 2003. It was found in the Sudan and western Yemen by 2006, and in Iran by 2007 (Nazari, 2008).

There is a precedent for predicting the future incursions of Ug_{99} and its derivatives into other countries. This was the international migration of the Yr_9 virulent pathotype of stripe rust (Expert Panel, 2005). This pathotype was first detected in eastern Africa (Kenya and Ethiopia) in 1986 (Pretorius *et al*., 2000). It then reached Yemen and Syria by 1991, Iran by 1994 and was into northern India by 1997. Modelling of the prevailing winds during the wheat growing season suggests that the stem rust pathotype Ug99 will go beyond its current distribution and migrate in the general direction taken by the Yr_9-attacking pathotype of stripe rust (Singh *et al*., 2006, 2008).

Other international migrations of rust fungi include the movement of barley yellow rust from South America to North America (Dubin and Stubbs, 1986).

Sexual recombination

As discussed under *Life cycle*, sexual recombination in all *formae speciales* of *Puccinia graminis* requires the presence of barberry to be completed. Barberry is a European plant species that was introduced into North America for use as hedgerows (Peterson *et al*., 2005a. It has since invaded natural ecosystems. The sexual cycle can only be completed in areas with cold winters, such as northern USA and Canada (Leonard, 2001a). Barberry is not grown in areas of Canada in close proximity to the major wheat growing areas (Roelfs, pers. comm.).

Sexual recombination among pathotypes of wheat stem rust has been demonstrated to produce new combinations of virulence (Roelfs and Groth, 1980; Wilcoxson and Paharia, 1958). Sexual hybridization has been demonstrated between pathotypes of *Puccinia graminis* f.sp. *tritici* and of rye stem rust, *Puccinia graminis* f.sp. *secalis*. This has resulted in the development of recombinant pathotypes with ranges of pathogenicity differing from those in the existing stem rust flora (Stakman, Levine and Cotter, 1930; Watson and Luig, 1962).

The new pathotypes produced in northern USA can be wind transported to southern USA, where they infect winter wheat crops that then provide inoculum to be wind transported north via the 'Puccinia path' in the following spring (Roelfs, 1982). It is also considered that the presence of barberry in wheat growing areas would act as a focus of inoculum for the initiation of epidemics in areas where stem rust would not otherwise survive cold winters (Leonard, 2001a).

In 1916, a stem rust epidemic in northern USA and in the Canadian Prairies resulted in a production loss of 300 million bushels (Roelfs, 1982). These losses curtailed food supply during wartime and provided the impetus for a major investment by the USA and Canada to control wheat stem rust. A component of this programme was to eradicate barberry in the USA. It is interesting to note that the disadvantage of growing barberry and small grains in near proximity had long been understood in Europe, where in 1660 a law had been passed in Rouen, France, to outlaw the growing of barberry.

By 1933, over 18 million barberry plants had been destroyed, with about 75% of these being self-sown plants away from the original plantings (Leonard, 2001a). North American species, including Allegheny barberry (*Berberis canadensis*) and Fender's barberry (*Berberis fendleri*), were also included in the programme as they were found to be infected by stem rust.

About 98% of the barberry eradication area had been cleared by 1972, and by 1990 all funding for this programme ceased despite scientific concerns that barberry would re-establish itself if the programme was discontinued, as small pockets of barberry remained (Leonard, 2001a).

The effect of the eradication programme was to reduce the frequency of stem rust epidemics. In the first decade of the eradication programme 5.1 of the thirteen northern states that participated in the programme experienced a loss due to wheat stem rust of 1% or greater each year (Figure 10). The frequency of rust epidemics had dropped to about three states per year in the next decade and continued to drop for the remainder of the millennium (Figure 10).

Table 4. Average number of stem rust pathotypes found per year during the barberry eradication programme (1917 to 1990).

Decade	Number of pathotypes[(1)]
1918–1927	17.5
1928–1937	10.7
1938–1947	6.5
1948–1957	7.7
1958–1967	7.5
1968–1977	5.2

NOTES: (1) Pathotypes that comprised more than 0.6% of the isolates collected. SOURCE: Reproduced from Roelfs, 1982.

Another beneficial outcome of the barberry eradication programme was the reduction in diversity in the North American wheat stem rust flora (Table 4). Roelfs (1982) suggested that the extent of the loss of diversity in the USA stem rust flora demonstrated in this table may be an underestimate of the actual loss of diversity as a smaller number of collections were made during 1918–1927 (1 541) than during 1968–1977 (18 403). He argued that the number of pathotypes found will increase with the number of samples studied. It is also argued that the eradication programme has stabilized the composition of the rust flora and consequently decreased the number of pathotypes to which a wheat variety must be resistant.

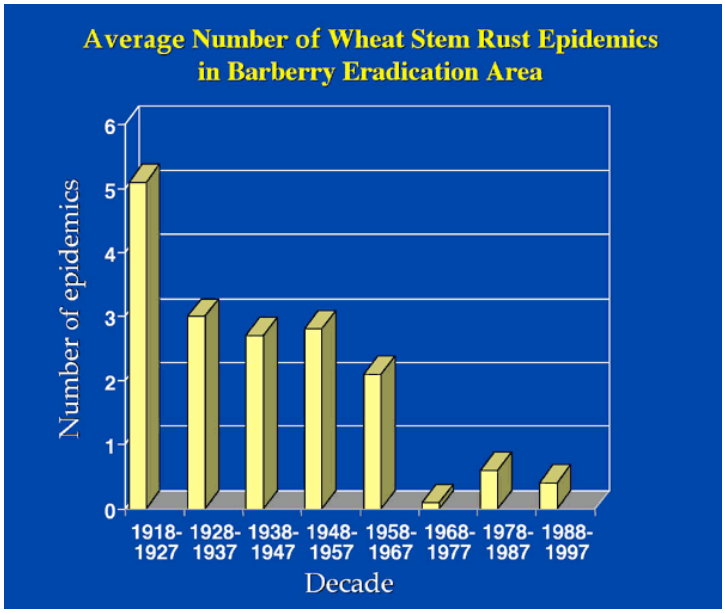

Figure 10. Frequency of wheat stem rust epidemics; average number of states within the barberry eradication area in which wheat yield losses to stem rust exceeded 1% in any given year. Reprinted from Roelfs, 1982.

In the last three decades the number of isolates collected using a standard sampling methodology has declined substantially, reflecting the great reduction in the amount of stem rust inoculum in North America (Figure 11; Leonard, 2001a). Consequently the probability of an epidemic will also decline sharply.

It must be stressed that, although a major source of genetic variation for the stem rust flora of North America had been eliminated by 1933, a new pathotype (15B) emerged in that year, which caused major epidemics in 1953 and 1954. This pathotype was isolated from barberry in Iowa, a considerable distance from any wheat growing area.

The continued threat to the North American wheat crop due to rust on barberry was highlighted by the constant magnitude of the variation in pathotype diversity in isolates obtained from barberry from 1912 to 2002 in Minnesota, USA (Peterson *et al.*, 2005b). This study also noted an increase in pathotype diversity in isolates from wheat between 1990 and 2000. This might suggest that following the cessation of the barberry eradication programme there has been an increase in the North American barberry population and a consequent increase in the contribution that sexual recombination makes to pathotype diversity in the North American stem rust flora.

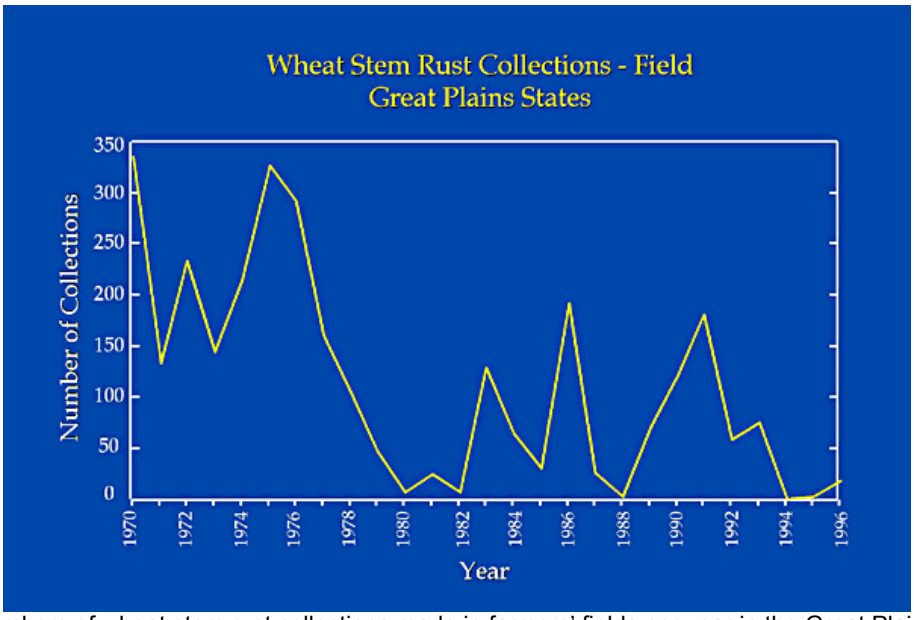

Figure 11. Numbers of wheat stem rust collections made in farmers' fields per year in the Great Plains from Texas to North Dakota. Reprinted from Leonard, 2001a.

Conclusions: sexual recombination

The availability of the sexual cycle to the wheat stem rust not only greatly enhances pathotype diversity but also is a source of inoculum in early spring in areas where stem rust would not normally overwinter due to freezing temperature. These two factors greatly enhanced the frequency with which damaging stem rust epidemics have occurred, and made the breeding of new varieties more difficult due to the range of virulence in the stem rust flora and the rapid loss of effectiveness of genes for resistance to stem rust.

It is also stressed that the stem rust fungus has not lost the ability to generate new pathotype variability. Also, there are indications that the cessation of the barberry eradication programme may have allowed barberry to spread again and increase its contribution to the pathotype diversity of the North American stem rust flora. While the evidence for this is tentative at this time, it seems probable. This is based on the near impossibility of eradicating a plant that has extensively invaded natural ecosystems, and the ability of barberry to invade other areas. This has been documented for Minnesota (Peterson *et al.,* 2005b).

Mutation

In many areas of the world, wheat rusts have demonstrated the capacity to acquire apparently new virulence where the alternate host is not available (stem and leaf rust), or where there is no known alternate host (stripe rust). This would suggest that one or more of migration, somatic hybridization or mutation is a mechanism for generating this variability. Selection on hosts that have the corresponding resistance gene will most often ensure that the new pathotype establishes in the stem rust flora.

It has been possible to infer the evolutionary drivers for pathotype diversity, where sexual recombination is not available, by comparing the virulence phenotypes with those of other

members of the rust flora. In Australia, using this methodology, it was noted that the evolution of wheat stem rust was largely manifest through variation within standard stem pathotypes, rather than among them, although over fifty different pathotypes had been identified (Luig and Watson, 1970). For example, the standard pathotypes 21 and 34 tended to develop a change in virulence in response to the commercial production of a variety or varieties with the corresponding gene(s) for resistance. New standard races did not develop.

Somatic hybridization has been found to result in genetic recombination that often involves more than one change in virulence. Luig and Watson (1970) concluded that the most probable explanation for their observation was mutation, as only single changes were most often observed, followed by strong selection pressure for the mutation due to the commercial production of varieties with the corresponding genes for resistance.

One exception to this observation has been rare incursions, which will be considered later (Watson and de Sousa, 1983).

Another exception in the evolution of the Australian stem rust flora was the isolation of pathotypes with a single virulence change for virulence against genes for resistance that had not been deployed commercially. These resistance sources were einkorn (*Triticum monococcum*) and vernal emmer (*Triticum turgidum*) (Luig and Watson, 1970). They concluded that the identification of virulence on einkorn and emmer was due to ongoing mutation for virulence on these wheats but the resulting pathotypes remained at a low frequency as there was no selection pressure for them. These virulences stayed in the stem rust flora due to iterative mutation or as there was little or no fitness to loss the pathogen in having this pathogenicity (Thrall and Burdon, 2003; Stearns, 1992; Bell, 1997).

Compelling evidence for mutation as a powerful mechanism for generating pathotype variability was obtained from an analysis of the stripe rust flora in Australia (Steele *et al*., 2001). Following the incursion of stripe rust into Australia in 1979, more than 20 pathotypes had evolved. A sample of pathotypes that had evolved between 1979 and 1991 was examined for polymorphisms using sixty random amplified polymorphic DNA (RAPD) markers. No polymorphisms were found. This study was conducted on pathotypes that evolved prior to the putative incursion of pathotype 134E16A+ from Africa (Wellings, 2007). The same primers applied to two pathotypes from the United Kingdom showed that three percent to be polymorphic. This lack of polymorphism across the stripe rust genome for Australian stripe rust pathotypes provided compelling evidence that the virulence changes were generated through natural mutation.

It is considered that all genes within an organism will change due to mutation. The frequency of these changes varies greatly among loci. This suggests that there is an iterative process for the generation of virulence against genes for resistance to the wheat rusts. With very few exceptions, pathotype surveys do not detect specific virulence changes unless the corresponding gene for resistance has been deployed commercially. The exceptions include the virulences in the rust flora prior to the deployment of resistance genes through breeding commercial wheat varieties (Waterhouse, 1929; Stakman and Levine, 1922). Another example is the infrequent identification of virulence of einkorn and emmer considered above. It is concluded that such virulences cause little loss of fitness for the pathogen (Burdon and Thrall, 2003; Stearns, 1992; Bell, 1997). The converse is that those virulences that are detected by pathotype surveys only, or mainly, subsequent to the commercial deployment of the corresponding gene for resistance result in a substantial loss of fitness for the pathogen.

Virulence against the gene for stem rust resistance, Sr_{26} has been identified in glasshouse studies but has never been identified from field collections, although this gene has been commercially deployed over substantial areas and during severe epiphytotics. It appears plausible to conclude that virulence against Sr_{26} may result in such a large genetic loss of fitness for the pathogen that the virulences cannot be sustained in the pathogen flora notwithstanding the significant commercial deployment of Sr_{26}.

Fitness loss due to the expanding of virulence in a pathogen has been demonstrated for a number of non-rust-host-pathogen co-evolutionary systems (Cruz *et al.*, 2000; Jenner *et al.,* 2002; Huang *et al.,* 2006). A negative correlation has been demonstrated between virulence and spore production for flax rust (Thrall and Burdon, 2003). More recently, significant

variation in loss of fitness due to different genes for virulence in the absence of the corresponding genes for resistance has been demonstrated for wheat stripe rust (Bahri, et al., 2009). In general, the fitness cost is insufficient to preclude the rapid multiplication of a pathotype that has a selective advantage due to its ability to parasitize a previously resistant host (Frank, 1993).

Under epiphytotic conditions stem rust can produce such huge amounts of inoculum (Stakman, 1957) that it must be concluded that, even with the lack of knowledge of natural mutation rates for avirulence to virulence in functional diploids, it is probable that natural mutations for virulence against all race-specific resistance genes do occur regularly. Luig and Watson (1970) noted that the development of varieties with broadly based resistance (number of genes for effective race specific resistance) did not give permanent control of stem rust in Australia. This may suggest that, under epiphytotic conditions, there is sufficient opportunity for the simultaneous generation of virulence against more than one resistance gene to occur.

The persistence at undetectable frequencies of pathotypes with 'new' virulences in the rust flora where there is no variety-based selection advantage will depend on the loss of fitness in the pathogen due to the virulence gene.

Intervention options: mutation

The actual breakdown event (when a new pathotype is detected on a previously resistant cultivar) will require the congruence of a number of events: the development of the new pathotype by mutation, the deposition of the new pathotype on the variety for which it has the required virulence, successful infection, multiplication on the previously resistant cultivar and spread to many other individual plants. The probability of all these events occurring concurrently is the product of the probability that individual events occur. The probability of the simultaneous breakdown of two effective genes for resistance in the one variety is the product of the probability for the breakdown of each gene for resistance separately. This suggests that the commercial deployment of varieties with two or more effective genes for resistance will reduce the probability of a breakdown event (Watson and Singh, 1952).

Consequently, the probability of a breakdown event occurring is dependent on the rust population size (the number of urediniospores produced) as large populations are necessary to increase the probability that the breakdown event will occur.

Intervention that greatly reduces the population size of the pathogen will have a concomitant impact of the probability of the low frequency breakdown event occurring. Such a strategy has been in place in northeastern Australia over the last forty hears. This is based on having varietal release conditional on meeting high standards of resistance to all three species of rust and advocating to farmers that they cease growing a variety once it has become susceptible. No substantial rust epidemics have been experienced in that time and the rate of breakdown of resistant varieties has been greatly reduced (Zwer, Park and McIntosh, 1992; Platz and Sheppard, 2007; Park, 2007).

Somatic hybridization

Johnson (1953) suggested that new pathotypes of stem rust may be developed through the fusion of dikaryotic hyphae and subsequent re-assortment of the nuclei in vegetative hyphae. This was partially confirmed through the mixing of pathotypes of stem rust differing in urediniospore colour and pathogenicity (Watson, 1957). Pathotypes with different combinations of urediniospore colour and pathogenicity were recovered. The capacity of stem rust to generate new pathotypes through the fusion of hyphae has now been demonstrated for a range of pathotypes in North America as well as Australia (Nelson, Wilcoxson and Christensen, 1955; Nelson, 1956; Ellingboa, 1961).

A number of the above authors reported the identification of a number of different pathotype from the culture of two parental pathotypes. The re-assortment of whole nuclei was considered inadequate to explain the diversity in the derivative population (Watson,

1957; Ellingboa, 1961). They suggested a parasexual cycle in which there is exchange of whole chromosomes. This would require fusion of the dikaryotic nuclei followed by chromosomal re-assortment when the diploid cell divided to re-establish the dikaryotic condition. Hartley and Williams (1971) suggested, on theoretical grounds, that chromosomal re-assortment may arise by accident due to the close proximity of the nuclei during their synchronized mitotic division.

The efficacy of somatic hybridization in generating new pathotypes was examined by comparing the frequency and type of recombinants that arose from sexual and asexual recombination (Roelfs and Groth, 1980). The frequency of recombinant pathotypes observed for sexual reproduction was 23.5% while that for somatic hybridization was 0.07%. Re-assortment among alleles for pathogenicity was random, suggesting chromosomal synapses. Non-random assortment of pathogenicity was observed for somatic hybridization, suggesting that there had not been exchange of genetic material between homologous chromosomes.

Somatic hybridization in wheat stem rust has also been a factor in the evolution of new pathotypes in nature (Watson, 1981). The Australian pathotype 34-2, 11 was first detected in 1957 and gave rise to many pathotypes of economic significance. Its origin by somatic hybridization was determined on the basis of its virulence gene combination, as the only plausible explanation for its origin was that it was derived from two other pathotypes. Also, a distinctive group of stem rust pathotypes largely found on the grass *Agropyron scabrum* was determined to have been derived from somatic hybridization between *Puccinia graminis* f.sp. *tritici* and *Puccinia graminis* f.sp. *secalis* (Luig and Watson, 1972). Additional evidence for these conclusions in both cases was provided by isozyme phenotyping (Burdon, Marshall and Luig, 1981; Burdon *et al*., 1982).

Somatic hybridization has been demonstrated under controlled conditions for stripe rust (Little and Manners, 1969; Goddard, 1976; Wright and Lennard, 1980). However, the results, under controlled conditions for leaf rust had been equivocal at best (Bartos *et al*., 1969; Barr, Caldwell and Amacher, 1964).

More recently, a new pathotype of leaf rust was isolated in Australia whose origin could not be attributed to natural mutation in an existing pathotype (Park, Burdon and Jahoor, 1999). The combination of virulescences in the pathotype suggests somatic hybridization between two pathotypes prevalent at the time the new pathotype was found. Its hybrid origin from the two pathotype was confirmed through isozyme and molecular marker analysis.

Detailed examinations of the evolutionary processes for several rust floras in response to the development and cultivation of rust resistant varieties have failed to identify somatic hybridization as a significant evolutionary force. This suggests that, providing that sexual recombination on barberry is not a factor, natural mutation is the predominant generator of new variability for pathogenicity. Also, migration plays a significant global role and, as such, will require global cooperation and investment to minimize its impact.

Options for controlling the wheat rusts

Historically the preferred option for controlling the wheat rusts has been through the development of resistant varieties. It is apparent that the outcome from this breeding investment has not been the rust-free wheat growing environment that was envisaged. This is due to the ability of the rust to change genetically to parasitize most of the resistant varieties released. While the expectations have not been met, valuable lessons have been learned and some very encouraging options for the future control of wheat rust have been identified.

Eradication of barberry and the consequent minimization of the sexual stage as a factor in pathotype evolution in North America had a very large impact in reducing the damage done to wheat by stem rust in North America. One factor that appears to have been missed by policy-makers (thought not by the scientific community) is the need for continued vigilance (and investment) to ensure that the North American barberry population does not increase and establish in close proximity to wheat growing areas (Leonard, 2001a; Peterson *et al*., 2005b).

There is now compelling evidence that rust resistance genes for which the rust fungus has the capacity to overcome can be deployed in such a manner as to provide high and continuing levels of protection to the wheat crop in areas that have, historically, been very prone to damaging stem rust epidemics (Park, 2007; Platz and Sheppard, 2007; McIntosh, 2008). Implicit in these strategies are the use of minimum rust resistance standards for release, promptly removing varieties from commercial production as soon as they become susceptible, and deploying varieties with at least two effective genes for resistance. The first two strategies greatly reduce the population size of the local rust flora so that the probability of a new mutation establishing in the rust flora is very low. Deploying varieties with at least two effective genes for resistance to a particular wheat rust species will greatly reduce the probability of the development of pathotypes that can parasitize previously resistant varieties.

The examination of the mechanisms for the evolution of new pathotypes of wheat rusts and the historical impact of these mechanisms has identified that there are some resistances against which the stem rust pathogen has not developed virulence. Examples of this are the Australian cultivars Hopps (released in 1957) and Kite (released in 1973) (Table 3).

Varietal deployment

Varietal deployment to minimize the probability of the breakdown of resistance has two important components: minimum rust resistance standards for release, and removal from commercial production of varieties when they become susceptible.

Minimum rust resistance standards

The purpose of minimum disease resistance standards (MDRS) for varietal release is to contribute to minimizing the size of the pathogen population and, as a consequence, reduce the probability that the rust fungus will mutate naturally and establish in the rust flora virulent pathotypes that can parasitize previously resistant varieties.

Action to develop a national set of MDRS in Australia for many wheat diseases, but particularly the wheat rusts, commenced at the national conference for wheat breeders and pre-breeders (Fourth Assembly of the Wheat Breeding Society of Australia) in 1984. The development of voluntary guidelines for the rusts continued through meetings of the Steering Committee of the National Wheat Rust Control Program. The standards agreed to were based on the adult-plant reaction of a particular variety to a specific pathotype and varied among regions depending on the level of risk in each region (Wallwork, 2007). The standards were a voluntary code and compliance with them varied. There was complete compliance with this code for release in the most rust-prone region of Australia (the northeast wheat growing region). This region has, over the 25 years, experienced little loss in production or quality due to rust (Park, 2007; Platz and Sheppard, 2007). This is partly due to the implementation of MDRS for release; partly due to the removal from commercial cultivation of varieties when they become susceptible; and partly due to the high level of cooperation between the wheat breeders in the region and the officers of the National Wheat Rust Control Program, conducted by Sydney University (Brennan, 1994). The success in north-eastern Australia has knock-on benefits for other areas of Australia, including Victoria and South Australia (Wallwork, 2007).

Concern has been voiced that the movement of Australian wheat breeding from the public sector to the private sector may reduce commitment to MDRS for release (Park, 2008).

Removal of susceptible cultivars from cultivation

The rigorous development and implementation of MDRS for variety release will go a long way to reducing inoculum levels, but much of the value of this may be lost when varieties are permitted to continue in commercial production once they have become susceptible (Wallwork, 2007). One example of this is the continual commercial production of the variety Oxley after it became susceptible to stem rust in northeastern Australia in 1976 (Platz and Sheppard, 2007). The pathotype with virulence against Oxley had developed in southern Australia in 1973, but did not migrate to northeast Australia until 1973. This resulted in a significant rust epidemic in northeast Australia on Oxley in 1983, and subsequently a pathotype that had an additional gene for virulence that allowed the Oxley pathotype to parasitize cv. Cook was found in the spring of 1984.

Samples of the stem rust on Cook were despatched to the Sydney University National Cereal Rust Control Program (NCRCP) for pathotype identification and the officers of the NCRCP advised immediately that they could confirm that a Cook-attacking pathotype had evolved. Also, seed merchants who were selecting seed crops to plant the 1985 crop were advised. Confirmation of the loss of effective resistance in Cook triggered a concerted media campaign to advise wheat growers of the dangers of continuing to grow Cook. Seed merchants, at considerable cost, divested themselves of obligations to acquire Cook seed and sought to acquire seed of alternative varieties. The area grown to Cook in Queensland dropped from 25% of the total wheat acreage in 1984 to 5% in 1985, and to almost nothing in

1986. At the same time the area sown to Cook across the whole of northeastern Australia dropped from 20% to 11%, virtually disappearing by 1986 (Platz and Sheppard, 2007).

The result of cooperation between the wheat breeders in northeast Australia and the NCRCP in developing new varieties; understanding the pathotype flora; compliance with realistic MDRS for release; and the removal from cultivation of varieties once they become susceptible, has been that there has been no loss of production due to stem rust and no loss of effectiveness of genes for stem rust resistance for 25 years in northeast Australia. This is in stark contrast to experiences in the first two decades of the commercial production of stem rust-resistant varieties in Australia (Table 3).

Durable disease resistance

The ability of many plant pathogens to change to be able to parasitize previously resistant varieties has long been known (Ward, 1902). This phenomenon has received critical analysis in potatoes bred for resistance to late blight (caused by *Phytophthora infestans*) due to the catastrophic loss of production and consequent social dislocation due to this disease. It was noted that there was considerable variation in the extent of yield loss among potato varieties during epiphytotics of late blight (van der Plank, 1963). Many varieties with seedling resistance to late blight became susceptible to this disease and sustained substantial productivity losses (Black, 1960). Varieties that had been bred for resistance that was not based on seedling reaction tended to experience far less loss of productivity and the resistance was maintained (van der Plank, 1968). Van der Plank (1963) called this resistance 'horizontal' because it was effective against all pathotypes of the disease-causing organism. Resistances that could be overcome were termed 'vertical', as they were effective against specific pathotypes only. Horizontal resistance was considered to remain effective indefinitely, not be evident in seedlings, to be under polygenic control, manifest itself as a slow rate of disease built up in adult plants, and to be partial in nature (van der Plank, 1968).

It was suggested that the horizontal resistance in potatoes to late blight would be overcome, but it would appear to be long lasting, as a single mutation for virulence will only overcome a portion of the resistance (Toxopeus, 1956; Niederhauser, 1962). History does not appear to have sustained the view that horizontal resistance in potatoes would be overcome by the late blight pathogen.

Difficulties with relating the nomenclature proposed by van der Plank (1963, 1968) to the phenomena and the concern of assumption of indefinite efficacy of 'horizontal' resistance has led to the general acceptance of the nomenclature proposed by Johnson (1981). He proposed that apparent long-lasting resistance should be called 'durable' and the resistance that can be broken down as 'race-specific' resistance. This convention will be here adhered to. The definition applied here for durable resistance is the one proposed by Johnson (1981, 1988):

> 'resistance that remains effective when deployed over extensive acreage and time, in an environment favourable for the disease'.

Much of the emphasis in breeding for stem rust resistance through much of the twentieth century has focused on seedling reaction as a predictor of adult-plant resistance. There has been limited deployment of varieties whose rust resistance is based on seedling susceptibility and adult-plant resistance and, as a consequence, there is limited information on which to base the identification of durable resistance.

Adult-plant resistance to the wheat rusts was documented in the nineteenth century for stem rust (Farrer, 1898). Systematic studies to understand durable resistance in wheat to leaf rust were undertaken by Calwell and co-workers early last century. They postulated the following mechanisms for durable resistance: reduced infection (Caldwell and Stone, 1936), smaller pustule size, and longer period between infection and sporulation (Caldwell *et al*., 1957; Caldwell, 1968).

There has been a tendency to use adult-plant resistance (coupled with a susceptible seedling reaction) as a surrogate definition for durability. History would suggest that there is a strong correlation between adult-plant resistance and durability, but there are exceptions.

The solely adult-plant leaf rust resistances Lr_{12}, Lr_{22a} and Lr_{22b} have been found to be race specific (Williams *et al*., 2006). Also, some adult-plant stripe rust resistances have also been race specific when deployed commercially (Johnson, 1992, 2000). Consequently, some caution must be exercised in claiming that a resistance will be durable when the only evidence is the solely adult-plant manifestation of the resistance.

It has been observed that race-specific adult-plant resistance can be clearly distinguished from known durable resistance by reaction type, as race-specific resistance has a resistant reaction on the adult plant while durable adult-plant resistance has a susceptible type reaction (Williams *et al*., 2003).

This is not so clear cut for stripe rust, as durable stripe rust-resistant varieties tend to exhibit a less than fully compatible reaction type as adults (Singh, *et al*., 2001). However, the first stripes to develop on durable resistant varieties tend to have a susceptible to moderately susceptible reaction type.

Durable leaf rust resistance

The gene for leaf rust resistance on chromosome 7D, Lr_{34} (Dyck, 1987) has met the criteria proposed by Johnson (1981) for durability (Singh, 1993). It tends to produce fewer, smaller uredinia, as well as having a longer latent period (Singh, Huerta-Espino and Williams, 2001). Lr_{34} is either very closely linked or pleiotropic to the slow-rusting stripe rust resistance gene Yr_{18} (Singh, 1992; McIntosh, 1992). Lr_{34}has been deployed in a number of CIMMYT-bred varieties, including cv. Parula. Genetic analysis of the leaf rust resistance of this variety has established that there are, in addition to Lr_{34}three other loci concerned with leaf rust resistance (Table 5). Together, these loci account for 53% of the variation in the mapping population for leaf rust resistance (Singh, Huerta-Espino and Williams, 2001).

Another named gene, Lr_{46}, is included in the genes for leaf rust identified for cv. Parula. This gene is also found in another CIMMYT variety, Pavon 76 (Singh, Huerta-Espino and Williams, 2001). Pavon 76 is also considered to have durable leaf rust resistance (Singh, Huerta-Espino and Williams, 2001). Cv. Pavon 76 has another two loci for leaf rust resistance in addition to Lr_{46}. Two of these loci also have stripe rust resistance associated with them, as do three of the loci in Parula (Table 5).

Williams *et al*. (2006) assert that the presence of one or two of the genes for slow rusting would be insufficient to provide adequate protection against loss of productivity. They recommend that four to five genes for slow rusting are required to provide the near immunity that has been noted for some CIMMYT wheats (Singh and Rajaram, 1994).

The apparent wealth of genetic resources to develop wheat varieties with adult-plant leaf rust resistance has been demonstrated by the location of QTLs for this trait at five loci in a cross involving a synthetic wheat and a bread wheat breeding line, although one locus differed from that reported by Williams *et al*. (2006) (Chu *et al*., 2009).

A recent review of the progress in identifying adult-plant leaf rust resistance compiled a list of eighteen apparently different QTLs for adult-plant leaf rust resistance in wheat (Roswarne *et al*., 2008). These authors identified an additional two loci in the Mexican cultivar, Attila.

In durum wheat, the varieties Playero, Planeta and Trile had three independently inherited additive genes for slow rusting, while Piquero, Amic, Bergand, Taqua and Knipa had two genes with additive effect (Herrera-Foessel *et al*., 2008).

The abundance of apparently different QTLs for slow rusting and adult-plant resistance in wheat and the progress in developing molecular tools to facilitate breeding suggests that the prospects for sustained global wheat leaf rust resistance are very good.

Table 5. QTLs for slow rusting to leaf and stripe rust in cvs. Parula and Pavon 76 (adapted from Singh, Huerta-Espino and Williams, 2001).

Cultivar	Chromosome	Marker	Disease Severity Reduction (%)		Genes	Source
			Leaf Rust	Stripe Rust		
Pavon 76	1BL	Wms259	35	27	*Lr46, Yr29*	Pavon
	4B	Wms495	18	15		Pavon(2)
	6A	Wms356	14	18		Avocet
	6B	PaggMcaa	-	18		Pavon(3)
	3BS	PacgMcgt	-	11	*Yr30, Sr2*	Pavon
Parula	7DS	Wms295, Ltn(1)	56	46	*Yr34, Yr18*	Parula
	7D or 7B	Pcr156	29	-		Parula
	1BL	Wms259	15	16	*Lr46, Yr29*	Parula
	Unknown	PaagMcta	22	14		Parula
	3BS	Glk2	-	12	*Yr30, Sr2*	Parula

NOTES: (1) Leaf tip necrosis, a morphological marker linked to Lr34. (2) Williams et al. (2006) appear to attribute this to 4BL of Avocet. (3) Williams et al. (2006) appear to attribute this to 6BL.

Durable resistance to stripe rust

The relatively short effective life of most stripe rust resistance genes and the relative abundance of putative durable resistance have encouraged breeders to breed for field resistance (a synonym for adult-plant resistance; Table 6; Boyd, 2005). There are a considerable number of wheat varieties where stripe rust resistance has remained effective over an extended period in climatic conditions favourable to stripe rust. These include the French variety 'Camp Remy' (Mallard *et al*., 2008); 'Cappelle des Prez' and 'Hybride de Bersee' (Johnson and Law, 1975); 'Gains', 'Nugaines' and 'Luke' (Milus and Line, 1986); as well as a number of CIMMYT varieties (Williams *et al*., 2006). The adult-plant resistance in the Australian breeding line CSP44 is also considered as a candidate for a durable stripe rust resistance (Khanna, Bansal and Saini, 2005).

Information is available for a number of specific resistance genes, notably *Yr18* and *Yr29*, that appear to fit the combined criteria of Johnson (1981) and Williams *et al.* (2006) for durability. The genetic and molecular analysis of these sources of resistance has been summarized (Boyd, 2005; Table 6). This summary suggests that a minimum of 20 different loci appear to contribute to slow rusting for stripe rust. This figure is derived by making the conservative assumptions that there is only one locus per chromosome arm for durable stripe rust resistance and the loci identified to the whole chromosome are the same as those located on one of the arms of the chromosome. This is quite a large number considering so few cultivars have been analyzed.

The genetic control for a number of durable resistances that have been identified in cultivars grown in the USA, namely for cvs. Gaines, Nugaines, Luke, Duchamp and Stephens, has been studied (Chen *et al*., 1998). These have not been located on chromosome arms. The resistance in these cultivars is mainly quantitative, additive and recessive.

This suggests that, based on the opinion of Williams *et al.* (2006) that 4 to 5 loci for slow rusting are required for high levels of protection from stripe rust, there are abundant genetic resources to provide effective protection from stripe rust for the global wheat crop. This allows for some of the identified loci to confer race-specific resistance.

Durable stem rust resistance

Slow rusting in wheat was reported very early in wheat breeding (Farrer, 1898). Observations of this nature have continued throughout the history of wheat breeding. In Australia, although the resistance to stem rust in most varieties failed to last, two varieties have remained resistant and would appear to meet the Johnson (1981) criteria for durability. These are cvs. Hopps and Kite (Macindoe and Walkden-Brown, 1968; Zwer, Park and McIntosh, 1992).

Hopps is derived from the American variety Hope, whose seedling resistance (*Sr17*) became ineffective in Australia in 1959 (McFadden, 1930; Macindoe and Walkden-Brown, 1968).

The stem rust resistance in variety Kite, *Sr26*, is derived from an *Agropyron elongatum* parent by introgressing a large segment of *Agropyron elongatum* chromosome into bread wheat (Knott, 1961). There is no recombination in this segment, so it is not possible to determine if *Sr26* is a single gene or more than one gene for resistance (McIntosh, Dyck and Green, 1977). This resistance has been deployed in a number of Australian wheat varieties and was exposed to the significant stem rust epiphytotic in 1973 (Zwer, Park and McIntosh, 1992). Virulence has been identified against *Sr26* in the greenhouse, but not in commercial crops, which may suggest that virulence against the gene causes a very substantial selective disadvantage to the pathogen, which has prevented it from establishing in the Australian stem rust flora.

The stem rust resistance in Hope has also been reported to be long lasting in commercial production. The Hope/Chinese Spring chromosome substitution lines were used to determine the chromosomal location of the adult-plant stem rust resistance in Hope (Brennan, 1975). Lower numbers of uredinia were observed for Hope substitution lines 3B, 6B and 7B. The short arm of Hope chromosome 3B was later found to be the location of the durable stem rust resistance gene *Sr2* (Hare and McIntosh, 1979).

The varieties and breeding lines Hope, Hopps, Marquillo, Thatcher, Chris, Bonza, Minnesota II-50-17 and MRFY were considered to have long-lasting stem rust resistance in north America (Knott, 1982). These lines were intercrossed and selections tested for stem rust resistance in an international set of environments. Three of these lines exhibited resistance against a wide range of rust pathotypes. Genetic studies suggested that the adult-plant stem rust resistance was recessive and a number of loci were involved. The adult-plant resistance in Thatcher was attributed to *Sr12* (3BS; McIntosh, Partridge and Hare, 1980) and *SrTc* (Nazareno and Roelfs, 1981).

Table 6. QTLs for adult plant stripe rust resistance in wheat (adapted from Boyd, 2005).

Chromosomal location of QTL	Source of resistance	QTL Reference	Chromosomal location of QTL	Source of resistance	QTL Reference
2D (*Yr16*)	Hybride de Bersee	Worland and Law, 1986	7DS (*Yr18*)	Parula	
3BS	Opata85	Singh, Huerta-Espino and Rajaram, 2000	2BS	Kariega	Ramburan *et al.,* 2004
3DS	Opata85		7DS	Kariega	
5DS	Opata85		3BS	Oligoculum	Suenaga *et al.,* 2003
7DS (*Yr18*)	Opata85		4BL	Oligoculum	
2DS(1)	Opata85		4DL	Oligoculum	
7AL	Opata85		5BL	Oligoculum	
2BS#	Opata85	Borner *et al.,* 2002	6BS	Oligoculum	
3DS	Opata85	Boukhatem *et al.*, 2002	7BS	Oligoculum	
5AL	Opata85		7DS	Oligoculum	
6DL	Opata85		1BL (*Yr29*)	CD87	Bariana *et al.,* 2001
7DS	Opata85		2DS	Katepwa	
2AL	Camp Remy		7DS (*Yr18*)	CD87	
2BL	Camp Remy		1BL (*Yr29*)	Attila	Roswarne *et al.,* 2008
3BS (*Yrns-B1*)	Lgst79–74	Borner *et al.,* 2000	2BS	Attila	
1BL (*Yr29*)	Pavon76	Williams *et al.,* 2006	2BL	Attila	
3BS (*Yr30*)	Pavon76		7BL	Attila	
4B	Pavon76		Not assigned	Attila	
6A	Avocet S		6AS	Express	Lin and Chen, 2009
6B	Pavon76		3BL	Express	
1BL (*Yr29*)	Parula	Singh, Huerta-Espino and Williams, 2001	1BL (*Yr29*?)	Express	
3BS (*Yr30*)	Parula				

NOTE: (1) The QTL on 2BS coincides with the location of *Yr27* in the ITMI population (R. McIntosh, pers. comm.).

The current upsurge in global concern at the threat posed by stem rust to food security has resulted in increased searching for adult-plant stem rust resistance. A number of breeding lines and varieties from the CIMMYT programme have exhibited high levels of adult-plant resistance that cannot be explained by the current understanding of the genetics of adult-plant stem rust resistance (Singh, 2008; Singh *et al.*, 2009). Also, the released Canadian cvs. AC Cadillac and Pearce had effective resistance against the Ug99 lineage, which is probably due to a combination of an unidentified gene on chromosome 6DS in combination with Lr_{34} and/or Sr_2 (DePauw *et al*., 2009). It is not known if this is due to seedling-expressed resistance or adult-plant resistance only.

A number of Iranian wheat land races were evaluated for seedling susceptibility and adult-plant resistance to wheat stem rust (Nazari *et al*., 2008). A number of these exhibited useful levels of adult-plant stem rust resistance.

Progress in the genetic dissection of adult-plant stem rust resistance has demonstrated that there may be a considerable number of loci involved (Table 7; Bansal *et al.,* 2008; Kaur *et al.,* 2009). Also, one of the varieties identified by Knott (1982), cv. Chris, has two genes for adult-plant resistance (Singh and McIntosh, 1987). This suggests that there may be considerably more genetic variation for adult-plant rust resistance as the genetic analysis has so far been limited to only a few sources of resistance.

Global threat of wheat stem rust

The evolution around 1998 in east Africa of a highly aggressive pathotype of wheat stem rust with the ability to parasitize many previously resistant varieties has emphasized the threat wheat stem rust poses to global food security (Pretorius *et al.,* 2000). This pathotype, known as Ug99 and TTKSK, has virulence against the following genes for stem rust resistance (Sr) genes: 5, 6, 7b, 8a, 8b, 9a, 9b, 9d, 9e, 9g, 11, 15, 17, 30, 31 and 38 (Expert Panel, 2005). The most significant aspect of the pathogenicity of Ug99 is its virulence against Sr_{31}, which has been used globally except in Australia, where there are concerns over the impact of the translocation of the rye chromosomal segment from 1RS to the wheat chromosome arm, 1BL (called IBL 1BL/IRS 1RS and includes Sr_{31}) on product quality (Martin and Stewart, 1986). Consequently, Ug99 has the potential to parasitize many of the current wheat varieties that the world depends upon for food security (Singh, 2006, 2008)

Ug99 was well established in Uganda and southwest Kenya by 1999, in western Kenya by 2001 and in Ethiopia by 2003 (Singh, 2008). The possible future migration of Ug99 was determined by analogy with the movement of the stripe rust race virulent against Yr_9 (Singh *et al*., 2006) and the prevailing wind directions during the growing period of wheat crops in the region (Hodson, Singh and Dixon, 2005). A Yr_9 virulent stripe rust pathotype moved from east Africa, where it was first isolated in 1986, across the Arabian Peninsula into the Near East by 1991 (Singh *et al*., 2004). It was isolated in Iran in 1992/3, Afghanistan by 1993/4 and northern India by 1997/8. The future migration path for Ug99 may not exactly mirror that taken by the Yr_9 virulent pathotype as this pathotype tended to be found in stripe rust-prone areas. Airborne particle trajectories from east Africa suggest that the Arabian Peninsula, Iraq, Iran Pakistan and India are at risk from the migration of Ug99 in the short term. This prediction has been partly fulfilled, with reports of Ug99 in Yemen in 2006 (Singh *et al*., 2008) and in Iran in 2007 (Nazari, 2008).

The potential of Ug99 to affect wheat production in the countries where it is currently found and to where it could migrate in the short to medium term has been determined by considering the susceptibility of current commercial varieties to Ug99 (Expert Panel, 2005). In Ethiopia, three of the 22 wheat varieties grown are moderately resistant (less than 20% infection), none are resistant, 2 are moderately susceptible and the remaining 17 are susceptible. There is a similar situation in Kenya, as 9 of the current 19 varieties are resistant, 2 are moderately resistant and the remainder are susceptible. Stem rust was considered a major concern in South Africa, but few resources to support breeding and monitoring are now available. No information was available from other southern African countries.

Table 7. QTLs for adult-plant to stem rust resistance in wheat

Chromosomal location of QTL	Genomic Region	Marker	Gene	Source of resistance	QTL Reference
5BL	QSr.sun-5BL	Xglk0354		Arina	Bansal *et al.*, 2008
7BS	QSr.sun-7BS	csLV34, swm10		Forno	
1AS		wmc0333		Forno	
7BL		gbxGb218	Sr_{17}	Forno	
3BS	QSr.sun-3BS		Sr_2	HD2009	Kaur *et al.*, 2009
5DL	QSr.sun-5DL		Sr_{30}	HD2009	
7A(1)	QSr.sun-7A			HD2009	
1D(1)	QSr.sun-1D			HD2009	
2B(1)	QSr.sun-2B			HD2009	
4B(1)	QSr.sun-4B			WL711	
5B(1)	QSr.sun-5B			HD2009	

NOTES: (1) small and inconsistent effects.

In North Africa, Egypt and Morocco are considered to be highly vulnerable to the incursion of Ug99, as most locally-used varieties are susceptible.

The west Asian countries, Turkey, Saudi Arabia, Iraq, Iran and Afghanistan, were identified as high risk as stem rust is of historically significant concern, and a high proportion of the current commercial varieties are susceptible to Ug99.

Wheat growing in India and Pakistan is dominated by a single variety in each country, PBW343 (has Sr_{31}) and Inquilab 91, respectively. Both varieties are susceptible to Ug99, as are other important varieties in both countries.

Stem rust is considered a significant problem in some areas of China. Approximately 60 percent of current varieties carry $Sr_{31,}$ and studies on the resistance of Chinese varieties suggest that many would be susceptible to Ug99. China is not considered to be at risk from an airborne incursion of Ug99, based on experience with the migration of the Yr_9 virulent stripe rust (Singh *et al.,* 2004). It should be stressed that human-mediated incursion has been implicated in rust migration, and must be considered as a threat to the Ug99-free status of the country.

In Europe, stem rust is only of concern to southern European countries, for climatic reasons, and little emphasis has been placed on the development of stem rust-resistant varieties in most European countries (Saari and Prescott, 1985). Historically, stem rust has been a concern over more of Europe and was a problem in the UK in Napoleonic times (Johnson, pers. comm.). Also, climate change is predicted to result in climatic warming at higher latitudes and thus change the spectrum of plant diseases and pests in these regions (Rosenzweig *et al*., 2000). This would favour the spread of stem rust in a northerly direction in Europe. Human-mediated incursion of Ug99 into many parts of Europe may occur quite readily, given the geographical proximity of Europe to east Africa.

Breeding for stem rust-resistant wheat varieties has been a long-term priority in eastern Australia (Park, 2008). There has not been the same emphasis on breeding for stem rust resistance in Western Australia, where a significant proportion of the Australian wheat crop is grown. The genes deployed for stem rust resistance in Australia have not included Sr_{31} and have emphasized Sr_{26} and Sr_{24} (Park and Bariana, 2008). The durable gene for stem rust resistance, Sr_2, has also been deployed in combination with race-specific genes. More recently, emphasis has been given to increasing the diversity of the genes for rust resistance, using *inter alia* Sr_{22} (Schomburgk), Sr_{33} (Lorikeet) and Sr_{45} (Thornbill), which are all effective against Ug99. This suggests that an incursion of Ug99 into Australia would have minimal impact except in Western Australia, which is the closest part of Australia to east Africa. This impact would be more significant from the incursion of the Ug99 variant TTKST,

which is virulent against Sr_{24}. Migration of stem rust (Watson and de Sousa, 1983) and stripe rust (Steele *et al*, 2001; Wellings *et al.,* 2002) into Australia from east Africa has been documented, as has the human-mediated incursion of stripe rust from Europe (Wellings, 2007).

It has been questioned whether the shift from publicly funded wheat breeding to private sector wheat breeding over the last decade will result in the same emphasis on releasing rust-resistant wheat varieties (Park, 2008).

A number of Canadian-bred wheat cultivars, including AC Barry (Singh *et al.*, 2008), AC Cadillac and Peace, as well as the durum varieties Napoleon and Commander, have expressed high levels of resistance when challenged with Ug99 (DePauw *et al.*, 2009). It is assumed that other Canadian varieties (of which there were 64 in general use in 2007) will not have the same level of resistance (Canadian Wheat Board, 2007).

The gene for stem rust resistance derived from the 1BS/1RS wheat/rye translocation, Sr_{31}, has been used extensively in the USA. Some 450 USA varieties and breeding lines were tested for resistance to Ug99 (TTKSK) to determine the vulnerability of the USA to an incursion by this pathotype. Resistance was identified in 16 percent of hard red spring varieties, 48 percent of hard red spring varieties and 27 percent of soft winter wheat varieties (Jin and Singh, 2006). The resistance in hard red winter wheat is considered to be due to Sr_{24}, while the resistance in soft winter wheat is probably due to Sr_{36}. The resistance derived from the 1AL/1RS translocation present in many winter wheats and breeding lines provided effective resistance against Ug99 (TTKSK).

It is difficult to envisage an incursion of Ug99 or any of its derivatives into North America through particle transfer with air movement. However, a human-mediated incursion, whether accidental or malicious (Hugh-Jones, 2002), cannot be discounted. *Puccinia graminis* is considered to have entered North America at least twice, which suggests it could do so again (Abbasi, Goodwin and Scholler, 2005).

The evolution of Ug99 to produce new races has been quite rapid, with virulence against Sr_{24} detected in 2006 and against Sr_{36} detected in 2007 (Table 8; Jin *et al*., 2008a, 2009). The genetic relationship of TTKST and TTTSK to Ug99 was examined using eighteen molecular markers. No polymorphisms were detected among the pathotypes, suggesting that mutation is the driver of this evolution (Jin *et al*., 2008b).

A different range of mutations has occurred in South Africa, with the loss of virulence against *Sr31* and the subsequent evolution of the resulting pathotype to acquire virulence against Sr_{24} (Visser, Herselman and Pretorius, 2009; Table 9).

Table 8. The evolution of pathotype virulence from Ug99.

USA Designation	Pathogenicity			First Isolated	
	Sr_{31}	Sr_{24}	Sr_{36}	Location	Year
TTKSK (Ug99)	+(1)			Uganda	1999
TTKST	+	+		Kenya	2006
TTTSK	+		+	Kenya	2007
TTKSF				South Africa	2000
TTKSP		+		South Africa	2007

NOTES: (1) Virulence

Conclusion

The risk to global wheat production posed by the development of Ug99 in Uganda and its subsequent transnational migration and evolution to gain virulence against widely used genes for stem rust resistance is a global problem, not a regional problem. This is a consequence of the virulence of Ug99 and/or its lineage against a large proportion of the world's current commercial varieties and the high probability of migration through air currents or through human mediation to many wheat growing countries over time.

The primary short- to medium-term impact, unless effective amelioration measures are rapidly implemented, will be in Africa and west and south Asia.

It is estimated that over 50% of the 75 million hectares planted to wheat in the 18 countries where Ug99 and its derivatives are currently found or are predicted to migrate to in the short or medium term are planted to varieties that are susceptible or moderately susceptible to these pathotypes. An epidemic in these areas will result in gross undersupply of food and substantial social dislocation. These are situations that would require global intervention for emergency amelioration, such as through chemical control (Singh *et al.,* 2008).

The way forward – Stem rust

Insight gained from the understanding of the evolution of the wheat rusts through pathotype surveys and on the expectations of host resistance longevity through deployment of resistance genes has provided the technologies necessary to develop wheat varieties with long-lasting stem rust resistance. There will be temporal and spatial variation in the solutions necessary because of the crisis circumstances in east Africa and West Asia and the international variation in the risk posed by stem rust. Short-term strategies to minimize the impact of stem rust in high-risk regions will probably, by necessity, not be sustainable. The solutions could include chemical control and would include genetic solutions that will probably not meet all consumer requirements.

Chemical control is too costly, particularly for small-scale and subsistence farmers. It is potentially not an environmentally sustainable option.

Crisis management

East Africa has been described as a 'hot spot' for wheat stem rust as the prevailing conditions allow for wheat to grow year round and stem rust is commonly found all year round (Saari and Prescott, 1985). This provides optimum conditions for the development of epiphytotics. The probability of an epiphytotic occurring is increased due to the high proportion of current varieties in Kenya and Ethiopia that are susceptible to Ug99 (Expert Panel, 2005).

There is also a high probability of epiphytotics due to Ug99 and/or its derivatives in countries to where these pathotypes have or will have migrated, due to the susceptibility of current commercial wheat varieties and the history of stem rust epidemics in these countries (Expert Panel, 2005).

Chemical control of rust to protect food production is the appropriate option if stem rust epiphytotics develop before adequate genetic solutions have been implemented, as losses due to stem rust often range up to 50% (Rees and Syme, 1981; Dill-Mackay, Rees and Platz, 1990, 1991). Folicur® has been found to provide good control of stem rust and minimize crop loss, provided it is applied early (Loughman, Jayasena and Majewski, 2005). The best results were obtained if spraying commenced when rust was first observed at heading. Folicur® was not very effective if the plants were heavily infected.

Monitoring for stem rust presence would be an essential component of any chemical-based strategy to control stem rust, due to the limited utility of chemical control once stem rust is well established.

Short term

The costs of chemical control, particularly for developing world farmers, and in terms of environmental and occupational concerns (and costs), require that genetic options are implemented as soon as possible. A genetic solution based on race-specific resistance, particularly in east Africa, may not be sustainable for very long given the favourable climate and abundance of susceptible host plants, which might rapidly generate pathotypes capable of parasitizing any newly deployed variety.

Varieties and breeding lines that display adult-plant resistance to infection by Ug99 and its derivatives should be identified as a matter of urgency. This material should be evaluated for utility in areas currently infested with Ug99 and its derivatives, and in those countries lying in the predicted migration path, and be released to growers as quickly as possible. The evaluation should emphasize all required traits, not just stem rust resistance. This is being undertaken and the results look very promising (Singh *et al.,* 2006, 2008). It is unlikely that this approach will be sustainable, as varieties identified in this way will most often fail to meet

all the expectations of growers and consumers, which will limit the market share for these varieties in the longer term. It will nevertheless provide short-term relief.

Long term

Detailed QTL analysis of leaf and stripe rust resistance in wheat has demonstrated considerable genetic variation for durable rust resistance, although a relatively limited amount of germplasm has been evaluated (Rosewarne *et al.,* 2008; Singh, Huerta-Espino and Williams, 2001; Suenaga *et al.,* 2003; Williams *et al.,* 1997, 2003). The genetic resources located so far would meet the Williams *et al*. (1997) requirement that 4 to 5 genes for slow rusting would be required to provide high levels (near immunity) of resistance (Singh and Rajaram, 1994).

Similarly, a comprehensive evaluation of soybean (*Glycine max* L.) germplasm for resistance to tropical soybean rust (*Phakopsora pachyrhizi)* was successful in identifying a considerable number of accessions with adult and seedling resistance (Miles, Frederick and Hartman, 2006; Miles *et al.*, 2008).

The success in locating genetic variation for putative durable resistance for leaf and stripe rust and tropical soybean rust provides confidence that sufficient durable resistance will be found for stem rust. Some priority candidates have already been identified. These include the Australian cvs. Hopps and Kite (Sr_{26}). Knott (1982) identified eight varieties and breeding lines.

This suggests that it is quite probable that wheat varieties with high levels of putative durable resistance to stem rust could be developed from the genetic resources readily available, or by using those that would be available in global genetic resource collections.

A considerable number of operational, management and technical matters would have to be addressed. The technical matters will be considered below under ‘Pre-Breeding’.

Operational

Genetic solutions, though not all the necessary tools are currently available, offer the best hope of sustainable stem rust resistance. They also have an impact on all levels of the value chain for plant variety-based industries. The level of intervention necessary to ensure that the value chain functions optimally will vary spatially. A value chain is a sequence of activities through which a product passes and gains value (Porter, 1985). A value chain is commonly a group of institutions who participate in the value chain for their mutual advantage (Trip, 2001). Functioning of the value chain is enhanced through innovation by and incentive for the individual institutions, and by effective communication among all institutions in the value chain.

The long-term commercial deployment of wheat varieties with durable stem rust resistance will be predicated on those varieties satisfying all the requirements of all institutions in the value chain. The importance of the wheat consumer cannot be over emphasized and it is illustrated by the commercial history of the rice variety KDML105 in Thailand (Sarkarung, Somrith and Chitrakorn, 2000). KDML105 was developed by selection for the aromatic properties of the cooked grain from a heterogeneous population by a farmer in 1945. In 1997 it was very low yielding, photoperiod sensitive, tall and susceptible to a range of pests and diseases. However, in 1997 it was the main aromatic rice variety grown in Thailand with a 42% market share. High yielding pest and disease resistant, photo period insensitive and short varieties are available to growers of KDML105, but these lacked consumer appeal and did not attract the high price paid for KDML105 in 1997. The incentive for consumers is to obtain a product that meets their culinary needs, and KDML105 does this.

The incentive for growers to select a particular variety will depend on a number of factors, providing that consumer preference and the consequent returns from this do not override other considerations, as they have done with KDML105. Important considerations when selecting a variety may include yield potential, drought tolerance, biotic and abiotic stress

resistance/tolerance, and agronomic features that facilitate production, such as flowering time, height or lodging resistance. The incentive for the farmer will vary, reflecting requirements for family sufficiency and income generation. The communication requirements include the feedback from consumers, making breeders aware of the traits required, and an understanding of the features of the available varieties.

Seed suppliers commonly have responsibility for seed multiplication and distribution (sale) to growers. The incentive for private sector entities will be profit, which will drive efficiency and inventory. Public sector entities are often not driven by profit and may not be as focused on efficiency and the relevance of inventory. Seed suppliers will require information from the breeder on variety characteristics and from the growers on their experience with a variety.

The incentives for the breeder include enhancing food production (altruism), releasing varieties and making a profit. The relative emphases will vary according to whether the breeder is public sector or private sector. The breeder will be a major contributor to the innovation in the value chain. The information required by the breeder includes understanding consumer wants, grower requirements and products from the pre-breeding community. The breeder is the prime source of information on research priorities for the pre-breeding community and the attributes and availability of new and current commercial varieties.

The function of the pre-breeding community will be to provide all the new technology required for the breeder to produce varieties that maximize value to the whole value chain. The incentive for the group will include improving the welfare of their target constituents, discovery of new information and products, and scientific recognition. The last-named is often fundamental to remuneration and promotion. The international currency by which scientists are judged is publication in refereed scientific journals. Often, technology development by pre-breeders is less amenable to scientific publication than is more fundamental research. This provides a real dilemma for scientists engaged in pre-breeding research, for their administrators and for investors—a dilemma for which an adequate solution is not apparent (BBSRC, 2004; Brennan *et al.,* 2008). This dilemma may result in technology push rather than outcome pull.

Investors in breeding (as opposed to pre-breeding) include the public and private sectors and philanthropic organizations. The incentive for the private sector will be to generate a profit.

The incentive for the public sector and philanthropic organization investors is to address public good issues in areas where there is market failure. Many governments consider that plant varieties are a private good and that intellectual property instruments have been developed to allow plant breeding to generate an equitable return on their investment in breeding and such governments seek to disinvest in plant breeding. Also, discretionary funds available to governments and philanthropic entities are often limited and in great demand. As a consequence, governments and philanthropic organisations tend to be invested in crisis situations.

This can result in such funding being directed to those issues that are causing current community concern. Investment maybe re-directed away from priority activities that address important issues and require ongoing investment, but are not obvious priority community concerns. The cessation of the funding results in the loss of intellectual capacity, corporate knowledge and infrastructure in the short term, and permits issues that were being addressed through breeding to resurrect in the medium to longer term.

Consequently, the incentive to invest in breeding by public sector and philanthropic organization may be transitory.

These concerns apply to pre-breeding, except that, in the absence of genetically modified varieties, a number of countries have recognized (USA, Canada and Australia) or are recognizing (UK and Nordic countries) that the profit margins from plant breeding are usually too small to be able to support the necessary quantum of pre-breeding research.

National Plant Variety Rights (PVR) or Plant Breeders' Rights (PBR) acts of parliament are the instruments that were designed to facilitate the breeder obtaining an equitable return on investment in plant breeding, and hence to encourage private sector investment in plant

breeding. Intellectual property rights for plant varieties (i.e. PVR/PBR) were agreed on by a significant portion of the global community at the UPOV convention in 1961, and have been revised a number of times since, with 68 countries having legislation that is harmonized with this convention.

Although some significant examples of increased private sector investment in plant breeding have been documented (UPOV, 2005), there is concern that a number of aspects of the UPOV convention often preclude breeders receiving an equitable return on investment in breeding. It is considered that the US Plant Variety Protection Certificate (PVPC) (USA version of the UPOV convention) is ineffectual in protecting PVR for soybean (Kesan and Gallo, 2005) and has had little impact on improving the yield of wheat (Alston and Venner, 2002), soybean (Perrin, Hunnings and Ihnen, 1983) or canola (Carew and Devadoss, 2003). Janis and Kesan (2002) concluded that PVPCs are difficult to enforce. A considerable number of limitations to the enforcement of the Australian PBR Act have been documented and solutions to these have been proposed (ACIP, 2007, 2008, 2010; Brennan, 2009). The utility of a number of options to enhance the enforceability of UPOV harmonized plant breeder's rights are dependent on the structure of the value chain for a particular plant variety-based industry in a specific country. The efficiency of changes to PBR/PVR to enhance enforcement will vary from country to country and from crop species to crop species.

The incentive requirements for private sector investment in plant breeding, provided there is an equitable return on investment, will be less subject to the pressure to change than those for the public and philanthropic sectors.

The communication requirements for investors include detailed proposals of the breeding or research to be undertaken and the progress and impact of that undertaking. Investors generally have a reporting obligation to their boards or government, or both, on the impact of the investment.

Summary

Genetic solutions for sustainable stem rust resistance are predicated on the development and wide-scale deployment of wheat varieties with long-lasting resistance. It is important that the breeding effort for such varieties is sustained in the long term. Government and philanthropic agencies would not appear to be dependable sources for the required long-term investment as they are often compelled to direct their limited resources to current community concerns. Also, governments are increasingly accepting the view that plant varieties are a private good and, as such, should not be an area for government investment. International experience with UPOV harmonized plant variety intellectual property rights (IPR) is that it is difficult to obtain an equitable return for private sector investment. This concern is being addressed, but not globally. Long-term private sector investment in the breeding of wheat varieties with sustainable resistance to stem (and other) rust diseases would appear to be more probable, providing that an equitable return on investment could be achieved. This will require developing a form of plant variety IPR that facilitates the required return. The exact nature of the IPR will vary from country to country, species to species, and may not be possible in some developing economies.

The uptake and long-term deployment of a plant variety is predicated on it having the necessary traits and IPR to generate the necessary incentive for all levels of the value chain to use the variety. Prominent among these will be the consumer preference traits and those that maximize productivity and minimize production costs. A key process in the development of sustainable rust resistant varieties will be developing a full understanding of the incentive drivers for the whole value chain and how they will be implemented. A priority component will be to understand the range of traits required in a variety and the development of the breeding technology and infrastructure necessary for efficient and effective breeding of such varieties.

One of the outcomes of this process will be priorities for pre-breeding research and development.

Management

Minimize inoculum pressure

Limiting the amount of inoculum is an intrinsic component of reducing the probability that the rust organism can change genetically to parasitize previously resistant varieties. Strategies that have worked well in the past to reduce the amount of inoculum include the removal of volunteer wheat that could harbour rust in the off-season, using diversity in the resistances deployed, using multiple effective genes for rust resistance in the one variety, implementing minimum rust resistance standards for release, and removing cultivars from commercial production whose resistance has broken down.

Minimizing the 'out-of-season' rust will have significant impact on limiting the development of subsequent epiphytotics. One of the major effects of the barberry eradication programme in the USA was minimizing the over-wintering of stem rust, which greatly reduced the frequency and magnitude of rust epidemics in regions where barberry was an effective alternate host for wheat stem rust (Leonard, 2001b). Government intervention and farmer education are key to minimizing the carryover of rust from one season to the next. Such activity has congruence with FAO Policy Objective 5 (FAO, 2008: 36).

Genetic vulnerability through the deployment of varieties in which there is little or no variability for disease resistance would appear to be a major global risk factor. Southern corn blight provides a historical precedence of the crop loss that can be sustained through genetic uniformity for resistance to disease. In 1970, Southern corn blight caused a loss of production valued at approximately one billion US dollars (Tatum, 1971). Diversity in the genetics of the resistances deployed will also reduce the probability of survival of natural mutations for virulence as it will reduce the probability of a mutant depositing and multiplying on a variety on which it is virulent and has a selective advantage. This has congruence with FAO Policy Objective 3 (FAO, 2008: 32).

Minimum rust resistance standards (MDRS) for release prevent the release of varieties that would facilitate the build up of rust (Wallwork, 2007). The full implementation of MDRS for release has been effective in contributing to preventing rust epiphytotics in the most rust prone areas of Australia (Park, 2007, 2008). The mandated implementation of such standards would only be possible through government intervention. This is congruent with FAO Policy Objective 3 (FAO, 2008: 32).

The failure to remove from cultivation a variety that had become susceptible to stem rust probably resulted in the loss of effectiveness of Sr_{36} in Australia (Park, 2007). The rapid removal from production of the Sr_{36}-carrying variety is credited with the maintenance of the stem rust resistance in other varieties (Park, 2007; Platz and Sheppard, 2007). Mandating that susceptible varieties should be removed from production may only be achieved through government intervention, which has congruence with FAO Policy Objective 3 (FAO, 2008: 32). The removal of susceptible varieties is facilitated when growers have creditable alternative varieties. This highlights the need for genetic diversity for rust resistance in wheat varieties for a specific geographical region, and for an effective breeding programme that delivers varieties that meet all the expectations of the value chain.

There will be a lower probability of the rust resistance in a variety 'breaking down' when the variety is protected by multiple effective genes for resistance to specific rusts.

Pathotype monitoring

The significance of having the capability to monitor the population dynamics of a pathotype flora effectively cannot be overestimated. Knowledge of the virulence in the rust flora provides guidance on the pathotype-specific genes for resistance that can be deployed and the long-term sustainability of a particular strategy for sustainable rust resistance. It also provides information on which to base minimum rust resistance standards for release, when a variety should be removed from commercial production, and what material in a breeding programme needs to be discarded as a result of changes in the pathotype flora.

Pathotype monitoring has also provided knowledge of the importance of migration, mutation and recombination (sexual and asexual) in generating new pathotypes (Watson and de Sousa, 1983; Wellings, 2007; Steele *et al.*, 2001). These authors have demonstrated, *inter alia*, the importance of trans-national movement of rust pathotypes by wind movement and through inadvertent human facilitation. The possibility of malicious human facilitation has also been noted (Hugh-Jones, 2002).

The need for national monitoring and transborder information exchange on pathotype population structure is included in FAO Policy Objective 2 (FAO, 2008: 30).

Pre-breeding research

Genetic resources

A priority activity to address the risk posed to global food production by Ug99 and its derivatives is the location of genetic resources that provide effective resistance to these pathotypes. The priority must be to locate putative durable sources of resistance. Such resistance would be identified as having long lasting effectiveness when widely deployed in environments favourable to the target rust species (Johnson, 1988) or as providing resistance at the adult stage with a compatible or near-compatible host reaction (Williams *et al*., 1997). A number of sources of stem rust resistance that have maintained effectiveness in the long term have been identified, and should be investigated as a matter of priority.

Emphasis should also be given to searching global genetic resources. Priority in this group should be given to sources of resistance in well developed backgrounds, as these could be transferred into commercial cultivars with minimal or no genetically linked deleterious (in terms of commercial acceptability) traits. Some genetic resources may be suitable as varieties, at least in the short term.

The exploitation of global genetic resources, particularly for wheat, is difficult and expensive due to the large numbers of accessions held and the large number of *ex situ* genetic resource centres in which they are held (Street *et al.,* 2008). Techniques have been developed to allow the targeting of genetic resources that have a higher probability of including the required trait (Street *et al.,* 2008; Bhullar *et al.,* 2009; Bonman *et al.*, 2007). A preliminary application of the Focused Identification of Germplasm Strategy (FIGS) technology (Street *et al*., 2008) for the location of rust resistance in a landrace collection suggest that FIGS was almost twice as effective as other strategies (random selection and core collections) in identifying sources of stem rust resistance (Street, pers. comm.). Similar results were obtained for the USDA Small Grains Collection using the known geographical distribution of stem rust-resistant germplasm (Bonman *et al.,* 2007).

The application of FIGS and similar technologies to the location of new sources of stem rust resistance should be given high priority due to the efficiency gains.

Genetic resources from landraces are often difficult to deploy directly in a breeding programme due to deleterious (for commercial purposed) linkages and background. Priority needs to be given to introgressing the desired gene(s) into commercially acceptable breeding lines. The monitoring of recombination adjacent to the target gene(s) would be facilitated using molecular markers.

Wheat relatives have long been recognized as a rich source for the genetic improvement of wheat. The durable stem rust resistance gene Sr_2 was derived from *Triticum turgidum*

cultivar Yaroslav emmer (McFadden, 1930) and the long-lasting resistance in the variety Kite was derived from *Agropyron elongatum* (Knott, 1961).

The use of wheat relatives poses similar but more extreme problems for wheat improvement in comparison with those posed by landraces, as they are very prone to have deleterious linkages that cannot be easily broken due to failure of alien chromosomes to recombine readily with wheat chromosomes (The *et al.,* 1988). Chromosomes of the wheat progenitors *Triticum turgidum* (n=14) and *Triticum tauschii* (n=7) do combine readily with wheat chromosomes. These species can and have been used extensively for wheat improvement by developing allohexaploids, called synthetic wheats, which are completely or almost completely fertile in crosses to bread wheat. These species should be given priority over other wheat relatives and can be exploited by evaluating genetic resources of each species and either crossing them directly to wheat or through the production of allohexaploids (Mujeeb-Kazi, Rosas and Roldan, 1996; van Ginkel and Ogbonnaya, 2006a, b).

Deleterious linkages in this material can be dealt with in the same way as for wheat landraces, *viz* using molecular markers to identify the required recombinants during the introgression of genes of interest into agriculturally elite backgrounds by backcrossing either limited or complete.

Other wheat relatives have been exploited in the past for sources of resistance to wheat stem rust (Table 9). These resistance sources have generally proved to be associated with a range of traits that compromise their utility in wheat breeding (The *et al*., 1988). Technologies have been developed to promote recombination between alien and wheat chromosomes with the appropriate recombinants identified using molecular markers. In this way versions of Sr_R (from Imperial rye) and Sr_{31} (from Petkus rye) on reduced alien chromosome segments, that may have eliminated the yield and quality defects associated with these genes, have been developed (Rogowsky *et al*., 1991; Lukaszewski, 2000, 2003; Dundas *et al*., 2007; Anugrahwati *et al*., 2008). Sources of Sr_{26} (from *Agropyron elongatum*) on reduced alien chromosome segments are also available (Dundas and Sheppard, 1994, 1996). Work is underway on developing similar stocks of stem rust-resistance genes Sr_{32}, Sr_{39}, Sr_{37} and Sr_{40} (Dundas *et al*., 2007).

Table 9. Originating genus and species and usefulness of designated *Sr* genes in conferring seedling and/or adult-plant resistance to Ug99 race of stem rust pathogen *P. graminis* f.sp. *tritici*.

Origin of *Sr* genes	Stem rust resistance (*Sr*)	
	Ineffective	**Effective**
Triticum aestivum	5, 6, 7a, 7b, 8a, 8b, 9a, 9b, 9f, 10, 15, 16, 18, 19, 20, 23, 30, 41, 42, Wld-1	28, 29, Tmp
Triticum turgidum	9d, 9e, 9g, 11, 12, 17	2, 13, 14
Triticum monococcum	21	22, 35
Triticum speltoides		32, 39
Triticum timopheevii	36	37
Triticum tauschii		33, 45
Triticum comosum	34	
Triticum ventricosum	38	
Triticum araraticum		40
Thinopyrum elongatum	24	25, 26, 43
Thinopyrum intermedium		44
Secale cereale	31, 27	1A.1R

SOURCE: Singh *et al*., 2006, and updated.

There is evidence that gives confidence that, with the appropriate technologies, sufficient sources of durable resistance can be identified in wheat to meet the global requirements for this genetic resource. There would be considerable long-term social dislocation if this confidence were misplaced. It appears prudent to continue the exploitation of these more distant wheat relatives, but with less priority than that for the other genetic resources documented above.

Predicting durability

The determination that a particular source of rust resistance is durable is based on the *a posteriori* criteria suggested by Johnson (1981, 1988). These appear to work well, but predictive, or *a priori*, criteria are necessary for stem rust as there has been less commercial deployment of adult-plant resistance. It has been suggested that adult-plant resistance where the host reaction is compatible or near compatible is a predictor of resistance durability (Williams *et al.,* 2002). There is evidence for this in leaf and stripe rust but it would not have predicted the durability of the *Agropyron elongatum* resistance gene Sr_{26} in cv. Kite.

Progress has been made in the development of tools for predicting the durability of disease resistances (Leach *et al*., 2001; Cruz *et al*., 2000; Burdon and Thrall, 2003; Bahri *et al.*, 2009). It is suggested that these and other approaches should be investigated to develop robust criteria for predicting durability.

Molecular markers

The application of molecular marker-assisted breeding offers many logistic efficiencies. These include increasing heritability. This is particularly relevant for those attributes for which the phenotype is an imprecise indicator of the genotype, or when large genotype × environmental interactions are involved (Koebner and Summers, 2003). Molecular markers would also improve the cost efficiency of a breeding programme if expensive phenotyping could be replaced by less expensive laboratory-based genotyping. The cost efficiency would be further enhanced if genotyping for multiple loci for the traits required in the breeding programme could be undertaken simultaneously. The development of chip-based systems offer relatively inexpensive simultaneous multi-trait genotyping (Gupta, Rustgi and Mir, 2008; Akbari *et al*., 2006; Jaccoud *et al*., 2001).

There are a number of examples where marker-assisted selection has been applied to wheat improvement (Kuchel *et al*., 2007; Howes, Woods and Townley-Smith, 1998; Frisch, Bohn and Melchinger, 1999).

Constraints on the application of molecular markers in plant improvement include recombination between the marker and trait, which generates false positives, and failure of a specific marker to segregate in a particular breeding population. Also, a gene for an undesirable trait may be located between the marker and the locus of interest if the recombination distance between the marker and gene of interest is relatively large. This gene will be selected for by selecting for the marker.

Molecular markers for the linked genes for rust resistance, $Yr_{17}/Lr_{37}/Sr_{38}$, were found to be completely linked and segregate in a large number of breeding populations (Sharp *et al.*, 2001). This result would be anticipated as these resistances and these markers are located on an alien chromosome segment from *Aegilops ventricosa* that is unlikely to recombine with homeologous wheat chromosomes (Robert, Abelard and Dedryver, 1999; Seah *et al*., 2000). In these circumstances, the marker identified would appear to have utility across breeding populations.

Conversely, markers identified for Sr_2 and flour colour were not diagnostic and were not located in all sources of Sr_2 and low flour colour (Sharp *et al.,* 2001). Also, the marker for Lr_{28} (Naik *et al.,* 1998) was considered to be too far from the gene to be useful (Sharp *et al*., 2001). The linkage for Sr_{39} (Gold *et al*., 1999) was also 'loose' (Sharp *et al.,* 2001). The marker for Lr_{35} (Seyfarth *et al*., 1999) was considered close enough to be of value in breeding.

The efficiency of a plant breeding programme would be enhanced if the constraints listed above could be avoided. One option for this is to identify closely linked markers for all loci of interest to the breeding programme, and that are available for all or many breeding populations.

Marker systems that facilitate the location of a number of markers for each locus of interest would increase the number of populations where at least one marker would provide valuable inference to the breeder. Single nucleotide polymorphisms (SNP) markers are abundant and relatively evenly distributed over the genome and would allow the discovery of

multiple markers per locus of interest (Gupta, Rustgi and Mir, 2008). The ready availability of rapid and relatively inexpensive genome sequencing has facilitated their discovery. Also, SNP markers are amenable to array technology, which facilitates the genotyping of many thousands of markers in parallel (simultaneously). High throughput, low-cost allelotyping SNPs are available commercially, and SNPs are being used for wheat (Gupta, Rustgi and Mir, 2008).

Diversity array technology (DArT) offers advantages similar to those of SNPs. It is based on abundant polymorphic segments of genomic DNA that are present in particular genome representation and do not require a knowledge of the genome sequence for their identification (Gupta, Rustgi and Mir, 2008; Jaccoud *et al*., 2001; Wenzl *et al.,* 2004). These markers also allow high throughput at low cost and have been developed for a number of crop species (Kilian *et al*., 2005). They are now widely used by wheat workers and, like SNP, would appear to have considerable utility for wheat breeding (Akbari *et al*., 2006; Semagn *et al*., 2006; Wenzl *et al*., 2007; Crossa *et al.,* 2007; White *et al*., 2008; Mantovani *et al.,* 2008; Badea *et al*., 2008; Huynh *et al*., 2008). There are currently in excess of 3000 DArT markers for wheat (Gupta, Rustgi and Mir, 2008).

The requirement for a plant variety to meet the needs of the whole value chain has been emphasized to ensure that it likely to be deployed widely. Consequently, the development of marker-assisted selection should focus on all required traits, not rust resistance alone. It would be cost effective to employ the marker technology to be used in breeding programmes for the initial marker discovery.

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Sustainable wheat rust resistance - Learning from history

This publication was the result of a competitive call for papers to review the state of knowledge on breeding for durable resistance, and prospects for improvement of resistance to wheat (*Triticum aestivum* L.) and soybean (*Glycine max* L.) rusts. The purpose of these studies was to support efforts directed to climate change adaptability and mitigation through enhanced and sustainable use of genetic variability. Topics covered include: 1. worldwide rust threats with high impact to food security (current and potential); 2. general approaches to breeding for resistance to these diseases; 3. relevance of vertical resistance approaches; 4. relevance of horizontal, durable resistance; and 5. the way forward.

GIPB is pleased to present the review papers that resulted from this initiative: Sustainable wheat rust resistance - Learning from history by P. S. Brennan (Plant Production and Protection Paper Series 203) and State of Knowledge on Breeding for Durable Resistance to Soybean Rust Disease in the Developing World by P. Tukamuhabwa and M. Maphosa (Plant Production and Protection Paper Series 204).