BREEDING STRATEGIES FOR RESISTANCE TO THE RUSTS OF WHEAT
BREEDING STRATEGIES FOR RESISTANCE TO THE RUSTS OF WHEAT
The International Maize and Wheat Improvement Center (CIMMYT) is an internationally funded, nonprofit scientific research and training organization. Headquartered in Mexico, the Center is engaged in a worldwide research program for maize, wheat, and triticale, with emphasis on food production in developing countries. It is one of 13 nonprofit international agricultural research and training centers supported by the Consultative Group on International Agricultural Research (CGIAR), which is sponsored by the Food and Agriculture Organization (FAO) of the United Nations, the International Bank for Reconstruction and Development (World Bank), and the United Nations Development Programme (UNDP). The CGIAR consists of 40 donor countries, international and regional organizations, and private foundations.

CIMMYT receives support through the CGIAR from a number of sources, including the international aid agencies of Australia, Austria, Brazil, Canada, China, Denmark, Federal Republic of Germany, France, India, Ireland, Italy, Japan, Mexico, the Netherlands, Norway, the Philippines, Saudi Arabia, Spain, Switzerland, the United Kingdom and the USA, and from the European Economic Commission, Ford Foundation, Inter-American Development Bank, International Development Research Centre, OPEC Fund for International Development, Rockefeller Foundation, UNDP, and World Bank. Responsibility for this publication rests solely with CIMMYT.


ISBN 968-6127-23-2
# Table of Contents

**Preface**  
iv

**Chapter 1—The Role of Specific Genes in Breeding for Durable Stem Rust Resistance in Wheat and Triticale, R.A. McIntosh, Plant Breeding Institute, Castle Hill, Australia**  
1

**Chapter 2—Resistance to Leaf and Stem Ruts in Wheat, A.P. Roelfs, Cereal Rust Laboratory, U.S. Department of Agriculture Research Service and the University of Minnesota, St. Paul, Minnesota**  
10

**Chapter 3—Pathogenicity Analysis of Yellow (Stripe) Rust of Wheat and Its Significance in a Global Context, R.W. Stubbs, Research Institute for Plant Protection, Wageningen, The Netherlands**  
23

**Chapter 4—Using Polygenic Resistance to Breed for Stem Rust Resistance in Wheat, D.R. Knott, Department of Crop Science and Plant Ecology, University of Saskatchewan, Saskatoon, Canada**  
39

**Chapter 5—Strategies for the Utilization of Partial Resistance for the Control of Cereal Ruts, J.E. Parlevliet, Department of Plant Breeding, Agricultural University, Wageningen, The Netherlands**  
48

**Chapter 6—Durable Resistance to Yellow (Stripe) Rust in Wheat and Its Implications in Plant Breeding, R. Johnson, Plant Breeding Institute, Cambridge, England**  
63

**Chapter 7—Current Thinking on the Use of Diversity to Buffer Small Grains Against Highly Epidemic and Variable Foliar Pathogens: Problems and Future Prospects, J.A. Browning, Department of Plant Pathology and Microbiology, Texas Agricultural Experiment Station, College Station, Texas**  
76

**Chapter 8—The Use of Variety Mixtures to Control Diseases and Stabilize Yield, M.S. Wolfe, Plant Breeding Institute, Cambridge, England**  
91

**Chapter 9—Current CIMMYT Approaches in Breeding Wheat for Rust Resistance, S. Rajaram, R.P. Singh, and E. Torres, Wheat Program, CIMMYT, Mexico**  
101

**Chapter 10—Synthesis: The Strategy of Rust Resistance Breeding, N.W. Simmonds, Edinburgh School of Agriculture, Edinburgh, Scotland**  
119

**Abstracts in Spanish**  
137

**Abstracts in French**  
144
Preface

An international workshop on Breeding Strategies for Resistance to the Rusts of Wheat was held at CIMMYT/Mexico June 29-July 1, 1987. It brought together an eminent panel of internationally recognized specialists charged with the task of reviewing the current situation and defining a broad breeding strategy that could be implemented in the future to incorporate the necessary level of resistance to control leaf, stem, and yellow rusts (*Puccinia recondita* Rob. ex Desm. f.sp. *tritici* Eriks, *Puccinia graminis* Pers. f.sp. *tritici*, and *Puccinia striiformis* Westend) in wheat.

In the first seven chapters, authorities from Australia, Canada, Europe, and the United States focus exclusively on the three rusts and discuss among other points the role of specific genes, diversity, and the use of polygenic, partial, and durable resistances.

Chapter 8 addresses disease management through variety mixtures, based on experience with barley powdery mildew—a concept that has obvious application to the rusts. Chapter 9 outlines current approaches at CIMMYT in breeding wheat for rust resistance.

Chapter 10 summarizes the discussions in the preceding chapters in the context of a defined strategy. The outcome represents a general consensus on future breeding strategies that should be employed to incorporate resistance to these three serious wheat diseases.

We hope that this document sheds new light on wheat rust resistance breeding and, as a result, will be a very significant contribution to the scientific literature.

We should like to thank the International Development Research Centre (IDRC) of Canada for the support it provided to make this workshop possible.

Norman W. Simmonds
Sanjaya Rajaram
Technical editors
Chapter 1

The Role of Specific Genes in Breeding for Durable Stem Rust Resistance in Wheat and Triticale

R.A. McIntosh, Plant Breeding Institute, Castle Hill, Australia

Abstract

Breeding for resistance to stem rust in wheat has been successful. The resistances deployed in agriculture have depended on single identifiable genes and combinations of identifiable genes. The adult plant resistance gene, Sr2, has contributed to durable resistance in many areas. Stem rust resistant wheats for the northeastern wheat areas of Australia have depended on the use of resistances which are replaced following the detection of virulent pathotypes. The deployment of genes in this way depends on relevant pathogenicity surveys, a knowledge of the genes present in wheat cultivars, and industry cooperation in rapid cultivar replacement. Genetic vulnerability to stem rust in the CIMMYT triticale program can be reduced by using information generated in Australia. The narrow genetic base for resistance can be widened by the use of European triticales, rye, and wheat. However, genetic diversity between wheat and triticale should be maintained.

Introduction

Disease resistances and, in particular, resistance to one or more of the three rust diseases of wheat (and triticale) represent a small part of the genotype package that must be delivered by the wheat breeder. Whereas it is not difficult to find or to produce rust resistant materials, it is difficult to combine high levels of resistance to multiple diseases with other desired characters. One only has to refer to recent issues of the CIMMYT Review in order to gain an appreciation of what these characters are and some indication of what they may involve. Clearly, many of them are more elusive and more difficult to achieve than rust resistance. Thus it is imperative that the rust resistance objective be kept relatively simple.

Breeding wheat, and presumably triticale, for resistance to rusts is relatively easy. The problems come with the genetic plasticity of the pathogens. So often, we no sooner have resistance when virulent pathotypes increase in frequency and either render the resistant cultivar(s) vulnerable to disease or actually cause crop losses. Thus, we get the “boom and bust” cycles of which we are often reminded. The recent occurrence of stem rust in triticale in Australia provided a timely reminder of the potential of this disease. On the other hand, we should acknowledge the success of wheat breeders in reducing the fear of widespread stem rust epidemics. This has been achieved by a combination of disease escape mechanisms such as earlier maturity and alternate host eradication as well as genetic resistance. This success has been supported by the continuing research effort that has been devoted to stem rust in contrast to many other disease problems addressed by intermittent effort as individuals and funding organizations make short-term
contributions and then abandon them as researchers gain promotions, or are distracted, or retire.

Breeding for rust resistance continues to be largely experimental in approach and it involves:

- The identification of potential sources of resistance.
- Assessment of their effectiveness over sites, seasons, and pathotypes.
- Incorporation of them into cultivars.
- Introduction of them to agriculture with cultivar release and recommendation.

The eventual test of a successful resistant cultivar will involve some measure of how widely it is grown, and the time for which it remains resistant.

**Features of Resistance**

There are two important criteria of resistance, namely durability and diversity.

**Durability**

The time for which a cultivar will maintain its resistance in a particular area, or at a particular time, cannot be predicted. Various strategies to extend the period of effectiveness of a resistance source have been suggested but, on a global basis, experience seems to vary. If we could identify a source of resistance with adequate effectiveness and total durability, then only that source of resistance would be required by all wheat breeders.

**Diversity**

There is a diverse range of rust resistance genes. Many of them have been used, and continue to be used, in various ways. In practice, genetic diversity is used as insurance against a lack of durability and thus as a means of reducing genetic vulnerability.

On the assumption that we have no durable sources of stem rust resistance giving adequate protection under all conditions, we must attempt to increase or prolong the effectiveness of the resistances that we have. This will be supported by:

- A knowledge of the epidemiology of the pathogen in relation to the prevailing agricultural practices.
- A relevant pathogenicity survey.
- A continuing research effort on host resistance.

**Epidemiology**

Stem rust occurs in various epidemiological regions in the warmer wheat-growing areas. Epidemics are contingent on conditions of favorable moisture, high levels of initial (source) inoculum (by implication, virulent pathotypes), and by susceptible hosts. Epidemics can be continental or local.

**Pathogenicity Surveys**

Over the last 60 years, it has been customary for rust workers to conduct pathogenicity or race surveys. There is no doubt that these have proved useful in epidemiological and evolutionary studies, but they have been of only limited value to breeders.

Several factors influence the distribution and frequency of pathogen genes and genotypes in a particular area.
Migration/introduction

New genes can be introduced from outside the area. Clearly distinctive pathotypes of Puccinia graminis f.sp. tritici were found in Australia in 1926, the early 1950s, and in 1968 (15). These presumed exotic introductions became established in different regions, but subsequently spread to other areas. Comparative studies of the putative 1968 introductions with collections from southern Africa, and meteorological data, indicated thaturedospores had been wind-transported to Australia from Africa (16).

The evidence for periodic introduction to Australia of new pathotypes is supported by various introductions of new diseases. These have included Puccinia graminis f.sp. secalis in the 1950s (14) and Puccinia striiformis f.sp. tritici in 1979. Once the new diseases arrived in Australia, they became established and spread quite rapidly, and evolved to form new pathotypes. Within a year of its occurrence in Australia, yellow rust was found in New Zealand where it subsequently formed pathotypes different from those found in Australia. However, yellow rust has not been reported in Western Australia.

The original yellow rust introduction came from Europe (8), presumably transported to Australia by man (17). Thus both natural factors and man are significant elements in the movement of diseases or particular pathotypes to new areas.

Mutation

Surveys worldwide have provided ample evidence indicating the role of mutation in the origin of new pathotypes. This has been demonstrated in Australia for the various historic groups of the wheat rust pathogens (8, 15). Mutations are the most likely and most predictable events that contribute to short-term rust pathogen variability. Such changes can be anticipated provided there is genetic knowledge of resistance and can be simulated by mutation experiments in the laboratory.

Recombination

The sexual cycles of rust pathogens have obvious implications both for the evolution of new pathotypes and for the seasonal carry-over of inoculum. However, in the absence of alternate hosts, there are established mechanisms of asexual variation. In Australia, somatic hybridization between P. graminis f.sp. tritici and P. graminis f.sp. secalis was probably involved in the origin of a group of rusts commonly found on Agropyron scabrum and barley (Hordeum vulgare). In addition, both pathogenic (6) and isozyme data (1) point to a somatic hybridization origin for one evolutionary pathway of P. graminis f.sp. tritici characterized by pathotype 34-2,11. However, somatic hybridization probably plays only a minor role in the evolution of rust pathogens.

Selection

The genotypes of the predominant commercial cultivars as well as those of the wheats, barleys, and grasses on which rust survives between seasons will influence the pathogen genotypes that survive.

Chance

Pathogen populations go through mammoth boom and bust cycles between crop seasons or, in the longer term, between epidemics. The survival of pathotypes in particular areas and between seasons is influenced by seasonal and
agronomic factors. After Cook wheat was infected by _P. graminis_ f.sp. _tritici_ pt. 343-1,2,3,4,5,6 in 1984, it was rapidly withdrawn from cultivation. The frequency of this "Cook" pathotype also quickly declined, despite the fact it had the pathogenic abilities of its widespread progenitor, 343-1,2,3,5,6. Was this decline due to chance because it was relatively localized and failed to establish or was the mutation event contributing to its origin associated with reduced fitness such that it had a survival advantage only when present on Cook wheat?

**Uses of pathogenicity surveys**

For breeding purposes, a relevant pathogenicity survey should do the following things:

- Indicate what pathotypes are present, where they occur and, with cautious interpretation, in what frequencies. The information will be in pathotype codes or as pathogenicity formulas from which avirulence/virulence frequencies for single genes or gene combinations can be determined. Single gene frequencies are not adequate.

- Indicate and/or confirm when pathogenic changes relate to commercial cultivars. Thus the value of surveys will be enhanced by a knowledge of the genes deployed in those cultivars.

- Act as an early warning to extension and advisory services involved in cultivar recommendation. There is usually a lag period between the detection of a new pathotype and the occurrence of crop losses as a consequence.

- Provide the pathotypes to be used in the breeding nursery. A new pathotype can be used in the breeding nursery before it causes damage in agriculture.

With a relevant pathogenicity survey and a reasonable knowledge of the genetics of resistance, induced field epidemics required for testing can be based on one or few released pathotypes. The use of a mixture of all available pathotypes is an insurance against ignorance. Multi-pathotype nurseries have problems in that pathogen components probably do not increase to equivalent levels; the breeder is then uncertain as to which components are present. In practice, each pathotype in the field nursery should be increased on a host genotype to which it is specialized.

**Genetics of Resistance**

At any particular time, breeders have access to resistant cultivars, resistant materials at various stages of development and potential resistance sources. The primary objective of a genetics program is to understand the expression and inheritance of resistance and to know the range of genetic diversity that is present in agriculture and in breeding programs. Most resistance breeders will demand resistance that is sufficiently effective and stable and that can be selected by means of a single assessment in the disease nursery.

I recognize two types of resistance, that effective at the seedling stage and that coming into effect at post-seedling stages. Obviously, potential resistance sources must confer resistance at growth stages corresponding to those when damage is likely to occur in the field situation. Some genes that are effective in seedlings do not confer
adequate levels of adult plant resistance (e.g. Sr8a, Sr25, and possibly Sr13).

There appears to be some misunderstanding of the processes of selection and the use of genes effective at the seedling stage. Genes effective in this respect usually also confer adult plant resistance. Genes for adult plant resistance cannot be detected in standard seedling tests. However, the breeding approach is different. The breeder is interested in resistance sources. These are initially detected or confirmed in field disease nurseries. If such sources also display seedling resistance and, if genes responsible contribute to resistance at both growth stages, there seems no reason for not combining seedling and adult assessments in the breeding exercise. It is usual to manipulate adult plant resistances in field nurseries, although they could be selected in the greenhouse.

**Methods for Establishing Genetic Diversity**

Long-term resistance to stem rust is dependent on a continuing availability of resistance sources. Various procedures assist in establishing that potential resistance sources carry new or different genes for resistance.

**Pedigree**

While useful for postulating genetic diversity, the breeder must be aware that apparently unrelated sources may carry the same gene(s) and that pedigrees might not be as stated.

**Response**

Different seedling or adult plant responses are indicative of different genes. Experienced rust workers can frequently recognize individual genes and can accurately postulate genes from an array of low infection types.

**Specificity**

A practical method for identifying genes in resistance sources is multi-pathotype testing. Genes can be postulated from the correlation of the responses of selected resistance sources with those of controls. However, this classic application of the gene-for-gene relationship has involved problems which include misinterpretation of the concept, misinterpretation of the data because genetic control lines developed in one geographic location were not applicable in another, use of poor data, and the use of pathotypes inappropriate for breeding purposes.

Multi-pathotype tests often accurately identify those genes which are useless for breeding because the important field pathotypes are virulent. However, they may not identify the potentially useful genes because these will provide resistance to the entire array. Moreover, the tests are usually performed on seedlings and important adult plant resistances may not be recognized. Despite such shortcomings, multi-pathotype tests, combined with relevant field monitoring, remain reliable and efficient means of distinguishing among potential resistance sources for use as parents in breeding programs.

**Genetic studies**

The most accurate way of demonstrating diversity is by conventional genetic analysis. However, this method is time consuming because up to four generations may be required. In addition, tests of allelism may be necessary. Known associations of
genes can be important in aiding the identification and manipulation or resistance genes. For example, wheats with *Sr24* will always carry *Lr24* and, if derived from cv. Agent, will be red-seeded; hexaploid wheats with *Sr9* usually will carry *Yr7*; wheats with *Sr31* will carry *Lr26* and *Yr9*, will display two instead of four chromosome satellites in mitotic chromosome preparations, can be identified by a unique isozyme pattern, and cannot have brown chaff. These associations can be useful when attempting to identify or manipulate the individual components of multigene resistances.

Genes of greatest potential will be those that are effective against all or most pathotypes in the area of interest. Some genes likely to be of current worldwide interest are discussed below:

- **Sr24**—The "Agent" gene produces LIT"2" to "2", Australian white-seeded wheats possessing *Sr24/Lr24* were derived from Dr. E.R. Sears' 3D/3Ag transfers Nos. 3 and 14. Isolates of *P. graminis* f.sp. *tritici* virulent for *Sr24* appeared in South Africa in 1984 (4).

- **Sr26**—This gene on chromosome 6A/6Ag produces LIT"1". It is present in several Australian wheats and has been deployed over a large area since 1967.

- **Sr30**—The Webster/Festiguay gene with LIT"2" to "3" became ineffective in Australia after several years of use. However virulence in the pathogen has declined and certain of the newer wheats including Banks, Vulcan, and Sunstar, probably possess this gene. Singh and McIntosh (10) identified it in Klein Cometa and its presence is suspected in certain CIMMYT-produced wheats such as Inia 66, Pavon, and Cheel. Virulence for *Sr30* has been relatively common in South Africa (5).

- **Sr31**—The Kavkaz/Aurora gene in chromosome 1BL/1RS conferring LIT"1" is very common in European winter wheats and in spring wheats developed by CIMMYT. It occurs in approximately 60% of lines distributed in the 17th International Bread Wheat Screening Nursery. Virulence in *P. graminis* f.sp. *tritici* has not been reported. This gene is present in one Australian biscuit wheat but is unlikely to be exploited further because of fear of the dough stickiness problem that appears to be associated with the presence of chromosome IRS.

Several other genes conferring resistance to a wide array of pathotypes are being investigated. These include *Sr22, Sr32, Sr33, Sr35*, and a gene present in VPM1 and its English derivative, Rendezvous. It appears that few additional highly effective resistance genes will be found in hexaploid wheat but, if required, further genes could be obtained from non-hexaploid wheats and related genera.

**Adult plant resistance**

Probably the most important and most durable source of stem rust resistance in hexaploid wheat is that transferred from tetraploid wheat resulting in Hope and H44. Many studies in the 1930s and 1940s, plus more recent work (2, 3) showed that both seedling and adult plant resistance genes were involved. The seedling resistance genes *Sr9d* and *Sr17* provided resistance only to certain pathotypes. On the other hand, the adult plant resistance gene *Sr2*, although less effective, proved...
to be a durable source of resistance for many parts of the world. \(Sr_2\) is present in many spring wheats and some winter wheats and its presence is shown by its association with head and stem melanism known as false black, or pseudo-black, chaff. This melanism can become excessive in some environments and may be confused with other disease problems.

\(Sr_2\) is very common in wheats developed by CIMMYT. These include Sonalika, Inia 66, Lerma Rojo, the Bluebird series, Pavon, and the Veery series. Consequently, it is present in wheats grown throughout the world. \(Sr_2\) is recessive and its slow-rusting response permits the development of variable levels of disease. There appears no doubt that \(Sr_2\) provides a desirable genetic background into which more effective, but less durable resistance genes can be placed.

### Multiple gene resistance

During the 1960s, Rajaram (9) conducted a genetic study of several wheats displaying low coefficients of stem rust infection on a worldwide basis. Resistance in these wheats to Australian pathotypes was determined by combinations of known and unknown genes. More recently, Singh and McIntosh (11) reported that Kenya Plume possessed eight genes (\(Sr_2, Sr_5, Sr_6, Sr_7a, Sr_8a, Sr_9b, Sr_12,\) and \(Sr_{17}\)). Its field resistance to the predominant Australian field pathotype was determined by \(Sr_2\) and possibly by an interaction of \(Sr_7a\) and \(Sr_{12}\). These authors (12) also attributed field resistance in Chris wheat to \(Sr_7a: Sr_{12}\) interaction. In order to test this conclusion, the Australian National Wheat Rust Control Program has initiated the transfer of \(Sr_7a\) to wheats known to carry \(Sr_{12}\).

While wheats with low coefficients of infection in multilocalational tests frequently possess multiple genes for resistance, the number of genes or type of gene interaction operating at each site has not been determined. However, information of this type will be essential if breeders are to assemble a diversity of such resistance as a means of achieving greater stability.

### Stem Rust on Triticale in Australia

#### General observations

Stem rust first appeared on triticale (x *Triticosecale* Wittmack) in Australia in 1981 but it was not until 1982 that we showed that a unique *P. graminis* f.sp. *tritici* pathotype was involved. The gene present in the affected triticales was \(Sr_{27}\) which had originated from Imperial rye. Comparative studies with selected *P. graminis* f.sp. *tritici* cultures showed that 67% of entries of the 12th International Triticale Screening Nursery (ITSN) possessed this gene. Moreover, Australian commercial cultivars and CIMMYT lines with \(Sr_{27}\) were extremely susceptible to pt. 34-2,12 (*--*12") refers to virulence on seedlings of cv. Coorong) as adult plants (7). In 1984, a further mutational change resulting in pt. 34-2,12,13 (*--*13") refers to virulence on Satu) caused severe rusting on Satu and Toort and moderate rusting on Ningadhu (Drina), Venus (Beagle), Currency, and Samson (Ram). In seedling tests, the IT"12" response of Ningadhu was distinguishable from IT"1 +3 + " displayed by Venus, Currency, and Samson and the IT"3 + " of Satu and Toort.

In 1984, a grant from the Australian Rural Credits Development Fund enabled the appointment of Dr. S.J. Singh to investigate the genetics of rust resistance in triticale, to provide
rust screening services to triticale breeders, and to initiate a backcrossing program to transfer resistance genes to rust susceptible genotypes. The genetic findings are listed below:

- The genes Sr27 in Coorong and SrSatu in Satu are allelic (13).

- A second allelic series involves genes in Tejon-Beagle (IT' ;1 + N"), Ningadhu (IT' '12"), and Juanillo 100 (IT' '23").

- A gene in 14th ITSN No. 64 and 15th ITSN No. 99 (IT' '1") and a gene in 14th ITSN No. 122 (IT' '1-2") appear to be independent of the above groups.

- A highly effective gene occurs in 17th ITSN No. 78 (IT' "1"). This gene may be derived from a Polish triticale. Other studies have indicated high levels of resistance in European triticales to pt. 34-2,12,13.

- Wheat genes Sr9b (13th ITSN No. 33) and Sr36 (University of New England) have been identified.

- There was no evidence for the presence of Sr31 in triticale.

Additional studies have shown that Satu triticale carries a leaf rust resistance gene, LrSatu (IT' '1") showing 11% genetic recombination with SrSatu. Preliminary evidence suggests that this gene contributes to the adult plant leaf rust resistance of Satu and many CIMMYT lines. Whereas CIMMYT materials are generally resistant to leaf rust in Australia, European triticales are often very susceptible. Thus, the use of European triticales as sources of stem rust resistance will require close monitoring in order to prevent loss of resistance to leaf rust.

Conclusion

Despite the spectacular break-down of stem resistance in triticale, several further genes for resistance are available in CIMMYT lines and materials developed elsewhere. An awareness of the high frequencies of Sr27 and SrSatu in CIMMYT triticales and the knowledge that virulent pathotypes are present in Australia should enable rapid progress in broadening the genetic base for stem rust resistance in the CIMMYT triticale population. Although some wheat resistance genes occur in triticale, genetic diversity for resistance between the two crops should be maintained.

Acknowledgements

Financial support from the Australian Wheat Industry Research Council and the Rural Credits Development Fund is gratefully acknowledged.

References


Chapter 2

Resistance to Leaf and Stem Rusts in Wheat

A.P. Roelfs, Cereal Rust Laboratory, U.S. Department of Agriculture Research Service and the University of Minnesota, St. Paul, Minnesota

Abstract

Although the cereal rusts have been able to overcome many of the resistant cultivars developed during the past 80 years, many other cultivars have been successfully grown on large areas. Stem rust has been controlled by the use of resistance combinations that include Sr2 transferred to Hope and H-44 from emmer by McFadden in 1923. Sr25 (from Agropyron elongatum), Sr31 (Secale cereale), and Sr36 (Triticum timopheevii) seem to be the most effective single gene resistances worldwide. Thatcher (resistance from T. durum) developed by Hayes et al. in 1934, also has a useful level of resistance in most areas. Leaf rust has been successfully controlled by combination of Lr13 and 34. These resistances were first used in the cultivars Frontana (Brazil 1934) and Americano 44D (Uruguay 1918). This gene combination continues to be used in recent durable cultivars Chris, Era, Ciano 67, Pavon 76, etc. Assumptions about the genetics and durability of some types of resistance has hindered selection and development of resistance cultivars.

Introduction

This chapter contains a plant pathologist's view of resistance in wheat to P. recondita f.sp. tritici and P. graminis f.sp. tritici. The specific work cited in this manuscript is referenced. However, many of the ideas and concepts were developed over a period of years of experience and through numerous contacts with fellow workers. The latter are difficult if not impossible to cite. I gratefully acknowledge co-workers, technicians, and graduate students who stimulated these ideas.

Breeding for Resistance

Although exceptions exist, most breeding for rust resistance has been done using a series of field tests, with disease notes taken near the peak disease severity. This type of evaluation of disease has produced many resistant cultivars some of which proved to have a durable resistance over a range of environments for many years. However, many other cultivars were not as successful over the range. The failures or disappointments have often been emphasized by both breeders and pathologists. This had led to a series of suggestions that another 'type' of resistance was needed. Programs for breeding using slow rusting, minor genes, horizontal resistance, etc. were proposed and some were undertaken. In the discussions of these alternate mechanisms of resistance, it was assumed or perhaps hoped that they were somehow different and therefore also better than the resistance previously used.

Field Evaluations

Field evaluations for rust intensity in the nursery consist of taking a severity reading (% of tissue of a tiller or flag leaf infected) and the host response (the size of the lesion). The percent disease severity and the host response were combined into a single value, the average coefficient of infection. To do this, the disease severity was multiplied by a numerical notation for host response where immunity = 0.0, resistant =
0.2, moderately resistant = 0.4, mixed = 0.6, moderately susceptible = 0.8, and susceptible = 1.0. Thus, a 60MR (60 x 0.4 = 24) and 20S (20 x 1.0 = 20) give a similar average coefficient of infection value. Are these equal? Combining severity and host response can thus obscure differences in susceptibility and resistance. Also, differences in host response are based on lesion size and characteristics, not on spore production potential. The severity on a resistant line in a nursery may depend more on its neighbors than on its genotype for rust resistance (23). Perhaps it is time to redesign our nurseries for some types of resistance by blocking similar material together in the nursery or inserting a border row between test lines or planting three-row plots and limiting notes to the center row. For some types of resistance it will be necessary to redesign completely our nurseries for disease evaluation (see Rollow and McVey (26)). Thus, to test for resistance due to a longer latent period or lower receptivity (fewer lesions) it may be necessary to inoculate uniformly a nursery over a short period (1-7 days) and then score host response 14 to 21 days later. To be effective this nursery must be isolated from other inoculum sources.

Components of Resistance

Perhaps the first stage in breeding for resistance is the careful observation of the proposed resistance. We must ask why do we want this as a resistant parent? Under what conditions were we able to detect the resistance? Then we must design experiments or nurseries (using the same or similar conditions) to allow for the detection of this particular resistance in progenies. It may be easier to do this if we look at the mechanisms (components) of resistance. Four factors that can be measured are the number or lesions per unit of leaf or stem area (receptivity), size of sporulating area of the uredium, length of latent period (time from infection to sporulation), and length of sporulating period. The genes for resistance that have been studied in detail may affect one or more of these components. Sr2 reduces the number of lesions but not evenly throughout the plant life span nor on all host tissues (8, 31). Sr8a reduces the size but not the number of lesions. Sr36 lengthens the latent period and, with most cultures, also reduces the number of pustules (24, 25). Resistances, such as Sr23, that are expressed with chlorosis or necrosis often have shorter periods of sporulation for a given uredium. Early telia formation is another example of this type of resistance mechanism. Thus, knowing how the resistance is expressed should make it easier to design the proper test and to follow it through a breeding program. Breeding for resistance may not be made easier; in fact, more complicated notes may be required but the end result may be better. Notes may have to be taken at different times or in different ways depending on the cross. A uniform test or level of resistance across all crosses may be neither desirable nor possible.

Gene-for-Gene Relationship

Although all resistances may not be on a gene-for-gene basis, many are. Understanding the complexity of the interaction is important in breeding for resistances. It is often assumed that genes for resistance are dominant. Our experience would indicate that most resistance genes are expressed as incomplete dominants; a few are almost recessive. Thus, in a breeding program the heterozygous individuals will probably be more susceptible than the resistant parent. Temperature, inoculum density, and
host growth stage usually have some effect on the expression of resistance. Genes for virulence, likewise, are assumed to be recessive but this is not always the case. The typical gene-for-gene interaction is shown in Table 1. The representation is over-simplified in that the heterozygous genotypes of both host and pathogen are omitted. Additionally, the disease response is characterized only as a high or low infection type. Low infection types result (assuming resistance is dominant and avirulence is recessive) from homozygous and heterozygous avirulent pathogen cultures with either a homozygous or heterozygous resistant host genotype. The homozygous virulent culture with any host genotype or any pathogen with the susceptible host results in high infection types (13).

The simple gene-for-gene model in Table 1 is expanded in Table 2, showing the interaction between Sr7b in the host and the corresponding virulence/avirulence locus in the host. Note that, in Table 2, when the heterozygous pathogen or heterozygous host is involved in the interaction, detectable change in the low infection types occur (21). This is probably the usual case. A change in infection type often occurs when the temperature is altered (4). The P7bP7b pathogen genotype interaction with the three possible host genotypes in a segregating F2 population could be fitted to a 1:2:1 or 3:1 ratio. The P7bP7b pathogen genotype interaction could also be scored as a 1:2:1 or 3:1 ratio; however, the 3± and 4 infection types might be combined giving a 1:3 ratio of resistant to susceptible plants. This variation in infection type due to heterozygous individuals could cause the apparent loss or reduction in effectiveness of resistance when a heterozygous host genotype was evaluated or when a heterozygous culture was substituted for the homozygous avirulent culture. A range of phenotypes in the F2 is assumed to indicate polygenic control. However in a F2 population with a single host-pathogen gene pair responding to the pathogen, at least five different infection types

### Table 1. The gene-for-gene interaction as frequently shown with infection types represented as highs and lows

<table>
<thead>
<tr>
<th>Host</th>
<th>Pathogen</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P_</td>
</tr>
<tr>
<td>&lt;sub&gt;R_&lt;/sub&gt;</td>
<td>&lt;sub&gt;Low&lt;/sub&gt;</td>
</tr>
<tr>
<td></td>
<td>&lt;sub&gt;rr&lt;/sub&gt;</td>
</tr>
</tbody>
</table>

### Table 2. The gene-for-gene interaction for Sr7b and p7b

<table>
<thead>
<tr>
<th>Host</th>
<th>P7bP7b</th>
<th>P7bP7b</th>
<th>P7bP7b</th>
</tr>
</thead>
<tbody>
<tr>
<td>R7bR7b</td>
<td>2</td>
<td>23</td>
<td>4</td>
</tr>
<tr>
<td>R7bR7b</td>
<td>2+</td>
<td>3±</td>
<td>4</td>
</tr>
<tr>
<td>7b7b</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>
usually occur. If the test were performed under field conditions, additional phenotypes would be induced by an environmental effect on the host-pathogen interaction. The range of phenotypes is even more evident when two genes in both the host and the pathogen interact (Table 3). Many wheat cultivars in the rust-prone areas of the world have many genes for resistance, e.g. Marquis released in 1911 (Sr7b, 18, 19, 20, X), Selkirk in 1953 (Sr6, 7b, 9d, 17, 23, 2) and Centurk in 1972 (Sr6, 8a, 9a, 17); thus a single cultivar can result in a wide range of infection types and interactions if a wide spectrum of cultures is used.

**Interaction between Genes and Genomes**

Many accessions of wheats or wheat relatives with lower ploidy levels have often been looked to as potential sources of resistance to rust. Derivatives of some of these sources have been very useful, e.g. Thatcher and Hope to stem rust. However, many such attempts have been disappointing. As the resistance is transferred to successively higher ploidy levels, the expression of the resistance decreases (6). This would seem to be a ‘dilution’ effect. Perhaps this can be overcome in the future by transferring the resistance to another homoeologous pair so that

**Table 3. The gene-for-gene interaction for Sr6 and Sr7b**

<table>
<thead>
<tr>
<th>R6 R7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
<th>P6 P7b</th>
</tr>
</thead>
<tbody>
<tr>
<td>R6 R7b</td>
<td>0:</td>
<td>0:</td>
<td>0:</td>
<td>:1</td>
<td>:1</td>
<td>:1</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R6 R7b</td>
<td>0:</td>
<td>0:</td>
<td>0:</td>
<td>:1</td>
<td>:1</td>
<td>:1</td>
<td>23</td>
<td>32</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R6 R7b</td>
<td>0:</td>
<td>0:</td>
<td>0:</td>
<td>:1</td>
<td>:1</td>
<td>:1</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R6 R7b</td>
<td>1+</td>
<td>1+</td>
<td>1+</td>
<td>X-</td>
<td>X-</td>
<td>X-</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R6 R7b</td>
<td>1+</td>
<td>1+</td>
<td>1+</td>
<td>X-</td>
<td>X-</td>
<td>X-</td>
<td>23</td>
<td>32</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R6 R7b</td>
<td>1+</td>
<td>1+</td>
<td>1+</td>
<td>X-</td>
<td>X-</td>
<td>X-</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R6 R7b</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R6 R7b</td>
<td>3</td>
<td>32</td>
<td>4</td>
<td>23</td>
<td>32</td>
<td>4</td>
<td>23</td>
<td>32</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R6 R7b</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
four alleles of a resistance gene could be present in a cultivar. The 'dilution' effect is especially important for wheat leaf rust where broadly effective genes for resistance are few in hexaploid wheats and high levels of resistance exist in *Triticum monococcum* and *T. durum*. The resistance in these species is apparently effective and durable.

Recently, Dyck (unpublished) has described a suppressor gene for wheat stem rust on the 7D chromosome. This suppressor gene appears to be the same as, or very closely linked to, the *Lr34* locus. Fortunately, the *Lr34* resistance allele is an indication of the presence of the non-suppressed stem rust genotype. A great deal of work needs to be done in this area. Are there other suppressors? To what extent do they affect leaf rust resistance? Are they primarily on the D genome where they affect leaf rust resistance transferred from *T. durum* and *T. monococcum*?

When *Lr13* (adult plant resistance) was combined with *Lr16* (resistant at all stages), cultures virulent to *Lr16* were avirulent to seedling plants with *Lr13* and *Lr16* (29). In fact the low infection type produced appeared to be similar to that of a culture avirulent to *Lr16*. The enhancement of *Lr16* resistance by *Lr13* is an exciting discovery and should lead to the evaluation of many specific gene combinations. Generally, combinations of resistance genes result in an effect similar to the more resistant of the genes in the combinations. However, combinations of a gene that produce few pustules, i.e. *Sr2* combined with a gene that produces small pustules, i.e. *Sr24* results in fewer and smaller lesions.

**Durable Resistance**

Durable resistance is that resistance which has been adequate against the disease for a number of years over a range of environments and pathogen cultures. It should not be assumed that it will always be adequate in the future nor that it will be effective against all cultures. However, the use of a resistance that has been effective over a range of environments, cultures, and years is certainly more likely to lead to a resistant cultivar than untested resistance and certainly more apt to succeed than resistances that are known to have failed. In case of stem rust, there are several known sources of durable resistance related to a 'single' gene while, for wheat leaf rust, most durable resistance is associated with gene combinations.

**Wheat Stem Rust**

**Sr2**—This is an adult plant resistance (not effective until around the boot stage) which was derived from Yaroslav emmer by McFadden (17) and is generally available through the cultivars Hope and H-44 and their derivatives (Table 4). In North America, the spring wheat Selkirk and the hard red winter wheat Centurk have this gene in combination with others. This gene does not provide immunity and, under high inoculum densities, is often characterized by susceptible-type lesions near the nodes and in the spike and awns (8, 31). Cultivars with *Sr2* in combination with other genes have been grown on millions of hectares in the northern Great Plains of North America without serious disease for nearly 30 years. Eagle, a hard red winter wheat from Kansas with *Sr2*, was grown on over a million hectares for 5 years without stem rust losses. In combination with other effective resistances, *Sr2* is difficult to follow
in progenies. The brown necrosis associated with Sr2 has often been used to follow the resistance.

Sr26—This resistance, which was derived from *Agropyron elongatum*, has been an effective resistance against cultures obtained worldwide. It has been widely used in Australian cultivars (Table 4) which have been grown on a million hectares annually for over 10 years (14). This resistance should be easy to follow using standard breeding techniques.

### Table 4. Cultivars with selected genes for resistance to wheat stem rust

<table>
<thead>
<tr>
<th>Sr2</th>
<th>Sr31</th>
<th>Sr36</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eagle (Ks)</td>
<td>Advokat</td>
<td>Viri</td>
</tr>
<tr>
<td>Hartog</td>
<td>Agra</td>
<td>Weique</td>
</tr>
<tr>
<td>Kalkee</td>
<td>Ahmus</td>
<td>Wentzel</td>
</tr>
<tr>
<td>Kenya Page</td>
<td>Alondra</td>
<td>Winnetou</td>
</tr>
<tr>
<td>Kenya Phume</td>
<td>Aurora</td>
<td>Yan 7770-4</td>
</tr>
<tr>
<td>Lancer</td>
<td>Balkau</td>
<td>Yi 78-4078</td>
</tr>
<tr>
<td>Lawrence</td>
<td>Benno</td>
<td>Zorba</td>
</tr>
<tr>
<td>Lerma Rojo 64</td>
<td>Bezostaya 2</td>
<td></td>
</tr>
<tr>
<td>Madden</td>
<td>Burgas 1</td>
<td></td>
</tr>
<tr>
<td>Newthatch</td>
<td>Burgas 2</td>
<td></td>
</tr>
<tr>
<td>Xuri 70</td>
<td>Cebeco 97</td>
<td></td>
</tr>
<tr>
<td>Ottawa</td>
<td>Clement</td>
<td></td>
</tr>
<tr>
<td>Pembina</td>
<td>Cordillera</td>
<td></td>
</tr>
<tr>
<td>Renown</td>
<td>Danubia</td>
<td></td>
</tr>
<tr>
<td>Resene</td>
<td>Disponect</td>
<td></td>
</tr>
<tr>
<td>Scout</td>
<td>Feldkrone</td>
<td></td>
</tr>
<tr>
<td>Scout 66</td>
<td>Feng Kang 2</td>
<td></td>
</tr>
<tr>
<td>Selkirk</td>
<td>Feng Kang 8</td>
<td></td>
</tr>
<tr>
<td>Sonalika</td>
<td>Feng Kang 15</td>
<td></td>
</tr>
<tr>
<td>Songlen</td>
<td>Fundulea 29</td>
<td></td>
</tr>
<tr>
<td>Suneca</td>
<td>Fundulea 262</td>
<td></td>
</tr>
<tr>
<td>Sunkota</td>
<td>Gamtoes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Genaro F 81</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Glennison M81</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Goetz</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Granada</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hamlet</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Helios</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Huangfuien</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Iris</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Istra</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Jing Dan 106</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Jugoslavitsaya</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kaloyan</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kavkaz</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kronjuwel</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Licanka</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lima 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Linos</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Loeric</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lovrin 10</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lov:in 12</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lovrin 13</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lovrin 24</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Magister Cebeco</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mamut</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Merkur</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mildress</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Millalcan Inia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mironovskaya 10</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nizkoroskava</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nautica</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Odessa</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Odessa 4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Odesskaya 66</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Odilo</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Orlando</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pakistan 81</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Perseus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Poleskaya 71</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Predgornaya 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Roxana</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sabina</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Saladin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salzmunde 14/44</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Selektia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Seri 82</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Serie</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Shlorm</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Siouxland</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Skorospelka</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Skorospelka 35</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Slavia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Solaris</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sutjeska</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Transilvania 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ures T81</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Urban</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Very'S</td>
<td></td>
</tr>
</tbody>
</table>

From Bartos (1), Hu and Roelfs (10), Luig (14), and MeVey (unpublished)
screening either seedling or adult plants for resistance. Its disadvantage is the narrow agronomic background of the cultivars in which the resistance currently exists.

**Sr31**—This gene was derived from Imperial rye. It is currently widely spread in the world population in many wheats (Table 4). This gene is on the 1B/1R translocation which also carries Yr9 and Lr26, as well having a sticky dough, a poor mixing characteristic. Although the resistance is useful, further work is needed to remove the undesirable character.

**Sr36**—This gene has been successfully used in much of the United States but failed once in Australia (15). It reduces the number of lesions and increases the latent period (24, 25). This resistance loses its effectiveness at or near maturity. Sources are shown in Table 4.

**Thatcher**—This resistance was derived from lunillo durum by Hayes and others (9). In Thatcher a resistance exists in addition to that provided by the combination of Sr5, 9g, 12, and 16. Brennan (2) thought that was due to two recessive genes. Nazareno and Roells (19) indicated that the resistance was often

<table>
<thead>
<tr>
<th>Sr gene</th>
<th>Sourceb/</th>
<th>Seedling infection typec/</th>
<th>Adult plantd/</th>
<th>Chromosome locatione/</th>
<th>Effective</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>T. dicoccum</td>
<td>4</td>
<td>S (few uredia)</td>
<td>3BS</td>
<td>worldwide</td>
</tr>
<tr>
<td>5</td>
<td>T. aestivum</td>
<td>0.0;</td>
<td>R</td>
<td>6Da</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>T. aestivum</td>
<td>1.1</td>
<td>R</td>
<td>2Da</td>
<td></td>
</tr>
<tr>
<td>7a</td>
<td>T. aestivum</td>
<td>1C, 23C</td>
<td>MS</td>
<td>4BL</td>
<td></td>
</tr>
<tr>
<td>7b</td>
<td>T. aestivum</td>
<td>2.2;</td>
<td>MS</td>
<td>4BL</td>
<td></td>
</tr>
<tr>
<td>8a</td>
<td>T. aestivum</td>
<td>2</td>
<td>MS</td>
<td>6Ba</td>
<td></td>
</tr>
<tr>
<td>8b</td>
<td>T. aestivum</td>
<td>X</td>
<td>MS</td>
<td>6Ba</td>
<td></td>
</tr>
<tr>
<td>9a</td>
<td>T. aestivum</td>
<td>2.2;</td>
<td>MS</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>9b</td>
<td>T. aestivum</td>
<td>2</td>
<td>2BL</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>9d</td>
<td>T. dicoccum</td>
<td>1.2;</td>
<td>R-MR</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>9e</td>
<td>T. dicoccum</td>
<td>1.2;</td>
<td>R-MR</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>9f</td>
<td>T. aestivum</td>
<td>1.2</td>
<td>?</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>9g</td>
<td>T. durum</td>
<td>2</td>
<td>MR</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>9h</td>
<td>T. aestivum</td>
<td>1.1; N, 23C</td>
<td>MR-MS</td>
<td>6BL</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>T. aestivum</td>
<td>2.2; c3</td>
<td>MR</td>
<td>6BL</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>T. durum</td>
<td>0.8;</td>
<td>MR</td>
<td>3BS</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>T. durum</td>
<td>2.2;</td>
<td>MR</td>
<td>6Ab</td>
<td>worldwide</td>
</tr>
<tr>
<td>14</td>
<td>T. durum</td>
<td>12C, 23C</td>
<td>MR-MS</td>
<td>1BL</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>T. aestivum</td>
<td>1.1; X, XN</td>
<td>MS-S</td>
<td>7AL</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>T. aestivum</td>
<td>2.2;</td>
<td>MS</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>T. dicoccum</td>
<td>1.1, X, XN</td>
<td>MR</td>
<td>7BL</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>T. aestivum</td>
<td>2</td>
<td>?</td>
<td>1DL</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>T. aestivum</td>
<td>1</td>
<td>?</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>T. aestivum</td>
<td>2</td>
<td>?</td>
<td>2BL</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>T. monococcum</td>
<td>1.2;</td>
<td>MR</td>
<td>2AL</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>T. boeoticum</td>
<td>0.2;</td>
<td>R-MR</td>
<td>7AL</td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>T. aestivum</td>
<td>23C, 1; C</td>
<td>MS-S, MR</td>
<td>4A</td>
<td>worldwide</td>
</tr>
</tbody>
</table>
associated with Sr12 in lines derived from Thatcher. This resistance is more effective in the field under low to moderate rust epidemics where it is generally rated susceptible, with a low (5-30\%) severity on the modified Cobb scale; however, in severely diseased nurseries, severities of 80-90\% are common. On seedlings, it produces an X' or X" infection type at 18°C but is susceptible at 20°C. This resistance has been effective in the northern Great Plains since Thatcher was released in 1934 although, in the 1953 and 1954 epidemics, it was damaged when grown in conjunction with the more susceptible Lee and durum wheats. It is probably present in most of the Thatcher derivatives grown in this area. Chris, Era, Neepawa, and Columbus may be examples of cultivars with this resistance. Many of the CIMMYT cultivars from CIANO 67 onwards may have some resistance from Thatcher, but this has not been proved.

In Table 5 the designated genes for stem rust resistance are listed, along with infection types produced on seedlings by avirulent cultures chromosomal locations, sources of resistance, and where the gene is effective against the natural population of \textit{P. graminis}.

Table 5. (continued)

<table>
<thead>
<tr>
<th>Sr gene Source(^b)</th>
<th>Response to an avirulent culture (^a)/</th>
<th>Seeding infection type(^c)</th>
<th>Adult plant(^d)</th>
<th>Chromosome location(^e)</th>
<th>Effective</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 A. elongatum</td>
<td></td>
<td>2.2</td>
<td>MS</td>
<td>3DL</td>
<td>not in S. Africa</td>
</tr>
<tr>
<td>25 A. elongatum</td>
<td></td>
<td>2.2</td>
<td>MS</td>
<td>7DL</td>
<td>worldwide</td>
</tr>
<tr>
<td>26 A. elongatum</td>
<td></td>
<td>2</td>
<td>RMR</td>
<td>6A</td>
<td>worldwide</td>
</tr>
<tr>
<td>27 S. cereale</td>
<td></td>
<td>0</td>
<td>R</td>
<td>3A</td>
<td>not in Australia</td>
</tr>
<tr>
<td>28 T. aestivum</td>
<td></td>
<td></td>
<td>R</td>
<td>2BS</td>
<td>South Asia</td>
</tr>
<tr>
<td>29 T. aestivum</td>
<td></td>
<td>2.2</td>
<td>MR</td>
<td>6D</td>
<td>worldwide</td>
</tr>
<tr>
<td>30 T. aestivum</td>
<td></td>
<td>2</td>
<td>MR</td>
<td>5D</td>
<td>worldwide</td>
</tr>
<tr>
<td>31 S. cereale</td>
<td></td>
<td>0.2</td>
<td>R</td>
<td>1B</td>
<td>worldwide</td>
</tr>
<tr>
<td>32 Ae. squarrosa</td>
<td></td>
<td>2</td>
<td>MR</td>
<td>2AS</td>
<td>worldwide</td>
</tr>
<tr>
<td>33 Ae. squarrosa</td>
<td></td>
<td>2</td>
<td>MR</td>
<td>1DL</td>
<td>worldwide</td>
</tr>
<tr>
<td>34 T. aestivum</td>
<td></td>
<td>5C,1 + C</td>
<td>MS,SM</td>
<td>2A</td>
<td></td>
</tr>
<tr>
<td>35 T. monococcum</td>
<td></td>
<td>0</td>
<td>R</td>
<td>3A</td>
<td></td>
</tr>
<tr>
<td>36 T. timophevii</td>
<td></td>
<td>0.0.1 + 0.1</td>
<td>R,S (few uredia)</td>
<td>2BS</td>
<td></td>
</tr>
<tr>
<td>37 T. timophevii</td>
<td></td>
<td>0</td>
<td>R</td>
<td>4AB</td>
<td>worldwide</td>
</tr>
<tr>
<td>Ru2 1. R. austivum</td>
<td></td>
<td>2</td>
<td>'</td>
<td>2B</td>
<td></td>
</tr>
<tr>
<td>17 T. aestivum</td>
<td></td>
<td>21CN,1 + C</td>
<td>MR</td>
<td>2D</td>
<td></td>
</tr>
<tr>
<td>HR T. dicoccum</td>
<td></td>
<td>2C</td>
<td>MS</td>
<td>'?</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\) Updated from Roebs and McVey (22), Loug (14), Roebs (20) \(^b\) Triticum T. Aegoporum A. Argoblos A. Secale \(^c\) Infection types at 18°C (plants with Sr6, 10, 12, 15, and 17 are more susceptible at higher temperatures, whereas plants with Sr13 are more resistant); variation is encountered with host genotype background and ploidy level: 55 (Knott, 11; Loug and Rajam, 10) \(^d\) Infection types at 18°C (plants with Sr6, 10, 12, 15, and 17 are more susceptible at higher temperatures, whereas plants with Sr13 are more resistant); variation is encountered with host genotype background and ploidy level: 55 (Knott, 11; Loug and Rajam, 10) \(^e\) Many host resistances are less effective at high temperatures, high inoculum densities, and at plant maturity. Variations also occur with different host genetic background. R = resistant, MR = moderately resistant, MS = moderately susceptible and S = susceptible \(^f\) Updated from McIntosh (18) \(^g\) Gene from 13, 14, other than Sr7, 9d, and 17. SrH was the cause of the differences in Canadian and United States survey data in the 1970s (7)
Wheat Leaf Rust

Leaf rust is probably the most important disease of wheat on a worldwide basis (28). Durable resistance to leaf rust is thought to be more difficult to obtain than with stem rust but some successes have been recorded. Leaf rust is more diverse for virulence than stem rust. This diversity may be the result of one or more factors. First, the population that survives between wheat crops probably is much larger for leaf rust. Second, the pathogen population size is currently much larger during the crop season. Third, resistance deployed against leaf rust has often been a single gene at a time. Thus population sizes are large, which results in a greater probability of mutants (30) and a greater probability that a greater diversity of virulence/virulence combinations can survive the non-wheat growing period. The use of cultivars with single effective genes for resistance permits mutations at single pathogen loci to render resistances ineffective. The sexual cycle of _P. recondita_ f.sp. _tritici_ is not generally thought to have a major effect (5, 27). Evidence that parasexual recombinants survive in nature, if they occur at all, is lacking. Although _P. recondita_ has a wide range, the host range of _P. recondita_ f.sp. _tritici_ seems to be limited to _Triticum_ and perhaps a few very closely related genera. However, additional research is desirable on the role of non- _Triticum_ species on survival of _P. recondita_ f.sp. _tritici_, especially during the non-wheat growing period.

Leaf rust resistance provided by the single genes (Table 6), with the exception of _Lr19_, are inadequate by themselves. _Lr19_ resistance has yet to be tested on commercial acreage so its durability is not proven. Unfortunately, _Lr19_ has usually been associated with a yellow flour color. The other genes listed are useful only in combinations of two or more.

Durable Resistance to Leaf Rust

The most durable resistance to leaf rust is associated with a few gene combinations (Table 7). It appears that _Lr13_ and perhaps _Lr12_, both adult plant resistances, in combinations with _Lr34_, are the basis of most of this resistance. The original source of these genes is unknown but apparently _Lr13_ and _Lr34_ were present in Alfredo Chaves, a land cultivar found in Brazil about 1921. _Americano_ 44D was selected in 1918 from a land cultivar in Uruguay. We have not determined its genotype for resistance, but it probably includes _Lr12_ and/or _Lr13_ and _Lr34_. Thus, these two land cultivars, which may be very similar, have been the resistance source of most of the durably resistant cultivars. It is assumed that these land cultivars had a southern European origin but no European cultivars are known to have this level of resistance. Resistance conditioned by _Lr13_ or _Lr12_ is sometimes inadequate under conditions that are very favorable for the disease, in nurseries in which inoculum levels are very high, in areas where wheat is grown at high temperatures, and in cultivars without other resistance.
Table 6. Known host genes for resistance to wheat leaf rust and their response to *P. recondita f.sp. tritici*

<table>
<thead>
<tr>
<th>Lr gene</th>
<th>Sourceb/</th>
<th>Seedling infection typec/</th>
<th>Adult plantd/</th>
<th>Chromosome locatione/</th>
<th>Effective</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td><em>T. aestivum</em></td>
<td>0:</td>
<td>R</td>
<td>5DL</td>
<td></td>
</tr>
<tr>
<td>2a</td>
<td><em>T. aestivum</em></td>
<td>0:</td>
<td>R</td>
<td>2Da</td>
<td></td>
</tr>
<tr>
<td>2b</td>
<td><em>T. aestivum</em></td>
<td>0:</td>
<td>R</td>
<td>2Da</td>
<td></td>
</tr>
<tr>
<td>2c</td>
<td><em>T. aestivum</em></td>
<td>0/1N</td>
<td>R</td>
<td>2Da</td>
<td></td>
</tr>
<tr>
<td>3a</td>
<td><em>T. aestivum</em></td>
<td>C:</td>
<td>R</td>
<td>6BL</td>
<td></td>
</tr>
<tr>
<td>3(bg)</td>
<td><em>T. aestivum</em></td>
<td>C:23</td>
<td>MR:MS</td>
<td>6BL</td>
<td>in combinations</td>
</tr>
<tr>
<td>3(ka)</td>
<td><em>T. aestivum</em></td>
<td>C:12C</td>
<td>MR</td>
<td>6BL</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td><em>Ac. umbellulata</em></td>
<td>0:</td>
<td>R</td>
<td>6BL</td>
<td>in combinations</td>
</tr>
<tr>
<td>10</td>
<td><em>T. aestivum</em></td>
<td>0:2</td>
<td>R:MS</td>
<td>1AS</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td><em>T. aestivum</em></td>
<td>Y:</td>
<td>MR</td>
<td>2A</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td><em>T. aestivum</em></td>
<td>-</td>
<td>R</td>
<td>4A</td>
<td>in combinations</td>
</tr>
<tr>
<td>13</td>
<td><em>T. aestivum</em></td>
<td>-</td>
<td>R</td>
<td>2BS</td>
<td>in combinations</td>
</tr>
<tr>
<td>14A</td>
<td><em>T. dicoccum</em></td>
<td>X</td>
<td>MS</td>
<td>7BL</td>
<td></td>
</tr>
<tr>
<td>14B</td>
<td><em>T. aestivum</em></td>
<td>X</td>
<td>MS</td>
<td>7BL</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td><em>T. aestivum</em></td>
<td>C:</td>
<td>R</td>
<td>2Da</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td><em>T. aestivum</em></td>
<td>0:N1</td>
<td>MS:MR</td>
<td>4A</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td><em>T. aestivum</em></td>
<td>0:1+N</td>
<td>MR:MS</td>
<td>2AS</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td><em>T. aestivum</em></td>
<td>2:</td>
<td>MS</td>
<td>5BL</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td><em>A. intermedium</em></td>
<td>0:</td>
<td>R</td>
<td>7DL</td>
<td>worldwide?</td>
</tr>
<tr>
<td>20</td>
<td><em>T. aestivum</em></td>
<td>0:</td>
<td>R</td>
<td>7AL</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td><em>Ac. squarrosa</em></td>
<td>0:</td>
<td>R</td>
<td>1DL</td>
<td></td>
</tr>
<tr>
<td>22A</td>
<td><em>Ac. squarrosa</em></td>
<td>-</td>
<td>R</td>
<td>2Da</td>
<td></td>
</tr>
<tr>
<td>22B</td>
<td><em>T. durum</em></td>
<td>-</td>
<td>R</td>
<td>2Da</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td><em>A. elongatum</em></td>
<td>0:1</td>
<td>MR</td>
<td>2B</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td><em>S. cereale</em></td>
<td>:N</td>
<td>R</td>
<td>4Ab</td>
<td>in combinations</td>
</tr>
<tr>
<td>26</td>
<td><em>S. cereale</em></td>
<td>0:</td>
<td>R</td>
<td>1BL</td>
<td></td>
</tr>
<tr>
<td>27</td>
<td><em>T. aestivum</em></td>
<td>0:</td>
<td>MR</td>
<td>3BS</td>
<td>only with Lr31</td>
</tr>
<tr>
<td>28</td>
<td><em>Ac. speltoides</em></td>
<td>0:</td>
<td>R</td>
<td>4HL</td>
<td></td>
</tr>
<tr>
<td>29</td>
<td><em>A. intermedium</em></td>
<td>0:</td>
<td>R</td>
<td>7DS</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td><em>T. aestivum</em></td>
<td>:1</td>
<td>R</td>
<td>4HL</td>
<td></td>
</tr>
<tr>
<td>31</td>
<td><em>T. aestivum</em></td>
<td>-</td>
<td>MR</td>
<td>4Ab</td>
<td>only with Lr27</td>
</tr>
<tr>
<td>32</td>
<td><em>Ac. squarrosa</em></td>
<td>0:1</td>
<td>MR</td>
<td>3D</td>
<td></td>
</tr>
<tr>
<td>33</td>
<td><em>T. aestivum</em></td>
<td>1:</td>
<td>MR</td>
<td>1BL</td>
<td></td>
</tr>
<tr>
<td>34(T2)</td>
<td><em>T. aestivum</em></td>
<td>23C</td>
<td>MS</td>
<td>7D</td>
<td>in combinations</td>
</tr>
</tbody>
</table>

a/ Updated from Browder (3) and Long (unpublished)
b/ *Triticum* = *T., Agropyron* = *A., Aegilops* = *Ac., and Secale = *S.*
c/ Infection types are at 20°C, can be more or less resistant at other temperatures (4)
d/ Many host resistances are less effective at high temperatures, high inoculum densities, and at plant maturity. Variations also occur with different host genetic background.

R = resistant, MR = moderately resistant, MS = moderately susceptible, and S = susceptible

e/ Updated from McIntosh (18)
Table 7. Bread wheat cultivars with durable leaf rust resistance

<table>
<thead>
<tr>
<th>Name</th>
<th>Habit</th>
<th>Source</th>
<th>Released</th>
<th>Probable source of resistance</th>
<th>Lr gene(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Americano 41D</td>
<td>spring</td>
<td>Uruguay</td>
<td>1918</td>
<td>land race</td>
<td>13, +</td>
</tr>
<tr>
<td>Frondo,,</td>
<td>spring</td>
<td>Brazil</td>
<td>1934</td>
<td>Alfredo Chaves (land race)</td>
<td>13, +</td>
</tr>
<tr>
<td>Fronteira</td>
<td>spring</td>
<td>Brazil</td>
<td>1934</td>
<td>Alfredo Chaves (land race)</td>
<td>13, +</td>
</tr>
<tr>
<td>Surpreza</td>
<td>spring</td>
<td>Brazil</td>
<td>1934</td>
<td>Alfredo Chaves (land race)</td>
<td>13, +</td>
</tr>
<tr>
<td>Frontana</td>
<td>spring</td>
<td>Brazil</td>
<td>1943</td>
<td>Frondo</td>
<td>13,4, T3</td>
</tr>
<tr>
<td>La Prevision 3</td>
<td>spring</td>
<td>Argentina</td>
<td>1935</td>
<td>Americano 41D</td>
<td>13,4, +</td>
</tr>
<tr>
<td>La Prevision 25</td>
<td>spring</td>
<td>Argentina</td>
<td>1937</td>
<td>Americano 41D</td>
<td>13,4, +</td>
</tr>
<tr>
<td>La Prevision 28</td>
<td>spring</td>
<td>Argentina</td>
<td>1937</td>
<td>Americano 41D</td>
<td>13,4, +</td>
</tr>
<tr>
<td>Klein Aniversario</td>
<td>spring</td>
<td>Argentina</td>
<td>1945</td>
<td>Americano 41D</td>
<td>13,3ka, +</td>
</tr>
<tr>
<td>Klein Cometa</td>
<td>spring</td>
<td>Argentina</td>
<td>1942</td>
<td>Americano 41D</td>
<td>13, +</td>
</tr>
<tr>
<td>Klein Lucero</td>
<td>spring</td>
<td>Argentina</td>
<td>1950</td>
<td>Americano 41D</td>
<td>13, +</td>
</tr>
<tr>
<td>Klein Progress</td>
<td>spring</td>
<td>Argentina</td>
<td>1937</td>
<td>Americano 41D</td>
<td>13, +</td>
</tr>
<tr>
<td>Klein Rendidor</td>
<td>spring</td>
<td>Argentina</td>
<td>1954</td>
<td>Americano 41D</td>
<td>13, +</td>
</tr>
<tr>
<td>Klein Sinvallecho</td>
<td>spring</td>
<td>Argentina</td>
<td>1943</td>
<td>Americano 41D</td>
<td>13, +</td>
</tr>
<tr>
<td>Klein Titan</td>
<td>spring</td>
<td>Argentina</td>
<td>1925</td>
<td>Americano 41D</td>
<td>13, +</td>
</tr>
<tr>
<td>Klein Vencedor</td>
<td>spring</td>
<td>Argentina</td>
<td>1925</td>
<td>Americano 41D</td>
<td>13, +</td>
</tr>
<tr>
<td>Ciano 67</td>
<td>spring</td>
<td>CIMMYT</td>
<td>1967</td>
<td>Ciano 67'S</td>
<td>13, +</td>
</tr>
<tr>
<td>Pavon F76</td>
<td>spring</td>
<td>CIMMYT</td>
<td>1976</td>
<td>?</td>
<td>10,12,34</td>
</tr>
<tr>
<td>Miner</td>
<td>winter</td>
<td>USA</td>
<td>1949</td>
<td>Klein Sinvallecho</td>
<td>3, +</td>
</tr>
<tr>
<td>Study</td>
<td>winter</td>
<td>USA</td>
<td>1960</td>
<td>Surpreza</td>
<td>13, +</td>
</tr>
<tr>
<td>Gage</td>
<td>winter</td>
<td>USA</td>
<td>1963</td>
<td>Frondo</td>
<td>13, +</td>
</tr>
<tr>
<td>Redcoat</td>
<td>winter</td>
<td>USA</td>
<td>1960</td>
<td>Frontana</td>
<td>13,34, +</td>
</tr>
<tr>
<td>Atlas 66</td>
<td>winter</td>
<td>USA</td>
<td>1948</td>
<td>Frontana</td>
<td>10,13,34, +</td>
</tr>
<tr>
<td>Chris</td>
<td>spring</td>
<td>USA</td>
<td>1965</td>
<td>Frontana</td>
<td>13,34, +</td>
</tr>
<tr>
<td>Era</td>
<td>spring</td>
<td>USA</td>
<td>1970</td>
<td>Frontana</td>
<td>10,13,34, +</td>
</tr>
</tbody>
</table>

References


Chapter 3

Pathogenicity Analysis of Yellow (Stripe) Rust of Wheat and Its Significance in a Global Context

R.W. Stubbs, Research Institute for Plant Protection, Wageningen, The Netherlands

Abstract

Yellow (stripe) rust of wheat (Puccinia striiformis Westend. f.sp. tritici) is studied by the Research Institute for Plant Protection (IPO) on an international scale. Races (virulences) are identified on seedlings of a broad set of 'old' and 'new' differential cultivars with some known, but mostly unknown, resistance genes under controlled conditions. Virulence related to race-specific mature plant resistance is analyzed in race nurseries (separate field plots). The relationship between the distribution of pathogen virulences and host resistances is evident but yet understudied. Results are presented of an analysis of yellow rust infecting triticale and cultivars with resistance derived from rye. The zonal distribution of yellow rust races in Europe, Africa, Asia, and South America is described. A continuous survey of changes in pathogenicity in race populations is highly recommended with regard to breeding for resistance and the evaluation of host resistance under different environments.

Introduction

Yellow (stripe) rust, caused by Puccinia striiformis Westend., is one of the major rust diseases of wheat; it also attacks barley, rye (Secale cereale), and other grasses. As far as is known, the fungus does not attack oats (Avena sativa), rice (Oryza sativa), or maize (Zea mays). The form infecting wheat is referred to as P. striiformis f.sp. tritici and the one infecting barley as P. striiformis f.sp. hordei. Yellow rust has no alternate host and mutation and somatic recombination are the mechanisms of variability. It is assumed that Transcaucasia is the center of origin of the fungus. From this center, yellow rust dispersed in all directions, reaching Australia only in 1979 (20). Yellow rust is considered to be a low-temperature pathogen and is serious in areas in which cool, moist weather prevails, as in northwestern Europe and mountainous regions of South America and East Africa. The minimum, optimum, and maximum temperatures for spore germination are 0°C, 9-12°C, and 20-26°C, respectively (24). Yellow rust is also characterized by its systemic spread in the leaf. Severe leaf and head infections may cause total losses in yield. Of the three wheat rusts, yellow rust appears to be the most sensitive to environmental factors, such as air pollution, which reduces germination of urediospores (25). Resistance of the host is much influenced by temperature and light which, in turn, influence disease assessment of the infected plant (26). Since 1956, the Research Institute for Plant Protection (IPO) in Wageningen, The Netherlands, has been engaged in studying the pathogenicity of yellow rust on an international scale. The present collection of yellow rust specimens contains 5000 cultures from 60 countries.
Pathogenicity Analysis

Methodology in the seedling stage

Techniques of handling the pathogen—The techniques of handling yellow rust in the glasshouse in the IPO were developed by Zadoks (41). These techniques, slightly modified, are as follows. Yellow rust cultures are grown separately in plastic cages in a glasshouse held at 15 + 2°C. Daylength is kept constant at 16 hours by supplementing light with fluorescent tubes giving a light intensity of 7500 lux. Yellow rust is difficult to grow in late autumn and winter, due to light deficiency which reduces sporulation intensity (18) and to air pollution being relatively high at the time. Spores, which are normally viable when samples are received within 2 weeks of collection, are transferred to seedlings of a susceptible cultivar. Older samples are placed in moist Petri dishes in a refrigerator to induce sporulation of the fungus. After inoculation, the seedlings are kept for 48 hours in a dew chamber held at 9°C and 16 hours light of intensity of about 500 lux and 8 hours dark. Urediospores appearing 10-14 days after inoculation are collected every 2 days, dried, and then stored in glass ampules in liquid nitrogen (-196°C). In contrast to leaf and stem rusts, urediospores of yellow rust stored in liquid nitrogen do not need to be reactivated by thawing in a water bath at a temperature of 40°C. No differences were found in germination when spores were thawed between 5 and 40°C.

The wheat cultivars used for growing *P. striiformis* f.sp. *tritici*, *P. striiformis* f.sp. *hordei*, and yellow rust on grasses (15), is commonly used for transferring the rust samples. However, it should be noted that this species is resistant to *P. striiformis* f.sp. *hordei* race 57, prevalent in the Indian Subcontinent.

The generation time (time between date of inoculation and date of sporulation) can be race-dependent as observed by Fuchs (8) who grouped races into slow, normal, and fast. The difference between the first and last group is 4 days.

Inoculation of large numbers of cultivars is done by atomizing spores suspended in mineral oil (Soltrol 170) which is somewhat toxic to barley. For barley, therefore, the urediospores are mixed with spores of *Lycopodium*. As the temperature in the pre-inoculation phase influences resistance expression (4), the plants are grown in the pre- as well as in the post-inoculation phase in growth chambers under a day/night regime of 18/15°C and 18 hours/6 hours. The light intensity is around 20,000 lux produced by a combination of high-pressure mercury lamps and high-pressure sodium lamps. Depending on the generation time of the races, the infection types are observed 14-17 days after inoculation and scoring is done on the 0-9 scale (19).

Identification of races (virulences)—The study of the physiologic races of yellow rust is not yet far advanced. A limiting factor is the inadequate information on resistance genes in the host which are necessary for identification of virulence genes in the pathogen. Attempts have been, or are being, made to develop isogenic lines of known resistance genes.
Physiologic specialization was first demonstrated in 1930 (1). Gassner and Straib (9) introduced a system of race identification and nomenclature which was revised by Fuchs (8), and was used until a new system was proposed in 1972 (13). The old system is still in use in the Soviet Union (27, 38) and Iran (2). The set of differential cultivars introduced by Gassner and Straib consisted of 11 wheat cultivars, six barleys, one rye, and one *Triticum dicoccum* var. *tricoccum*. In the nomenclature, no distinction has been made in the specialized forms. The reaction types of 66 races on these differentials have been summarized by Stubbs (35). Noteworthy is the inclusion of Petkus rye which contributed resistance to yellow, stem, and leaf rusts in chromosome 1R (42) to many wheat cultivars presently grown or being developed. Straib (28) found only one race (race 34), giving a susceptible reaction on Petkus rye, but a resistant reaction on all wheat differentials. This race may have belonged to f.sp. secalis. Petkus rye unused for many years is now included in our study of the pathogenicity of yellow rust on triticale.

In our routine work on race identification, wheat differentials are used, as proposed by Johnson et al. (13). 'Old' differential cultivars such as Blé Rouge d'cosse and Holzapfel's Früh have been abandoned, which is regrettable because they possess resistance genes that may still be present but unrecognizable in cultivars presently grown or being developed. In recent tests, virulence and avirulence for Blé Rouge d'Écosse was clearly shown by non-European yellow rust cultures. The question is whether this virulence is 'necessary' or 'unnecessary' in the rust population. The same applies to the virulence to the cultivars which have supplemented the standard set of differentials. These cultivars are, among others, Mexipak = Kalyansona and Giza 155 (31) and Anza (4+), among others.

Registration of these virulences is important in the evaluation of host resistance. However, the consequence of adding new supplementals is that hundreds of old yellow rust cultures have incomplete virulence formulas.

At present, much attention is given to the analysis of samples from triticale and from wheat cultivars with resistance derived from rye (1B/1R substitution or translocation). The resistance gene Yr9 is a rye gene and is present in differential cultivars Riebesel 47/51 (Crielwener 104/Petkus rye) and Clement (Yr9 + 2?). The resistance of the latter was overcome in The Netherlands in the second year of its cultivation (1974). Riebesel 47/51 is susceptible in the seedling stage but so far shows a high degree of mature plant resistance. Table 1 exemplifies the analysis of samples from wheats with resistance derived from rye and from triticale. Considerable complexity is evident. Race 6E0 was found in Ecuador in 1985, infecting both wheat with Yr9 and triticale (P. Fox, pers. comm.). Race 6E150 severely attacked triticale in Rwanda (E. Torres, pers. comm.). Race 140E12 did the same in Zambia. Race 134E150 was also found in Kenya on triticales (D. Daniel, pers. comm.) which were much less infected than those in Rwanda (E. Torres, pers. comm.). Race 234E171 overcame the resistance of Granada (SR/GB//Triticale/ Thatcher/Taca/Jubilaer) in The Netherlands in 1985, in the second year of its cultivation. Race 191E206 infected Lovrin 13 in China (Yang Hua-an, pers. comm.). All these races are virulent for Yr9 but react differently on Clement, Granada, and Delphin. The latter
two may possess resistance genes differing from Yr9. In field tests, the triticale cultivars Mapache and Rosner showed resistance to Dutch races with avirulence for Yr9 and susceptibility to races virulent for Yr9. This indicates the possible presence of Yr9 in triticale. Thus much useful information can be acquired from maintenance of a "bank" of pathogen samples.

Methodology in the mature plant stage

The analysis of pathogenicity of yellow rust in the post-seedling stage is done by the IPO in race nurseries (41). The nurseries are inoculated in the second half of April and, depending on the disease development, 3-5 observations are made at intervals of 8-10 days. The severity of infection is assessed on a 0-100% scale and the infection type on a 0-9 scale (19). The disease progress in each race-or isolate-cultivar combination is expressed as a compatibility index (41). The compatibility index varies from 0 to 100 and, from the epidemiological point of view, a compatibility index above 15 is considered to be dangerous (41). The value 100-compatibility index is used as a parameter of the so-called 'rest resistance' which is assumed to be race-non-specific (32, 41). The susceptible checks are Michigan Amber and Itana/PI 178383 selection 111 (S111) having one temperature-sensitive minor gene (26). The latter is used to detect change in infection type and, consequently, change in the disease progress with change of temperature in the race nurseries.

In Table 2, examples are given of isolates that perform similarly on the standard set of differential cultivars in the greenhouse or growth room, but differently on cultivars having mature plant resistance. Accordingly, the isolates have been named 'greenhouse races' and 'field races,' respectively (41). Field races are usually named after the cultivars from which they were obtained. Most of the cultivars hardly show their race-specific mature plant resistance in the seedling stage or, at least, do not.

Table 1. Seedling reactions\(^a\) of wheat cultivars with Yr9, and triticale after inoculation with yellow rust races with virulence to Yr9

<table>
<thead>
<tr>
<th>Race</th>
<th>Virulence formula(^c)</th>
<th>Origin</th>
<th>Clement (^d) 9 + 2?</th>
<th>Granada 9 + 2?</th>
<th>Fed.4 Kvak.d/9</th>
<th>Fink's se/9</th>
<th>Triticale Salvo?</th>
<th>Delphin?</th>
</tr>
</thead>
<tbody>
<tr>
<td>6E0</td>
<td>6.7.9.A</td>
<td>Ecuador</td>
<td>R</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>R</td>
</tr>
<tr>
<td>6E150</td>
<td>2.6.7.8.9.A</td>
<td>Rwanda</td>
<td>R</td>
<td>R</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td>1+PE12</td>
<td>3.6.9.A</td>
<td>Zambia</td>
<td>S</td>
<td>R</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td>13E150</td>
<td>2.6.7.8.9.A</td>
<td>Kenya</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>R</td>
</tr>
<tr>
<td>23E171</td>
<td>2.3.4.7.8.9.Su.C5</td>
<td>Netherlands</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td>175E142</td>
<td>1.2.3.6.7.9.P</td>
<td>China(^f)</td>
<td>S</td>
<td>R</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>R</td>
</tr>
</tbody>
</table>

\(^a\) Susceptible (types 7-9); R = resistant (types 0-6); = no data
\(^b\) Nomenclature after Johnson et al. (13)
\(^c\) According to resistance genes Yr 1.2.3 etc., A = Avocet (39); Su = Sw on 92, C5 / Carsten's "S" V.
\(^d\) Federation's Kvakaz developed by the Plant Breeding Institute, Castle Hill, Australia
\(^e\) Data obtained by D.L. Daniel (CIMMYT, Kenya) in the IPO
\(^f\) Data obtained by Yang Hua-an (Institute for Plant Protection, Beijing) in the IPO
Table 2. Infection spectrum of races of *P. striiformis* f.sp. *tritici* on wheat cultivars in the mature plant stage

<table>
<thead>
<tr>
<th>Cultivar (genotype)</th>
<th>32E0</th>
<th>32E0/Alba</th>
<th>Triumph</th>
<th>32E128</th>
<th>Heine's7</th>
<th>Heine's4</th>
<th>32E128</th>
<th>Leda</th>
<th>36E132</th>
<th>Flamingo</th>
<th>106E139</th>
<th>Lely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Check</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>Heine's VII (2 + ?)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>48</td>
<td>49</td>
<td>50</td>
<td>34</td>
<td>40</td>
<td>50</td>
<td>50</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>H. Kolben (6 + ?)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>73</td>
<td>64</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Alba (Ab + ?)</td>
<td>54</td>
<td>1</td>
<td>7</td>
<td>0</td>
<td>31</td>
<td>8</td>
<td>6</td>
<td>0</td>
<td>40</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>D. Triumph (DT + ?)</td>
<td>3</td>
<td>1</td>
<td>63</td>
<td>3</td>
<td>5</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Heine's IV (H4 + ?)</td>
<td>0</td>
<td>0</td>
<td>68</td>
<td>0</td>
<td>43</td>
<td>46</td>
<td>0</td>
<td>39</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Leda (2 + Ab + H4)</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>23</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Flamingo (2 + 6 + H4)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>10</td>
<td>39</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Lely (2 + 7 + Ab)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>55</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*a/* Nomenclature based on seedling reactions of the standard set of differential cultivars (13)
b/ Trivial name
c/ Cultivar of the standard set of differential cultivars
not do so at the temperature used for identification of races. A few cultivars may do so at other temperatures which indicates that their resistance is temperature-sensitive as well as race-specific (29). It is worthwhile to mention here the causes of the break-down of the resistance of Lely which had a life of 6 years, relatively long as compared to other cultivars (which often last only 1 or 2 years). Lely had two effective genes (Yr7 and YrAb) when first cultivated. Then, in France, race 106E139 appeared and was virulent to Yr7 but avirulent to YrAb. In France, this race overcame the resistance of those cultivars having Yr7 as the only protection against the prevailing race 104E137. The progeny of race 106E139, the Lely-race, completed the break-down of resistance of Lely. The question is whether the life-time of Lely would have been prolonged if those French cultivars had not been grown within the epidemiological zone of yellow rust in northwestern Europe. Virulence to Alba has also been observed outside Europe and its presence there is probably due to cultivars possessing the resistance gene(s) of Alba. Cultivars with the same specific resistance as Alba (such as Mado and Falco) have one common ancestor, Juliana (33), which is a progeny of a cross with Wilhelmina, also a common ancestor of many cultivars now or formerly grown outside Europe. Investigating the history of resistance is a matter of time as well as of money, but worthwhile to do. In this connection, Gäumann (10) may be quoted: “the forms persisting to the present day might well be regarded as living fossils.” Yellow rust is one of these fossils, not only revealing its own history but also that of the resistance of its hosts.

Geographical Distribution of Yellow Rust Races

In Europe before World War II, studies of the geographical distribution of races of *P. striiformis* were mostly done by Gassner and Straib in Braunschweig, West Germany. Starting in 1955, these studies were resumed by Fuchs (7) in cooperation with Zadoks (41) who conducted the Yellow Rust Trials Project. These trials, now named "trap nurseries," were sown in almost all European countries and in a few places outside Europe. A rust survey on a worldwide basis was initiated in 1968 when a resolution was taken at the First International Congress in London to conduct a worldwide survey of virulence in plant pathogens. This survey is now being made through CIMMYT's International Disease Trap Nurseries. In 1932 Gassner and Straib (9) stated that the distributions of races are related to the local host cultivars and that changes in cultivars will be followed by changes in race composition. Their statement remains valid to this day. The following description of race distribution therefore presents a momentary snapshot of a changing, or co-evolutionary process of two biological entities.

The world distributions of yellow rust virulences have been reviewed elsewhere (35) and will now be treated by areas.

Europe (Figure 1)

Based on data collected in 1932-1955, Oort (21) distinguished three zones in Europe, one of which extends from England to Turkey, a distance of about 2500 km. The latter zone has been omitted in
Figure 1 as many races in central and eastern Europe have originated in northwestern Europe. An example of this is race 104E137 which was observed in England in 1969 for the first time (5) and became dominant in northwestern Europe in the following 4 years. It gradually disappeared from the west but moved to the east and became prevalent in eastern Europe in the late 1970s. The same race travelled to Australia in 1979 (20).

Northwestern Europe is considered as a source of new races and virulences because it is the scene of intensive breeding for resistance. Almost all resistance genes, either singly or variously combined in cultivars have been overcome by the pathogen. The evolution of *P. striiformis* f.sp. *tritici* in northwestern Europe has been described by Stubbs (35).

The races of south-western Europe (Spain and Portugal) have been designated by Zadoks (41) as the Iberian population, based on their performance in the trap nurseries. A characteristic was the absence of Yr7 (Hope/Timstein)-virulence. This virulence is now present as a result of the immigration of race 6E16 from northwestern Africa. It is prevalent in zone 2 and differs from other 6E16 races by being avirulent to YrA.

![Figure 1. Distribution of races of yellow rust in Europe.](image_url)
(Anza), but virulent to Sonalika also possessing YrA. Races in zones 1 and 5 are avirulent to both Anza and Sonalika. Data on the races of zone 2 have been used in the analysis of the 1978 epidemic of yellow rust in Spain (17).

The rusts of zone 3 have been designated by Zadoks (41) as the Grecian population having a similar infection spectrum to that of the Iberian population. Yr7 virulence is now also present but the data on races now prevalent in Greece are insufficient to link zone 3 to zone 4.

Rust in zone 1 has been designated as the Levantine population, differing from the previous two populations by virulence on Hope/Timstein (Yr7) and Selkirk (41). The dominating race of zone 4 is 6E16 with virulence or avirulence to Anza, Sonalika, Giza 155 (previously resistant in Egypt), or Miriam (previously resistant in Israel). Yr10 (Moro)-virulence, being absent in zones 1, 2, 3, and 5, is represented by race 82E16. Yr10-resistance is indigenous in this zone (35) and was used in breeding for resistance in the USA but was soon overcome by the pathogen.

Zone 5, comprising Norway, Sweden, Finland, and the western part of the Soviet Union, has not been described previously because the first samples were only received in 1979. The races identified, 4E0 among others, are similar to those described by Shchekotkova (26) and by Tsikaridze et al. (38) in the Soviet Union. A characteristic of these races is the virulence for Yr6 which is common in spring wheats such as those grown in Finland (Stubbs, unpublished). Zone 5 overlaps zone 7 (shown in Figure 3), as both zones have the same races, 6E16 among others.

East Africa (Figure 2)

The data of Zadoks (41) showed few differences in pathogenicity between the Levantine and the Kenyan populations. A common characteristic was their compatibility with Selkirk, being resistant in Europe. The data on races identified in Wageningen and Braunschweig also revealed few striking differences. The two populations, however, do differ in virulence for Yr9, something which has not yet been observed in zone 4. Race 134E150, having Yr9-virulence (Table 1), is widespread in Kenya and is also present in Ethiopia. It may be expected that race 134E150 will migrate to zone 4. For Kenya, the evolution of virulence in relation to host resistance has been studied by Honthuis (3) in Wageningen. The relationship of Yr9, 7, 6 and/or 2-virulence in races with resistance genes in wheat cultivars selected in Kenya has been shown by D.L. Danial (pers. comm.). Yr10-virulence represented by race 82E16 is present in zones 4 and 6. According to D.L. Danial (pers. comm.) the cultivar Kenya Popo possesses Yr10, but the origin of this gene may be different from the one in the USA cultivar. Moro. The presence of race 6E150 only in Rwanda and of race 140E12 in Zambia (Table 1), both unknown elsewhere and both infecting the triticale Delphin, indicate a separate divergent evolution of the rust in zone 6.

Asia (Figure 3)

Zones 4, 5, 7, and 8 overlap because they have a few races, (6E16, 38E16, and 70E16, among others) in common. The west (zone 4) to east (zone 8) movement of virulences, and its causes have been described by Saari and Prescott (22) and Nagarajan (16). At present, zone 8 is characterized by the widespread presence of race 7E150 in Pakistan.
Figure 2. Distribution of races of yellow rust in Africa.
India (S. Nagarajan, pers. comm.), and Nepal. According to IPO's race data (unpublished), race 7E150 appeared in Afghanistan in 1981 and then moved eastwards. This race, which is new to zone 8, infects the commercially grown cv. Sonalika, which appears to be more susceptible at high elevations than at low elevations, as observed by H.J. Dubin and R.W. Stubbs in Nepal in 1986. Race 7E150 seems to occur more often than other races which are also virulent on Sonalika. The first race may be more aggressive or virulent, or may have a wider temperature adaptation than the others.

Cultivars Sonalika, Inia 66, and Anza = WW15 = Karumu have gene YrA (39), but the last-named cultivar may possess an additional resistance gene, as suggested by tests with cultures from North Africa. In Iran, the resistance of Inia 66 was overcome by race B20A2 (2), which is not identical to race 7E150.

Zone 9 is an isolated spot in the Indian Subcontinent with a separate evolution of the pathogen.

Data recently collected by Yang Hua-an (Institute for Plant Protection, Beijing, China) in the II-O indicate that yellow rust in China (zone 10) has evolved in some isolation from the rest of Asia. The difference is

Figure 3. Distribution of races of yellow rust in Asia.
mainly due to the fact that indigenous as well as foreign resistance genes have been utilized in breeding. The indigenous genes are ineffective in China but effective in other parts of the world as shown in tests with Chinese and non-Chinese yellow rust races (Yang Huan-an, pers. comm.).

The Americas (Figure 4)
According to Humphrey et al. (11), yellow rust entered the American continents by way of the Aleutians and Alaska. The western mountain ranges of the two continents provided the route to southern Chile. The Andean-Patagonian valleys are thought to have been the paths to Argentina (39). In South America, the route described was followed by P. striiformis f.sp. hordei race 24 when it was introduced to Colombia in 1975 (6). Most of the barley grown at that time were susceptible to race 24, which was ideal for registering the routes and the speed of dispersal of the pathogen.

Rajaram and Campos (22) suggested six epidemiological zones for wheat rusts in the Western Hemisphere. Yellow rust is important in two of these zones, namely, the Pacific Northwest in the U.S. and the Andean countries (Figure 4). The pathogenicity of the fungus in the first zone is being analyzed by the Regional Disease Laboratory in Pullman, Washington, and that in the second zone principally by the IPO in Wageningen. Regrettably, uniformity of procedures with regard to race nomenclature and the use of differential wheat cultivars (34) has not been achieved. A few races from the U.S. have also been identified by the IPO but the data are too limited to indicate whether zone 1 overlaps zone 2 (Figure 4). However, it can be said that virulence on the resistance genes Yr2, 3, 6, 7 and on the differential cv. Suwon 92/Omar occurs in both zones. So far, virulence on Yr9 has been reported in zone 1 but it is interesting to note that the U.S. race CDL-21 was collected from rye and triticales as well as from wheat (14). The U.S. differential Riebesel 47-51 possessing Yr9 is resistant to this race but, like Clement, it does not always indicate the presence of virulence on Yr9 in the races (Table 1). In Mexico this virulence is represented by race 138E10.

In the review given on the distribution of virulences in South America, the epidemiological zone for yellow rust has been divided into two sub-zones (35) (as shown in Figure 4). Zone 1 includes southern Mexico and Guatemala (32), which overlaps zone 2 because the same races occur (0E0 and 8E0 among others). These races may have travelled from one zone to the other, but they may also have developed separately in the two zones. Zones 2 and 3 are distinguishable as shown in Table 3. The zones all overlap in Peru, where races of zone 1 as well as those of zone 2 have been observed. This fact suggests a north to south as well as a south to north movement ofurediospores.

According to Tollenaar (37), southerly winds predominate in central Chile but are not very effective in carrying urediospores from south to north. However, this view is somewhat contrary to the race data from Chile and Peru.

Differences in races between zones 2 and 3 may be related to differences in host resistance genes. In Chile, western European wheat cultivars are, or have been, grown, for example, Vilmorin 27 (Yr3). Intermedio = Orca (Yr3c + 2 + 6), Manella (Yr2 + Ab) and Cappelle Desprez (Yr3a + 4a). The latter, possessing a durable resistance against yellow rust (12), was severely
Figure 4. Distribution of races of yellow rust in North and South America.
attacked by race 104E9. Evidently, the durable resistance of Cappelle Desprez is environmentally bounded, as it was adequate in England, but inadequate in Sweden (12). Race 108E141 was found on Intermedio. The appearance of race 110E143, a progeny of race 108E141, can be related to the introduction of cultivars with Yr7, as Victoria Pavon 76 (Yr6 + 7) (40). Race 236E141 was found in Clement (Yr9 + 2?), although this cultivar was not grown on a commercial scale.

Race 134E0, a progeny of race 6E0, appeared in Ecuador and Colombia in 1985, and infected cultivars with Yr9 (P. Fox, H.J. Dubin, pers. comm.). As in Chile and in other countries, virulence for Yr9 was readily present in the pathogen population to infect cultivars with this resistance gene.

The presence of race 104E9 in two continents and that of race 108E141 in three continents are a typical example of a parallel evolution of races.

**General Remarks and Recommendations**

The data presented in this paper are but a fraction of those collected by the IPO in the past 15 years. They present a situation which was described in 1979 as follows: "the distribution of the factors of virulence of yellow rust in the world is directly related to the factors of resistance of the cultivated host varieties, either indigenous or foreign, and that the evolution of yellow rust develops in the same stages and places as the man-guided evolution of resistance" (29). This statement is a variant of the one

### Table 3. Distribution of races of *P. striiformis f.sp. tritici* in South America, Europe, and Australia

<table>
<thead>
<tr>
<th>Race (virulence formula)</th>
<th>Colombia</th>
<th>Ecuador</th>
<th>Peru</th>
<th>Chile</th>
<th>Argentina</th>
<th>Uruguay</th>
<th>Brazil</th>
<th>Europe</th>
<th>Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td>2E0 (7)</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>64E0 (Su)</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40E0 (3)</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6E0 (6,7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>134E0 (6,7,9)</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>104E9 (Su,3,4)</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>108E141 (Su,2,3,4,6)</td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>109E141 (Su,1,2,3,4,6)</td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>110E141 (Su,2,3,4,6,7)</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>236E141 (Su,2,3,4,6,9)</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
</tbody>
</table>
presented in 1932. We may expect that other variants will be stated in the future but may hope that it will be possible to add that the evolution of the rust had been better guided than in the past.

In breeding for resistance, whether nationally or internationally, knowledge of the stages of evolution of the pathogen is essential and should be continuously updated. The stage of evolution of the rust differs from place to place and the recognition of these differences is a basis for an effective evaluation of host resistance under different environments. International cooperation is clearly essential.

It is relevant to the objectives of the workshop to enquire whether, in the many wheat collections screened in the IPO, cultivars have been found with resistance that is effective against the rust. There are a few, but it is uncertain whether their resistance is determined by a single unidentified gene or by combinations of known genes. In the former case, the cultivars could be classified as new sources of resistance and used as such in breeding.

References


Chapter 4

Using Polygenic Resistance to Breed for Stem Rust Resistance in Wheat

D.R. Knott, Department of Crop Science and Plant Ecology, University of Saskatchewan, Saskatoon, Canada

Abstract

Multigenic resistance to stem rusts has been known for many years. Non-specific resistance to disease has been hypothesized but it is difficult to prove. Partial resistance and slow rusting are often controlled by several genes having small effects and are sometimes thought to be non-specific. In studies at Saskatoon, lines of wheat were developed that lacked seedling resistance to race 15B-1 but had good field resistance to the same race. Their resistance proved to be controlled by three to five recessive genes, each having a small effect. The genes reduced the latent period and pustule number and size. Resistance that is controlled by several genes having small effects is likely to be relatively durable, regardless of whether it is specific or non-specific. Polygenic resistance is difficult to use in wheat breeding programs but its use could be very worthwhile.

Introduction

A polygenic character is one that is controlled by a number of genes each having a small effect. Just how many genes should be involved to make a character polygenic is not clear. For the purpose of this paper, I will assume that if a character is controlled by several genes and it is difficult or impossible to identify the effects of individual genes, then the character is polygenic.

The occurrence of polygenic resistance to the rusts has been known for many years. In 1946, Ausmets et al. (1) cited a number of reports of multigenic resistance to leaf rust (P. recondita f.sp. tritici) and stem rust (P. graminis f.sp. tritici). However, in 1971, in an extensive survey of genetic studies on host-parasite interactions, Person and Sidhu (10) found that 875 papers reported that resistance to various pathogens was due to major genes and only 60 reported resistance that was due to minor genes or polygenes. Much of the early work was concentrated on genes that had major effects and usually proved to be race-specific. The situation has changed. As a result of the stimulus provided by Vanderplank (11) in the last 20 years, there have been many studies on types of resistance that have proved to be complex in inheritance—partial resistance, slow disease development, etc.

Non-specific Resistance

Vanderplank (11) first hypothesized that there are two distinct types of disease resistance, vertical and horizontal, now more commonly called specific and non-specific. He presented evidence for the existence of the two types, particularly for the potato late blight system (Solanum tuberosum-Phytophthora infestans). Basically, specific resistance is effective against only certain genotypes of the pathogen while non-specific resistance is effective against all genotypes.

Since then, the concept has undergone various modifications. First, in 1968 Vanderplank (12) concluded that, if data from the
interaction of a set of host and pathogen genotypes were analyzed by an analysis of variance, the presence of a significant mean square for the interaction between host and pathogen genotypes indicated the operation of specific resistance. The presence of significant main effects due to differences among host genotypes and among pathogen genotypes supposedly indicated the presence of non-specific resistance. However, it can easily be demonstrated that resistance that results solely from the action of genes for specific resistance can generate significant main effects (Table 1). The data in Table 1 are based on field tests of near-isogenic lines of Marquis with four races of stem rust. Typically, a gene gives the same rust severity with each race to which it is resistant. Although the resistance is entirely due to the action of genes for specific resistance, there are sizeable mean squares for the two main effects which result from differences among lines and among races.

Table 1. Theoretical results from testing Marquis and three near-isogenic lines of Marquis with four races of stem rust (rust severity in percent based on actual field tests but adjusted so that Marquis is rated as 100% in each case)

<table>
<thead>
<tr>
<th>Cultivar or line</th>
<th>Race 56</th>
<th>Race 15B-1</th>
<th>Race 29-1</th>
<th>Race 11-1</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marquis</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100.0</td>
</tr>
<tr>
<td>Marquis-Sr6</td>
<td>10</td>
<td>10</td>
<td>100</td>
<td>10</td>
<td>32.5</td>
</tr>
<tr>
<td>Marquis-Sr7</td>
<td>100</td>
<td>40</td>
<td>100</td>
<td>40</td>
<td>70.0</td>
</tr>
<tr>
<td>Marquis-Sr9a</td>
<td>30</td>
<td>100</td>
<td>30</td>
<td>100</td>
<td>65.0</td>
</tr>
<tr>
<td>Mean</td>
<td>60.0</td>
<td>62.5</td>
<td>82.5</td>
<td>62.5</td>
<td>66.9</td>
</tr>
</tbody>
</table>

Analysis of variance (mean square [DF]): lines 3056 (3), Races 440 (3), Interaction 1473 (9)
Table 2. A model for non-specific resistance in which the five hosts carry from 0 to 4 genes for resistance, each reducing disease severity by 25%, and the five pathogens carry 0 to 4 genes for aggressiveness, each increasing disease severity by 25% (A), and the genes each affect disease severity by only 10% (B).

### A.

<table>
<thead>
<tr>
<th>Pathogens and genotypes&lt;sup&gt;a/&lt;/sup&gt;</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Host and genotype* | Effect  | 0  | +25 | +50 | +75 | +100 | Mean |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0</td>
<td>50</td>
<td>75</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>85</td>
</tr>
<tr>
<td>B</td>
<td>-25</td>
<td>25</td>
<td>50</td>
<td>75</td>
<td>100</td>
<td>100</td>
<td>70</td>
</tr>
<tr>
<td>C</td>
<td>-50</td>
<td>0</td>
<td>25</td>
<td>50</td>
<td>75</td>
<td>100</td>
<td>50</td>
</tr>
<tr>
<td>D</td>
<td>-75</td>
<td>0</td>
<td>0</td>
<td>25</td>
<td>50</td>
<td>75</td>
<td>30</td>
</tr>
<tr>
<td>E</td>
<td>-100</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>25</td>
<td>50</td>
<td>15</td>
</tr>
<tr>
<td>Mean</td>
<td>15</td>
<td>30</td>
<td>50</td>
<td>70</td>
<td>85</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

Analysis of variance (mean squares [DF]): Pathogens 4063 (4); Hosts 4063 (4); Interaction 156(16).

### B.

<table>
<thead>
<tr>
<th>Host and genotype*</th>
<th>Effect</th>
<th>0</th>
<th>+10</th>
<th>+20</th>
<th>+30</th>
<th>+40</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0</td>
<td>50</td>
<td>60</td>
<td>70</td>
<td>80</td>
<td>90</td>
<td>70</td>
</tr>
<tr>
<td>B</td>
<td>-10</td>
<td>40</td>
<td>50</td>
<td>60</td>
<td>70</td>
<td>80</td>
<td>60</td>
</tr>
<tr>
<td>C</td>
<td>-20</td>
<td>30</td>
<td>40</td>
<td>50</td>
<td>60</td>
<td>70</td>
<td>50</td>
</tr>
<tr>
<td>D</td>
<td>-30</td>
<td>20</td>
<td>30</td>
<td>40</td>
<td>50</td>
<td>60</td>
<td>40</td>
</tr>
<tr>
<td>E</td>
<td>-40</td>
<td>10</td>
<td>20</td>
<td>30</td>
<td>40</td>
<td>50</td>
<td>30</td>
</tr>
<tr>
<td>Mean</td>
<td>30</td>
<td>40</td>
<td>50</td>
<td>60</td>
<td>70</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

Analysis of variance (mean squares [DF]): Pathogens 1250 (4); Hosts 1250 (4); Interaction 0(16).

<sup>a/</sup> A + indicates the presence of a gene for either aggressiveness or resistance, respectively, in homozygous condition.
range of severity, there is an interaction mean square. However, if the effect of each gene is reduced to 10% so that the range is only from 10 to 90% severity, then the interaction mean square is zero (Table 2B). A host genotype with no genes for resistance shows a considerable range in disease severity, as do all host genotypes. Resistance is no longer horizontal in Vanderplank's original sense. Similarly, a pathogen genotype with no genes for aggressiveness shows a range in disease severity, as do all pathogen genotypes. Interestingly, there seems to be no theoretical reason why a pathogen cannot develop increasing levels of aggressiveness to the point where all host genotypes are fully susceptible. On the other hand, it might be possible for the host to develop resistance of a type that the pathogen could not overcome.

A major problem with the concept of non-specific resistance is the difficulty in proving its occurrence. A resistance may appear to be non-specific or effective against all races until a race of the pathogen is discovered to which it is susceptible. In other words, resistance is non-specific until it is found to be specific.

**Slow Rusting and Partial Resistance**

In 1968 Caldwell (2) drew the attention of wheat breeders to general resistance to rusts, emphasizing its durability. Later he was particularly interested in slow rusting and pattern rusting in leaf rust in which the rust was heavy only on certain areas of the leaf, e.g., the leaf tip.

In recent years, many rust workers have emphasized slow rusting or partial resistance. These types of resistance involve host-pathogen combinations in which the rust develops slowly and never reaches a high degree of severity. As a result, damage is slight. The degree of resistance is often measured as the area under the disease progress curve (AUDPC) when the disease severity has been measured several times during the development of the epidemic. There is now ample evidence that minor genes for resistance can affect rust development at various stages, for example: receptivity, length of the latent period, pustule size, and spore production. Each gene has a relatively small effect but when several of them are combined, satisfactory resistance can result.

Parlevliet (7, 8) has done a thorough analysis of partial resistance to leaf rust (*Puccinia hordei*) in barley, particularly of the inheritance of the length of the latent period. He found that up to five genes were involved. Their total effect was to increase the latent period from 8 to 16 days and the cumulative effect on a leaf rust epidemic in the field was sufficient to reduce considerably the final rust severity. Parlevliet (8) also found that the effects of at least one of the genes was specific.

**Studies on Adult Plant Resistance at Saskatoon**

Some years ago, I made four crosses each involving four parents that had been selected because it was thought that they had resistance to stem rust that was not due to major genes for specific resistance. The F2 progeny of the crosses were selected for seedling susceptibility to race 15B-1. This should have eliminated any major genes for specific resistance to race 15B-1. The progeny of the susceptible F2 plants were then selected for several generations for adult plant resistance to the same race in the field. Lines with good
resistance to race 15B.1 were easily obtained (4). Although only the one race had been used in the selection, the lines proved to be resistant to multi-race mixtures and resistant also in tests throughout North America. Of 20 lines tested in the 1976 International Spring Wheat Rust Nursery, 17 were susceptible at at least one location while three had at least some resistance at all locations. Thus, it appeared that some degree of specificity was involved.

When the resistant lines were crossed to a susceptible, the F1 progeny were in most cases similar to the susceptible check in percentage rust readings in field tests. The F2 populations gave distributions that were fairly normal when the epidemic was moderate. However, if the epidemic was heavy, the distributions were skewed, with the plants concentrated at the susceptible end of the curve. In either case, few were as resistant as the resistant parents. The results suggested that the resistance was due to several recessive genes having small, cumulative, or perhaps multiplicative, effects.

Woodend (15) tested five of the lines with race 15B.1 in the field. Compared to a susceptible check, they showed (with one exception) reduced rust severity, pustule size, and areas under the disease progress curve and reduced apparent infection rates per day. In tests under greenhouse, growth chamber, and field conditions, the lines showed longer latent periods and reduced pustule sizes compared with the susceptible check. Resistance increased as plants got older.

Recently, Padidam (6) completed a genetic study of seven of the lines. Each line was crossed to a susceptible parent and a single seed descent procedure was used to produce a random set of F5 plants from each cross. The seed from these plants was increased and F5-derived F7 lines were tested with race 15B.1 in field nurseries from 1982 to 1984. The results varied somewhat depending on the severity of the rust epidemic from year to year. However, the readings on the F7 lines showed high correlations from one year to the next. The results for resistant line 91 will be used as an example (Figure 1). In 1982, a

![Figure 1. Results of field tests in 1982 and 1984 with race 15B.1 on 135 F5-derived F7 lines from the cross, line 91 x a susceptible line.](image-url)
susceptible check showed 81% rust severity; line 91 showed 13% and only 7 of 135 F7 lines were considered to be similar to it. In 1984 a susceptible check showed 66% rust severity, line 91 showed 11%, and 20 of the 135 lines were similar to line 91. In 1982, for six of the seven resistant lines, estimates of the number of genes involved in resistance ranged from three to five. The seventh line proved to be segregating for Sr6 as well as polygenes. The F5-derived F7 lines were tested for seedling resistance to other races to see if such resistance might be related to the adult plant resistance in the field. No association was found.

The results confirm that resistance is due to several genes, but not a large number. The frequent negative skewness of the distributions suggests that individual genes tend to have little effect by themselves but that their effects are multiplicative. There is little doubt that these genes are similar to those that have been reported by other workers to control resistance described as partial resistance or slow rusting. They can provide adequate field resistance. In fact, the lines tended to give better resistance against multi-race mixtures than against 15B-I alone. Undoubtedly, this was because they carried additional genes for specific resistance to some races in the mixture.

**Durability of Polygenic Resistance**

A key question is whether resistance of this type will be durable and, if so, why. Resistance can be durable for only two reasons. First, the pathogen cannot develop a highly virulent or aggressive race, for whatever reason, does not come into contact with the resistant host.

In North America, under normal circumstances, there is little reason to think that, if a virulent race develops for a particular type of resistance that is in common use, it will not eventually become established. Nevertheless, it is possible that a virulent race may develop where a particular resistance is being used but never become established in the overwintering area.

In general, however, it must be assumed that, if a virulent race develops, it will become established. If this is so, then durability must depend on the inability of the pathogen to develop virulence. It is well-known that genes for specific rust resistance are rapidly overcome by the pathogen (although there can be exceptions). Gene Sr26 derived from *Agropyron elongatum* has been used in Australian wheats since 1970. It is present in at least seven cultivars and has remained effective. Luig (5) reported that all attempts to find a susceptible infection in the field or to produce one by mutation had failed but he later found virulence in a laboratory culture from the USA. Vanderplank (14) states that, "resistance genes that the pathogen cannot match are more likely to be found in foreign species."

I doubt that resistance genes in the relatives of wheat will somehow be physiologically different from those in wheat itself. Resistances derived from relatives of wheat have frequently been overcome.

An important question is whether resistance controlled by polygenes is somehow different physiologically from resistance controlled by major
genes for specific resistance. For the barley-barley leaf rust system, Clifford et al. (3) argue that there are separate mechanisms governing the two types of resistance. I agree that they are genetically separate, but it is not clear that they are physiologically different. Many genes controlling specific resistance have intermediate effects and produce results similar to slow rusting. Is the mechanism different from that governed by polygenes which results in slow rusting? Even if the mechanisms are different, does this mean that one type can be overcome by the pathogen and the other cannot? Parlevliet (9) argues that genes for partial resistance do operate on a gene-for-gene basis. I would be surprised if they did not, but the question is still open.

The final question is whether selection pressures are different on resistance controlled by major genes for specific resistance compared to resistance controlled by polygenes. Consider, for example, two cases: a cultivar with five genes for specific resistance compared to one with five polygenes for resistance. In the first case, a pathogen genotype with no genes for virulence would require five mutations to overcome the resistance. The probability of the simultaneous occurrence of five separate mutations is essentially zero. Unfortunately, what usually happens is that the five genes are released singly in cultivars over a period of time. The presence of any one single resistance gene exerts strong selection pressure on the pathogen to overcome it. A virulent mutant can attack the cultivar, the avirulent genotype cannot do so or, at least, can only do so much less effectively. Thus, as the genes are released singly, the pathogen overcomes them by stepwise mutation.

In the second case, a cultivar with five polygenes, the situation is different. If the genes are specific, a polygenic mutant that overcomes one or them would undoubtedly increase. As additional mutations occur, they would also increase slowly in frequency. Eventually, the resistance would be overcome but the process could be much slower than for specific resistance. If, on the other hand, the resistance were non-specific, there is no reason to think that it could not eventually be overcome by mutations for aggressiveness in the pathogen. It really goes back to the original question, "Are some types of resistance actually uniform or horizontal against all races of a pathogen?" Certainly there is good evidence in a few cases of resistance that have been effective over long periods.

For both stem and yellow rust (F. strumarum) of wheat, there is increasing evidence that polygenic resistance is recessive and it may take several genes to produce appreciable resistance. The gene effects appear to be multiplicative rather than additive, as follows:

First gene—3% reduction; 97% severity
Second gene—6% reduction; 91% severity
Third gene—12% reduction; 87% severity
Fourth gene—24% reduction; 55% severity
Fifth gene—48% reduction; 7% severity

In this model, only combinations of four or five genes could have easily detectable effects. If a host carried all five genes, then a pathogen mutant that overcame one of them would have a sizable effect, increasing rust severity from 7% to 55%. It would
have a considerable selective advantage. Additional mutants would have less selective advantage, but still might appear over time. Some degree of resistance could remain for a long time.

The major difference between the two situations that I have discussed is that major genes for specific resistance act independently of each other but that polygenes act additively or multiplicatively. With genes for specific resistance, a virulent mutant in the rust attacks only those cultivars that carry the matching gene for resistance and no other unmatched gene. Cultivars are either resistant or susceptible to the mutant. With polygenes, a mutant in the rust overcomes only part of the resistance of a cultivar regardless of whether the resistance is specific or not. Polygenic resistance is much more likely to be durable.

Using Polygenic Resistance in Breeding

The durability of polygenic resistance makes it of considerable interest in wheat breeding, particularly in areas in which specific resistance is usually rapidly overcome by the pathogen. As yet, however, polygenic resistance has been studied more than it has been used. There is no doubt that polygenic resistance is difficult to use in a routine breeding program. First, it is impossible to select for if genes for specific resistance to the races being used are present. Second, the frequency of resistant plants in crosses is low and selection must be carried out over several generations and finally on a family basis. If breeders want to use polygenic resistance, they must be prepared to put considerable effort into it.

The masking effects of genes for specific resistance can be overcome in two ways:

1) A race can be used that is virulent on all of the genes for specific resistance present in the parents being used in the program (assuming that such a race is available). I thought of transferring resistance from some of my lines to our most important cultivars such as Manitou and Neepawa. Unfortunately no race virulent on either Manitou or Neepawa was available.

2) The alternate is to test seedlings of a cross with a highly virulent race and eliminate all resistant plants. The race can then be used to select for field resistance in the progeny of the seedling susceptible plants.

Because of the expected low frequency of resistant plants in crosses involving polygenic resistance, the breeder will have to concentrate on a few, well planned crosses. If the source of polygenic resistance is poorly adapted to the breeder's area, then probably at least one backcross will have to be made. Since some evidence suggests that three to five genes may be involved, it should be possible to make the cross and the backcross, and then start selecting for resistance. Fairly sizable populations will be needed and several generations of selection will be required. Since moderate levels of resistance may be overwhelmed by heavy spore loads, particularly in single plants, a pedigree system involving the testing of families will probably be most successful.

Breeders have probably shied away from using polygenic resistance because it would require considerable effort and a substantial change in breeding procedures. However, the effort could well be very worthwhile.
References


Chapter 5

Strategies for the Utilization of Partial Resistance for the Control of Cereal Rusts

J.E. Farleveleit, Department of Plant Breeding, Agricultural University, Wageningen, The Netherlands

Abstract

In cereals all resistance to cereal rusts is of the species-specific type, i.e., the resistance is effective to one rust species only. Against each rust pathogen two types of species-specific resistance can be recognized: i) A major gene, hypersensitive type of resistance, characterized by low infection types, race-specificity and lack of durability; ii) a quantitative type of resistance (partial resistance), characterized by a reduced rate of epidemic build-up despite a high, susceptible infection type, by absence of large race-specific effects (although small ones do occur) and by durability. In the absence of major genes, selection for partial resistance is easy. Even a mild selection against susceptibility, if applied consistently, is highly effective in accumulating genes for partial resistance. This mild selection enables the breeder to select for other characteristics at the same time. If one wishes to increase partial resistance in the presence of major genes that have not been fully neutralized by the pathogen, the efficiency of selection is considerably less. If possible, the breeder should expose the host population to a single race of the pathotype, a race that neutralizes a maximum number of major genes. In this host population, the breeder should remove the most susceptible genotypes at each stage of selection and also those genotypes that show a low infection type. If it is too difficult to score infection types reliably, the breeder should remove the most resistant genotypes together with the most susceptible ones as the former are assumed to carry major genes. In some situations, the pathogen population to which the host is exposed cannot be controlled and exists as a mixture of races. Selection for partial resistance is very difficult in this case. Continuous removal of the most susceptible lines together with those lines that are nearly unaffected will tend to favor partial resistance, but the progress may be slower than hoped for.

Introduction

Wheat, barley, oats, and rye are hosts for several different rust species. These rusts collectively represent the biggest disease threat for these crops worldwide.

The most important rusts are the stem rusts of wheat, oats, rye, and barley (Puccinia graminis f.sp. tritici, avenae, and secalis and P. graminis), leaf (brown) rust of wheat (P. triticina = P. recondita f.sp. tritici), rye (P. recondita = P. recondita f.sp. secalis), and barley (P. hordei).

Resistance to these rusts has been widely used and is often a major gene type. Characteristic is the large number of major genes that have been found—over 40 Sr-genes (wheat stem rust), over 30 Lr-genes (wheat leaf rust), over 40 Pm-genes (oats crown rust)—and the numerous races of each pathogen. With few exceptions these major, race-specific
resistance genes are not durable: when exposed over large areas for long periods, races develop that neutralize the effect of the resistance genes. In order to obtain longer lasting resistance, two approaches are possible. Either another type of resistance is looked for and used or the non-durable resistance genes are used in ways that reduce the threats of new races. This chapter concentrates upon the former, i.e., on partial resistance.

**Terminology**

The terminology of host-pathogen systems is far from consistent. In order to be clear, the most important terms and concepts used in this paper are discussed and defined.

The cereals are hosts to several rust pathogens. These rusts are able to invade the host plant. The tissues invaded can be indicated as the tissues *affected*. The affected areas can be recognized as sporulating and discolored areas. High growth and reproduction rates of the pathogen, measured as rapid expansion of the host tissue area affected, are indicative of a high aggressiveness of the pathogen, or of a high susceptibility and thus low resistance of the host or of both. With an increase in resistance or a decrease in aggressiveness, the rate of tissue area expansion diminishes.

With immunity, complete resistance or with non-aggressiveness, the rate of fungal growth and/or reproduction is zero. *Incomplete resistance* (with rusts often designated *slow rusting*) or reduced aggressiveness allows some growth and reproduction of the pathogen. *Partial resistance* is a form of incomplete resistance characterized by a susceptible or high infection type. Despite this susceptible infection type, the tissue area affected remains less than that of a very susceptible genotype: the epidemic is retarded. The *infection type* describes a resistance reaction to the rust pathogen on a 0-9 scale, where 0 = no sporulation, tiny necrotic flecks and 9 = large urediosori, abundant sporulation, and no host tissue reaction around the sori. Low infection types indicate the presence of one or more genes for hypersensitivity. Nearly all major gene resistances belong to this category of hypersensitive or low infection type resistance.

Resistances can be effective against a broad range of parasites, for example, tannins in various crops or glucosinolates in Cruciferae. This is *broad resistance*. If the resistance is effective to one parasite species only, it is *species-specific resistance* (17). All known forms of resistance in cereals to the rusts have to be classified thus. Parasites in turn may have a parasitic ability directed to a wide range of host species, *generalists*, or to a narrow range of host species, *specialists*. The cereal rusts are specialists.

Resistance is often sub-divided into race-specific and race-non-specific resistance. *Race-specific resistance* is resistance that is effective only to certain races of the pathogen. There are host cultivar-pathogen genotype interactions and the ranking order of the host cultivars for resistance depends on the race of the pathogen used. Races of the pathogen at the same time vary in their *cultivar-specific aggressiveness* (= *virulence*). *Race-non-specific resistance* operates against all genotypes of the pathogen.
The resistance level may vary depending on the cultivar-non-specific aggressiveness* of the pathogen, but there are no interactions between host and pathogen genotypes and the ranking order of the cultivars for resistance is independent of the pathogen genotype used. Race-nonspecific resistance is almost invariably used within the context of resistances that are of the species-specific types as in the case of the cereals/cereal rusts. Whether it is rightly used in this group of host pathogen systems is discussed later (see section on Specificity below).

**Partial Resistance**

As defined above partial resistance is characterized by a reduced rate of epidemic development despite a high or susceptible infection type. It is therefore not identical with slow rusting, as all incomplete resistance to rusts results in slow rusting including resistances with intermediate infection types.

The reduced rate of epidemic development is a result of the combined effects of reduced infection frequencies, longer latent periods, and reduced rates of spore production per urediosorus (16, 20).

---

* In the literature, virulence, aggressiveness, and pathogenicity are used inconsistently. If virulence is the cultivar-specific and aggressiveness the cultivar-non-specific counterpart of race-specific and race-non-specific resistance, one needs a counterpart of resistance. Pathogenicity is not suitable as it means more than aggressiveness. Therefore the terms used here are proposed to replace the present usage of virulence and aggressiveness. Virulence could be used to indicate symptom-inducing ability of pathogens such as viruses, which incite true diseases.

**Measuring partial resistance**

Partial resistance must be evaluated in the field. Basically, the proportion of host tissue affected is measured, either once near the end of epidemic development or several times during the development of the epidemic. The former is assumed to represent the cumulative result of the components of partial resistance over time (26). The latter makes it possible to calculate the area under the disease progress curve (AUDPC) (36) or the apparent infection rate, r (34). This r-value approach, however, is clearly inferior to the other two methods mentioned (20, 29, 30).

Assessment of partial resistance is normally done on cultivars grown in small plots adjacent to one another, whereby cultivars differing in earliness and in susceptibility are often exposed to unusually high levels of inoculum. This situation, quite different from that of the former, may result in what Vanderplank (34) called "representational errors." These errors may result in under- or overestimating the partial resistance or even ranking the cultivars wrongly.

**Inoculum pressure**—The use of small plots exposed to spreader rows of a very susceptible cultivar tends to reduce apparent differences in partial resistance considerably. It is a form of interplot interference.

**Interplot interference**—In the case of windborne pathogens such as the cereal rusts, differences in partial resistance can be considerably reduced. Highly susceptible cultivars produce far more spores than the partially resistant ones in the trial and many of these are exported to adjacent plots. In general, the partially resistant cultivars receive more spores from the surrounding plots than they export. Partial
resistance is best-measured in the central parts of not-too-small plots that are sufficiently separated from others to reduce the import of inoculum to insignificant levels relative to their own spore production. The partial resistance measured in small adjacent plots can be greatly underestimated. For barley leaf rust, the underestimation varied from 5 to 16 times if the adjacent plots were 4.5 m wide, from 14 to 30 times in case of 1.5-m plots and from 75 to 130 times for single-row plots (27). Partial resistance in wheat to leaf rust in small adjacent plots appeared to be underestimated to a similar extent (Broers, pers. comm.). In barley, partial resistance is strongly underevaluated in small adjacent plots but the rank order remains constant (26). Measurements from adjacent plots can easily be translated into representative values if one includes cultivars that represent a wide range of partial resistance (27).

Earliness—If the genotypes to be compared vary greatly in maturity and time of heading, partial resistance may be difficult to evaluate. If observed on the same day, early genotypes tend to be underestimated and late genotypes overestimated. If one observes the amount of rust present at the same developmental stage, the partial resistance of the late cultivars is underestimated because of infection from the early genotypes. This problem can be solved by planting genotypes of similar earliness together in the same block and evaluating the blocks at different times. Also, one should not assess the flag leaf only, but at least the three upper leaves.

Time of evaluation—The best time to assess infection is when the most susceptible genotypes in the trial are not yet fully affected. Later assessments reduce the differences between genotypes while earlier evaluations tend to be more laborious or less accurate. If the epidemic has not developed very well, one should assess it when at least three leaf layers are still green.

Presence of major genes—If the genotypes to be assessed contain major genes and the rust population is a mixture of races to which these major genes are only partially effective, it may be very difficult to discern partial resistance (Table 1) (19). Where possible one should avoid using race-mixtures. One should use a single race, the one with the highest number of cultivar-specific aggressiveness factors.

Components of partial resistance
For detailed reviews the reader should see references 16 and 20. The basic components are infection frequency, latent period, and spore production per urediosorus. Of the last component, the spores produced early in the life of the pustule are especially important for the development of the epidemic. The importance of the components may vary with the rust species. Latent period is the most important component in barley and wheat leaf rusts, pathogens with little systemic activity. In yellow rust, partial systemic within the leaf, t infects, infection frequency and spore production may be the most important components. The components tend to vary in an associated way. Partially resistant cultivars tend to have reduced infection frequencies, longer latent periods, and reduced sporulation rates compared with more susceptible cultivars.

Generally it is assumed (though not proved) that these components are controlled by different genes. In
barley, however, the partial resistance to barley leaf rust seems to be largely controlled by minor genes with pleiotropic effects on infection frequency, latent period, and spore production (1, 21). As to wheat leaf rust, the components are less strongly associated with one another and with partial resistance (Jacobs, Broers, pers. comm.).

Genetics

The genetics of slow-rusting in cereals were reviewed by Wilcoxson (35). In many of the studies reviewed, slow rusting and partial resistance were taken to be identical. Often, progenies of crosses between highly susceptible cultivars and partially resistant ones were investigated. The segregation patterns were quantitative in nature and transgression was often observed. The number of genes assumed to be involved varied from a few—in maize/P. sorghii (7), in wheat/P. triticina (8) and in oats/P. coronata (10)—to several—in wheat/P. triticina (5) and P. graminis f.sp. tritici (33) and in barley/P. hordei (14, 15, 23).

In wheat, temperature-sensitive minor genes against P. striiformis have been reported. They act additively and together give a low infection type. They appear to have durable effects (31).

Specificity

Partial resistances to the cereal rusts, even when they are typically polygenically inherited are species-specific (17); i.e. the partial resistance genes are effective to only one Puccinia species. Partial resistance is not only species-specific because race-specific effects have also been reported for several pathogens, including barley leaf rust (20). These effects tend to be small, or at least insufficiently large to be of use in identifying races. It is questionable whether true race-non-specific resistance occurs within species-specific resistance.

Durability

Partial resistance is considered to be durable, but pertinent information is hard to obtain. The wheats Thatcher and Lee have been known to rust slowly for 55 and 30 years.

Table 1. Percentage of host tissue affected if cereal cultivars carrying different race-specific resistance (R) genes were exposed to a mixture of rust races, where the races vary in cultivar-specific aggressiveness (= virulence) genes (a-genes)

<table>
<thead>
<tr>
<th>R-genes</th>
<th>a2 (30)</th>
<th>a3a (40)</th>
<th>a4 (25)</th>
<th>a2a3a4 (5)</th>
<th>Percentage of host tissue affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>R1</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>70</td>
</tr>
<tr>
<td>R3</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td>50</td>
</tr>
<tr>
<td>R2R4</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>40</td>
</tr>
</tbody>
</table>

a/ Percentage of each race in the initial inoculum in parentheses.
b/ A + sign indicates that the host cultivar is susceptible for that race, a - sign that it is not.
respectively (35). Red Rustproof, an oat, has been reported to be slow rusting to crown rust for over 100 years (9), while the wheat Knox has been partially resistant to wheat leaf rust for over 20 years (12).

Most western European barley cultivars carry low to fair levels of partial resistance to barley leaf rust. There are no indications that this partial resistance, although exposed on large areas over many years, has diminished to any significant extent (6, 18, 20). The wheat cultivars grown in The Netherlands before 1930 all showed durable (mostly partial) resistance to yellow rust (4).

Data of this type are also available from a wide range of other host-pathogen systems. All these observations together suggest that partial resistance, provided it is not clearly monogenically inherited, is of a durable nature.

Selection for partial resistance
If the aim is to increase partial resistance, various approaches are possible depending on the situation. The most important restrictive factor is the presence of effective resistance of the major gene, low-infection type. Also important is the degree of association between the components of partial resistance. If this association is high and of a pleiotropic nature (as in barley to leaf rust) selection for one of the components of partial resistance can be as effective as field selection. If, however, the association between components is low then it is not advisable to use selection for a single component. On the other hand, one could try to improve the plant material for each of the components separately, which would increase the partial resistance strongly.

A third aspect is the expression of partial resistance in different environments. The reaction of barley to its leaf rust appears to be rather independent of environment. Resistance is expressed under a wide range of temperature conditions and the ranking order of the cultivars for partial resistance does not vary over years or test locations (13, 25, 26). This stable expression of partial resistance over a wide range of environments, however convenient for the breeder, is not necessarily characteristic of all cereal-rust pathosystems. In wheat leaf rust, for instance, the important component of partial resistance, latent period, appeared highly temperature-sensitive. The long latent periods of most partially resistant cultivars were best expressed at low temperatures and hardly at all at high temperatures (Broers, pers. comm.). However, when tested in totally different environments (The Netherlands, two soil types; southern Brazil; and Mexico), the partially resistant cultivars ranked in the same order and the partial resistance was well expressed in all four environments (Broers, pers. comm.).

Partial resistance in the absence of major genes
Absence of effective major genes in commercial cultivars is not common. Within the small-grain cereals, barley with respect to barley leaf rust is the only clear-cut example (25). Maize—Puccinia sorghi and peanut—P. arachidis are other examples.

How to proceed in such a case can be shown by reference to the barley-leaf rust system. All cultivars grown in western Europe appear to carry some partial resistance (25). Even some such as Akka, which are considered to be extremely
susceptible, carry a little resistance. Most cultivars, however, carry considerably more and it is this that prevents the rust from becoming a major pathogen in western Europe.

Parlevliet et al. (25) showed that one can select for this resistance in any stage of a breeding program, and at any stage of the barley plant. They selected with success in the seedling stage in the greenhouse by taking seedlings which had slightly longer latent periods and reduced infection frequencies. The latent periods and infection frequencies were not measured; from each box of seedlings, the 10% with relatively long latent periods and the fewest uredia were selected. Selection among genetically diverse genotypes in the field was successful when each genotype was represented by a single plant and even more successful if the genotypes were represented by small plots. In both cases, the selection consisted of taking the 5% or 25% of units (single plants or plots) with the lowest level of leaf area affected. Either selection intensity gave excellent selection response.

In the field, the plants or plots to be selected received their inoculum from spreader rows. These consisted of plants of a highly susceptible cultivar and infection was initiated several weeks before the seasonal epidemic was expected to begin. The single plants were well spaced and two rows were separated by one spreader row. The spreader rows in the case of small plots ran perpendicularly to the plots on both sides. The inoculum used in all experiments consisted of a single race.

This experiment clearly indicated that both selection in the greenhouse, based on components, and selection in the field, based on levels of leaf rust, were effective in raising the level of partial resistance. The following two experiments confirmed this.

**Selection for one component in the greenhouse**—Partial resistance in barley is strongly correlated with latent period in the adult plant stage (25, 26). However, the European cultivars seem to carry much the same minor genes for latent period (15). To increase partial resistance, other minor genes must be added to those of the European barleys. It was observed that the fairly primitive cultivar Cebada Capa carried, behind the hypersensitivity gene Pa7, a high level of polygenic partial resistance. Cebada Capa was therefore crossed to Vada and the F2 seedlings segregated in a 3:1 ratio for the Pa7 gene. All plants carrying Pa7, characterized by a low infection type, were removed and the 25% of the plants with a susceptible infection type were grown on. The adult plants were re-inoculated. They showed a relative latent period ranging from about 150 to over 260 (L94 = 100, Vada = 185). The plants with the longest latent period were kept and F3 lines evaluated the next year. From the selected F3 lines, plants with the longest latent period were again kept and so on. In this way F6 lines were obtained with a relative latent period in the adult plant stage approaching 300 (23). These lines were evaluated in the field and the results were beyond expectation. The partial resistance of the selected lines (Table 2) was 100 times higher than that of Vada and some 5000 times higher than that of Akka. The prevailing conditions were extremely favorable for the rust; there was still some interplot interference and the epidemics in the individual plots had started early. Under normal western European farming conditions, these lines would remain virtually free of rust.
This procedure was very effective but it placed no pressure on agronomic characters and is therefore suitable only for selecting parental material or for improving base populations from which further breeding will be done.

Selection in the field—As mentioned above, it is not sufficient to show that one can increase partial resistance. Agronomic characters must be, at least, maintained. To study this, an experiment was set up to see whether partial resistance could be improved while selecting for agronomic traits as well. Two genetically variable populations, quite different from each other, were taken as the starting point. One population (A) was produced by intercrossing eight two-rowed European spring barley cultivars with each other for the three consecutive generations to recombine their genes thoroughly. The eight cultivars carried no known major genes for rust and varied in partial resistance from hardly any (Mamie, as susceptible as Akka) to good (Vada). The other population (B) was taken from Composite Cross (CC) XXI (32). This extremely variable population had been multiplied in isolation for more than 10 years. This resulted in a population consisting of a mixture of widely different, fairly homozygous lines, most of which were four- to six-rowed. For partial resistance to rust, this population was also quite variable (11). The idea was to accumulate resistance genes, while selecting for agronomic traits as well in both populations, using a recurrent selection procedure. The selected lines of each population would then be crossed to try to get renewed response to selection.

The selection practiced consisted of discarding in each cycle approximately the 30% most susceptible plants or lines. Among those remaining, selection for desirable agronomic traits (grain yield, 1000-grain weight, lodging resistance, earliness, and partial resistance to powdery mildew) was carried out. The selection for partial resistance to the two fungi was therefore very weak.

Table 2. Leaf area affected (percent) and number of uredia per tiller of three barley cultivars and three lines selected for high partial resistance to barley leaf rust about 6 weeks after the start of the epidemic. There were plots of 1.0 m², separated by 4 m of rye. The latent periods given are relative to those of the very susceptible L94 (after 24)

<table>
<thead>
<tr>
<th>Cultivar or line</th>
<th>Leaf area affected on 4/7/1983</th>
<th>No. of uredia per tiller</th>
<th>Relative latent period (L94 = 100%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Akka</td>
<td>40</td>
<td>5000</td>
<td>113</td>
</tr>
<tr>
<td>Sultan</td>
<td>10</td>
<td>1200</td>
<td>137</td>
</tr>
<tr>
<td>Vada</td>
<td>1.0</td>
<td>100</td>
<td>185</td>
</tr>
<tr>
<td>26-6-11</td>
<td>0.011</td>
<td>1.1</td>
<td>291</td>
</tr>
<tr>
<td>17-5-9</td>
<td>0.009</td>
<td>0.9</td>
<td>281</td>
</tr>
<tr>
<td>17-5-16</td>
<td>0.004</td>
<td>0.4</td>
<td>281</td>
</tr>
</tbody>
</table>
The experiment started in 1976 with 5000 plants of each population. After removing approximately 30% of the plants most affected by rust and the 30% most affected by mildew, the remainder was selected for the other agronomic traits. From each population, 400 plants were thus selected. The selection applied in the subsequent generations was essentially the same. The 800 progenies of these plants (S1) were grown and evaluated in the next year, together with various controls. Of each population the 12 best F3 lines (S2) were selected and crossed in as many combinations as possible within each population. In 1979, again, 5000 plants of each population were evaluated, as in 1976. In 1980, the 400 best F3 lines of each population (S3) were evaluated as in 1977, together with suitable controls. The field appeared very heterogeneous and therefore only very weak selection was practiced. About one third of the lines were retained and retested as F4 lines in 1980. The 12 best F4 lines of each population (S4) were selected and used to recombine the genes of both populations. The 12 lines of population A were crossed in as many different combinations with the 12 lines of population B as possible. Of the resulting F2, 10,000 plants were grown in 1983 and selection carried out as in 1976 and 1979. In this population, two-rowed and four- to six-rowed plants occurred. Selection was carried out independently within these two phenotypes. As in the earlier cycles, the 400 best plants were selected and their F3 lines grown and evaluated in 1984. From the F3 lines, the best 20 within each phenotype were selected and this end product of selection (S7) was compared with a series of checks derived from the various stages of the recurrent selection procedure. This evaluation was done at two sites near Wageningen, on a sandy soil and a clay soil.

Results are summarized in Table 3. The data are averaged over replications and sites. The selection for resistance resulted in an almost identical response in both populations. The starting level was the same, a level slightly below that of the fairly susceptible cultivar Sultan. At the end of the second cycle (S5), the amount of rust was greatly reduced, to less than 10%. After the S5, selected lines were intercrossed and from this population S7 lines were selected with partial resistance significantly better than that of Vada. Due to the testing situation, there was a considerable interplot interference. The 60-fold increase in partial resistance in this experiment (Table 3) from the S0 to the best lines clearly underestimated the real progress. Corrected for interplot interference, the real progress is estimated at about 900-fold. This is comparable with a gain from the level of a very susceptible cultivar to the level of a cultivar sufficiently resistant to prevent any significant yield damage in western Europe, even in years conducive to rust (28).

Very interesting was the case with which lines could be obtained that were considerably more resistant than Vada, which represents the highest level of partial resistance among commercial cultivars. Only mild selection against susceptibility was needed and the remarkable gain in resistance was accompanied by a gain in yield as well (Table 3).

Partial resistance in the presence of major genes
In cereal-rust systems, major genes are frequent and we have already seen that their presence seriously
confounds the identification and use of partial resistance. As a rule, one can state that, if major and minor genes are both present, to select strongly will tend especially to select the major genes, while mild selection favors both types (18). If breeders wish to select exclusively for the minor genes, they should ascertain that they first remove the major ones. This implies the need to distinguish clearly between the two resistances, though this is, unfortunately, not always easy.

In the cereal rust pathosystems the major gene resistance is of the hypersensitive type, characterized by low infection type. If such genes caused only very low infection types (say 0 to 3) and partial resistance only very high infection types (say 7 to 9), it would be easy to classify the observed resistances. However, the distinction is rarely so clear. More often there is a more or less continuous distribution of infection types and it is then difficult to separate one type of resistance from the other. This continuous distribution is caused by various factors, as follows:

- Hypersensitive resistance genes may have, even under optimal conditions, different infection types. Major genes with intermediate infection types are not rare.
- The genetic background of a major gene may affect its infection type.

Table 3. Numbers of barley leaf rust uredia per tiller and relative grain yields of two barley populations, A and B, at three stages of selection. Two barley cultivars that went into population A are included as standards (after 28)

<table>
<thead>
<tr>
<th>Generation</th>
<th>Uredia</th>
<th>Yielda/b</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>Mean</td>
<td>A</td>
</tr>
<tr>
<td>S0, unselected</td>
<td>5200</td>
<td>5200</td>
<td>5200</td>
<td>114</td>
</tr>
<tr>
<td>S5, after 2-3 b/</td>
<td>350</td>
<td>500</td>
<td>425</td>
<td>139</td>
</tr>
<tr>
<td>S7, after 3-2 c/</td>
<td>275</td>
<td>250</td>
<td>260</td>
<td>127</td>
</tr>
<tr>
<td>Best S7 line d/</td>
<td>75</td>
<td>100</td>
<td>90</td>
<td>124</td>
</tr>
<tr>
<td>Best S7 line e/</td>
<td>150</td>
<td>100</td>
<td>125</td>
<td>143</td>
</tr>
<tr>
<td>Manic</td>
<td>11000</td>
<td>-</td>
<td>-</td>
<td>76</td>
</tr>
<tr>
<td>Vada</td>
<td>500</td>
<td>-</td>
<td>-</td>
<td>123</td>
</tr>
</tbody>
</table>

a/ The yields are relative to the mean of the starting populations, S0
b/ "After 2-3" means the third stage of selection in the second cycle of recurrent selection
c/ After the S5, A and B were crossed into one population, but the two-rowed and the four- to six-rowed plants or lines were treated as if belonging to two different populations, A and B
d/ Best line, based on partial resistance to barley leaf rust
e/ Best line, based on yield
gene dosage can be of importance. 
It is often reported that major 
gen_s are inherited as dominants. 
If, however, the infection types of 
homozygotes (double-gene dosage) 
and heterozygotes (single-gene 
dosage) are compared, there is 
often a difference, indicating 
incomplete dominance. Thus, Pa7 
in an heterozygous condition gives 
on average a higher infection type 
(range 2 to 6) than in a 
homozygous condition (range 1 to 
4) (22).

The development of the plant is of 
importance as well. The infection 
type may become lower at more 
advanced development stages.

The race may affect the infection 
type.

The environmental conditions are 
of great importance in the 
expression of these major genes. 
Browder (2) says that, for the 
expression of the active event, 
incompatibility resulting in low 
infection type, a specific 
environment is required. In other 
environments, the activity [and so 
the expression] is less. Examples 
of resistance genes giving low 
infection type at a given 
temperature and a higher one at 
other temperatures are numerous.

The moment of assessment can 
affect the infection type 
assessment. After infection, it 
takes some time for the full 
expression of the infection type. 
With a high infection type, the 
optimal moment to assess is often 
a few days after the lesions start 
to sporulate. If one waits, the 
infection type tends to become 
lower probably because of 
exhaustion of the tissue directly 
surrounding the lesion. So, as a 
rule, one should not wait too long.

With non-hypersensitive 
resistance, the infection type is 
not always uniform. Actually, the 
greater the level of partial 
resistance the lower the infection 
type tends to become. In 
seedlings, the partial resistance 
tends to be only weakly expressed, 
so the infection type remains high. 
But, in adult plants, where the 
partial resistance is fully 
expressed, the infection type may 
be somewhat reduced. So, with 
high levels of partial resistance, 
the infection type can be in the 
same range as the infection type 
of some major genes.

Clearly it is impossible to 
discriminate unambiguously between 
major gene and partial resistance on 
the basis of infection type. But one 
can discard at least some of the 
major genes by assessing the 
infection type. How efficient this is 
depends on the host-pathogen 
system. In the barley leaf rust 
system, most major genes can be 
recognized in the seedling stage. If a 
genotype has an infection type lower 
than 8, one should assume the 
presence of a major gene. In adult 
plants it is less easy to discriminate 
as the infection types may overlap. 
However, in case of an intermediate 
infection type with a very long latent 
period, one may think of partial 
resistance, while an intermediate 
infection with a moderate latent 
period may suggest a major gene.

In other crop-pathogen systems, it is 
less easy. Yellow rust in wheat, for 
instance, shows a fully continuous 
spectrum of infection type that is 
well correlated with a resistance 
measured as spore production (3). In 
this pathosystem it is very difficult 
to use infection types in order to 
identify partial resistance.
How should one proceed if major gene and partial resistance are difficult to discriminate? A seedling screening with a single race, which can neutralize as many major genes as possible, is the best approach. Very susceptible and moderately susceptible seedlings serve as standards. Seedlings with low or intermediate infection types can be removed and, among the ones with a high infection type, those which are less affected or carry less and fewer lesions that are slightly smaller could be selected. Major genes that are expressed only in the adult plant stage, such as the wheat Sr2 and wheat Lr12, Lr13, and Lr22 cannot be removed in this type of test.

In many cases, the screening is done solely in the field, in which case only general guidance is possible. Much depends on the host, the pathogen and the local conditions. If the breeder thinks he can obtain reasonable infection type assessments, the advice is to select those plants or lines that have an infection type as high as possible with an amount of tissue affected as low as possible. Plants or lines that are virtually clean of the pathogen should not normally be considered as carrying an extremely high level of partial resistance, but of probably carrying an effective major gene. Table 4 tries to show this. The lines 2, 5, 18, and 20 are assumed to carry a major gene. Those with infection types of 7 or more and a relatively low leaf area affected are considered to carry partial resistance (lines 3, 8, 17, and 22).

If no infection type data are collected at all, the possibility of selecting partial resistance declines. To give partial resistance in such a case a fair chance, one should select mildly

<table>
<thead>
<tr>
<th>Line</th>
<th>IT</th>
<th>LAA</th>
<th>Line</th>
<th>IT</th>
<th>LAA</th>
<th>Line</th>
<th>IT</th>
<th>LAA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>60</td>
<td>C2</td>
<td>8</td>
<td>35</td>
<td>16</td>
<td>9</td>
<td>75</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>0(0)</td>
<td>9</td>
<td>6</td>
<td>15</td>
<td>C2</td>
<td>8</td>
<td>25</td>
</tr>
<tr>
<td>3</td>
<td>*</td>
<td>25</td>
<td>10</td>
<td>9</td>
<td>50</td>
<td>17</td>
<td>7</td>
<td>15</td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td>5</td>
<td>11</td>
<td>7</td>
<td>55</td>
<td>18</td>
<td>1</td>
<td>(0)</td>
</tr>
<tr>
<td>C2</td>
<td>9</td>
<td>60</td>
<td>12</td>
<td>7</td>
<td>30</td>
<td>19</td>
<td>8</td>
<td>60</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>5</td>
<td>C1</td>
<td>9</td>
<td>70</td>
<td>20</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>7</td>
<td>30</td>
<td>13</td>
<td>9</td>
<td>80</td>
<td>C1</td>
<td>9</td>
<td>50</td>
</tr>
<tr>
<td>7</td>
<td>9</td>
<td>70</td>
<td>14</td>
<td>5</td>
<td>10</td>
<td>21</td>
<td>7</td>
<td>40</td>
</tr>
<tr>
<td>8</td>
<td>8</td>
<td>25</td>
<td>15</td>
<td>8</td>
<td>50</td>
<td>22</td>
<td>8</td>
<td>20</td>
</tr>
</tbody>
</table>

a/ (0) = trace
against susceptibility (the lines 1, 7, 10, 11, 13, 15, 18, and 19), but also against too strong levels of resistance (the lines 2, 4, 5, 14, 18, and 20). In this way, at least some of the major genes are removed, while some accumulation of partial resistance is ensured.

In short, the previous discussion shows that it is difficult to select for partial resistance in the presence of major gene resistance but not, in general, impossible. The experiment summarized in Table 3 illustrates the matter. The two barley populations were selected for resistance to two pathogens, rust and mildew. For mildew, population B carried various major genes that were partly effective, while the natural mildew population to which these populations were exposed consisted of mixtures of races that varied in composition over the years. Although the same selection procedure was practiced as against leaf rust, progress in resistance to mildew was small compared with progress in resistance to leaf rust. Progress in the former was probably due to both major gene and partial resistance while progress in the latter was solely due to partial resistance.

References


Chapter 6

Durable Resistance to Yellow (Stripe) Rust in Wheat and Its Implications in Plant Breeding

R. Johnson, Plant Breeding Institute, Cambridge, England

Abstract

Yellow (stripe) rust, caused by the obligate parasite, *Puccinia striiformis*, is found wherever wheat is grown in cool climates. Several race-specific resistance genes effective in wheat seedlings have been identified but more remain to be identified. Such resistance genes may be dominant or recessive and some are strongly influenced in expression by environment and genetic background. Resistance developing after the seedling stage is also frequently race-specific. Combining together race-specific resistance genes has not been successful in controlling yellow rust in Britain. However, some resistance developing after the seedling stage does not show race-specificity even after prolonged and widespread testing. Such durable resistance can only be distinguished from race-specific adult plant resistance by prolonged testing. Although it may be under complex genetic control, such resistance can be used in breeding programs as described here but the durability of resistance produced in such programs cannot be guaranteed. All new resistant cultivars, whatever the breeding method used, should therefore be monitored for evidence of pathogen races with matching pathogenicity.

The Distribution of Yellow Rust on Wheat

Yellow (stripe) rust is potentially a damaging disease in all cool climates in which wheat is grown. Surrounding or contiguous with most of these areas are other wheat growing zones where the climate is marginal, usually too warm and dry, for yellow rust. Occasionally the disease spreads into such areas and its distribution may be thought to be extending. Such events are sometimes attributed to climatic change or, perhaps, to adaptation of the pathogen to higher temperatures. There are several reports of experiments on optimum temperatures for spore production, germination and infection by the pathogen *Puccinia striiformis*. These show that many environmental factors can influence the viability and germinability of the urediospores, including the temperatures, light conditions, and humidity in which they are produced, as well as those in which germination and infection take place (4). It has been suggested that the temperature at which the spores are produced can influence the optimum temperature for germination (20). If so, it is perhaps not surprising to find that different temperature optima have been reported for germination of *P. striiformis* in different studies. What is less clear is whether such differences indicate the specialization of the pathogen into races genetically adapted to different temperatures. Dennis (3) suggested the possibility that races of *P. striiformis* capable of surviving high temperatures in the Australian summer might have high temperature optima for infection. However, he concluded that a race of *P. striiformis* that had evolved in Australia showed similar responses to temperature to those reported in other countries, with an optimum for infection of between 7°C and 10°C, maximum about 18°C and minimum below 0°C. He concluded that these temperatures were probably typical of *P. striiformis*. 
In general, I believe that occasional extended distribution of yellow rust on wheat usually occurs due to widespread cultivation of a highly susceptible cultivar in an area with an environment marginal for the disease, sometimes assisted by unusually favorable weather conditions, rather than adaptation of the pathogen to new climates.

**Specificity in Resistance**

As with the other rust pathogens of wheat, *P. striiformis* has been known since the 1930s to be specialized into races that differ in pathogenicity towards individual wheat cultivars, as well as to related species and genera. It is assumed that the specificity of pathogenicity towards individual wheat cultivars operates according to the gene-for-gene hypothesis. Resistance has frequently been demonstrated in wheat seedlings and eleven specific genes have been described, using the *Yr* symbol (*Yr1* to *Yr10* and *Yr15*) (13, 14, 15). Other such genes have been detected but remain to be identified and designated. Of the *Yr* genes so far identified, including those such as *Yr8* and *Yr9* from alien sources, all have proved to be race-specific, except the recently introduced and not yet exploited gene *Yr15* from *Triticum dicoccoides* (C. van Silfhout, pers. comm.).

The infection types of the different genes expressed in seedlings include some which produce very minute chlorotic flecks such as *Yr1*, *Yr8*, and *Yr10*, others that produce extensive necrosis with or without some sporulation such as *Yr7*, and others that give less consistent reactions sometimes ranging from a nonsporulating reaction to considerable sporulation and only slight chlorosis. I include in this group such genes as *Yr2* and *Yr6*, which may vary with environment and also in response to the genetic background in which they occur.

Some of the named genes are dominant but several are recessive at least in some crosses, including *Yr2*, *Yr6*, and *Yr9*.

In addition to specificity of resistance detected in seedlings, it is critical to note that, in the interaction of wheat with *P. striiformis*, race-specificity of resistance can also be found in resistance that develops after the seedling stage and is most readily detected in adult plants (8, 16, 18, 25). Four resistance genes (*Yr11* to *Yr14*) that provide such resistance were recently identified (15) and it is clear that other race-specific resistances detected in adult plants reported by Stubbs (21) are different from those controlled by these four genes (9). Specificity of resistance in adult plants is not unique to yellow rust, having certainly been observed also in resistance to brown leaf rust of wheat (2). However, I emphasize this aspect of resistance to yellow rust because of the widespread assumption implicit in some publications that resistance that is incomplete or moderate, rather than complete, is race-non-specific.

**Distribution of Races of *P. striiformis* in the World**

Stubbs (21) concluded from worldwide surveys of *P. striiformis* that "yellow rust in all parts of the world possesses the same genetic background of pathogenicity." This was based on observations of the distribution of pathogenicity for recognized race-specific resistance genes in many parts of the world and particularly where such resistance genes were deployed. Despite these observations, it should not be assumed that the distribution of pathogenicity is uniform, and some of the variation may be important in trying to propose a general approach to breeding for
resistance. The following examples suggest some significant differences in the distribution of specific pathogenicity. The gene \textit{Yr5} from \textit{Triticum spelta} var. \textit{album} was effective against all naturally occurring races in Europe for many years, but was matched in Australia within 4 years of the arrival of the pathogen there (C.R. Wellings and R.A. McIntosh, pers. comm.); it was not used widely in Europe nor in any commercial cultivar in Australia. The resistance gene \textit{Yr10} derived from a Turkish wheat PI No. 178383 was transferred to the cultivar Moro, where it was soon matched when this cultivar was used commercially in the USA (1). Pathogenicity was also reported from the Middle East (21) but, so far, no pathogenicity for this gene has been detected in the UK or Australia. It has not been used in commercial cultivars in either country but its present performance in Australia contrasts with the that of \textit{Yr5}. Pathogenicity for \textit{Yr8} from \textit{Aegilops comosa} was found in England from three different sources, including natural occurrence in the field, although the gene was not used in any commercial cultivar (7). Pathogenicity for \textit{Yr8} is found in some other areas, but not everywhere. A race-specific resistance recognized in the Australian wheat cultivar Avocet was detected shortly after the introduction of \textit{P. striiformis} to Australia (23). Attempts to recognize this specificity using UK races have shown that the race-specific resistance of the Australian Avocet is more effective against some UK isolates than others but, of the races so far tested, none completely matches the gene in the way shown by the race 104E137A+ from Australia (Table 1).

Table 1. Infection types (scale: 0 resistant to 4 susceptible) of selections of Australian wheats lacking (-) or possessing (+) race-specific resistance first identified in the Australian cultivar Avocet (YrAv)

<table>
<thead>
<tr>
<th>Line</th>
<th>YrAv</th>
<th>Leaf</th>
<th>104E137A+ by WYR 85-25</th>
<th>104E137A- by WYR 85-24</th>
<th>106E139 by WYR 81-24</th>
<th>45E140 by WYR 75-23</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avocet</td>
<td>-</td>
<td>1</td>
<td>3+-4*</td>
<td>4-</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avocet</td>
<td>+</td>
<td>1</td>
<td>4-</td>
<td>3-3+</td>
<td>3-3+</td>
<td>3-3+</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td>ON-3-</td>
<td>ON-1-N</td>
<td>2N-3-</td>
</tr>
<tr>
<td>Banks</td>
<td>-</td>
<td>1</td>
<td>3-3+</td>
<td>3+-4</td>
<td>4-4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Banks</td>
<td>+</td>
<td>1</td>
<td>3-4-</td>
<td>ON-3</td>
<td>ON-1-N</td>
<td>1+N-3+</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td>ON-2-N</td>
<td>ON-2-N</td>
<td>1+N-3+</td>
</tr>
<tr>
<td>Strubes Dickkopf</td>
<td>(Susceptible)</td>
<td>1</td>
<td>3-4</td>
<td>3+</td>
<td>3+</td>
<td>4</td>
</tr>
</tbody>
</table>

\textit{a/} Isolates from Australia via R.W. Stubbis, possessing (\textit{A+}) or lacking (\textit{A-}) pathogenicity for YrAv (UK MAFF license PHF 48/A80(76))

* No data
These aspects of the distribution of variation in pathogenicity of *P. striiformis* are referred to in the discussion of breeding for durable resistance.

**Durable Resistance to Yellow Rust**

In the climate of the UK, which is favorable to the pathogen in most years, combining as many as four race-specific genes, whether of the type effective in seedlings and throughout the life of the plant, or only in the adult stage, has not so far been a successful breeding strategy. Table 2 indicates the fate of some of the cultivars in which such genes were used. Combining race-specific genes, particularly those for which matching pathogenicity is rare, might provide a more satisfactory control of disease in climates that are less favorable to the pathogen.

Despite these examples of rapidly matched resistance, there were during the same period other cultivars that remained adequately resistant. Similar experiences are reported from the northwestern United States (12). Some cultivars thought to have displayed durable resistance to yellow rust in the UK and elsewhere are listed in Table 3. The list is not complete but all the cultivars included possess a type of incomplete resistance in the presence of races with pathogenicity matching their known race-specific genes. As yet there seems to be no simple test for distinguishing the phenotype presented by these

<table>
<thead>
<tr>
<th>Year</th>
<th>Race</th>
<th>Cultivar/Line</th>
<th>Yr genes in cultivarb/</th>
</tr>
</thead>
<tbody>
<tr>
<td>1966</td>
<td>37E132</td>
<td>Rothwell Perdix</td>
<td>1. 2(seg)c/</td>
</tr>
<tr>
<td>1968</td>
<td>41E136</td>
<td>Maris Templar</td>
<td>1. Cd/</td>
</tr>
<tr>
<td>1939</td>
<td>104E137</td>
<td>Maris Beacon</td>
<td>2. He/</td>
</tr>
<tr>
<td>1971</td>
<td>104E137(2)</td>
<td>Joss Cambier</td>
<td>2, 11</td>
</tr>
<tr>
<td></td>
<td>41E136(2)</td>
<td>Joss Cambier</td>
<td></td>
</tr>
<tr>
<td>1974</td>
<td>41E136(3)</td>
<td>Maris Nimrod</td>
<td>2, C, 13</td>
</tr>
<tr>
<td>1975</td>
<td>232E137</td>
<td>Clement</td>
<td>2??f, 9</td>
</tr>
<tr>
<td>1979</td>
<td>41E136(4)</td>
<td>CWW 916/26</td>
<td>1, C, 14</td>
</tr>
<tr>
<td>1980</td>
<td>171E138</td>
<td>CWW 1771</td>
<td>1, 9</td>
</tr>
<tr>
<td>1982</td>
<td>169E136</td>
<td>CWW 1645</td>
<td>1, 9, 13</td>
</tr>
</tbody>
</table>

a/ All cultivars were too susceptible for further use
b/ Postulated from interactions with races
c/ Segregating
d/ C indicates specificity like that of Cappelle Desprez
e/ H indicates specificity like that of Hybrid 46
f/ ?, probably present but not demonstrated with existing races
Table 3. Wheat cultivars that probably possess durable resistance to yellow rust, maximum level of infection to any race in a field trial 26 June 1980, and probable race-specific genes

<table>
<thead>
<tr>
<th>Cultivar</th>
<th>Origin</th>
<th>Maximum percent infection</th>
<th>Yr genes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anza</td>
<td>USA</td>
<td>8</td>
<td>Av&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>(WW15)</td>
<td>Australia</td>
<td>-</td>
<td>Av</td>
</tr>
<tr>
<td>(Karamu)</td>
<td>New Zealand</td>
<td>-</td>
<td>Av</td>
</tr>
<tr>
<td>Atou</td>
<td>Europe (E)</td>
<td>4</td>
<td>C&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>H. de Bersee</td>
<td>E</td>
<td>5</td>
<td>C</td>
</tr>
<tr>
<td>Bouquet</td>
<td>E</td>
<td>25</td>
<td>C, 14</td>
</tr>
<tr>
<td>Cappelle Desprez</td>
<td>E</td>
<td>37</td>
<td>C</td>
</tr>
<tr>
<td>Champlein</td>
<td>E</td>
<td>38</td>
<td>C</td>
</tr>
<tr>
<td>Elite Lepeuple</td>
<td>E</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td>Flanders</td>
<td>E</td>
<td>17</td>
<td>l(seg), C</td>
</tr>
<tr>
<td>Flinor</td>
<td>E</td>
<td>2</td>
<td>?&lt;sup&gt;c&lt;/sup&gt;/</td>
</tr>
<tr>
<td>Holdfast</td>
<td>E</td>
<td>6</td>
<td>?</td>
</tr>
<tr>
<td>Hybrid 46</td>
<td>E</td>
<td>25</td>
<td>H&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Jubilar</td>
<td>E</td>
<td>13</td>
<td>?</td>
</tr>
<tr>
<td>Little Joss</td>
<td>E</td>
<td>3</td>
<td>?</td>
</tr>
<tr>
<td>Luke</td>
<td>USA</td>
<td>22</td>
<td>?</td>
</tr>
<tr>
<td>Maris Huntsman</td>
<td>E</td>
<td>27</td>
<td>2, C, 13</td>
</tr>
<tr>
<td>Nugaines</td>
<td>USA</td>
<td>33</td>
<td>?</td>
</tr>
<tr>
<td>Starke II</td>
<td>E</td>
<td>17</td>
<td>?</td>
</tr>
<tr>
<td>Viln:orin 27</td>
<td>E</td>
<td>12</td>
<td>C</td>
</tr>
<tr>
<td>Maris Widgeon</td>
<td>E</td>
<td>4</td>
<td>C</td>
</tr>
<tr>
<td>Yeoman</td>
<td>E</td>
<td>20</td>
<td>13</td>
</tr>
<tr>
<td>Desprez 80</td>
<td>E</td>
<td>75</td>
<td>C</td>
</tr>
</tbody>
</table>

<sup>a</sup> Av specificity similar to Australian Avocet
<sup>b</sup> C, specificity similar to Cappelle Desprez
<sup>c</sup> ?, no recognized specificity
<sup>d</sup> H, specificity similar to Hybrid 46
durably resistant cultivars from cultivars that possess incomplete resistance that subsequently proves to be race-specific. For example, those we have identified do not appear to have a high infection type combined with a low disease incidence, as is sometimes indicated for slow rusting resistance or partial resistance that may be inferred to be race-non-specific to other rust pathogens. It is reported from the USA that the resistance of durably resistant cultivars such as Nugaines is temperature-sensitive, being more effective in adult plants at high temperatures (12). In our experience, however, high temperatures are not critical for the expression of resistance in cultivars that we consider to have durable resistance. Nevertheless, it is probable that the majority of the resistances, including those known to be race-specific, are to some degree sensitive to temperature. Thus temperature sensitivity per se cannot be considered to be diagnostic for durable resistance. This raises the problem of whether there are any simple tests that could help to indicate potentially durable resistance.

One necessary component of an active breeding program is the testing of selected lines at many locations to assess the stability of performance. This method may sometimes indicate vulnerability to diseases, thus helping to avoid risks in using a new cultivar. However, the method cannot reliably indicate the potential durability of resistance in a cultivar to variable pathogens such as P. striformis because the total area of the cultivar that can act as a selective screen for pathogenic races remains small in such tests compared with the area in large-scale commercial exploitation. Similarly, it is sometimes considered that successful performance in multi-locational testing can help to ensure that resistance is under complex genetical control. Combined with recycling of breeding material through crossing, as in CIMMYT programs it may help to permit the formation of longer-lasting combinations of resistance genes. However, it is also possible for a single gene, especially a newly introduced one, to give resistance in all test nurseries. The example of resistance to stem rust in triticale given by McIntosh (Chapter 1) illustrates this problem. The alternative, of testing new cultivars with many pathogen races suffers from similar limitations. Even where the cultivar remains resistant, this does not provide a strong test for potential durability of resistance, and it does not necessarily indicate its complex genetical control.

Lastly it should be noted that slow development of disease, that which may be referred to as slow rusting or dilatory resistance, can be due to one of at least three possible causes: 1) race-specific, adult plant or incomplete resistance; 2) a low frequency of pathogenicity for a race-specific gene in a mixed population of races, so that cultivars possessing the gene receive a low frequency of matching infection; 3) slow rusting of a durable, apparently race-non-specific type.

For these reasons, I consider that this still leaves the difficulty of being able to identify cultivars with durable resistance only after they have been widely grown. If this is true, such cultivars are all the more valuable both as potential sources of durable resistance and for studies of the genetical basis and mechanism of their resistance, as indicated for stem rust and leaf rust of wheat by McIntosh (Chapter 1) and Roelfs (Chapter 2).
Genetical Basis of Durable Resistance to Yellow Rust

Many of the cultivars identified as having displayed durable resistance, such as Cappelle Desprez and Maris Huntsman, show evidence of possessing some race-specific components, while others, such as Holdfast and Little Joss, have not displayed any race-specificity to *P. striiformis* in the UK. However, they have not been tested with exotic races of the pathogen. The question can be posed as to whether the resistance of the latter two cultivars and the residual resistance when race-specific components have been matched in the former class are race-non-specific. I think that there can be no absolute proof of non-specificity and, particularly, of the non-specificity of the genetical components of which their resistance can be shown to consist. For practical purposes, however, the resistance has not displayed race-specificity even after prolonged and widespread testing, which encourages the hope, though not the certainty, that it will not prove to be race-specific if used further or even transferred to a new cultivar by appropriate breeding.

For cultivars that possess durable resistance (and contain genes that are race-specific), it is worth enquiring whether those genes provide the residual resistance shown by the cultivars to races that are considered to have matching pathogenicity to the race-specific genes. I have no proof that they do not provide such residual resistance but the data of Table 3 show that cultivars with the same complement of race-specific genes can have very different levels of resistance to the races that have pathogenicity matching those genes. I interpret this to mean that the cultivars carry genes, other than the recognized race-specific genes, that provide these levels of resistance. Thus, in Cappelle Desprez and other similar cultivars, I do not consider that their durable adult plant resistance is, to any measurable degree, a residual or ‘ghost’ effect of their known race-specific genes.

There have been few detailed genetical analyses of cultivars identified as possessing durable resistance to yellow rust. However, some cytogenetical studies have been carried out on Cappelle Desprez and other cultivars at the Plant Breeding Institute (PBI). Using Cappelle Desprez as an example, it is known to possess race-specific resistance to certain races and was described as possessing genes *Yr3a* and *Yr4a* by Lupton and Macer (13). In addition, Cappelle Desprez possesses at the adult stage a moderate level of resistance, that remained effective during widespread use of the cultivar for almost 20 years in the UK (5). Evidence was obtained in many tests for a small race-specific component in this adult plant resistance (6). Races used to analyze the cytogenetic control of the adult plant resistance were those giving the highest known levels of infection on Cappelle Desprez. It was shown that a chromosome designated as 5BS-7BS carried an important component of the resistance since plants nullisomic for this chromosome were much more susceptible than the euploid and that this resistance was controlled by the 5BS arm (Table 4) (11). Families consisting of a mixture of monosomic and euploid plants, derived from plants monosomic for each of the chromosomes except chromosome 5BL-7BL, deviated from the euploid in their resistance. In particular, plants monosomic for chromosomes 5A and 5D were more resistant than the euploid (11). These results corresponded with the results
of studies with other cultivars (17) and suggested that the long arm of chromosomes of homoeologous group 5 often promote susceptibility while the short arms promote resistance. Also, using the technique of reciprocal backcross monosomics, Worland and Law (24) identified a gene (Yr16) on chromosome 2D of Cappelle Desprez that controlled part of its adult plant resistance.

These data indicate that the resistance of Cappelle Desprez is genetically complex: it includes components identified as race-specific and also other components that contribute to the level of resistance observed in adult plants, the effects of which have not been shown to be race-specific. These include various elements that both increase and decrease resistance, the observed resistance being the resultant product of these components. Given this complexity, it is not possible to conclude which are the particular genetic components that are critical for the durability of resistance displayed by Cappelle Desprez.

It is not suggested that this description of the different components of resistance to yellow rust in Cappelle Desprez is a model for other cultivars that possess durable resistance. However, no single genes or gene complexes have yet been identified that can be implicated in durable resistance and the methods I have proposed to transfer durable resistance in breeding programs therefore depend upon trying to ensure that resistance is derived from a known durable source and to include as many of the components of resistance from that source as possible.

Breeding for Durable Resistance to Yellow Rust

At the PBI I have tried to encourage the breeders to use sources of durable resistance, such as Hybride de Bersée and Cappelle Desprez, to provide resistance to yellow rust in new wheat cultivars. As noted above, however, some of the durably resistant wheats possess identified race-specific genes and many modern cultivars that breeders wish to use in crosses also contain race-specific genes. In order to select the more important components of resistance, selection should be carried out using a race of the pathogen with pathogenicity matching the known race-specific genes. If this is done, no special

<table>
<thead>
<tr>
<th>Line</th>
<th>Percentage infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euploid</td>
<td>12.6</td>
</tr>
<tr>
<td>Monosomic 5BS-7BS</td>
<td>17.3</td>
</tr>
<tr>
<td>Nullisomic 5BS-7BS</td>
<td>48.8</td>
</tr>
<tr>
<td>Ditelosomic 5BS</td>
<td>7.7</td>
</tr>
<tr>
<td>Ditelosomic 7BS</td>
<td>47.5</td>
</tr>
</tbody>
</table>

a/ Law et al. (11)
techniques are required, since mere visual selection for resistance suffices. However, it is possible, in some crosses, for recognized race-specific genes to come together in new combinations for which we have no currently available matching race. This can include combinations of genes effective in the seedling with those race-specific genes that are effective only after the seedling stage. These combinations give resistance, sometimes at a higher level than that derived from the durably resistant parent. It is therefore proposed that such combinations should be prevented, either by controlling the crossing so that they cannot be formed or, if they do occur, by eliminating whichever is the simplest gene to detect so that a race with pathogenicity matching the known remaining race-specific genes can be used for screening. An example of the method is indicated in Table 5.

In practice, breeders are reluctant to use old cultivars in crossing programs, and also to limit the range of crosses merely to prevent the formation of new combinations of race-specific resistance genes. My colleagues, Mr. A.J. Taylor and Mr. G.M.B. Smith, have therefore initiated a project to introduce resistance to yellow rust from durably resistant varieties into

Table 5. Pedigree of cultivar Bounty showing how resistance was inherited from the cultivar Ploughman, thought to possess durable resistance to yellow rust derived from Maris Widgeon

<table>
<thead>
<tr>
<th>Race</th>
<th>Seedling</th>
<th>Adult</th>
<th></th>
<th>Race</th>
<th>Seedling</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>41E136</td>
<td>r</td>
<td>r</td>
<td></td>
<td>41E136(3)</td>
<td>s</td>
<td>r</td>
</tr>
<tr>
<td>104E137</td>
<td>s</td>
<td>r</td>
<td></td>
<td>104E137</td>
<td>r</td>
<td>r</td>
</tr>
</tbody>
</table>

Note: Combination of YrH with YrI would have given complete resistance to all races available at the time, so YrH was eliminated and selection carried out in presence of race 41E136(3). The origin of Yr13 in Bounty is not clear, but it may have come from Ploughman, in which it was not detected due to the presence of YrH.

a/ H. race-specificity like Hybrid 46
b/ C. race-specificity like Cappelle Desprez
c/ Resistant
d/ Susceptible
advanced but susceptible lines from the main PBI winter wheat breeding program. In this project, selection has not been limited to resistance to yellow rust but has included resistance to other diseases and selection for agronomic characters (Chapter 5). Selection for resistance to rust takes place in the presence of races with pathogenicity for all the possible combinations of race-specific genes that could occur in the crosses. In order to limit the possible production of effective new combinations of race-specific genes, the opportunity has been taken to eliminate as many as possible of the race-specific genes that can be detected in seedlings. Owing to the recessive nature of some of the genes, this has not been simple. No attempt has been made to eliminate the race-specific genes effective in adult plants, owing to the difficulty of detecting them when races with matching pathogenicity are frequent in the pathogen population present in the breeding nurseries (9). The logistics of this small breeding program are quite different from those of the main winter wheat program, and it is most unlikely that any of the selected lines could compete as successful cultivars with those from the main program. It is intended only that they will be sufficiently attractive for the breeders to consider them as crossing parents, thus keeping in the program as much as possible of the genetic components derived from sources of resistance that are thought to be durable.

As noted, some of the lines from this special breeding program will contain genes for race-specific adult plant resistance, probably including Yr12, Yr13, and Yr14 for which we possess races with matching pathogenicity. However, the distribution of pathogenicity for these genes in the world is not monitored and, if the lines were sent to breeders in other countries, it may be that they would observe the resistance due to those race-specific genes. Likewise, the cultivar Anza and related (or synonymous) cultivars appear to possess a durable resistance to yellow rust. However, they also possess the race-specific component first recognized in the Australian Avocet (C.R. Wellings and R.A. McIntosh, pers. comm.) (Table 3). In Britain, the resistance we observe in these lines is probably due to this race-specific component, rather than to the genes controlling the durable resistance expressed in Anza in the presence of races that have matching pathogenicity. Therefore, if we attempted to use Anza as a source of durable resistance to yellow rust in our programs, we might only be successful in selecting the race-specific component of the resistance and producing cultivars with resistance similar to that of the Australian Avocet which became highly susceptible when its race-specific component was matched.

Because of the extensive nature of the breeding programs at PBI, which are typical of many wheat breeding programs, it cannot be expected that crosses will be limited, or even handled consistently in the way described, to those that will ensure that resistance to yellow rust in all new cultivars has been derived from the appropriate components of a source of durable resistance. It must therefore be expected that many vulnerable race-specific genes will survive in breeding populations and varieties.

**Transgressive Segregation for Resistance to Yellow Rust**

Numerous authors have reported that resistance at a higher level than in either parent can be selected in the progeny from crosses between susceptible or moderately resistant
parents (e.g., 10, 22). One such example concerns a cross between Maris Huntsman and Cappelle Desprez. These two cultivars possessed a similar level of resistance to a race of *P. striiformis* that overcame the recognized race-specific components of both and Cappelle Desprez was in the pedigree of Maris Huntsman. Surprisingly, despite their close relationship, there was wide segregation for resistance in the F2 progeny, indicating that the parents differed genetically in components of resistance (Table 6). This was confirmed in the F3 generation (22). These and other examples indicate that transgressive segregation for resistance to yellow rust can be selected in the progeny from many different wheat crosses. It could therefore be a useful way of increasing resistance by crosses among locally adapted wheats, rather than trying to transfer resistance from unadapted sources thought to possess durable resistance. However, I believe that the assumption that resistance accumulated in this way will be race-non-specific is not warranted. There is no proof that a race matching all potentially race-specific genes can be found to screen the segregating progeny, as proposed by Robinson (19). Furthermore, transgressive segregation for resistance could arise from interactions or additive effects of race-specific genes, or from the transfer of race-specific genes from suppressive to an expressive background (22). It could also arise from accumulation of resistance genes of the type associated with durable resistance. It would not be possible in advance to predict which of these possibilities had been achieved.

**Conclusions**

There is much resistance to yellow rust among existing wheat cultivars and even more is available from alien sources but the genetic basis of much of this resistance is not known. There are numerous examples of resistance that have proved, on exploitation, to be race-specific and as many again that have demonstrated great durability. Much more data should be accumulated on the scale of cultivation of cultivars and the evidence of durability of their resistance to rust diseases. It is proposed in this chapter that the best way to enhance the probability of achieving durable resistance in

---

**Table 6. Frequency distributions of parents and progeny from crosses between Cappelle Desprez and Maris Huntsman infected with race 41E136(3) of *P. striiformis*. (Modified from Wallwork and Johnson (22))**

<table>
<thead>
<tr>
<th>Lines</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>90</th>
<th>100</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cappelle Desprez</td>
<td>9</td>
<td>11</td>
<td>3</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>26</td>
</tr>
<tr>
<td>Maris Huntsman</td>
<td>14</td>
<td>9</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>23</td>
</tr>
<tr>
<td>F1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td>22</td>
<td>14</td>
<td></td>
<td></td>
<td>70</td>
</tr>
<tr>
<td>F2</td>
<td>5</td>
<td>29</td>
<td>33</td>
<td>41</td>
<td>27</td>
<td>21</td>
<td>28</td>
<td>31</td>
<td>18</td>
<td>6</td>
<td>50</td>
</tr>
</tbody>
</table>
new cultivars is to transfer resistance from sources already identified as durable. As yet there is no simple way to identify the precise genetic components that are associated with the durable resistance to yellow rust of wheat, so the method is designed to retain as much of the resistance as possible from durably resistant sources. Resistance could also be accumulated from crosses between moderately susceptible wheats; this can readily be achieved within pedigree programs and does not require resort to recurrent mass selection methods. Although there is no guarantee that such resistance would be durable, the method seems much more promising than the introduction of single major genes or combining already known race-specific genes into new groups. The accumulation of resistance from crosses of locally adapted cultivars could sometimes be attractive as an alternative to attempting to derive durable resistance from known but unadapted sources.

Because of the large-scale nature of major wheat breeding programs, it is unlikely that either of these methods could be applied consistently to all crosses. Furthermore, although the methods might increase the probability of producing cultivars with durable resistance, they are not such as to guarantee future durability. I have also indicated my opinion that no currently available test, including multilocation testing, can determine the potential durability of resistance. Under these circumstances, it remains vitally important to monitor the disease resistance of all new cultivars and to maintain systems for detecting changes in pathogen populations. The risks of genetic uniformity should be continually kept in view and systems to reduce widespread dependence on single uniform cultivars should be encouraged.

References


Chapter 7

Current Thinking on the Use of Diversity to Buffer Small Grains Against Highly Epidemic and Variable Foliar Pathogens: Problems and Future Prospects

J.A. Browning, Department of Plant Pathology and Microbiology, Texas Agricultural Experiment Station, College Station, Texas

Abstract

Indigenous co-evolved populations of small grains progenitors and their obligate parasites are stabilized by epidemiologic dilatory resistance. This is effected genetically by general resistance in which are embedded major-effect genes for specific resistance in different spacegenic configurations. The general resistance protects the plant; the specific resistance protects the population by minimizing the pathogen's aggressiveness by adjusting the finely tuned feed-back interaction of the interlocked host-pathogen genetic systems. Both general and specific resistance should be used in agricultural systems consistent with their evolutionary origin. But often agriculture has depended on genes for specific resistance to protect not the population, as in nature, but the plant from highly epidemic foliar pathogens; this has been experienced when the resistance gene was used in cultivars grown in homogeneous stands over extensive areas. In contrast, programs in Colombia, England, U.K.; Netherlands, India, and the USA (Iowa and Washington) have deployed genes for specific resistances in diverse stands and experienced population protection with as little as 1/3 resistance. The small amount of resistance has stabilized stands in a diverse natural ecosystem; thus, the figure seems valid. Corroborating data from natural and agroecosystems suggest that a holistic gene-management system that includes diversity, as in nature, portends truly durable resistance. In spite of its many benefits, relatively little diversity has been used, primarily because of i) a paradigmatic problem and ii) a cryptic error of experimentation that has frequently underestimated the potential value of resistance and discouraged its use. A suitable compromise between the legitimate needs for agronomic uniformity and the benefits of diversity are three-cultivar mixtures.

Introduction

The USA’s southern corn leaf blight (SCLB) epidemic of 1970 resulted in the greatest biomass loss of any biological catastrophe. The cause of the epidemic is commonly considered to have been Helminthosporium maydis race T, but it was not. It is obvious that H. maydis race T was present and that the environment was favorable but this was a man-made epidemic caused by excessive homogeneity of the USA’s tremendous maize hectarage (23, 33). A few plant pathologists and plant breeders had been concerned about genetic vulnerability to disease, arthropods, and other causes of plant stress prior to 1970. But the birth of the concept of genetic vulnerability in the popular consciousness stemmed from the SCLB outbreak and was certified by the National Academy of Sciences’ (NAS) 1972 tome “Genetic Vulnerability in Major Crops.” It stated, “If uniformity be the crux of genetic vulnerability, then diversity is the best insurance against it” (23, p. 295).
Diversity is considered desirable, even necessary, in many aspects of life, including the world of finance, but not in the crops and animals on which mankind depends. With them, most effort has been directed to minimizing diversity, rather than to reconciling the benefits of diversity with legitimate requirements for uniformity. Over a decade after the devastating SCLB epidemic and the warning of the NAS (23), breeders of five major crops (cotton, corn, sorghum, soybeans, and wheat) deemed USA cultivars not to be genetically vulnerable. The plant breeders, in a survey conducted by Duvick (12), assessed vulnerability relative to their perceived ability to adjust in a reasonable time to a threat detected anywhere in the world and announced expeditiously over their commodity’s international network. They are probably correct if one has the luxury of “time to adjust.”

Significantly, wheat breeders were least optimistic in their response to the survey, possibly because they work on an autogamous crop that, with other small grains, has a long and continuing history of vulnerability to disease. Wheat and other vulnerable crops were in Robinson’s (26, p.3) mind when he pleaded for a new, holistic approach to meeting disease challenges. He stated: “After some 70 years of scientific breeding of plants for disease resistance, we are no nearer control of many of those diseases than when we started. Ironically, we have been least successful with the diseases that have been most intensively studied. We still suffer savage epidemics of wheat leaf rust.” Recurringly, to the present time, small grains have been thrown out of genetic balance with their pathogens and into an epidemic. The intractable pathogens we have been unable to keep in genetic balance cause crowd diseases (diseases of dense populations) and they are our major concern. These are highly variable pathogens in which virulent and aggressive variants can increase at rates of up to 50% per day on new, previously resistant cultivars that give them opportunity. To manage these pathogens, we must manage holistically all aspects of the host population they mirror genetically. Truly, a new, more effective gene-management is indicated if we are to establish and maintain a yield-stabilizing genetic balance. This chapter is about gene management to thwart crowd diseases in crop populations. I discuss: i) genetic and epidemiologic concepts of resistance, concentrating on resistance principles that impact its management in diverse populations to achieve population balance and, therefore, portend durability; and ii) constraints against the use of diversity.

Duvick (12) took pride in plant breeders’ ability to effect future adjustment. As a small grains pathologist, my concern is not just future adjustment but also protecting the current crop: Texas’s 3 million ha of wheat sustained a 28% reduction in yield in 1985 from leaf rust, and the loss was 50% in 600,000 ha of its most productive central area (21). The question is not just how this could have been prevented in 1986 and thereafter (important as that question is), but how it might have been prevented before the 1985 crop was sown in the fall of 1984.

Theoretically, there are two possible strategies that are not mutually exclusive—fungicides and deployment of genetic host-plant resistance. Fungicides carry a cost to the producer and to the environment and pathogens may develop insensitivity to them. If used, they
are completely miscible with resistance. If resistance is adequate and well managed, however, no foliar fungicide should be needed. Theoretically, seed-applied systematic fungicides may be economical, environmentally safe and epidemiologically promising. Applied to the seed of some, but not all, components of a mixed population on a rotational basis as advocated by Wolfe (36), they promise to augment gene-management programs in a unique way. Deployment of genetic host-plant resistance is the main thrust of this paper.

Genetic and Epidemiologic Concepts of Resistance

In this chapter, the term host reaction expresses, simultaneously, reaction (i.e., resistance/susceptibility) of the host, avirulence/virulence and aggressiveness of the pathogen, and favorableness of the environment. Host reaction has been described in diverse ways. Vanderplank (35) advanced resistance theory and practice by dividing resistance into two contrasting types, vertical and horizontal. He said vertical resistance is that which is effective against some but not all races whereas horizontal resistance is that operative against all races. Then he reasoned that vertical resistance acts epidemiologically by decreasing the effective amount of incoming inoculum but not the rate of increase of virulent races, while horizontal resistance acts, not by selecting differentially among strains, but by reducing the rate of increase of all strains.

Thus, Vanderplank (35) not only defined vertical and horizontal resistance genetically, but he projected the consequences epidemiologically. The apparent correlation between the type of genetic resistance in an individual plant and the epidemiological consequences in a population frequently holds, but there are enough exceptions that confusion results. In attempts to help correct this, the terms and concepts have been analyzed and redefined. Unfortunately, some authors chose to retain the terms but with new definitions, which only increased confusion. This was reviewed by Browning et al. (9) and Browning (5). The former (9) saw the need not just to clarify terms but also to separate genetic and epidemiologic concepts of resistance. They retained "specific" and "general" resistance as having the right of priority to describe genetic concepts of resistance. They coined the terms "discriminatory" and "dilatory," to describe epidemiologic concepts. Discriminatory means "to distinguish and treat differently," dilatory means "to delay," and epidemiology deals with disease in populations. Thus, discriminatory resistance or susceptibility characterizes a population of host plants that affects the epidemic by discriminating among pathogen strains, i.e., favoring or rejecting components of the pathogen population. Dilatory resistance characterizes a host population that affects the epidemic by reducing the rate of development of the pathogen population. Tolerance, another useful term, was retained separate from resistance to describe a plant or a population that is rated susceptible visually but that is damaged less by the epidemic than another susceptible plant or population. The pathogen's counterpart to the host's genetic resistance/susceptibility is avirulence/virulence. The counterpart in the pathogen population to dilatory resistance in the host population is aggressiveness.
Management of Host Plant Resistance to Buffer Crop Populations Against Pathogen Populations

The natural ecosystem of Israel abounds in progenitors of cultivated wheat, oats, and barley. They are attacked by the same arrays of pathogens that plague these crops in agroecosystems, yet epidemics are rare; the co-evolved populations have reached a dynamic balance and crop traits (multiple disease resistance, large seed, excellent grain quality, high protein percentage, etc.), highly desired by breeders, have evolved. Such ecosystems yield not only valuable germplasm, but knowledge of how plants in natural ecosystems protect themselves. As these are the same biologic species as their respective cultivated counterparts, a century of literature aids immediate interpretation and application to agroecosystems. This has been reviewed by Browning (4) and by Segal et al. (29).

Conclusions are that natural ecosystems have identifiable protective mechanisms. These are primarily the pathological-ecological phenotype of the host population’s polygenic general resistance or tolerance, and of its necessary counterpart, the pathogen’s polygenic general pathogenicity-aggressiveness. Oligogenic specific resistance/susceptibility and avirulence/virulence probably became superimposed during evolutionary time over the basic polygenic systems primarily responsible for host-pathogen homeostasis in indigenous populations. Oligogenic systems can contribute in a significant way to maintaining homeostasis where both polygenic and oligogenic resistance occur and where a protective spaciogenic (D.S. Marshall, Texas Agricultural Experiment Station, Dallas, Texas, pers. comm.) population structure has co-evolved. Nature uses one resultant type of epidemiologic resistance—dilatory—to protect the population, but it uses many types of genetic resistance and population structures to achieve it. This had been called the protection of indigenousness (4) and it can be emulated in agroecosystems. The challenge that faces agricultural science is to develop agroecosystems that utilize specific and general genetic resistance to create cultivars and/or spaciogenically stable populations with dilatory epidemiologic resistance or tolerance.

This was illustrated (25) with an epidemiological test of different degrees of tolerance and of different types of resistance and population structures, which would tend to simulate a naturally diverse population. Foliowski and Browning (25) trapped urediospores of *Puccinia coronata* at the periphery of large, isolated oat plots and plotted cumulative spore yields as a function of time for their measurement of relative disease progress curves (Figure 1). They included: i) a susceptible check (C649, which is the recurrent parent of the midseason Multiline M73 and of the isoline X421-I) that carried two genes for specific resistance, both of which were matched by races in the experiment; ii) an immune check (X421-I) that only measured background noise because it carried an effective gene for specific resistance from the wild oat, *Avena sterilis*; iii) a cultivar (Portage) said to have “partial resistance,” possibly polygenically inherited; iv) seven pureline cultivars thought to have degrees of polygenic tolerance to crown rust; and v) two multiline cultivars, M73 and E74. The last two had the protection of population
Figure 1. Disease progress curves of *Puccinia coronata* increase on each of the 12 oat cultivars in the 1975 field experiments. Cumulative spore counts per 100 liters of air are an average of three replications fitted to the logistic equation. From ref. (25).
buffering from crown rust by way of the heterogeneity characteristic of multilines (14). The results showed that the multiline cultivars were highly mixed in reaction to crown rust (as in Israel’s indigenous population that was the source of several of the genes for specific resistance, some plants supported considerable crown rust while others were free); X421-I was immune; Portage was moderately resistant; and all other cultivars appeared susceptible. The epidemiologic response, as shown by the disease progress curves of all cultivars, both pure lines (except C649 and X421-I) and multilines, however, was that of a continuum of different degrees of dilatory resistance—a slowing of the rate of epidemic development relative to the susceptible check.

Several types of genetic resistance (pureline specific resistance/susceptibility, multiline specific resistance/susceptibility, and different levels of general resistance) were included in the experiment (25). Epidemiologically, the resistant and susceptible checks showed, respectively, discriminatory resistance/susceptibility against, or in favor of, the rust races in the experiment; all other cultivars (pureline and multiline) displayed only different degrees of dilatory resistance.

In several respects, this experiment (25) simulated aspects of a natural ecosystem and illustrated the principle that nature uses primarily a single type of epidemiologic resistance—dilatory resistance—to protect populations, but that it uses many different genetic systems and spaciogenic structures to achieve it. Specific resistance/susceptibility contributes to dilatory resistance in natural systems, as does the basic system for general resistance. The latter probably is the system most responsible for maintaining homeostasis in indigenous ecosystems. Specific resistance, superimposed on the general resistance system, provides a feedback mechanism and contributes to homeostasis in a major way (5, 24).

Much of our disillusionment with frequently ephemeral specific resistance has come from misunderstanding its role in nature and its epidemiological manifestation as discriminatory resistance. Hence, we have managed it ineffectively. Dilatory resistance is a common and effective phenomenon in nature. Discriminatory resistance, however, so widely used in agricultural systems, is an artifact of agricultural systems. Natural populations do not normally occur in the large homogenous areas required either to generate or receive large quantities of homogeneous inoculum. Mixed stands in nature, or a multiline cultivar, do filter with discrimination the small amounts of incoming “seed” inoculum, but the main effect on pathogen increase and spread will be that of dilatory resistance in the naturally heterogeneous plant populations. What then is the role of specific resistance/susceptibility in natural systems? Parlevliet (24) said that its role is not to protect the plant—that is the role of general resistance—but to prevent the pathogen from becoming too aggressive.

Vanderplank (35) considered that pathogen aggressiveness is the counterpart of host horizontal resistance. An epidemiologic concept, aggressiveness is also the counterpart of dilatory resistance. Thus, in Figure 1, host cultivar Multiline M-73 can be described as having more dilatory resistance than, say, Otec. Also the pathogen population can be described as being
less aggressive on Multiline M-73 than on Otee. Like resistance, aggressiveness is relative. The disease progress curves in Figure 1 give the ecological carrying capacity of the different cultivar entries. Measured epidemiologically, Otee had a carrying capacity for a larger pathogen population than had Multiline M-73; Otee enabled the pathogen population to express its aggressiveness more than Multiline M-73, regardless of the different genetic systems involved. The feedback mechanism of using the finely-tuned specific resistance/susceptibility system to reduce aggressiveness in the pathogen, as illustrated in Figure 1, undoubtedly co-evolved in the host-pathogen system and contributed survival advantages to both the host and the pathogen in a genetically homeostatic equilibrium.

How should the host-pathogen genetic systems that result in the expression of specific and general resistance be managed? Clearly, specific resistance should be used, as in nature, only in diverse populations and, preferably, embedded in an effective system for general resistance (4, 24). How much specific resistance is needed to protect a population? Our work (J.A. Browning and M.E. McDaniel, unpublished) with a 1-resistant: 2-susceptible, two-cultivar mixture showed that as little as one-third resistance is adequate to protect a population even in the long South Texas disease season, in an environment in which the homogeneous susceptible cultivar was killed prematurely by rust. In a coastal environment, even more favorable for the disease, one-third resistance to an individual race still seemed adequate, but the population required more diversity. In this experiment, the susceptible parent was killed by crown rust, but the two Iowa multilines seemed untouched even though half their components were susceptible to strains of the undefined pathogen population.

Similarly, dramatic protection of winter barley from powdery mildew was experienced in the long disease season in England with three-cultivar mixtures, making each population two-thirds resistant (37). Clearly, not nearly as much resistance is required to protect a population from highly epidemic, variable, airborne, foliar pathogens as was formerly—and is still very commonly—thought. That these disease data are corroborated by data from the natural ecosystem (29) gives me great confidence that the one-third figure is real (4). The degree of resistance desired can be obtained, as in nature, by deploying genes for specific resistance in diverse populations and, preferably, by embedding them in an effective second basic system—that which tends to result in general resistance.

Use of Diversity

Borlaug's (3) early wheat multiline development for the Rockefeller Foundation Agricultural Program in Mexico (that later was incorporated into CIMMYT) readied two backcross series for release in the early 1960s. However, they were withheld because the agronomic type was outclassed by CIMMYT's new high-yielding, semidwarf, photoperiod-insensitive wheats. The first multilines used commercially were the highly successful wheats Miramar 63 and Miramar 65 released by the Rockefeller Foundation Agricultural Program in Colombia. Meanwhile, the semidwarfs became widely accepted and, by 1970, CIMMYT Cross 8156 had been proved in commercial production worldwide. To continue its multiline
interest and diversify 8156, CIMMYT developed mixture components based on the 8156 genotype for use by national wheat breeding programs. Wheat multilines are researched in at least three such programs in India and Multiline KSML3 was released in the Punjab in 1979. Unrelated to CIMMYT or 8156, yellow rust-resistant multilines Tumult and Crew were released in, respectively, The Netherlands in 1979 and in Washington and Idaho, USA in 1982 (22).

Our Iowa, USA, program released 13 multiline cultivars of oats in two maturity classes. These were grown on up to 0.4 million ha in Iowa and contiguous states without report of damage from crown rust even in severe crown rust years. Tested also in the long disease season of the Texas coastal plain and in Israel, they responded similarly. In 1983-84, Iowa released a new oat multiline on a higher yielding recurrent parent with needed resistance to the barley yellow dwarf virus (13, 22).

Wolfe (36, 37) has advocated and researched three cultivar mixtures of barley in the UK to buffer primarily against powdery mildew.

In the light of the foregoing, a question obviously arises as to the relative usefulness of multilines and broader (e.g., varietal) mixtures. Multilines, as in Iowa, give agronomic uniformity, as industry required when the first Iowa multilines were released. But these were developed also for research purposes; there are so many benefits from diversity (16, 30) that a uniform agronomic background was necessary to research the role of diversity per se itself in controlling disease. Since that question has been answered, I see no additional biological advantage to growing multilines. They have excessive uniformity, some of it for merely cosmetic traits where diversity would benefit the crop population. A suitable compromise between the legitimate needs for agronomic uniformity and the benefits of diversity against biotic and abiotic sources of stresses are the three-cultivar mixtures developed and researched in England (36, 37).

Constraints on the Use of Diversity

Diversity per se has been reviewed adequately (7, 30) and more recently (22, 35). Each review established a clear need and rationale for diversity. The Rockefeller Foundation (27) recognized that "The multiline theory for the production of composite varieties is one of the truly new concepts of the century in breeding self-pollinated crops." For a long time, diversity has been known to buffer against all of the most highly epidemic foliar pathogens of small grains (7) except *Pyricularia oryzae* that causes rice blast. Finally Chin (11) showed that rice blast, too, could be controlled with mixtures if the right host genotypes were chosen. This illustrates that, to effect control of any crowd disease, the resistance genes used must be functional (28) and relevant (36) to the pathogen population. Diversity has been shown to be effective against a non-specific pathogen (*Septoria nodorum*) of wheat (15) and against a soilborne pathogen of oats (2). It has protected soybeans from the cyst nematode (10) and wheat from Hessian fly (32). It has even benefited yield when different barley cultivars were mixed that carried the same major-effect gene against powdery mildew (J. K. M. Brown, Plant Breeding Institute, Cambridge, pers. comm.).
After the 1970 SCLB pandemic, the NAS (23) targeted uniformity as the "crux of genetic vulnerability" and Ullstrup (33, p. 46) warned "Diversity must be maintained in both the genetic and cytoplasmic constitution of all important crop species" (his emphasis). The section on "Genetic and Epidemiology Concept of Resistance" above built a case for the use of diversity in some form if it is indicated, i.e. if disease or other sources of stress are serious enough to justify it, and the genes for the job are available. The section that followed compared diverse agricultural populations with an indigenous ecosystem and demonstrated its epidemiological naturalness, which portends truly durable resistance. Experience, as in Iowa and Colombia, has proved its success.

With the abundance of theory, of "bio-logic," of data, and of experience from both natural systems and agroecosystems supporting the use of diversity, why has so little diversity actually been used? What are the problems? I suggest that they are these three:

- A paradigmatic one.

- Our failure to communicate convincingly that diversity can buffer a crop population against disease or another source of stress and especially to explain adequately and logically its mechanism of action.

- Other, including unjustified fear of potential superraces and optimism (some of it justified) for the eventual success of current efforts to effect durable resistance by accumulating minor-effect genes, pyramiding major-effect genes, etc.

Space will only allow me to discuss the first two constraints to the use of diversity, which I will do from the perspective of a professional career as one of its advocates. Of the three constraints, the first may be the most significant and the least readily solvable.

The paradigmatic problem

The collective thinking of a peer group of scientists or the state of the art of an industry may be described as their paradigm (19), and it is very difficult to move beyond it. Thus, a paradigm is much more than a clear example or pattern. It is an underlying basic idea of what we are trying to understand. And our paradigm our underlying basic idea, may be so strong and fixed that it may actually prevent us from understanding what we purport to be trying to understand. Our idea of the nature of our subject prompts us to make certain inquiries, not to make others, and to interpret all from the perspective of our paradigm. As Kuhn (19) wrote, even in the sciences, fundamental ideas about the way things work guide our seeing, including what we see from the results of our experiments, rather than simply emerging from what we have seen.

To take a now-ridiculous example, a person convinced that the world is flat (or round) would ask questions and interpret answers from the paradigm of his underlying basic belief about that world. If a person and his peers are sufficiently convinced, albeit subconsciously, that the world is flat, say, even serious inquiry is probably going to result in reinforcing that belief. The individual must take a determined, conscious, sometimes painful effort to keep this from being true. The USA's creation-evolution controversy continues to be a current and relevant example. In both examples,
two irreconcilable paradigms operate. What Kuhn (19) called a *paradigm conversion* is indicated.

Our view of diversity versus uniformity *per se* is another example of irreconcilable paradigms in which a paradigm conversion is indicated. I experienced a personal and very relevant paradigm conversion in my early professional years. I was enamored of a pure-culture, pureline philosophy as any other plant pathologist or plant breeder. Then, while still in graduate school at Cornell University, plant breeder Neil Jensen (16) and phytopathologist and major professor George Kent challenged this. A long, slow, and painful transition started and had to run its course before I could accept diversity in principle. Before that, all my inquiries reinforced my underlying, basic idea of the need for genetic purity; subsequently, in the need for diversity. For many years while I was on the Iowa State University faculty researching and developing multiline cultivars as a member of the oat project and, especially after I was privileged to study disease development in Israel's indigenous populations, I referred to what I called an "agroecosystem bias" or mind-set that helped prevent many excellent scientists from seeing the need and potential benefits that we in Iowa (and a few others) saw in favor of more genetic diversity in agricultural crops. Nowadays, I call that an agroecosystem paradigm. The agroecosystem paradigm is very real and a number of concepts basic to plant pathology, such as the fitness and formae speciales concepts, have been expressed as we know them because of the agroecosystem paradigm. These would have to be expressed very differently to be consistent with observations on pathogen development in both natural ecosystems and agroecosystems (6). Thus, the agroecosystem paradigm governs most thinking in phytopathology and in plant breeding. It also governs most thinking and activity about western-type agriculture and agricultural research in general, including what inquiries are made, what are the current grant-funding areas, what grant proposals are recommended to be funded by peer-review panels, and what we see as a result of our inquiries. I believe that the agroecosystem paradigm of uniformity for uniformity's sake is the greatest constraint to researching, understanding, and using diversity in agroecosystems. "The lesson to be learned seems to be that society in general, and scientists in particular, must realize that some changes of attitudes and emphasis will be required..." (23, p. 13).

**How diversity protects a population**

Probably the most sustained and successful program developing heterogeneous cultivars is Iowa's oat multiline program (8, 13). Yet, according to current thinking, Iowa's multilines cannot effect control of an epidemic foliar pathogen in Iowa and they cannot possibly do what they have done! This illustrates the extent to which we scientists have failed to discern and communicate the means by which diversity helps to control disease. I will discuss this.

As analyzed by Vanderplank (35), an epidemic is controlled by reducing disease either at the outset \((X_0)\) or later by reducing its apparent rate of increase \((r)\). Recent reviewers (22, 36) stated, for example: "if one compares a mixed population with the mean of its components grown separately and exposed to the same pathogen population, there should be no reduction in \(X_0\)" (22, p. 533). And, relatively to \(r\), Wolfe (36) added...
that "... with rust diseases of cereals in ... the northern USA, epidemic levels may be determined almost directly by the exogenous inoculum from further south; the number of pathogen generations during development may be only three or four. The mixture may thus provide little more protection than by simple diversification, with almost no restriction in disease spread." Thus, eliminating Xo and r leaves no known mechanism to explain the dramatic success of the Iowa multilines, and little reason to encourage others to try diversity. Obviously, either the data have not been obtained, or the correct concepts have not been formulated, or communication has failed.

That the above quotation from Mundt and Browning (22) could not be correct defies logic. The statement uses the same criterion for yield of the pathogen or the amount of disease that is the standard measure of success of diversity, namely: compare data from the mixture with the mean of the components in pure stands.

But the matter is academic. If a farmer grows, say, a three-cultivar mixture, he probably doesn't grow or observe the components; a common complaint by scientists is that farmers generally won't grow check-treatments. If the cultivars have different resistances and susceptibilities, the incoming inoculum will be only 1/3 as effective as in a pure stand of one of the component cultivars. The number of early subfoci are similarly reduced from the outset and this will contribute to reducing r and x (amount of disease) and increasing profit. And, as Wolfe (36) has emphasized for yield, the farmer could not have predicted which, if any, cultivar grown in a pure stand would have been protected. In the three-way mixture, all benefited from the outset.

For a diverse population to outyield the mean of its components (at least with near-isogenic lines), there must have been less disease, doing less damage, on the susceptible plants in the mixture than in pure stands; the whole becomes an entity that is greater than the sum of the parts. But the amount of disease is commonly deemed proportional to the percentage of susceptible plants. This traces at least from Leonard (20) who found that "The amount of rust in mixtures... in noninoculated check plots was proportional to the percentage of (susceptible) plants in the mixture" (20, p. 1846). As this result does not mesh with my expectation and is inconsistent with my observations on the Iowa multilines, I am compelled to ask "why?" It seems to me that data leading to this conclusion are an unrecognized artifact of experimentation and that they result when one tests the spread of highly epidemic pathogens in mixtures of host genotypes and i) uses small plots and extraneous inoculum, ii) moves and works among the plants often repeatedly, unwittingly "reading inoculum with one's clothing and hands, and/or, iii) inoculates the mixtures with unnaturally large quantities of inoculum.

Take movement through plots and handling of plants to count uredia, for instance. Fingers are recognized as excellent for making greenhouse inoculations. But when they are used to handle plants in the field to take data on rust increase that is presumed to result from natural spread of the pathogen, they can give spurious results. Once when my project personnel were counting oat stem rust uredia repeatedly in pure stands and mixtures of oat isolines, I
observed that "workers handling plants supplemented natural air currents in disseminating spores. Resistant plants served as effective barriers to air dissemination of inoculum but not to worker dissemination. This was most obvious near the pcci and along entry paths where worker activity was greater..." (7, p. 363-364). The increased amount of rust was visibly and dramatically greater where people worked through the plots. We had no choice but to discard those data. That was the last year we entered plots to take data on rust increase in tests of diversity. Thereafter, we used Rotorod Spore Samplers outside the plots, trapped spores 2 hours daily, counted the spores, and plotted cumulative spore counts/100 liters of air as our quantitative measure of the carrying capacities of different populations and the effectiveness of diversity in buffering against an airborne pathogen (17). The spore yield curves in Figure 1, which are our measure of disease progress, resulted from using this technique (25).

The effect of each of the above three practices is to telescope time, hastening in an unnatural way the time taken by inoculum to reach susceptible plants, so that inoculum quickly ceases to be limiting. With naturally windborne inoculum such asurediospores of the rust fungi (pathogens with r of up to 50% per day) one cannot influence time and expect a fair assessment of the finely-tuned buffering effect of diversity. Both Browning and Frey (7) and Wolfe (36) analyzed the importance of time in the functioning of diversity. Telescoping time gives a monomolecular growth curve, not a sigmoid curve. Experimental techniques that give a monomolecular curve are excellent for the qualitative identification of specific resistance in a disease nursery. But one must achieve sigmoid growth curves to assess dilatory resistance, horizontal resistance in the sense of Vanderplank (35), or the benefits of mixtures. Yet more authors continue to concern themselves with minimizing inter-plot contamination than with other aspects of managing experiments to test diversity: one or more of the three practices are still used, and with the same conclusion of proportionality (1). Vanderplank (34) cautioned against using field techniques that would lead to the so-called "cryptic error" and thus underestimate the value of horizontal resistance. Experimental designs and techniques can lead also to cryptic errors that underestimate the value of diversity which epidemiologically, can be considered "synthetic horizontal resistance." Three experimental techniques that should avoid the cryptic error and evaluate diversity fairly are: i) estimate pathogen yield by trapping spores at the periphery (or down mowed walkways) of large, isolated plots (17); ii) estimate disease remotely with multispectral sensors (D.S. Marshall, pers. comm.); and iii) estimate host yield conventionally.

Conclusions

Diversity is the only defense against the unknown, as against a future disease threat. Use of adequate diversity in some form is also the only way to justify the prediction of genetic protection of an extensively grown crop in the current year. With the ease of mixing genotypes rather than breaking linkage groups, combining many polygenic systems, or pyramiding major-effect genes, diversity is also an easy and effective way of effecting future adjustment such as Duvick (12) anticipates. Furthermore, diversity also offers a means to confront exotic pathogens with high hitchhiking potential and to ensure against an epidemic following their introduction (23, 31).
Clearly, diversity can benefit small grains greatly but more scientists must discover this for themselves... experiments that avoid cryptic errors. This will contribute also to overcoming the serious paradigmatic constraint to using diversity. Grain yield response of mixtures of near-isogenic lines of wheat or oats affected by disease is commonly nonlinear when compared to the means of the components in pure stands. Similarly, the yield of spores or the amount of disease should be nonlinear. This would be expected because disproportionately less rust, causing disproportionately less damage, is generally present on susceptible plants in mixtures than in pure stands. If this is not so and the amount of disease is strictly proportional to the percentage of susceptible plants, I fear it may have resulted from an artifact of experimental technique. This may result especially if “the observer inescapably became part of the observed system” (18), as by superimposing a spore distribution system different from that which one purports to be studying. The all-too-frequent reporting of linear response is probably the second major deterrent to using diversity.

A degree of diversity whereby one-third of the population is resistant seems to effect adequate protection. This goal is safely exceeded with simple three-cultivar mixtures. Cultivar mixtures offer a better compromise than multilines between the needs for uniformity and the benefits of diversity and are, in principle, recommended. Diversity portends an extended useful life expectancy for a valuable natural resource—genes for specific resistance. Of great current importance, diversity portends the same for the even more valuable specific resistance genes that one day will be incorporated via biotechnology. Diversity offers considerable additional benefits to pathology and breeding programs alike, at least if containing a highly epidemic disease is a resource-consuming objective of the programs (8). Once this economy of genes and other resources is recognized, I predict a greatly expanded use of diversity. Diversity is strongly indicated for centers of cultivar improvement. “One cannot help being thrilled at their prospects for helping to feed a hungry world, nor alarmed at their potential for guiding the evolution of major pathogens on a global scale” (7). Thus, diversity should be among the gene-management strategies in the repertoire of responsible breeding programs of international centers and of multi-national corporations.

References


Chapter 8

The Use of Variety Mixtures to Control Diseases and Stabilize Yield

M.S. Wolfe, Plant Breeding Institute, Cambridge, England

Abstract

The major factors leading to loss of effectiveness of disease resistance and fungicides in current European agriculture are briefly described. Among the options available to the breeder and farmer to improve the situation, the use of variety mixtures is discussed in some detail. Considerable evidence from field trials points to the advantages of disease control, yield increase, and yield stability from this simple system, which can be added to any other method of disease control.

Introduction

Within the advanced agricultural system of northwestern Europe, there is continuous erosion of the effectiveness of qualitative and quantitative host resistance and of fungicides by the cereal mildew pathogens. The main reasons for this are summarized in the following paragraphs. First, there are uncontrollable factors intrinsic to the situation. The disease is endemic throughout most of the area, the pathogen has a regular and functional sexual cycle, and the spores are widely distributed by the wind system. Then there are several controllable factors listed as follows:

- **Agronomic features**—Monoculture of cereals, particularly of wheat, tends to be predominant, and is based mostly on the cultivation of very few, highly purified varieties, often in large fields. Large amounts of inorganic nitrogen fertilizer are used, greatly increasing the potential susceptibility of the uniform crops. The general trend to earlier sowing of winter wheat also encourages most diseases.

- **Resistance breeding**—Breeders depend largely on rapid replacement of qualitative resistances that are rapidly overcome because of the factors noted above. Quantitative resistance is also used but the durability of such resistance is often unknown simply because of the technical difficulty of confirming its erosion by the pathogen.

- **Fungicide use**—Because of the problem of obtaining and maintaining effective host resistance, farmers have been persuaded to use fungicides extensively. Unfortunately, there is little diversity among the chemicals available and little more in prospect. As a result, they, too, are steadily losing effectiveness under intense selection.

To progress from this depressing European picture, the following discussion first summarizes the range of options available for disease control. The second part considers specifically the option of using mixed varieties in cereal cropping.

Current Options for Disease Control

**Options for the breeder**

Cereal breeders have to reconcile many different breeding objectives. Within such a framework, the
resources available for improving disease resistance are limited and the following methods prevail.

**Reserve strategy**—The continuous introduction of simply inherited qualitative resistances probably represents the simplest and most reliable procedure for the breeder since the resistance is easy to recognize and causes least disturbance to the achievement of other breeding objectives. This strategy depends on a ready supply of different resistance genes of which many are available for control of barley mildew, but not for wheat mildew. It also contributes to yield variation for the farmer because of pathogen response to the resistances.

**Durable resistance**—Among the simply inherited genes for qualitative resistance to mildew, the ml-o gene in barley has proved to be unusually durable. A pathogen response to the gene was not detected in the field until 1986 (8), about 8 years after its introduction. Even then, it was small; apparently, several genes are needed in the pathogen to overcome the single host gene. Most other single resistance genes have begun to lose effectiveness within 2 to 5 years of introduction.

Partial or quantitative resistance, usually in the form of adult plant resistance, may have been more variable in durability. For example, several wheat varieties with Pm2 mildew resistance became highly susceptible at the seedling stage shortly after introduction, but they still provide adequate resistance at the adult stage. There are many other instances of varieties with quantitative resistance that became more susceptible within a short period of general use. Unfortunately, there are no rigorous tests available either of the mode of inheritance of quantitative resistance or of any pathogen response to it.

**Re-cycling resistance genes**—Except in rare circumstances, re-cycling a variety that has become susceptible is unlikely to be successful. However, if the resistance is hybridized into a new variety with a genetic background selected in the presence of the matching virulence, then the combination of the resistance and the new background may provide useful protection (10). This assumes that, at the time of introduction of the new variety with the re-cycled resistance, the frequency of the matching virulence has declined to a low level.

**Combining resistance genes**—The procedure in Australia and North America, of releasing varieties with combined resistance based on knowledge of the structure of the pathogen population, can be highly successful. The procedure of simply recombining defeated resistance genes that have been used separately is disastrous (11). In this case, the matching virulence genes are already in adapted backgrounds, so that only a simple recombination is needed for the pathogen to overcome the combined host resistance and to cause increased infection of the varieties with single resistance characters.

In summary, the methods described are used to produce varieties intended for large-scale use in intensive agriculture. The first, the reserve strategy, assumes that the resistances will not be durable, but that they may last as long as the expected commercial lives of varieties. However, a system of variety use that prolongs the effectiveness of these resistances may be more economical of resources and of greater benefit to the farmer in terms of stability of production.
The other methods are used in the hope that resistance will persist. However, we are still unable to predict the durability of resistance of any variety ahead of the only known test for this character, of major exposure of the variety for a long period (4). A system of varietal use that protects the variety from this stringent test will therefore help to maintain the effectiveness of its resistance.

Options for the farmer

The breeder produces varieties for use in existing systems of agriculture on which he has little direct influence. The systems that are used tend to arise as compromises between conflicting needs: they vary considerably in their influence on diseases:

Reserve strategy—This depends on a readily available supply of resistant varieties and the assumption that none is likely to remain in commercial production for more than a few years. However, resistance genes may be in short supply, as for wheat mildew, and the claims that new varieties are always better than older ones may sometimes be dubious. Constant change is not liked by the farmer, partly because of cost and partly because he wishes to learn how best to grow a particularly variety in order to restrict the variation in its performance. This process may take several years.

Crop rotation—This can be an effective strategy, particularly for soilborne diseases. The longest possible rotation will provide diversification of cropping both in time and space, particularly if field size is small, thus limiting the size of potential sources of inoculum. Unfortunately, demand for different crops is uneven and, where a major cereal such as wheat or rice becomes dominant, this restricts the possibilities for rotation. Interestingly, the average yield of wheat in Europe has risen to a level that now allows the possibility for improved rotations; it is not certain, however, that this opportunity will be exploited.

Varietal diversification (between crops)—Whether or not rotations are feasible, a farmer should grow different varieties of any crop species in different fields, ensuring as far as possible that the varieties differ in resistance to a particular disease. This form of diversification has the obvious merit of insurance; it is unlikely that a new race of a pathogen able to overcome several different varieties will emerge and increase immediately.

It is also argued (7) that diversification among fields will slow down epidemic development relative to cultivation of a single variety, but the extent of such an effect is debatable. It has been argued that interaction between pathogen populations in adjacent fields occurs only during the early period of establishment of an epidemic (12). However, more recent data suggest that migrating spore clouds may have a considerable influence over a large area during the whole crop cycle (5).

Varietal diversification (within crops)—Mixing varieties with different levels and kinds of resistance offers a simple method of ensuring diversification and interaction between neighboring plants on disease progress. The method can be added to any of the other forms of disease control to obtain the benefits of complementary interaction. This option is discussed in the section below on "The Variety Mixture Option."
Use of fungicides—Farmers are advised not to use fungicides prophylactically but, because they are averse to risk and commonly find it difficult to spray on the correct day, they often do not follow the advice. Unfortunately, the tendency to prophylactic treatment increases selection for fungicide insensitivity in pathogen populations. Farmers are also encouraged to diversify their treatments among the available fungicides but: a) there are very few distinct fungicide groups among which to diversify; b) the major triazole group has a broad spectrum of activity thus encouraging repeated use and continuous selection for insensitivity; and c) manufacturers now encourage the use of mixtures of fungicides that have already been widely used separately. This may lead to rapid selection of pathogen genotypes with complex combinations of fungicide insensitivity characters (15).

However, farmers are becoming more conscious of the cost of pesticides and of public concern about their use. The consequences of these attitudes could be to relax selection for fungicide insensitivity and to place a higher premium on inherited disease resistance.

The Variety Mixture Option

Some of the work on variety mixtures and multiline has been reviewed recently (9). This section highlights some of the more important features of the review, relevant to disease control in mixtures and their potential use in wheat cultivation.

The mechanisms of disease control in mixtures

To restrict the spread of an airborne pathogen in a variety mixture relative to the mean spread in the components grown as pure stands requires only a difference in resistance between the components. Such comparisons should involve geometric rather than arithmetic means (3), but even then, spread in the mixture can be reduced. A simple explanation is that the reaction of a more resistant component is less affected by variation in inoculum density than that of a more susceptible component. Thus, the more susceptible component becomes less infected to a greater degree than the more resistant component becomes more infected. The net effect of reduced infection will become more pronounced as the distance between the susceptible plants is increased.

The phenomenon can be exploited more effectively in restricting pathogens that exhibit specific adaptation to resistant hosts, assuming that the pathogen tends to adapt singly to different hosts rather than to combinations of hosts (14). Under these conditions, the susceptibility of host A to one fraction of the population is restricted by the presence of host B, which is resistant to that fraction. Conversely, the susceptibility of host H to a different fraction is restricted by the presence of host A which is resistant to that fraction. The addition of other resistant hosts increases the effect, principally because of the increased separation of plants with the same genotype. Each addition, however, has a smaller effect and there is probably little to be gained from increasing the number of components beyond five.

Disease control

The net effect of interaction between several hosts can be considerable; disease may be reduced to less than 5% of expectation under favorable conditions (16). Commonly, in a
three-component mixture of spring barley varieties, the level of powdery mildew infection is about half that of the mean of the components grown alone.

Trials with spring barley and mildew infection indicated the necessity for intimate mixing of the components. Indeed, there were indications that a high seed rate was desirable for the mixture so as to reduce the average tiller number and thus increase the interaction between neighboring plants of different resistance genotype. More recently, it has been suggested (6) that the appropriate spatial arrangement of the components of a mixture depends on whether or not the pathogen initiates the epidemic in separate foci. If it does so, implying a slow start from few initial points in the field, then the spatial arrangement of the hosts is not critical; relatively large host unit areas will be as effective as small areas. If foci are not evident, as is true of barley mildew, implying a rapid start to the epidemic from many points, then intimate mixing of the host varieties is essential. Since the focal nature of epidemics is likely to be environmentally variable and therefore unpredictable, the safest procedure may be to opt for intimate mixing.

Most of the evidence for disease control in mixtures has come from trials with crown rust of oats (2) and powdery mildew of spring barley (13). However, evidence from elsewhere suggests that some control of airborne pathogens may be expected from appropriate mixtures of any crop.

Mixtures and durability of disease control
It has often been argued that variety mixtures or multilines would select rapidly for recombinants in the pathogen population that would overcome all components. This may be true for multilines in which the components are near-identical and the resistance genes have been exposed previously to the pathogen population. However, if the pathogen race adapted to all components is at some competitive disadvantage on a particular component against the race adapted only to that component, then the outcome is not predictable (1). Indeed, there has so far been no field evidence for a consistent increase in a complex race on a variety mixture (10).

Furthermore, if a single race able to infect all components does become predominant, it is unlikely to attack all components to the same degree; there may be residual resistance in some. In this case, the level of infection will tend towards that of the most resistant component.

To help to guard against a possible loss in effectiveness of a widely-grown mixture, it is strongly recommended to diversify the composition of mixtures. This requirement adds to the desirability of using mixtures with only few components since these can be matched easily for quality and harvest maturity and new components can readily be introduced.

The effect of disease control on yield: mean yield
There are two options for comparing the yield of a mixture with that of pure crops: use either the mean of the components or the yield of the best component. The argument for the latter is supposedly practical; the comparison should be made rigorously between mixture yield and the farmer's best available option. However, this presupposes that it is possible to predict which component will be the best yielding in a future
environment. Statistical analysis, supported by practical experience, shows that this is generally not possible, even if large bodies of trials data were available. For most areas of the world, such data are not available, so that the only sensible option is to compare mixture yield with the mean of the components grown as pure stands.

During 11 years of trials at the Plant Breeding Institute, more than 150 mixtures, mostly with three components, were compared with their components (17). Among these, 122 were described as well-chosen mixtures in that they provided effective control of powdery mildew (other diseases occurred only occasionally and in small amounts). These mixtures provided a mean yield increase of 8%, with most of the individual yields distributed around those of the best components (Table 1).

The poorly chosen mixtures were defined as those composed of highly susceptible components such that the disease control that did occur was insufficient to protect the mixture from heavy infection. Even under these conditions, the yield distribution of the mixtures was better than that of the individual pure stands.

The effect of disease control on yield stability

Stability of yield is often said to be of more importance to a farmer than is high yield. It is therefore necessary to consider the stability of the high yields of mixtures. Among the trials summarized in Table 1, there were some in which the same mixtures and their components were compared over different environments, mostly among years (17). From these data, it was shown that the yields of three-component mixtures were about as stable as the mean yields of their components grown as sets of three pure cultures, equivalent to the strategy of variety diversification. Mixing and diversification provided more stable yields than pure culture of single varieties (Figures 1 a-c). A similar distribution of results was obtained for a set of 12 winter wheat mixtures in 1986 even though there was little disease. Individual trial effects were not significant but, in all comparisons, the mixture yield exceeded that of the mean of the components.

The lack of data on yield stability of a single mixture grown in diverse locations provided part of the stimulus for a project that is being undertaken by the author and Dr. H.J. Dubin. The intention is to run trials at many sites internationally over 3 years, using widely adapted

| Table 1. Comparisons of yield data for well-chosen and less well-chosen mixtures of three varieties; yields in t/ha |
|-----------------|-----------------|------------------|-----------------|-----------------|-----------------|
| Choice | Mixture No. | Av. yield | Av. yield of pure components | % gain | Mixture better than |
|        |                |           |                               |        | 0    | 1    | 2    | 3    |
| Good  | 122            | 5.61      | 5.20                           | 8***   | 0    | 12   | 50   | 60   |
| Poor  | 30             | 4.66      | 4.58                           | 2*     | 4    | 6    | 13   | 7    |
CIMMYT wheat varieties that differ in their disease resistance characters.

Summary of advantages of variety mixtures
The principal advantages of variety mixtures in relation to disease control, mean yield and yield stability have been described above. In addition, it should be pointed out that mixtures may provide some advantage relative to pure stands in response to non-airborne pathogens and abiotic stresses if there is some variation in the mixture with respect to the stress. This is because neighboring plants less affected by the stress may be able to compensate in terms of yield for those that are more affected; this cannot happen in a pure stand unless the stress only affects random individuals.

Figure 1a. Mean yields (t/ha) each of four spring barley varieties grown as pure stand in each of six trial years.

Figure 1b. Mean yields (t/ha) of four spring barley varieties grown as pure stands but expressed as the average for each of the four possible sets of three permuted from them. Each line thus expresses the effect of diversifying among three varieties.

Figure 1c. Mean yields (t/ha) of each of the four possible mixtures of three components that can be derived from four varieties.

Note: The horizontal line in Figs. 1a-c represents the mean yield over the whole period.
In relation to airborne pathogens, the composition of a mixture can be arranged to provide control of a range of diseases in a way that is difficult to achieve in a single host genotype (Table 2). From this table, the three-component mixtures appear to be especially suitable for mildew control because they are all reasonably resistant and the resistances are derived from different genes. Against each of the rust diseases, a different pair of components is resistant, so that the mixture should be highly resistant to both rusts.

By using mixtures, and particularly if a range of different mixtures is provided, the farmer is forced into variety diversification with little effort on his part. The greater the area that is occupied in such a strategy, the larger will be the overall benefit, relative to large-scale pure culture, as the size of each pathogen population is damped down.

The potential advantages of cultivating mixtures on a large-scale appear to have been recognized first in the German Democratic Republic (GDR) where 60% of that country’s malting barley crop is now produced from mixtures. The popularity of this approach has been influenced by the high cost of fungicides in the GDR; for a similar reason, it is likely that Poland will soon have a large area of variety mixtures. Interest has been slower to develop in western Europe, although the area of mixtures in Denmark is considerable, helped by public concern against the over-use of pesticides.

**Summary of disadvantages of variety mixtures**

Other than the reluctance, particularly on the part of the seed trade, to change any feature of the agricultural system, the main disadvantage of mixtures lies in the acceptance of the product by large-scale users of grain. However, the technical reasons are often not clear.

---

**Table 2. Disease resistance characters of three winter wheat varieties. A high number denotes resistance**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Brock</th>
<th>Norman</th>
<th>Rendezvous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Powdery mildew</td>
<td>8</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Yellow rust</td>
<td>7</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>Leaf rust</td>
<td>4</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Septoria nodorum blotch</td>
<td>7</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Septoria tritici blotch</td>
<td>6</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Eyespot</td>
<td>4</td>
<td>5</td>
<td>8</td>
</tr>
</tbody>
</table>

Data from the NIAB leaflet. *Recommended List of Cereal Varieties, 1987*
strong and may be removed if a limited range of mixed grain were to be produced in large quantity; this appears to be the case in the GDR.

Agronomically, there are few difficulties. It is important to ensure that the range of harvest maturity of the mixtures components is less than the potential range of harvest dates; where herbicides are used, the components must have the same range of sensitivities.

Unfortunately, it is usually necessary to reconstitute the mixtures after one or two cycles of production because of drift in their composition. Such changes often result from the competitive action of the most vigorous component, frequently unrelated to disease response.

References


Chapter 9
Current CIMMYT Approaches in Breeding Wheat for Rust Resistance
S. Rajaram, R.P. Singh, and E. Torres, Wheat Program, CIMMYT, Mexico

Abstract
Wheat varieties derived from CIMMYT germplasm are grown on more than 50 million ha in the developing world. Because these materials are grown on such a large area which probably will increase in the future, the CIMMYT breeding policy has been to maintain and enhance diversity of rust resistance in wheat germplasm. International multilocation testing (IMT) has made an important contribution in ascertaining this genetic diversity. In addition to IMT, CIMMYT employs some genetic analyses in its improvement strategy. CIMMYT believes that utilization of major gene, pathotype-specific, vertical resistance (VR) as described by Simmonds (Chapter 10) could lead to precarious situations; as an alternative to the endless incorporation of resistance genes or gene combinations, CIMMYT has pursued and recommends breeding for what Simmonds describes in Chapter 10 as polygenic, pathotype-non-specific horizontal resistance (HR) which portends durability of resistance. In a global context, durable resistance (or stability) and genetic diversity are of paramount importance in CIMMYT's breeding program. The ideal situation would be to identify a gene or set of genes that may prove to have provided durable resistance as a foundation and then continually combine additional genes for resistance to ensure genetic diversity. Stem rust resistance (Sr2 complex) derived from the variety Hope and leaf rust resistance (Lr13 complex) derived from the variety Frontana are the foundation of resistance durability to these two diseases in CIMMYT germplasm. For yellow (stripe) rust, the CIMMYT-bred variety Anza has been reported to have durable resistance to the disease. CIMMYT routinely identifies lines with partial resistance (slow rusting) in the field and believes this endeavor has made important contributions to sustainability of wheat yields worldwide. CIMMYT has pursued multiple major gene-based resistance only as a supplementary strategy and is actively looking into the revival of the development and use of multiline composites/cultivar mixtures.

Introduction
The CIMMYT wheat improvement program produces high-yielding, broadly adapted, rust-resistant germplasm for the less developed countries (LDCs). In 1967-68 high-yielding semidwarf wheats were grown on about 5 million ha in the LDCs; by 1982-83 this area had increased to 50.7 million ha (3). Most of these wheats are either CIMMYT materials or are lines derived from crosses with CIMMYT germplasm in national programs. Both yield potential and productivity of wheat have steadily increased in many agroclimatic environments with genetic advance for disease resistance playing a principal role. In the southern part of Sonora, Mexico, yield potential for bread wheats in this favorable environment doubled from 3000 kg/ha to 6000 kg/ha between 1950 and 1960. From 1960 to 1980, yield potential increased further by at least 100 kg per year. Most varieties released in the 1980s have yielded more than 8000 kg/ha in the Yaqui Valley of Sonora. In a favorable year, it is not unusual to realize as much as 9000 kg/ha.
Due to those yield gains and subsequent increases in production, especially in optimum growing environments, the sustainability of yield is a central and continuing objective of the CIMMYT wheat breeding programs. The potential genetic vulnerability to mutable pathogens, particularly the rust fungi, warrants top priority for research, especially in regard to which type of resistance should be chosen and which breeding methodology will be used to achieve that resistance. Although there have been sporadic, isolated epidemics of leaf rust on semidwarfs, it is noteworthy that major rust pandemics, phenomena that frequently occurred in the past on wheat land races in developing countries, have not been reported since the large-scale introduction of semidwarf varieties 20 years ago.

The durable resistance to stem rust and leaf rust has been achieved despite the fact that more than 50 million ha of semidwarfs have been planted in an environment that is very conducive to the development of these diseases. Furthermore, in the Indian Subcontinent approximately 7 million ha are grown to one variety, Sonalika, that is resistant to stem rust. Prior to the introduction of the semidwarfs, few observers would have predicted that Sonalika would have displayed the durability of resistance that it has considering that it occupies such a large area. Sonalika was introduced 20 years ago in India. Since semidwarfs now occupy approximately 50% of the wheat acreage in the developing world, it is opportune to evaluate the durability (stability) of rust resistance in semidwarfs (especially in regard to stem rust and leaf rust associated with Mexican-CIMMYT semidwarf bread wheats). This chapter highlights the critical factors that have provided stable rust resistance in CIMMYT germplasm worldwide.

**Incorporation of Genetic Diversity**

Recognizing that wheat varieties derived from CIMMYT materials are grown on such a large area and are exposed to different pathogens under conditions that may favor disease development, CIMMYT breeding policy has been to utilize sources of germplasm that are as diverse as possible for rust resistance. The current array of varieties in CIMMYT's bread wheat crossing block consists of varieties and lines with the following characteristics and geographic origins:

1. Stem rust- and leaf rust-resistant germplasm from the Southern Cone countries of South America.
2. Yellow rust- and leaf rust-resistant germplasm from the Andean region of South America.
3. Rust-resistant germplasm from Central America, including Mexico.
4. Rust-resistant wheat lines from North America:
   a) Yellow rust-resistant lines from the Pacific Northwest.
   b) Stem rust- and leaf rust-resistant lines from the Great Plains of the USA and Canada.
5. Stem rust- and leaf rust-resistant lines from the Indian Subcontinent.
6. Stem rust- and yellow rust-resistant lines from the eastern highlands of Africa, including Kenya.
7) Rust-resistant lines and varieties from North Africa, the Iberian Peninsula, and Middle East, including the Nile Valley region.

8) Yellow rust-resistant lines from western Europe.

9) Stem rust-resistant lines from southern Europe.

10) Stem rust-resistant lines from Australia and New Zealand (Oceania).

The flow of germplasm to and from the bread wheat improvement program is continuous and CIMMYT scientists are in contact with national program scientists to ensure this germplasm exchange. The sources of rust resistance in CIMMYT germplasm have, by intent, been kept very diverse, first by the exchange of germplasm and second by its use in the breeding program. When a new yellow rust race was introduced into Australia in the early 1980s, most of the locally developed varieties without CIMMYT germplasm in their pedigrees were highly susceptible, but CIMMYT-derived germplasm was highly resistant (R.A. McIntosh, pers. comm.). This situation permitted Australian plant breeders to use CIMMYT germplasm as a principal source of yellow rust resistance in their breeding programs.

**International Multilocation Testing: A System That Aids the Enhancement of Genetic Diversity in CIMMYT Germplasm**

Although multilocation testing is not a perfect system for identifying resistance sources, evidence accumulated in CIMMYT over many years would indicate that multilocational testing has greatly facilitated the confirmation of the existence of genetic diversity in CIMMYT germplasm. Indeed, the initiation of international testing came about because three people envisioned its use for identifying diverse and durable sources of resistance. Dr. E.C. Stakman proposed the USDA International Rust Nursery in 1950; Dr. N.E. Borlaug proposed similar testing for CIMMYT in 1958-59; and Dr. John Niederhauser initiated such testing for the Rockefeller Foundation’s Potato Program in the 1960s. This testing system is now fully in place for major crops whose improvement is dealt with at other International Agricultural Research Centers (IARCs) and at major agricultural universities in the United States with international programs.

It should be emphasized that international multilocation testing (IMT) is complementary to traditional genetic analysis and both should be parts of an overall improvement strategy. Some geneticists/plant pathologists have criticized the system perhaps without giving due consideration to its benefits, which have included the development of germplasm with the combined traits of high yield potential, broad adaptation, and resistance to the three rusts. CIMMYT has employed both IMT and genetic analysis in its improvement strategy, as will become clear in this chapter.

Genetic studies have suggested that wheat genotypes that are resistant to a given rust disease in many dissimilar locations—as indicated by low average coefficients of infection—often contain multiple factors for resistance (14). Irrespective of whether some of this resistance is race-non-specific, a line that contains several functioning resistance genes has a better chance to have stable resistance against a changing pathogen than one with a single gene resistance. By testing
lines at a number of epidemiologically dissimilar sites and exposing the lines to the greatest possible range of virulence factors, the probability of identifying lines that may prove to have had durable resistance should, in principle, be increased.

CIMMYT recognizes that, in certain situations, a single gene, such as Lr19, will give a low average coefficient of infection because no virulence has developed for that infection gene. But such a situation is rare and should be treated as an exception. The resistance of most lines showing low average coefficient of infections in international nurseries is polygenic (14).

A low average coefficient of infection may be associated, but not necessarily, with the presence of broad-based resistance. Analyses of patterns of reaction to diseases at diverse sites that possess different combinations of virulence factors allow grouping of lines with distinct sets of resistance genes. Although the individual genes cannot be identified thus, the method does provide a simple, rapid means of identifying lines with different resistance genes for use in the breeding program (4). Table 1 provides a list of the bread wheat nurseries that comprise part of CIMMYT’s multilocation testing system.

In Table 2, the genetic diversity of 188 advanced lines of bread wheat is shown for resistance to stem rust, leaf rust, and yellow rust. The lines are classed into eight arbitrary groups. Such noticeable differences suggest the existence of different groups of varieties where response to rust is under distinct genetic control.

### Table 1. Major international bread wheat nurseries distributed by CIMMYT for the evaluation of disease resistance. Different locations have different pathogen populations

<table>
<thead>
<tr>
<th>Nursery</th>
<th>No. Sets distributed (1986-87)</th>
<th>Approximate no. of entries a/</th>
</tr>
</thead>
<tbody>
<tr>
<td>International Bread Wheat Screening Nursery (IBWSN)</td>
<td>186</td>
<td>250</td>
</tr>
<tr>
<td>International Septoria Observation Nursery (ISEPTON)</td>
<td>90</td>
<td>100</td>
</tr>
<tr>
<td>Helminthosporium Resistance Screening Nursery (HRSN)</td>
<td>84</td>
<td>100</td>
</tr>
<tr>
<td>Scab Resistance Screening Nursery (SRSN)</td>
<td>61</td>
<td>80</td>
</tr>
<tr>
<td>International Disease Trap Nursery (IDTN)</td>
<td>245</td>
<td>200</td>
</tr>
<tr>
<td>Barley Yellow Dwarf Virus Screening Nursery (BYDVSN)</td>
<td>54</td>
<td>150</td>
</tr>
<tr>
<td>Drought Screening Nursery (DSN)</td>
<td>50</td>
<td>150</td>
</tr>
<tr>
<td>Aluminum Tolerance Screening Nursery (ATSN)</td>
<td>50</td>
<td>150</td>
</tr>
</tbody>
</table>

a/ Germplasm included in each nursery is different and serves different mega-environments; rust evaluation is made on each entry and ACI calculated on the basis of IMT data.
Additional genetic analysis is critically needed to interpret the genetic makeup of these groups but is not absolutely essential for plant breeding. The CIMMYT Wheat Program does perform some analyses on selected groups of genotypes, but this work should be expanded, preferably conducted as collaborative research with other centers of excellence.

It should be emphasized that "hot spot" locations (those locations with maximum variability of a pathogen and/or severity of a disease) are carefully selected for inclusion in the IMT program. Table 3 lists locations currently used as hot spots. Hot spot locations for rusts exist in Kenya, Ecuador, and Mexico.

### Durability of Resistance and Genetic Diversity

### General features

In a global context, durable rust resistance, along with genetic diversity for thwarting genetic vulnerability, is of paramount importance.

#### Table 2. Genetic diversity in 188 advanced lines of 18th International Bread Wheat Screening Nursery classified into average coefficient of infection (ACI) in respect to the three rusts in International Multilocation Testing (IMT)

<table>
<thead>
<tr>
<th>Disease</th>
<th>No. of locations</th>
<th>No. of entries in ACI classes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0-2</td>
<td>2.1-5</td>
</tr>
<tr>
<td>Leaf rust</td>
<td>31</td>
<td>38</td>
</tr>
<tr>
<td>Stem rust</td>
<td>12</td>
<td>21</td>
</tr>
<tr>
<td>Yellow rust</td>
<td>24</td>
<td>21</td>
</tr>
</tbody>
</table>

#### Table 3. Hot spot locations for various diseases currently utilized by CIMMYT for shuttle breeding and testing

<table>
<thead>
<tr>
<th>Location</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Njoro, Kenya</td>
<td>Stem rust</td>
</tr>
<tr>
<td>Quito, Ecuador</td>
<td>Yellow rust</td>
</tr>
<tr>
<td>Cd. Obregon, Mexico</td>
<td>Leaf rust</td>
</tr>
<tr>
<td>Rio Bravo, Mexico</td>
<td>Leaf rust</td>
</tr>
<tr>
<td>Poza Rica, Mexico</td>
<td>Helminthosporium leaf blotch</td>
</tr>
<tr>
<td>Holetta, Ethiopia</td>
<td>Septoria tritici blotch</td>
</tr>
<tr>
<td>Toluca, Mexico</td>
<td>BYDV</td>
</tr>
<tr>
<td>Nanjing, China</td>
<td>Fusarium head scab</td>
</tr>
</tbody>
</table>
importance in CIMMYT’s breeding program. It is essential that germplasm with different genetic makeup for rust resistance is available to breeders for deployment. However, as has been discussed in the literature, it is quite possible that a particular genetic resistance may succumb to a new biotype. Whenever this happens, the breeder develops materials with another kind of resistance or combination of resistances, until that is overcome also. The availability of genetically diverse materials has so far allowed breeders to keep on utilizing those resistances. CIMMYT believes that this major gene, pathotype-specific, vertical resistance (VR) as defined by Simmonds in Chapter 10 is a precarious situation and, in the event of the development of new pathotypes, could result in catastrophic epidemics.

An alternative to the continuous development of new sets of resistance gene combinations is to attempt to breed for resistance that has a better probability of being durable. In the literature, there are a number of instances of rust resistance associated with durability. Breeding exclusively for one set of resistance genes, even if they are of a stable nature, is likely to result in a narrowing of genetic variability—this is not satisfactory in a global context. The ideal situation would be to identify a gene or set of genes for probable durability of resistance to be used as a backbone, and then continually combine various other sets of genes to provide genetic diversity. This situation would result in germplasm that combined genes for durability of resistance with other resistance genes. We need these other genes even though we already have durable resistance genes because, in many instances, the gene or genes for durable resistance perform better in the presence of other genes. Historical evidence of durable resistance is an indication of polygenic, pathotype-nonspecific, horizontal resistance (HR) as described by Simmonds in Chapter 10.

Use of the Sr2 complex in CIMMYT germplasm for control of stem rust

The stem rust resistance derived from the variety Hope (Sr2 complex) seems to have provided the foundation for durable resistance to stem rust in CIMMYT germplasm. Durable resistance in CIMMYT germplasm to stem rust undoubtedly facilitated adoption of semidwarf germplasm in many developing countries. Before the Sr2 complex was bred into the semidwarfs, stem rust created periodic havoc in South America, Asia, and Africa. Analysis of comparative stem rust infection data on local land race, improved tall, and semidwarf varieties indicates that semidwarfs are, in general, more resistant than the local land races and unimproved tall varieties (Table 4). This finding is contrary to the opinion of critics of the green revolution.

As far as can be ascertained, the enhanced resistance to stem rust in CIMMYT semidwarfs is associated with the Sr2 gene complex (Sr2 gene in combination with various other genes), derived from the variety Newthatch, a Minnesota release, which inherited it from the variety Hope (Figure 1). It is unlikely that Sr2 alone would have imparted this durable resistance. It is CIMMYT’s contention that the Sr2 gene in combination with other genes is responsible for this durable resistance. Dr. A.P. Roelfs, University of Minnesota (unpublished) has postulated that the resistance of most of the CIMMYT semidwarfs to stem rust is associated with Sr2; however, he ascertained that there
were additional genes (sometimes three to four) in the background. The Sr2 complex is an excellent example of the combination of a durable resistance gene plus an array of additional genes which has compound durable resistance to stem rust in CIMMYT germplasm. Since varieties derived from CIMMYT germplasm have remained resistant to stem rust in worldwide testing, it is suggested that the Sr2 complex in CIMMYT wheats is a typical example of the HR character referred to by Simmonds in Chapter 10, not VR. It is important to note that stem rust resistance in CIMMYT germplasm has not been conferred through the

Table 4. Bread Wheat Regional Disease Trap Nursery (RDTN) data (average coefficient of infection) for stem rust (P. graminis f.sp. tritici) from approximately 50 locations in 30 countries

<table>
<thead>
<tr>
<th></th>
<th>Year 1978</th>
<th>Year 1979</th>
<th>Year 1980</th>
<th>Year 1981</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local</td>
<td>21.8</td>
<td>21.8</td>
<td>21.1</td>
<td>19.6</td>
<td>21.0</td>
</tr>
<tr>
<td>Improved Tall</td>
<td>9.2</td>
<td>8.6</td>
<td>9.9</td>
<td>9.3</td>
<td>9.0</td>
</tr>
<tr>
<td>Semidwarf</td>
<td>7.8</td>
<td>4.7</td>
<td>5.1</td>
<td>6.2</td>
<td>6.0</td>
</tr>
</tbody>
</table>

Source: CIMMYT, unpublished.

Figure 1. Major wheat cultivars of Mexico (*) from 1950 onwards that have remained resistant to populations of stem rust under field conditions. Their resistance is probably derived from the cultivar Newthatch (Sr2).
presence of a single gene (i.e., \( \text{Sr2} \)) but through a complex of genes in which \( \text{Sr2} \) plays a principal role. Drs. R.A. McIntosh, University of Sydney, and A.P. Roelfs (both contributors to these proceedings, Chapters 1 and 2 respectively) have confirmed this in personal communications.

As already mentioned, the original source of \( \text{Sr2} \) was the variety Hope (and the related line H44-24) which has remained resistant to stem rust for the last 70 years. University of Minnesota breeders used Hope in their program and produced Newthatch. Dr. N.F. Borlaug of CIMMYT used Newthatch in the Mexican/ Rockefeller Foundation breeding program in the early 1950s. Since then this complex has entered a substantial amount of germplasm. Knott (9) and Green and Dyck (6) have described genes \( \text{Sr2}, \text{Sr7b, Sr7d}, \text{Sr17}, \text{and Sr18} \) in the variety Hope. Hare and McIntosh (7) located \( \text{Sr2} \) in Hope and Hope derivatives in the short arm of chromosome 3B. These authors also found that the amount of rust development on varieties with \( \text{Sr2} \) was variable and may have been modified by alleles at additional loci.

CIMMYT (16) and Minnesota (18) studies have also provided some confirmatory evidence that \( \text{Sr2} \) alone gives only a slow rusting response when tested with races in Mexico; however, when combined with other resistance genes, this has resulted in enhanced levels of durable resistance.

Over the last 20 years (1965-1985), the CIMMYT breeding program has attempted to incorporate diversity in conjunction with \( \text{Sr2} \). Most of the genetic combinations displayed in the international nurseries sent to CIMMYT cooperators have \( \text{Sr2} \) plus two to four additional genes (A.P. Roelfs, pers. comm.). These additional genes, which may or may not be modifiers, include \( \text{Sr5, Sr6, Sr7a, Sr7b, Sr8a, Sr9b, Sr9d, Sr9e, Sr9g, Sr10, Sr11, Sr12, Sr17, Sr24, Sr26, Sr30, Sr31, and Sr36} \).

**Distribution of the \( \text{Lr13} \) complex in CIMMYT germplasm as a safeguard against leaf rust**

The South American variety Frontana has been judged to be one of the best sources of durable resistance to leaf rust (A.P. Roelfs, pers. comm.). The variety was first used in the Mexican-Rockefeller Foundation Program in the 1950s. The genetic analysis of this variety by Dyck et al. (5) has indicated the presence of \( \text{Lr13} \) and other genes. The current general opinion, especially of Drs. D.J. Samborski, Canada Department of Agriculture, Winnipeg, A.P. Roelfs, and R.A. McIntosh (pers. comm.) is that the \( \text{Lr13} \) gene, in combination with the other genes, may impart a high degree of durable resistance to leaf rust. Under Mexican conditions, this gene complex shows a slow rusting characteristic—similar to the \( \text{Sr2} \) complex phenomenon discussed above (i.e., pathotype-non-specific, horizontal resistance or HR as described by Simmonds in Chapter 10. CIMMYT recognized the importance of this gene complex in the early 1970s when it was transferred, along with other genes, into many wheat varieties. Table 5 provides a partial list of these varieties.

Again, it should be emphasized that \( \text{Lr13} \) alone confers only a measure of resistance, at least in Mexico, and only in conjunction with other genes does it provide a degree of resistance with high probability of being durable. The mode of action of the
LR13 complex in the Mexican-CIMMYT Program is non-specific resistance (HR as described by Simmonds in Chapter 10, and the most important aspect is that LR13 must be combined with other genes. Varieties such as Genaro 81, Pavon 76, and Torina 73 have remained resistant to leaf rust in Mexico for 6, 11, and 14 years respectively. Analyses of CIMMYT advanced lines in the rust laboratories at the University of Minnesota by Dr. A.P. Roelfs and at Castle Hill Australia by Dr. R.A. McIntosh suggest that most CIMMYT lines have the LR13 complex (unpublished data).

An analysis of the resistance spectrum to leaf rust in local landrace types, tall improved, and semidwarf wheats, compared over a 4-year period indicates that the semidwarfs are more resistant than the other two (Table 6). This reflects effective deployment of genetic resistance to leaf rust on a worldwide basis.

Table 5. CIMMYT varieties and CIMMYT derivatives from India and Pakistan with LR13 a/

<table>
<thead>
<tr>
<th>Variety</th>
<th>Country of Adoption</th>
<th>Variety</th>
<th>Country of Adoption</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sonalika</td>
<td>India</td>
<td>Kea&quot;S&quot;</td>
<td>Mexico</td>
</tr>
<tr>
<td>Inia 66</td>
<td>Mexico</td>
<td>Prl&quot;S&quot;</td>
<td>Mexico</td>
</tr>
<tr>
<td>Tobari 56</td>
<td>Mexico</td>
<td>Myna&quot;S&quot;</td>
<td>Mexico</td>
</tr>
<tr>
<td>Nuri 70</td>
<td>Mexico</td>
<td>Chilero&quot;S&quot;</td>
<td>Mexico</td>
</tr>
<tr>
<td>Yecora 70</td>
<td>Mexico</td>
<td>Kauz&quot;S&quot;</td>
<td>Mexico</td>
</tr>
<tr>
<td>Zaragoza 75</td>
<td>Mexico</td>
<td>Prl&quot;S&quot;/Vee&quot;S&quot;</td>
<td>Mexico</td>
</tr>
<tr>
<td>Pavon 76</td>
<td>Mexico</td>
<td>Punjab 81</td>
<td>Pakistan</td>
</tr>
<tr>
<td>Tonichi 81</td>
<td>Mexico</td>
<td>Pari 73</td>
<td>Pakistan</td>
</tr>
<tr>
<td>Genaro 81</td>
<td>Mexico</td>
<td>Lyallpur 73</td>
<td>Pakistan</td>
</tr>
<tr>
<td>Ures 81</td>
<td>Mexico</td>
<td>Koñinoor 83</td>
<td>Pakistan</td>
</tr>
<tr>
<td>Galvez 87</td>
<td>Mexico</td>
<td>Sandal 73</td>
<td>Pakistan</td>
</tr>
<tr>
<td>Trap No. 1</td>
<td>Mexico</td>
<td>Sarhad 82</td>
<td>Pakistan</td>
</tr>
<tr>
<td>Garuda&quot;S&quot;</td>
<td>Mexico</td>
<td>Zargoon 79</td>
<td>Pakistan</td>
</tr>
</tbody>
</table>

a/ Source: R.A. McIntosh. University of Sydney and R.P. Singh. CIMMYT
Advances made in durability of resistance to yellow (stripe) rust

Data on yellow (stripe) rust from the international nurseries for land race, improved tall, and semidwarf wheats indicate that there has been improvement for yellow rust in semidwarfs, but not to the extent exhibited for stem rust and leaf rust resistance (Table 7). The data indicate that local land race types are more susceptible than improved tails and semidwarfs.

In its crossing program during the mid-1960s, CIMMYT used varieties from the Andean region, which were highly resistant to yellow rust such as the Colombian variety Andes. The Californian variety Anza was bred by CIMMYT, and has been reported to be durably resistant to yellow rust by Johnson (Chapter 6). Anza was derived from the cross LR/N10B/3*AN8 and has been released in North Africa, Sudan, South Africa, and New Zealand. It is thought that the durable resistance of Anza to yellow rust is derived from the Andes; this hypothesis should be tested by genetic analysis. Although the genes conferring resistance in Anza are likely to be different from those in Cappelle Desprez and Little Joss, the effect on durability of resistance is the same. In both cases there is a need to

Table 6. Bread Wheat Regional Disease Trap Nursery (RDTN) data (average coefficient of infection) for leaf rust *(P. recondita f.sp tritici)* from approximately 50 locations in 30 countries

<table>
<thead>
<tr>
<th>Year</th>
<th>Local</th>
<th>Improved Tall</th>
<th>Semidwarf</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>1978</td>
<td>39.9</td>
<td>19.1</td>
<td>12.1</td>
<td>35</td>
</tr>
<tr>
<td>1979</td>
<td>31.1</td>
<td>11.4</td>
<td>7.1</td>
<td>13</td>
</tr>
<tr>
<td>1980</td>
<td>41.6</td>
<td>14.1</td>
<td>8.7</td>
<td>8</td>
</tr>
<tr>
<td>1981</td>
<td>28.1</td>
<td>7.8</td>
<td>6.0</td>
<td></td>
</tr>
</tbody>
</table>

Source: CIMMYT, unpublished.

Table 7. Bread Wheat Regional Disease Trap Nursery (RDTN) data (average coefficient of infection) for yellow rust *(P. striiformis)* from approximately 50 locations in 30 countries

<table>
<thead>
<tr>
<th>Year</th>
<th>Local</th>
<th>Improved Tall</th>
<th>Semidwarf</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>1978</td>
<td>11.9</td>
<td>7.9</td>
<td>9.2</td>
<td>18</td>
</tr>
<tr>
<td>1979</td>
<td>18.3</td>
<td>6.5</td>
<td>8.6</td>
<td>8</td>
</tr>
<tr>
<td>1980</td>
<td>22.4</td>
<td>9.2</td>
<td>10.0</td>
<td>9</td>
</tr>
<tr>
<td>1981</td>
<td>18.6</td>
<td>6.9</td>
<td>9.9</td>
<td></td>
</tr>
</tbody>
</table>

Source: CIMMYT, unpublished.
conduct genetic analysis to elucidate the genetic control of resistance. Durable resistance derived from the variety Anza is widely dispersed in CIMMYT advanced lines, and hot spot testing will be continued to verify the durable resistance to yellow rust in those advanced lines that have Anza in their pedigrees. CIMMYT also plans to continue collaboration with Dr. R. Stubbs of IPO, Wageningen, The Netherlands, who has a worldwide collection of yellow rust isolates in his laboratory to test CIMMYT germplasm against selected yellow rust races.

To accelerate the incorporation of a wide spectrum of yellow rust resistance genes into CIMMYT semidwarfs, a cooperative shuttle breeding program has been initiated involving CIMMYT and national programs in Kenya, Ethiopia, Ecuador, and Peru — each as an equal partner. This large international partnership venture in breeding for yellow rust should be beneficial to all the parties involved.

Breeding for Partial Resistance (Slow Rusting)

General features
Partial resistance is a manifestation of a host:parasite interaction in which infection occurs, but in which one or more steps in the infection process take place with lesser efficiency than in a susceptible host. As such, partial resistance has long been observed and utilized by potato breeders against the late blight fungus (10, 19). The first convincing case of partial resistance (slow rusting, general-type resistance) to wheat leaf rust was documented in 1968 (2).

The components of partial resistance are difficult to detect. However, the effects of partial resistance can be quantified by precise observations of the steps affected, and these data may be utilized for breeding purposes (11). Even when specific effects are not quantified, partial resistance can be detected by a marked reduction in the rate of epidemic development (smaller area under disease curve) (4, 20).

CIMMYT wheat breeders and pathologists have maintained that the crucial test for durability of resistance can only be conducted in the field over time (12). General resistance of a partial nature to stem rust in the adult plant stage was observed in a number of wheat varieties such as Yaqui 50, Bonza 55, and Penjamo 62 (12). Sartori et al. (16) showed in a genetic analysis of Penjamo 62, Hopps, and Mengavi that these varieties have general, partial resistance to stem rust, and that in Mengavi this resistance is due to a lesser receptivity to infection by the stem rust fungus. Skovmand et al. (17) in a different study found a similar situation in the variety Mengavi as reported by Sartori et al. (16).

Identification of components of partial resistance (slow rusting) at CIMMYT
As stated earlier, longer latent period, smaller pustule (uredium) size, and pustules per unit area all play strong roles in retarding disease development. Tables 8 and 9 list wheat varieties showing differences for these components of partial resistance to leaf rust. In Table 8, the varieties Juzco, Katahdin, and Favon 76 differed significantly from Inia 66 in terms of pustule size and pustule number in the seedling stage; and for pustule number in the adult plant stage.
In Table 9, the varieties Opata 85, Pavon 76, Genaro 81, Seri 82, Myna "S", and Kauz "S" are compared with the susceptible checks Morocco and Siete Cerros. The partially resistant varieties differed significantly from both checks for latent period, days to full infection, and in receptivity. It is noteworthy that this approach is similar to that described by Parlevliet (Chapter 5), i.e., selection for types of resistance characterized by a reduced rate of epidemic development.

Based on the above results, Figure 2 compares Pavon 76 and Genaro 81 to Inia 66 under field conditions. The pattern of infection rates of Pavon 76 and Genaro 81 (both partially resistant) has been shown now for 8 consecutive years. Both varieties carry the Lr13 complex (Table 10).

The procedure to identify partial resistance in the field (Figure 2) and studies of components (Tables 8 and 9) are routine activities at CIMMYT. It is believed that the studies of this nature, which have been going on for the last 17 years, have made important contributions in achieving sustainability of wheat yields on a global basis.

![Figure 2. Slow rusting resistance of Genaro 81 and Pavon 76 to leaf rust when compared to Inia 66 (Cao. Obregon 1984-85).](image)

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Seedling</th>
<th>Mature plant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Juzco</td>
<td>Pustule size (mm²)</td>
<td>0.1605a</td>
</tr>
<tr>
<td></td>
<td>No. of pustules per cm²</td>
<td>4.28a</td>
</tr>
<tr>
<td>Katahdin</td>
<td>Pustule size (mm²)</td>
<td>0.2028a</td>
</tr>
<tr>
<td></td>
<td>No. of pustules per cm²</td>
<td>7.14a</td>
</tr>
<tr>
<td>Pavon 76</td>
<td>Pustule size (mm²)</td>
<td>0.2098a</td>
</tr>
<tr>
<td></td>
<td>No. of pustules per cm²</td>
<td>9.28a</td>
</tr>
<tr>
<td>Dove</td>
<td>Pustule size (mm²)</td>
<td>0.2368b</td>
</tr>
<tr>
<td></td>
<td>No. of pustules per cm²</td>
<td>4.00a</td>
</tr>
<tr>
<td>Inia 66 (check)</td>
<td>Pustule size (mm²)</td>
<td>0.2625b</td>
</tr>
<tr>
<td></td>
<td>No. of pustules per cm²</td>
<td>20.28b</td>
</tr>
</tbody>
</table>

Source: Huerta-Espino and Rajaram (in press).

Genotypes followed by different letters are statistically different at P = 0.05 (Dunnett Test)
Breeding for Multiple Major Gene Resistance

Although the longevity of resistance based on multiple major genes (i.e., VR) may be limited and stepwise mutation can eventually lead to susceptibility, this strategy has been successfully employed in Australia for stem rust. However, given CIMMYT’s global mandate and the corresponding difficulty in deploying genes effectively in an international context, CIMMYT has pursued this as a supplementary strategy.

If multiple major gene resistance is to be pursued as a strategy, pathogenicity analysis is a prerequisite for maintaining control. The spectrum of virulence/avirulence genes for stem rust and leaf rust identified in Mexico are given in Tables 11 and 12, respectively. Resistance gene analysis can be conducted utilizing these races. When the varieties have complex combinations of genes, known or unknown, however, this method is not adequate. In that situation, continual genetic analysis is required.

Table 9. Analyses of slow rusting components such as latent period, infection period, and receptivity to leaf rust in eight varieties of wheats when tested with isolates 87.34A or 87.40B

<table>
<thead>
<tr>
<th>Isolate used</th>
<th>Days to latent period a/</th>
<th>Days to full infection a/</th>
<th>No. of pustules (10 cm²) a/</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morocco (check) 87.34A</td>
<td>5.00a</td>
<td>10.00a</td>
<td>228.9c</td>
</tr>
<tr>
<td>Morocco (check) 87.40B</td>
<td>5.00a</td>
<td>10.00a</td>
<td>204.6c</td>
</tr>
<tr>
<td>Siete Cerros (check) 87.40B</td>
<td>6.00b</td>
<td>10.00a</td>
<td>123.9d</td>
</tr>
<tr>
<td>Siete Cerros (check) 87.34A</td>
<td>6.00b</td>
<td>10.40b</td>
<td>119.8d</td>
</tr>
<tr>
<td>Opata 85 87.34A</td>
<td>7.60c</td>
<td>12.85d</td>
<td>74.0cd</td>
</tr>
<tr>
<td>Pavon 76 87.34A</td>
<td>7.65cd</td>
<td>12.00c</td>
<td>80.5cd</td>
</tr>
<tr>
<td>Genaro 81 87.40B</td>
<td>8.19d</td>
<td>14.00c</td>
<td>22.6ab</td>
</tr>
<tr>
<td>Serti 82 87.40B</td>
<td>8.80e</td>
<td>14.00c</td>
<td>8.7a</td>
</tr>
<tr>
<td>Myna“S” 87.40B</td>
<td>8.05cd</td>
<td>13.00d</td>
<td>61.1bc</td>
</tr>
<tr>
<td>Kauz“S” 87.40B</td>
<td>9.45f</td>
<td>13.00d</td>
<td>7.5a</td>
</tr>
<tr>
<td>LSD</td>
<td>0.53</td>
<td>0.19</td>
<td>47.40</td>
</tr>
</tbody>
</table>


a/ Different letters denote significant difference at P = 0.05 (Duncan’s New Multiple Range Test)
to study diversity and allelic relationships of resistant varieties. Such a study for leaf rust resistance in 10 varieties is presented in Table 13. The data indicate that these varieties possess as many as four effective genes and as many as nine different genes may be involved in conferring leaf rust resistance. Such a study on genetic diversity is important for pyramiding resistance genes and ultimately achieving multiple gene combinations.

**Revival of the Multiline Approach**

Another way to achieve stable resistance to diseases caused by obligate parasites is through the use of multiline composites as originally proposed by Jensen (8) and Borlaug (1). This means of manipulating rust resistance and the methods used by CIMMYT are discussed in greater detail elsewhere (13, 15). Multiline breeding is a very conservative and slow approach in regard to yield because newer varieties may rapidly supersede the recurrent parent. The multiline approach may offer considerable merit in maintaining yield stability, especially in areas at high-risk from disease. CIMMYT is currently in the process of generating multilines using the high-yielding varieties Seri 82 and Genaro 81 for stabilizing leaf rust resistance in the northwestern Mexican states of Sonora and Sinaloa. Resistance genes Lr9, Lr19, Lr24, and others yet to be identified are being used. Only a moderate allocation of CIMMYT resources is currently devoted to the multiline approach. However, this may be increased in the future depending on the progress with multilines and new collaborative research that is underway on varietal mixtures in conjunction with Dr. M. Wolfe at PBI in Cambridge, England.

Table 10. Genetic constitution of certain CIMMYT/INIFAP varieties released since 1976 in Mexico in relation to *P. reconditum* f.sp. *tritici*

<table>
<thead>
<tr>
<th>Cultivar</th>
<th>Year of release</th>
<th>LR genes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naecozi 76</td>
<td>1976</td>
<td>Lr10 + slow rusting genes</td>
</tr>
<tr>
<td>Pavon 76</td>
<td>1976</td>
<td>Lr11, Lr10, Lr13 + slow rusting genes</td>
</tr>
<tr>
<td>Ciano 79</td>
<td>1979</td>
<td>Lr16 + 1 gene</td>
</tr>
<tr>
<td>Tonichi 81</td>
<td>1981</td>
<td>Lr10, Lr17 + 2 adult plant genes</td>
</tr>
<tr>
<td>Genaro 81</td>
<td>1981</td>
<td>Lr3, Lr13, Lr26 + slow rusting genes</td>
</tr>
<tr>
<td>Opata 85</td>
<td>1985</td>
<td>Lr10 + 1 adult plant gene</td>
</tr>
<tr>
<td>Papago 86</td>
<td>1986</td>
<td>Lr16 + 1 gene</td>
</tr>
<tr>
<td>Cucurpe 86</td>
<td>1986</td>
<td>Lr10 + 2 adult plant genes</td>
</tr>
</tbody>
</table>

Source: R. P. Singh.
Ideal Situation: Combining Durable Resistance and Partial Resistance with Major Genes

CIMMYT's preferred scenario requires that the principal thrust must be a durable resistance expressed as partial resistance in conjunction with, major genes that confer additional security. For example the Sr2, Lr13, and Anza-type resistances are used as principal components to establish durability through partial resistance in conjunction with other genes.

The above approach has been pursued to develop resistance to leaf rust in the set of varieties listed in Table 10. The varieties Pavon 76, Ciano 79, Tonichi 81, Genaro 81, Opata 85, Papago 86, and Cucurpe 86 have shown a combination of these desirable and different types of leaf rust resistance. No leaf rust epidemic has occurred in Mexico since 1978 where the above varieties have been widely adopted. Large-scale epidemics of stem rust were thwarted wherever Sr2 plus other genes for stem rust resistance have

---

**Table 11. Virulence/avirulence combinations of *P. graminis* f.sp. *triticum* identified in Mexico during 1984-86**

|Sr5, 9a, 9d, 36/Sr7a, 7b, 8, 9b, 9c, 10, 11, 13, 24, 25, 26, 27, 30, 37|
|Sr5, 8, 9a, 9d, 36/Sr7a, 7b, 8, 9b, 9c, 10, 11, 13, 24, 25, 26, 27, 30, 37|
|Sr5, 7b, 8, 9a, 9d, 36/Sr7a, 9b, 9c, 10, 11, 13, 24, 25, 26, 27, 30, 37|
|Sr5, 9a, 9b, 9d, 36/Sr7a, 7b, 9c, 10, 11, 13, 24, 25, 26, 27, 30, 37|
|Sr5, 8, 9a, 9d, 11, 36/Sr7a, 7b, 9b, 9c, 10, 13, 24, 25, 26, 27, 30, 37|
|Sr5, 7b, 8, 9a, 9d, 11, 36/Sr7a, 7b, 9c, 10, 13, 24, 25, 26, 27, 30, 37|
|Sr5, 9, 9a, 9b, 9d, 11, 36/Sr7a, 7b, 9c, 10, 13, 24, 25, 26, 27, 30, 37|
|Sr5, 7b, 8, 9a, 9b, 9d, 11, 36/Sr7a, 9e, 10, 13, 24, 25, 26, 27, 30, 37|

Source: R. P. Singh.

---

**Table 12. Virulence/avirulence combinations of *P. recondita* f.sp. *triticum* identified in Mexico during 1984-87**

|Lr1, 3, 3b, 10, 13, 17, 27 + 31/Lr2a, 2b, 2c, 15, 23, 24, 26|
|Lr2c, 10, 17/Lr1, 2a, 2b, 3, 3b, 13, 15, 23, 24, 26, 27 + 31|
|Lr1, 3, 3b, 10, 13, 15, 23, 24, 26, 27 + 31/Lr2a, 2b, 2c, 17|
|Lr1, 2c, 10, (17), 23, 26, (27 + 31), /2a, 2b, 3, 3b, 13, 15, 24|
|Lr1, 2a, 2b, 2c, 3, 3b, 10, 13, 15, 17, 27 + 31/23, 24, 26|
|Lr1, 2c, 10, (17), /2a, 2b, 3, 3b, 13, 15, 23, 24, 26, 27 + 31|
|Lr1, 2a, 2b, 2c, 3, 3b, 13, 15, 26/10, 17, 23, 24|
|Lr1, 2a, 2b, 2c, 3, 3b, 13, 15, 23, 26, 27 + 31/10, 17, 24|
|Lr2a, 2b, 2c, 3, 3b, 10, 13, 15, 23, 27 + 31, /Lr1, 17, 24, 26|

100% virulence for genes: 14a, 14b, 18, 20, and 28

100% avirulence for genes: Lr3Ka, 9, 11, 16, 19, 21, 25, 29, 30, and 33

Genes in parentheses indicate intermediate virulence

Source: R. P. Singh.
been used: likewise, wherever $Lr13$ plus other genes for leaf rust resistance were used, large-scale epidemics of leaf rust have been prevented. When $Lr13$ has been used on its own, as in the variety Sonalika, there has been susceptibility. Where $Lr13$ has been absent, as in Mexico in 1977 with the variety Jupateco 73, there was a serious epidemic.

CIMMYT also contends that the widespread adoption of semidwarfs on more than 50 million ha worldwide is due, in part, to durable resistance against rust diseases. The high yield potential of semidwarfs would have been short-lived if stable resistance to rust diseases had not been simultaneously bred in. In the future, we must find new stable gene combinations to complement $Sr2$ and $Lr13$.

References


Table 13. Genetic relationship and genetic diversity present in 10 leaf rust-resistant parents in field conditions when crossed to susceptible variety Siete Cerros

<table>
<thead>
<tr>
<th>Variety</th>
<th>Parula “S”</th>
<th>Bow “S”</th>
<th>Tonichi “S”</th>
<th>Yaco “S”</th>
<th>Baeubuc “S”</th>
<th>Chiloero “S”</th>
<th>Junco “S”</th>
<th>Myna “S”</th>
<th>Hahn “S”</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kea “S”</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>(D)</td>
<td>(D)</td>
<td>(D)</td>
</tr>
<tr>
<td>Parula “S”</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>(D)</td>
<td>-</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Bow “S”</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>D</td>
<td>(D)</td>
<td>3</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Tonichi “S”</td>
<td>-</td>
<td>-</td>
<td>D</td>
<td>S</td>
<td>D</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Yaco “S”</td>
<td>-</td>
<td>-</td>
<td>D</td>
<td>-</td>
<td>-</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Baeubuc “S”</td>
<td>D</td>
<td>-</td>
<td>D</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Chiloero “S”</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>2</td>
<td>2</td>
<td>-</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Junco “S”</td>
<td>-</td>
<td>D</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Myna “S”</td>
<td>D</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Hahn “S”</td>
<td>-</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Source: R.P. Singh, F1, F2, and F3 data.
In body of Table: D = Completely different genetic basis, in the two varieties; (D) = Partially different genetic basis; S = Same genetic basis.


Chapter 10

Synthesis: The Strategy of Rust Resistance Breeding

N.W. Simmonds, Edinburgh School of Agriculture, Edinburgh, Scotland

Abstract

The purpose of breeding for disease resistance is to protect biomass and hence crop yield. Resistance is simply a state of "less disease"; and no disease (immunity) is rarely a realistic objective. Four kinds of resistance may usefully be recognized, namely: 1) major gene, pathotype-specific, vertical resistance (VR); 2) polygenic, pathotype-non-specific, horizontal resistance (HR); 3) pathotype-non-specific, major gene resistance (NR); and 4) interaction or mixture resistance (IR). VR is often effective against immobile pathogens, but is generally non-durable against mobile, airborne ones; continued disease control is sometimes possible, however, by the use of multiple (pyramided) VR genes deployed under tight genetical and pathological management. HR is durable and generally fairly highly heritable; most diseases are controlled thus in many crops. NR is valuable if available, but is rare. IR, due essentially to heterogeneous VR elements, is poorly understood, but probably more valuable than is yet generally realized.

Of the wheat rusts, stem rust has been well controlled worldwide for years by pyramided VR genes, but must be judged to retain potential for epidemic outbreak if tight genetic/pathological control were to lapse. Leaf rust is probably the most damaging of the three rusts at present, the genetic VR base is narrow and more epidemics must be expected. Yellow rust differs from the other two in that VR seems to have wholly failed, so that breeders in Europe have begun to abandon VR and effective HR is recognized and beginning to be exploited.

CIMMYT breeding has so far been mainly concentrated upon pyramiding VR for all three rusts, but hardly under tight genetic control. It is arguable that this is a risky strategy because small farmers in less developed countries can ill afford epidemics that farmers in rich countries could well tolerate. It is suggested that a reasonable/feasible shift of strategy would be a move towards research on and exploitation of HR and IR; in the context of stem and leaf rusts, both are greatly under-researched but must, with all reasonable certainty, have much potential. The shift from VR to HR/IR emphasis would not come rapidly but is of very great long-term potential and practical importance. A parallel shift of the grand strategy of the Center from breeding wheat varieties to strategic (not basic) research is implied and seems to be consonant with the policy of the Consultative Group for International Agricultural Research.

Introduction

This meeting was primarily concentrated upon this. The authorities in CIMMYT thought that, though nearly everyone present was an internationally-respected expert on some aspect of the cereal rusts, it would do no harm to have someone present to sum up, someone who
had rather wide experience of crops and disease resistance breeding but no specialized knowledge of the rusts. Hence my presence. The onlooker, they say, sometimes sees most of the game. I was specifically charged by the CIMMYT authorities to formulate some conclusions as to the Center’s breeding strategy. This I have done and thank my colleagues both in CIMMYT and abroad for their many comments; in the end, though, since there was rather less than total agreement, those views had better be attributed to me. But I hope that no one will feel that they are seriously unrepresentative of the general trend of discussion.

The material for this chapter largely comes from the preceding chapters and I have found it convenient to refer to them briefly thus: [5].

meaning Chapter 5 by J.E. Parlevliet. Few other references seemed necessary since we are mostly dealing with well known facts and arguments. A relevant reference to the general strategy of disease resistance breeding is (8) and a valuable compendium of papers on horizontal resistance will be found in (4). The tropical agricultural context of disease resistance is emphasized in two FAO publications (1, 2).

**General Context of Disease Resistance**

The paramount objective of all plant breeding is timely high yield in a chosen environment. True, yield as an objective is sometimes qualified by quality considerations and there are trade-offs. But, at a given quality level, then yield remains economically dominant. Yield (Y) is procured by enhancing biomass (B) (dry matter per unit area) and partition to desired product (a dimensionless fraction, P) such that

\[ Y = BP. \]

This is a perfectly general relation and the importance of plant diseases is that they act by reducing B and hence Y. Disease resistance breeding is therefore a yield-enhancing procedure that does so by protecting biomass. Disease resistance is not an objective in its own right, though it sometimes seems to be thought of thus. It matters only insofar as it protects yield. Ideally, one would like to have formal proof that disease resistance is economically worthwhile before embarking upon breeding. For the wheat rusts, of course, such proof is hardly necessary; but the point is relevant elsewhere because some diseases can look bad but do little damage.

**R. resistance** is simply a state of less disease: it is not a state of “no disease” and, if one means immunity, one should say so. **Susceptibility** is simply the complement of resistance but is not obviously bounded because a state of total susceptibility is hardly definable. Resistance scales often present difficulties at the bottom end and, as a general practical point, it is always helpful to have very susceptible standards in all experiments to help to define the lower bound.

In practice, most crop diseases are only partially controlled by one means or another, genetic, agronomic or chemical. Absolute control is rare so we generally have to live with a moderate level of any particular disease: annoying perhaps but economically tolerable. A state of **no disease** is rarely a realistic objective. Furthermore, it is not necessarily even a desirable one. The yield-disease intensity curve is rarely linear so that low, even moderate, levels of disease are often found to have effects so small as to be unmeasurable or economically trivial. Therefore “enough resistance is enough” is a good practical
maxim: how much is enough will of course, vary from place to place, according to disease intensity.

Finally, we should recall the point (that emerged several times in the meeting), that a good plant variety represents a balanced package of characters of which disease resistance is but one component. The farmer judges on a sort of weighted index and there are many examples of favored varieties that breeders or pathologists have proclaimed to be too susceptible; also of resisters that farmers did not like. Again, enough is enough, and how much that is has to be judged against that elusive quality of general worth.

Kinds of Resistance

General

Four kinds of resistance—The main features of the four broad kinds of resistance are summarized in Table 1. I shall now run through them briefly but avoid the terminological (sometimes almost theological) niceties that tend to obtrude in this area. Before doing so, it is well to point out that the list is broadly applicable to all classes of pathogen: virus, bacterial, fungal, and animal. But it would, admittedly, be hard to cite really clear cases of all the 4x4 combinations.

Table 1. The four main kinds of resistance (8, 9)

<table>
<thead>
<tr>
<th>Kind of resistance</th>
<th>Specificity</th>
<th>Genetics</th>
<th>Durability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pathotype-specific or vertical, VR/SR</td>
<td>very high</td>
<td>oligo-</td>
<td>(1) mobile pathogens, durability usually bad</td>
</tr>
<tr>
<td>2. Pathotype-non-specific major gene resistance, NR</td>
<td>nil</td>
<td>oligo-</td>
<td>(2) immobile pathogens, durability may be good</td>
</tr>
<tr>
<td>3. General or horizontal resistance, HR/GR</td>
<td>nil/low</td>
<td>poly-</td>
<td>high</td>
</tr>
<tr>
<td>4. Interaction, or mixture resistance, IR/MR</td>
<td>some</td>
<td>hetero-geneous oligo-al/</td>
<td>probably good</td>
</tr>
</tbody>
</table>

a/ Some authors [7] are inclined to attribute some weight to heterogeneity for polygenic systems, but this matter seems to me to be undecided.
First, *vertical resistance* (VR, Vanderplank's term) or *specific resistance* (SR) is due to major genes highly specific to matching pathotypes (gene-for-gene correspondence). VR genes are often dominants and often control seedling hypersensitivities but not always: semi-dominance (heterozygous expression), recessiveness and adult plant resistance are fairly frequent [1, 2]. VR genes, when looked for, are numerous, so numerous that estimates seem to be largely bounded by the patience of investigators. Given specificity and pathotype adaptability (by migration, mutation, recombination under host selection pressure [1]), VR often fails. Against highly mobile pathogens (rusts, mildews, downy mildews, many ascomycetes, some insects), failures have been very numerous but some instances of long-lived persistence of a vulnerable VR gene are known and some successes have been achieved by the use of multiple and/or sequentially deployed VR genes (see below). By contrast, against immobile (soil-inhabiting) pathogens, VR systems have often been very successful (as against potato wart, *Synchytrium*, and cyst eelworm, *Globodera rostochiensis*). Thus VR is typically (but not quite always) non-durable against mobile pathogens but may have very useful durability against immobile ones.

Second (Table 1), *horizontal resistance* of Vanderplank (HR) or, as preferred by many writers, *general resistance* (GR), or *field resistance* (not favored nowadays), or *partial resistance* (of Parlevliet [5]) are all terms that mean at least nearly the same thing, namely: a polygenic resistance that is pathotype-non-specific and acts, typically, by a rather complex mixture of inhibition of infection, long latent period, slow lesion growth, and reduced sporulation. HR is typically highly durable (4). It is the rule rather than the exception and most plants are protected thus from most diseases, a fact which often becomes apparent only by way of *new-encounter* events: a variety carried to a new place and meeting an unfamiliar disease or a new disease introduced to a population of unadapted varieties. Genetic and operational features of HR are discussed further below.

Fourth, interaction or mixture resistance (IR for the present purpose) (8) occurs when a heterogeneous pathotype population meets a heterogeneous crop and the result is a *clamping* interaction that reduces overall epidemic intensity (and see (5), (6)). The effect has been likened to the damping that seems to be characteristic of (heterogeneous) wild populations [7]. Typically, an effective IR looks like an HR epidemiologically and might be expected to be durable (though this can hardly be said to have been thoroughly tested).

**Complications**—The simplicity of Table 1 needs some qualification (Figure 1). The intersections of the figure make several useful points. First, if polygenic HR is based on
relatively few genes (which is probably often true because *poly* does not have to mean many [4, 5]), then a monogenic NR is the limiting case. Second a weak VR gene that allows some infection and sporulation (as with diverse *Sr, Lr, Yr* genes in wheat [1, 2, 4, 6]) can look very like an HR until investigated genetically and, conversely, there can be VR components of seeming HR that may be difficult to detect [6]. Third, if a VR gene remains effective for a long time and no virulent pathotype appears it will look like an NR and, indeed, had better be functionally classified as an NR even if the appropriate pathotype is known to exist but fails to multiply, for whatever (usually unknown) reason.

**Genetic features**

**Major genes**—The major genes, whether VR or NR offer no unusual features: they segregate in normal Mendelian fashion and numerous examples are available in the literature (e.g., (7)). For the wheat rusts we have an example in this volume (leaf rust, one or two genes

---

Figure 1. Relation between the four main kinds of disease resistance (see Table 1). Modified from (7).
segregating [9]) and hundreds of other such cases have been recorded. Linkages, of course, sometimes turn up [1, 2] and may be useful or not, depending on circumstances (e.g. the black chaff associated with Sr2 [1]) and the yellow flour associated with Lr19 [2]. Sometimes, the very refined cytology available in wheat permits exact allocation of genes to chromosome arms (e.g. for Yr genes [6]; and see also [2] Tables 2 and 3 for Sr and Lr genes).

**Polygenic inheritance**—Analysis of polygenic HR has, until recently, been rather little studied, as Knott [4] points out. However, with the increasing recognition of its importance in the past 20 years, studies multiply and examples demonstrating the key features will be found in [4, 7, 8, 9]. Those features are: continuous variation in segregating generations, environmental variation in expression (statistical error), necessity for biometrical rather than Mendelian analysis, evidence of a significant genetic component of variance (i.e. heritability) and, finally, the need (if starting from inbred lines at least) for four generations if the general nature of inheritance is to be unambiguously stated [see [4, 5]].

Two examples will illustrate these points. The first (Figure 2) is from durum wheat in North Dakota, USA. I have selected one cross out of three for illustration. It is between a susceptible variety A and a resistant one P. The F2 mean resistance (R) was almost exactly intermediate between the parents and the distribution continuous and very nearly normal. So far, we could be

![Figure 2](image-url)

**Figure 2.** Horizontal resistance to stem rust in durum wheat in North Dakota, USA. Ordinate f is frequency, abscissa R is resistance on a scale 0 (very susceptible) to 100 (immune). Parent A (Akrona) susceptible, parent P (Pentad) resistant. For details see the text. From ref. (10).
dealing with a semidominant gene (segregating 1:2:1) plus minor genes and error. However, the F3 was also continuously distributed, with no trace of the discontinuity that could surely have been detected had a major gene been segregating. There is no means of estimating the number of genes concerned; in general, there is no biometrically satisfactory way of doing so. As remarked above, poly does not have to mean many. In this case heritability is obviously high, as evidenced by: the calculated genetic range in F2 ($R \pm 2\sigma_G$), the top end of which is high but not transgressive of variety P; and the offspring-on-parent regression ($RF_3 = 23 + 0.66 RF_2$) which shows that excellent progress would be made by selecting among the F2. Progress by selecting among F3 lines would probably be even better.

The other example (Figure 3) refers to barley mildew and it shows broadly the same features, namely: hybrid generations with means intermediate between parents.

**Figure 3.** Horizontal resistance to mildew in spring barley in the UK. Ordinate R is resistance (Parent KU very susceptible, parent PR middling, parent GV resistant). Parental and F3 means and ranges with indications of genetic ranges. KU, Kuusamo; PR, Proctor; GV, Gloire du Vellay, From ref. (3).
continuous, more or less normal F3 distributions, evidence of wide genetic spans in the F3 and of good heritability, potential for roughly equaling the resistant parent (GV) but not transgressing it. These were UK barleys and variety PR has an HR roughly at the threshold of acceptability; clearly, GV and the better F3 lines from both crosses would be acceptable in respect of resistance.

The above examples were chosen for heuristic reasons as showing, with textbook clarity, the main features of more or less additive polygenic systems. Complications are possible of course: chosen scales may themselves be non-linear and therefore might need transformation; if appropriate scales were chosen, dominance/recessiveness would tend to produce skewness at F2 and later generations but the point about continuity still holds (see, for example [4, Figure 1]); some distributions defy any simple interpretation (e.g., [6, Table 5]), but still leave the fact of polygenic inheritance and heritability rather plain. (The data of [6, Table 5] look like additive recessive resistances from both parents giving a susceptible F1 and transgressive F2.)

**Strategies**

**General**

The general objective of all disease resistance components of plant breeding programs must be to provide adequate levels of resistance that are reliable over the years. If varieties are individually expected to survive in cultivation for many years, the need for durability is implied and this would suggest the need for HR or, perhaps, IR; if immobile pathogens were concerned, then reasonable durability could sometimes be provided by VR. For mobile pathogens, VR would sometimes simply be a foolish choice, as against leaf diseases of evergreen perennial crops in the wet tropics (2). Even for annual crops, VR is, in general a risky choice, if durability, in the sense of long survival of specific genotypes, is the object. The accumulation of several VR genes together (pyramiding) has sometimes been useful but often it has not (as in potatoes against blight, in rice against blast, and in other inbred cereals against rusts (e.g., [6]). It may take several years for a new virulent pathotype to appear but it is prudent to assume that it will appear, however many VR genes have been accumulated, and that it will do so sooner rather than later.

Thus VR per se, even pyramided, cannot be expected to provide durability but it may still be operationally fairly reliable if deployed effectively over time, new pathotypes being anticipated by appropriately resistant varieties. To do this implies a high degree of pathological and genetical understanding and control, as is evidently available for wheat rusts in Australia [1], but is by no means universally feasible. One should note that, although skillful deployment of VR genes can, in favorable circumstances, give long-continued protection of the crop at large, this situation cannot be described as an example of durability.

VR in single, specific genotypes remains, in general, non-durable. VR alone thus has both uses and limitations. If its use has to be abandoned or supplemented or if, as is true of many host-pathogen combinations, it is simply not available, what remains? The primary recourse is to HR and, as remarked above, this is the means by which, in fact, most minor plant
diseases are kept down to minor status and many major ones are controlled well enough. Despite much speculation to the contrary, there is no clear evidence of significant erosion of polygenic HR, that is, of pathogenic adaptation or increase in aggressiveness. There are innumerable examples of varieties that continue at an unchanged moderate to good level of resistance for decades, thus satisfying Johnson’s [6] practical criterion of durability, namely survival. Polygenic HR is a perfectly general source of durability that VR per se cannot offer (4, 7, 8, 9).

IR/MR [7, 8] remains an attractive possibility which is yet too little investigated to allow judgment as to general utility. Obviously, it offers a chance of using VR genes which would otherwise be useless or, at best, of transient utility. But better understanding is essential with particular reference to the following features (5, 6): optimal (or necessary) numbers of resistance genes; their disposition between and within component lines; the relative importance of VR and HR among components; durability; and agricultural features, such as seed supply. Whatever the problems, though, the prospects are exciting.

If durable resistance be the object, therefore, the general strategy is fairly clear: prefer polygenic HR as the basic approach and use VR per se only if (as against immobile pathogens) there is a clear prospect of durability. Consider the development of populations that exploit IR/MR as a supplement to a basic HR. For airborne pathogens, this means, in effect, that HR is fundamental and that VR should be avoided or deployed only under rigorous scientific control or exploited as an element of IR/MR. (Particular

Features of VR/SR breeding—A program committed to this approach would have to accept the risks incurred. That these risks are real in respect of the rusts of inbred cereals is abundantly documented in several chapters in this book [e.g., [1] for stem rust of triticale in Australia, [6] for yellow rust in Europe, [7] for recent leaf rust outbreaks in Texas]. The breeder would try to pyramid VR genes, recognizing the need for exact genetic control of host genes and good knowledge of pathotypes if this were to be done efficiently and safely [1, 2]. He would, further, try to enhance the genetic background against which the VR genes work, though this is, in general, a difficult, sometimes virtually impossible task [5, 6]. The breeder would make use of a battery of methods, including shuttle breeding selection, multilocational testing, hot spots, glasshouse tests, and so forth but he would recognize that, in the limit, none of these can guarantee prolonged survival of the products. Further, the breeder would hope to impose some discipline upon the users of his products, avoiding, above all, the deployment one-at-a-time of VR genes which might be much better pyramided (see example in [3]). Finally, the breeder committed to VR/SR would have to accept the idea of a potentially rapid turnover of varieties, sometimes under great pressure, and, along with this, the idea that a large part of his resources would have to be devoted to disease resistance, working hard to keep ahead of the pathogen’s evolution. (Features of HR/GR breeding—A program committed to this approach would, in general, try to get rid of VR genes in parental stocks or, if this were impossible in the short
term (which is likely), the breeder would take care to use appropriate virulent pathotypes for testing (as potato breeders have been doing for many years; see also examples in this book [4, 5, 6]). The breeder would accept slowness of progress relative to VR in the initial stages but he would recall that the heritability of HR usually turns out, when studied, to be high, even very high, so progress would be faster than many workers might imagine. High heritability is documented in (4, 7, 9), in Figures 2 and 3 above, and in the striking selection experiment on barley leaf rust described here by Parlevliet [5]. One recalls also that maize breeders have long recognized the high heritability of HR to the leaf diseases (rusts and Helminthosporium) with which they have to deal. Once several cycles of HR have been accomplished, the disease resistance problem declines, because parents with fair HR and more or less additive genetic variance jointly ensure good average levels of HR in progeny. In the absence of VR (or with its nullification by the use of virulent tester-pathotypes) breeding then simply becomes a matter of throwing away the worst in each generation, as emphasized by Parlevliet [5] and as has long been known to sugarcane breeders (2). Thus the longer term condition tends to stability, with only small resources being devoted to resistance per se. This is not the least attraction of a well-established HR system.

The breeder exploiting HR would have to accept the idea of enough resistance rather than immunity as the objective and his farmer-customers would usually have to accept the presence of at least some disease as being normal. In practice this is true of very numerous diseases and too much emphasis can, I believe, be laid on the farmer’s desire for an absolutely clean crop. Immunity is very rare. The breeder would generally have to be aware of the need for adaptation to varying local levels of disease, of the use (and misuse) of hot spots (it is all too easy to over-select), and of interactions between neighboring plantings such that variation of disease scores between genotypes tends to be diminished, the extremes of susceptibility and resistance being damped. But errors tend to be enhanced, thus depressing heritabilities.

Features of IR/MR breeding—
Little strategic thinking about this subject is yet possible because our knowledge is but rudimentary, relating mainly to oat multilines in the USA and barley mixtures in England [7, 8]. The very idea of non-uniformity of crop varieties, though not new, has only recently begun to gain wide acceptance, however sensible it might seem biologically [7]. Superficially, then, it looks as though the breeder of multilines would have to have access to diverse stocks at a good level of field performance, carrying diverse VR genes. He would have to accept that the backcrossing programs to form constituent lines would take time, might have to be extended to use new VR genes, and that multilines would therefore be liable to be overtaken in yield potential by later pure lines. He would also have to accept that there might be some (maybe not great) problems of maintenance and seed supply.

The breeder exploiting variety mixtures [8] would face somewhat different, and perhaps slightly lesser, problems. He would have to have access to good varieties carrying diverse VR genes, so such a program
would fit naturally into a program that already had a large stock of failed VR material (as for barley/mildew and wheat/yellow rust combinations in Europe [6, 8]). The breeder would have to ensure that constituents of mixtures were compatible as to maturity and quality (though the latter sometimes might not matter), would face heavy experimentation in choosing good combinations, and would have to work out maintenance and seed production methods (which are yet unclear).

In general, IR/IR looks particularly attractive as a means of exploiting VR genes that have already lost their effectiveness; thus it would help greatly in effecting the transition from a VR system to an IIR one. But further speculation is vain. The fundamental requirement is for better understanding (5, 6), for serious research, especially in agriculture at low latitudes and particularly in regard to durability. If one had to guess as to a choice between mixtures and multilines, I suspect (with Browning [7]) that the former might be preferred because: first, diverse mixtures could often be composed straight out of a breeding program without the necessity of much laborious backcrossing; second, there might well be merit in heterogeneity per se besides the disease control element; and, third, there could sometimes be opportunity to control two or more diseases simultaneously [8]. Potentially a feature of great importance with the wheat rusts. In connection with the second point above, one notes that, in Wolfe's work with barley [8], the proportion of the average yield gain of 8% that is to be attributed to mildew control is undeterminable (though possibly large).

**Wheat Rust Context**

In this section I attempt to summarize, from the information presented in preceding chapters, what I take to be the main features of the three wheat rusts in relation to their control by breeding.

**Stem (black) rust (P. graminis f.sp. tritici)**

Ecologically, stem rust is characteristic of areas where wheat matures in hot, dry conditions. It has, on occasion, been devastating but, in the last few decades, epidemics have been pretty well controlled (sometimes even averted [1]) by the systematic use of VR (Sr) genes built up over many years by a huge body of research in many countries. STR is probably the best researched of all plant diseases. Many of the 30-40 Sr genes known have failed, some of them more or less immediately, but a few have lasted longer. Sr2, isolated decades ago in Hope from an emmer cross, seems to be exceptional in having persisted usefully for a very long time (i.e. seems to be durable [1, 2]; it has an incomplete adult plant resistance type of reaction and is very widely spread (e.g., in CIMMYT wheats [9]). It has something of an NR about it and is presumably not a single locus but a linked block. It is normally not strong enough by itself but is valuable in a pyramided background. Of the other Sr genes, Sr24, 26, 30, 31, and 36 have been more than averagely useful [1, 2]. Many Sr genes are exotic, having been transferred from eight species other than T. aestivum [2], a sign of the superb cytogenetic control possible in wheat, unmatched in any other crop.

The multiple Sr system has worked pretty well, by virtue of excellent genetics and critical pathotype surveys especially well developed in
Australia, even to the point of identifying the next pathotype before it happened [1]. This is far from a random assortment of pyramided VR genes. Surveys show that multiple pathogenities are common in rust isolates, for example up to eight in Mexico in 1984-86 [9, Table 11]. Mere pyramiding, of itself, is clearly helpless in only the slightly longer run against stem rust (and indeed for the other two rusts, as well). That seemingly good resistances can go through extensive international testing successfully and still fall to an unexpected new pathogenicity is evident from recent Australian experience with Sr27 in triticale [1]. There is no evident reason to me why the same should not happen to durum and bread wheats and more such episodes must surely be expected.

The question for the future is whether continuation of the historically pretty successful VR program will be appropriate. Will the breeders run out of Sr genes? Or can one foresee an indefinite continuation of the current procedure, adding new Sr genes (often from alien sources) successively, as dictated by pathotype surveys? An alternative, not yet seriously entertained, I think, would be to initiate a move towards the use of HR. This would involve a major re-orientation but might develop into an important provision for the future. HR against stem rust is not yet deeply researched but there is clear evidence that it can be developed [4] and also a strong a priori presumption to that effect.

**Leaf (brown) rust**

*P. recondita*

Leaf rust is a disease of warm rather than hot places, worldwide in distribution, and generally judged to be, nowadays, economically the most important of the three rusts [2]. Some 30-40 *Lr* genes are known; they resemble the *Sr* genes in coming from diverse sources (seven species other than *T. aestivum* and three genera other than *Triticum* [2]). Pathotypic complexity seems to be as great as for stem rust [2, 9]. Of the *Lr* genes in use, *Lr13* associated with several other elements (especially *Lr34*) is prominent [9] and *Lr19* is is thought by some workers (but not all) to be promising [2], though not yet much exploited. The CIMMYT workers tend to regard *Lr13* in much the same light as *Sr2*, having something of an NR about it. The control exercised by breeding appears to be less securely established than for stem rust and the longer term durability of pyramided *Lr* genes must be regarded as dubious. It is unfortunate that this meeting accorded relatively little attention to the disease despite its importance and despite the fact that there is a good deal of information available about it, especially from North America (McIntosh, pers. comm.).

**Yellow (stripe) rust**

*P. striiformis*

This disease is characteristic of cooler places than the other two rusts. It is worldwide in temperate latitudes and at high altitudes in the tropics, but it only reached Australia in 1979 [3]. Somewhat fewer VR genes are known than for the other rusts; *Yr1-15* have been identified [6] but no doubt many more could be picked up or brought in from alien sources if required. The *Yr* genes have a long history of failure [3, 6] and no signs of either durability or sustained control by pyramiding are apparent [6]. The race structure is complex and, as for all the rusts, constantly shifting; it is repeatedly apparent that the new pathotypes are evoked by the *Yr* genes in the cultivars grown [3].
So complete has been the failure of VR breeding against yellow rust that serious attention has been paid to alternatives. The historical evidence in favor of durable resistance in wheats (mostly but not exclusively European) is set out by Johnson [6] and is, in my opinion, persuasive. The genetic nature is not well established but a substantial HR element seems clear, probably accompanied by miscellaneous residual effects connected with major genes. Some of the durable resistance occurs in European land race materials which would not normally be favored by breeders as parental material. Johnson [6] makes valuable observations on simple breeding strategies whereby such resistance could be built up into a form usable in modern breeding programs. He also makes suggestions as to how useless (or nuisance) VR genes can be got rid of or nullified, the better to exploit HR (see also [5]). Broadly, the prospect for practical levels of HR routinely incorporated in excellent varieties seem good.

An analogous situation holds in barley in relation to its leaf rust. Parlevliet's [5] observation of durable HR in established cultivars and of an outstandingly good response to joint selection for resistance and yield ([5], Table 3) points the way to managing the disease in barley and neatly complements Johnson's [6] observations on exploiting HR in otherwise unexciting parents.

**CIMMYT Rust Programs**

**Outline of practice**

Based on Chapter 9 and references therein, the bulk of the CIMMYT effort is founded upon pyramiding major genes (whether hyper-sensitivities or adult plant resistances) rather few of which are strictly identified by reference to pathogenicity tests on standard fungal isolates. The procedures used are essentially shuttles in Mexico (Toluca-Obregón, with special sites also for leaf rust), international shuttles, and multilocational testing of the more advanced stocks. So far as they are available, mixtures of complex pathotypes and spreader rows are used.

Some race surveys are done in Mexico for stem and leaf rusts (see [9] Tables 11 and 12) and considerable genetic complexity of the fungi is thereby revealed: but, as emerged in discussion, there is not, and cannot be, any guarantee that either shuttling or multilocation testing will always reveal all the (sometimes critical) specific pathogenities. The selection of materials on the basis of low average coefficients of infection and the absence of high average coefficients of infection, but with little specific knowledge of host genetics or pathogen specificities, means that the pyramiding process is essentially random.

Of the three rusts, leaf rust currently receives the most weight: yellow rust is a relatively recent addition to the program [9]: and the stem rust situation at present seems to be fairly stable [1]. At least a partial reason for the last point seems to be that many CIMMYT materials carry Sr2 and Lr13 which, unlike most of the other genes with which we are concerned, appear to have something of the character of an NR (see stem rust under the "Wheat Rust Context" section above) and work well with other components. As to the recent history of durability, one recalls that, in Australia and the USA, really damaging outbreaks of stem rust have largely been prevented in the past 30 years (but sometimes fairly narrowly [1]) by timely genetic analysis of wheat
varieties and of fungal races, followed by appropriate varietal substitution. There was, however, an outbreak in southern Australia in 1973 due, not to failure of VR, but simply to growing a susceptible cultivar (McIntosh, pers. comm.). On the whole, control has been rather good, but the recent [1] experience in Australia of triticale susceptible to stem rust with pathogenicity for Sr27 should surely serve as a reminder that much may depend upon unforeseen (usually unforeseeable) vulnerability of major genes (VR). It is by no means clear (to me, at least) that pyramided VR to stem rust should generate any confidence in durability, as distinct from some years survival followed by enforced varietal replacement.

As to leaf rust, this is clearly CIMMYT's greatest current preoccupation. As I read the literature and heard the discussions, resistance seems to depend much upon combinations of Lr13 with other genes (e.g., Lr34 [2]). The cultivars Ciano 79, Tonichi, Pavon 76, and Genaro 81 which have been standing up well to leaf rust [9] depend upon the above in part and in part upon other genes such as Lr16 and Lr26 (McIntosh, Roells, pers. comm.). The potential of Lr19 may be considerable [2], but it carries an undesirable association with yellow flour color. As to yellow rust, epidemics since the mid 1970s in wheats in California and Pakistan and in Andean barleys serve as reminders that this rust seems to be as adaptable to host VR as the others, a repeated experience elsewhere, as described by Stubbs [3] and Johnson [6].

As a complement to the pyramiding process, the CIMMYT program also seeks slow rusting (termed dilatory resistance) of a major gene (adult plant resistance) character mentioned in Chapters 1 and 2. Thus Pavon 76 and Genaro 81 have at least some VR genes between them [9, Table 10] and also slow-rusting components [9, Figure 2]. Some effort also goes into attempts to backcross VR hypersensitive genes into a slow-rusting (dilatory resistance) background.

There has, in the past, been some effort to construct multilines (e.g., materials based on backcrosses of Lr genes into 8156 derivatives such as Siete Cerros) and the work continues with the present use of Seri 82 and Genaro 81 as recurrent parents [9].

As to leaf rust, this is clearly CIMMYT's greatest current preoccupation. As I read the literature and heard the discussions, resistance seems to depend much upon combinations of Lr13 with other genes (e.g., Lr34 [2]). The cultivars Ciano 79, Tonichi, Pavon 76, and Genaro 81 which have been standing up well to leaf rust [9] depend upon the above in part and in part upon other genes such as Lr16 and Lr26 (McIntosh, Roells, pers. comm.). The potential of Lr19 may be considerable [2], but it carries an undesirable association with yellow flour color. As to yellow rust, epidemics since the mid 1970s in wheats in California and Pakistan and in Andean barleys serve as reminders that this rust seems to be as adaptable to host VR as the others, a repeated experience elsewhere, as described by Stubbs [3] and Johnson [6].

As a complement to the pyramiding process, the CIMMYT program also seeks slow rusting (termed dilatory resistance) of a major gene (adult plant resistance) character mentioned in Chapters 1 and 2. Thus Pavon 76 and Genaro 81 have at least some VR genes between them [9, Table 10] and also slow-rusting components [9, Figure 2]. Some effort also goes into attempts to backcross VR hypersensitive genes into a slow-rusting (dilatory resistance) background.

Alien sources of resistance genes are also being exploited, as they have been very extensively exploited in the past [2, Tables 5 and 6]. There is no reason to think [6] that they will provide genes that confer resistance any more durable than usual VR.

Observations

Social context—It would generally be conceded that, for small farmers in the Third World, and even for many not so-small ones, not only high yields but yields stable over seasons are important. Occasional disasters can be very damaging. Epidemics that the farmers of a developed country could bear or could afford to control by spraying could be economically hurtful, even disastrous, to the small farmer. Furthermore, in developed countries, an unforeseen epidemic can often be quickly met by variety substitution in the next season, so that the effect is transient. In the Third World, there can but rarely be any
guarantee that resistant successor varieties and the means to distribute them will be available quickly enough. In general, by intense scientific effort and much expenditure, the developed countries have kept the wheat rusts under fair to good control and when things have gone wrong (as they not infrequently have) rich agricultures could bear the losses. It is otherwise in the Third World; the same intensity of scientific effort is not there and the socioeconomic consequences of failure are worse. One must conclude, I think, that deliberate attention to long-term stability of disease control is well justified.

**Technical features**—Broadly, CIMMYT breeding produces a good flow of high-yielding wheats that are responsive to high inputs, well liked by growers, and reasonably rust-resistant at the time of release. However, the resistance breeding strategy has heretofore rested on pyramided VR genes with little genetic control of what genes are used, limited pathotype information, and therefore little chance of anticipating epidemics in the manner sometimes possible in, say, Australia [1]. Efforts to introduce more durable resistance also rest, as we have seen above, on the use of adult plant resistance-type major genes which, it would be prudent to assume, are likely to be as much pathotype-specific in effect as typical seedling hypersensitivities [1, 2]. One has to conclude, I think, that risks of unpredicted, indeed unpredictable, epidemics are real. Some, not so far very damaging ones, have happened in the past and more must be assumed possible in the future.

Of the strategies that might promote longer term stability of crop performance by promoting durability of resistance, one (IR/MR) is receiving some attention and the other (HR) apparently none.

**Conclusions**—I conclude that, in the interests of stability of cropping, some shift of emphasis would be desirable. The following elements are apparent:

1. Commit a substantial effort to building up polygenic HR in a proportion of breeding stocks by deliberately discarding major genes, by using appropriate land race materials [5, 6], and by testing with virulent races. These methods have worked in diverse crops (4) and are already being applied to wheat in relation to yellow rust [6]. The products would have to be worked up on a broad genetic base, under enhanced recombination, to a level of performance at which they would be acceptable as parents; that good progress is indeed possible without prohibitive labor is well known here by Parlevliet’s [5] experiments with barley. Wheat could be more difficult because of the complex background of ill-defined VR genes but cannot be impossible to handle, as indicated by Knott’s preliminary results [4].

2. Test the potential of IR/MR for controlling wheat rusts at low latitudes. Since some work is already in hand, little is implied here beyond an enhanced effort and a very deliberate effort to determine what has long been the subject of speculation but not yet of test, namely durability of resistance. Much research on this, on principles of construction of multilines and mixtures and on their deployment and maintenance is needed.
(3) Strengthen the genetic control exercised over the VR genes because this would be valuable in three respects, namely: in enabling more critical deployment of VR genes in relation to pathotypes during the period (which must extend over a good many years) in which the program is still dependent upon pyramided VR; in assisting in the elimination or nullification of unwanted VR genes in the HR programs; and in assisting in the construction of populations that exploit VR genes in multiline or mixtures.

In summary, the conclusions are that there should be a move away from the exploitation of pyramided VR towards research on the development of HR and IR/MR. In the long term, it would be socially favorable but, so great is the commitment worldwide to pyramided VR in wheat, that the shift could not come rapidly; there is therefore all the more reason to start soon.

Wider Context

The general style of the CIMMYT wheat program has so far been intensely practical and, overwhelmingly, the biggest output has been a stream of excellent new wheat varieties, having, in total, a huge practical impact. A strong advance in underlying yield potential having thus been secured, I believe that the time is ripe for a shift towards enhanced understanding of wheat breeding strategies, including, of course, a substantial element relating to stable disease resistance. The conclusions outlined on this point in the preceding section might seem far-reaching but are not in fact at all remote from current CIMMYT pre-occupations [9] with the importance of stability of resistance, the need for HR, and the exploitation and study of IR.

If these arguments be accepted, a considerable shift of emphasis, a moving of resources from practical breeding towards strategic research, would be implied: strategic research, not basic because no agricultural research can ever sensibly be described as basic.

Two implications follow (Figure 4). First, the practical breeding effort, the flow of varieties, would decline as resources shifted, leaving the national systems (Figure 4) with greater local responsibilities for domestic progress. This has long been foreseen by the international centers as a whole as a natural long-term progression. As national programs develop, the Centers' responsibilities would move, according to conventional doctrine, away from short-term practical endeavors towards becoming centers of scientific and training excellence, supportive of the national programs' activities. My suggestion would thus seem to be concordant with official doctrine. Second, the scientific tasks are substantial and it cannot be expected that even a great institute such as CIMMYT could cover the relevant field of strategic research on its own. CIMMYT already has numerous collaborative research projects with laboratories in developed countries. There must be very many scientists and skills in those laboratories that could be adapted to CIMMYT's emergent strategic research needs. Lest I be misunderstood here, I should emphasize that I am not talking about molecular biology or genetic engineering which is yet, I believe, irrelevant to plant breeding, whatever the long-term promise might be. The research, however, could well have biotechnological components since this field is starting to throw up powerful diagnostic techniques of great potential for routine applications.
The conclusion is plain: adapt the existing network quite explicitly to supporting and enhancing CIMMYT's in-house research program (Figure 4). At present, projects often come to CIMMYT from outside; all that is implied here is that, more often than heretofore, CIMMYT identifies an area needing study and goes out to find and use the expertise it needs in a laboratory overseas.

Figure 4. CIMMYT: The general context of breeding and research programs.
The change proposed is not, in fact, profound. Rather it represents a shift of emphasis, as Figure 4 makes clear. I am confident that, in the longer run, such a shift would benefit, not only the customers, wheat growers, and consumers, but also CIMMYT itself, adding scientific luster to a name already famous for great practical achievements.

References


Capítulo 1

La función de genes específicos en el mejoramiento para obtener en el trigo y el triticale resistencia durable a la roya del tallo

R.A. McIntosh, Instituto de Fitogenética, Castle Hill, Australia

Resumen

Han tenido éxito los intentos fitotécnicos para obtener resistencia a la roya. Los tipos de resistencia logrados en la agricultura han dependido de genes identificables únicos o de combinaciones de esos genes. El gen de resistencia en la planta adulta, Sr2, ha contribuido a la obtención de una resistencia durable en muchas zonas. Los trigos resistentes a la roya del tallo para las zonas de cultivo del cereal en el nordeste de Australia se han basado en el empleo de tipos de resistencia que se reemplazan después de detectar patógenos virulentos. Un manejo tal de genes requiere encuestas sobre la patogenicidad, el conocimiento de los genes presentes en las variedades de trigo y la cooperación de la agricultura para un reemplazo rápido de las variedades. La vulnerabilidad genética a la roya del tallo en el programa de triticale del CIMMYT se podría reducir utilizando información obtenida en Australia. La estrecha base genética de la resistencia se podría ampliar recurriendo a variedades europeas de triticale, centeno y trigo. No obstante, es preciso conservar la diversidad genética entre el trigo y el triticale.

Capítulo 2

Resistencia a las royas de la hoja y del tallo en el trigo

A.P. Roelfs, Laboratorio de Royas de los Cereales, Servicio de Investigaciones del Departamento de Agricultura de los Estados Unidos y Universidad de Minnesota, St. Paul, Minnesota

Resumen

Si bien las royas de los cereales han logrado dominar un gran número de las variedades resistentes obtenidas en los últimos 80 años, muchas otras variedades se han cultivado con éxito en grandes extensiones de tierra. Se ha combatido la roya del tallo usando combinaciones de resistencia que incluyen el gen Sr2 transferido por McFadden de la escanda a las variedades Hope y H-44 en 1923. Las resistencias conferidas por Sr26 (proveniente de Agropyron elongatum), Sr31 (de Secale cereale) y Sr36 (de Triticum timopheevii) parecen ser las resistencias causadas por un solo gen más eficaces en todo el mundo. La variedad Thatcher (con resistencia proveniente de T. durum), obtenida por Hayes et al. en 1934, tiene también un grado adecuado de resistencia en la mayoría de las zonas. Se ha combatido con éxito la roya de la hoja mediante una combinación de los genes Lr13 y 34. Se usaron por primera vez esto tipo de resistencia en las variedades Frontana (Brasil, 1934) y Americano 44D (Uruguay, 1918). Se continúa utilizando esta combinación de genes en variedades durables recientes como Chris, Era, Clano 67, Pavón 76, etc. Las suposiciones acerca de la genética y la durabilidad de algunos tipos de resistencia han obstaculizado la selección y obtención de variedades resistentes.
Capítulo 3

Análisis de la patogenicidad de la roya amarilla (lineal) del trigo y su importancia en el contexto mundial

R.W. Stubbs, Instituto de Investigaciones para la Protección de las Plantas, Wageningen, Países Bajos

Resumen
El Instituto de Investigaciones para la Protección de las Plantas (IPO) estudia a nivel internacional la roya amarilla (lineal) del trigo (Puccinia striiformis Westend. f.sp. tritici). En condiciones controladas, las razas (virulencias) se identifican en las plantulas de un amplio conjunto de variedades diferenciales "antiguas" y "nuevas" con ciertos genes de resistencia, algunos conocidos, pero en su mayoría desconocidos. En viveros para observar las razas (parcelas separadas del campo), se analiza la virulencia vinculada con la resistencia de la planta adulta, específica para cada raza. Aunque todavía no se ha estudiado suficientemente, es evidente la relación entre la distribución de la virulencia de los agentes patógenos y los grados de resistencia de los huéspedes. Se presentan los resultados de un estudio de la roya amarilla que infecta los triteles y variedades con resistencia derivada del centeno y se describe la distribución por zonas de las razas de roya amarilla en Europa, África, Asia y América del Sur. Se recomienda investigar en forma continua las modificaciones de la patogenicidad en las poblaciones de las razas con el propósito de mejorar la resistencia y evaluarla en huéspedes en distintos medios.

Capítulo 4

Empleo de la resistencia poligénica para mejorar la resistencia a la roya del tallo en el trigo

D.R. Knott, Departamento de Ciencia de los Cultivos y Fitoecología, Universidad de Saskatchewan, Saskatoon, Canadá

Resumen
Desde hace muchos años se conoce la resistencia multigénica a las royas del tallo. Se ha postulado la existencia de una resistencia no específica a la enfermedad, pero es difícil demostrarla. Varios genes que producen pequeños efectos a menudo determinan la resistencia parcial y la resistencia dilatatoria a la enfermedad, que a veces se consideran no específicas. En investigaciones realizadas en Saskatoon, se obtuvieron líneas de trigo que como plantulas carecían de resistencia a la raza 15B-1, pero que mostraron buena resistencia a la misma raza en el campo. Se comprobó que su resistencia estaba determinada por tres a cinco genes recesivos, cada uno de los cuales tenía un efecto pequeño. Los genes reducían el periodo de latencia y el número y tamaño de las pustulas. Es probable que la resistencia determinada por varios genes de efectos pequeños sea relativamente duradera, sin importar que esa resistencia sea o no específica. Aunque es difícil su empleo, la resistencia poligénica podría ser de gran utilidad en los programas de fitomejoramiento de trigo.
Resumen
En los cereales, toda resistencia a las royas que los afectan es de tipo específico para la especie, es decir, la resistencia es eficaz sólo en relación con una especie de raya. Se pueden distinguir dos tipos de resistencia específica para la especie contra cada agente patógeno de la raya: 1) un tipo hipersensible de resistencia, determinada por genes mayores, que se caracteriza por bajos niveles de infección, la especificidad para una raza y la falta de durabilidad; ii) un tipo cuantitativo de resistencia (resistencia parcial), caracterizado por una tasa reducida de acumulación epidémica a pesar de ser susceptible y presentar un alto nivel de infección, la ausencia de grandes efectos específicos para la raza (si bien se producen efectos pequeños) y la durabilidad. Cuando no intervienen genes mayores, es fácil la selección para obtener resistencia parcial. Aun una selección poco rigurosa para eliminar la sensibilidad resulta, cuando se aplica sistemáticamente, muy eficaz para acumular genes que determinan la resistencia parcial. Esta selección poco rigurosa permite al fitogenetista obtener al mismo tiempo otras características. Cuando se pretende aumentar la resistencia parcial en presencia de genes mayores que no han sido neutralizados por completo por el agente patógeno, la eficacia de la selección es considerablemente menor. Siempre que sea posible, el fitogenetista debe exponer la población huésped a una sola raza del patógeno, una raza que neutralice una cantidad máxima de genes mayores. En cada etapa de la selección, el fitogenetista debe eliminar de esa población huésped los genotipos más sensibles y también aquellos que presenten un bajo nivel de infección. Cuando es demasiado difícil clasificar de manera confiable los niveles de infección, el fitogenetista debe eliminar los genotipos más resistentes junto con los más susceptibles, ya que se supone que los primeros son portadores de genes mayores. En algunos casos, no es posible controlar la población patógena a la que está expuesto el huésped porque está constituida por una mezcla de razas. En estas circunstancias es muy difícil la selección para obtener resistencia parcial. La eliminación continua de las líneas más susceptibles y de las que casi no resultan afectadas favorecerá la resistencia parcial, pero el progreso puede ser más lento de lo esperado.
Capítulo 6

Resistencia durable a la roya amarilla (lineal) en el trigo y sus repercusiones en la fitogenética

R. Johnson, Instituto de Fitogenética, Cambridge, Inglaterra

Resumen
La roya amarilla (lineal), causada por el parásito obligado Puccinia striiformis, se encuentra dondequiera que se cultive el trigo en climas frescos. Se han identificado varios genes de resistencia específica para cada raza, eficaces en las plántulas de trigo, pero aún resta identificar otros. Ésos genes de resistencia pueden ser dominantes o recesivos y tanto el medio como el fondo genético influyen mucho en la expresión de algunos de ellos. La resistencia que se desarrolla después de la etapa de plántula a menudo es también específica para la raza. La combinación de genes de resistencia específicos para cada raza no ha tenido éxito como método para combatir la roya amarilla en Gran Bretaña. No obstante, a veces la resistencia desarrollada después de la etapa de plántula no muestra especificidad para las razas incluso después de pruebas prolongadas en diversas áreas. Esa resistencia durable sólo puede distinguirse de la resistencia de las plantas adultas específica para la raza mediante pruebas prolongadas. Si bien esa resistencia puede estar sometida a un control genético complejo, se puede usar en programas de mejoramiento como los descritos en este capítulo: sin embargo, no es posible garantizar la durabilidad de la resistencia producida en tales programas. En consecuencia, es preciso vigilar todas las variedades resistentes nuevas, cualquiera que sea el método de mejoramiento empleado, para detectar la existencia de razas patógenas con patogenicidad equivalente.

Capítulo 7

Ideas actuales sobre el empleo de la diversidad para proteger los cereales de agentes patógenos foliares muy epidémicos y variables: Problemas y perspectivas para el futuro

J.A. Browning, Departamento de Fitopatología y Microbiología, Estación Agrícola Experimental de Texas, College Station, Texas

Resumen
Las poblaciones autóctonas de progenitores de cereales y sus parásitos obligados, que han evolucionado paralelamente, son estabilizadas por la resistencia dilatoria epidemiológica, producida genéticamente por la resistencia general que incluye genes mayores de resistencia específica en distintas configuraciones génicoespaciales. La resistencia general protege a la
Capítulo 8

Uso de mezclas varietales para combatir enfermedades y estabilizar el rendimiento

M.S. Wolfe, Instituto de Fitogenética. Cambridge, Inglaterra

Resumen

Se describen brevemente los principales factores que conducen a la pérdida de eficacia de la resistencia a las enfermedades y de los fungicidas en la agricultura europea actual. Se analiza con cierto detalle el empleo de mezclas varietales, una de las opciones de c. se disponen el fitogenetista y el agricultor para mejorar la situación. Numerosos datos obtenidos en ensayos sobre el terreno revelan las ventajas que este sistema sencillo aporta al control de las enfermedades y al aumento y estabilidad del rendimiento; dicho sistema puede sumarse a cualquier otro método de lucha contra las enfermedades.

planta; la resistencia específica protege a la población al minimizar la agresividad del agente patógeno ajustando la delicadamente armonizada interacción de realimentación entre los sistemas genéticos del huésped y el agente patógeno, muy vinculados entre sí. Tanto la resistencia general como la específica deben usarse en sistemas agrícolas coherentes con sus orígenes evolutivos. No obstante, con frecuencia la agricultura ha dependido de los genes de resistencia específica no para proteger la población, como ocurre en la naturaleza, sino para defender la planta de patógenos foliares muy epidémicos, como ha sucedido cuando se empleó el gen de resistencia en variedades cultivadas en poblaciones homogéneas en zonas extensas. Por el contrario, en programas realizados en Colombia, Inglaterra, los Países Bajos, la India y los Estados Unidos (Iowa y Washington), se han incorporado genes de resistencia específica en poblaciones diversas y se ha logrado la protección de la población con una resistencia de escasamente un tercio. Esta pequeña resistencia ha estabilizado las poblaciones en un ecosistema natural variado y, por consiguiente, la cifra parece real. Los datos corroborativos obtenidos en ecosistemas naturales y agrícolas indican que un sistema de manejo global de los genes que incluya la diversidad, como sucede en la naturaleza, vaticina el logro de una resistencia verdaderamente durable. A pesar de los numerosos beneficios que aporta la diversidad, se ha utilizado relativamente muy poco, en esencia a causa de i) un problema paradigmático y ii) un error crítico de la experimentación, que a menudo ha hecho que se subestime el valor potencial de la resistencia y se desista de su empleo. Con las mezclas de tres variedades se satisface de manera adecuada la legítima necesidad de conservar la uniformidad agronómica y al mismo tiempo se aprovechan los beneficios de la diversidad.
Capítulo 9

Métodos actuales del CIMMYT para mejorar la resistencia a la roya en el trigo

S. Rajaram, R.P. Singh y E. Torres. Programa de Trigo, CIMMYT, México

Resumen

En más de 50 millones de hectáreas del mundo en desarrollo se cultivan variedades de trigo derivadas del germoplasma del CIMMYT. Como estos materiales se siembran en una superficie tan extensa, que probablemente incrementará en el futuro, la política de mejoramiento del CIMMYT ha consistido en conservar y aumentar la diversidad de la resistencia a las royas en el germoplasma de trigo. Las pruebas internacionales en sitios múltiples han representado una importante contribución para verificar esta diversidad genética. Además de esas pruebas, el CIMMYT emplea algunos análisis genéticos en su estrategia de mejoramiento. En el CIMMYT se piensa que la utilización de la resistencia vertical (RV) específica para el patógeno producido por genes mayores, como la describe Simmonds (Capítulo 10), podría llevar a situaciones precarias; como una alternativa para la interminable incorporación de genes o combinaciones de genes de resistencia, el CIMMYT ha intentado y recomienda el mejoramiento para obtener lo que Simmonds describe en el Capítulo 10 como resistencia horizontal (RH) poligénica, no específica para el patógeno, que promete durabilidad. En el contexto mundial, la resistencia durable (o estabilidad) y la diversidad genética tienen una enorme importancia en el programa de mejoramiento del CIMMYT. La situación ideal sería identificar como base un gen o un conjunto de genes que quizá haya proporcionado resistencia durable, y luego combinar continuamente genes de resistencia adicionales para asegurar la diversidad genética. La resistencia a la roya del tallo (complejo Sr2) derivada de la variedad Hope y la resistencia a la roya de la hoja (complejo Lr13) derivada de la variedad Frontana son la base de la durabilidad de la resistencia a esas dos enfermedades en el germoplasma del CIMMYT. En cuanto a la roya amarilla (lineal), se ha informado que la variedad Anza producida por el CIMMYT tiene resistencia durable a la enfermedad. El CIMMYT ordinariamente identifica en el campo líneas con resistencia parcial (avance lento de la enfermedad) y se opina que este esfuerzo ha contribuido mucho a mantener los rendimientos del trigo en todo el mundo. El CIMMYT ha buscado la resistencia basada en múltiples genes mayores sólo como una estrategia complementaria y ahora investiga activamente la posibilidad de volver a desarrollar y usar compuestos de multilineas y mezclas varietales.
Capítulo 10

Síntesis: La estrategia para mejorar la resistencia a las royas

N.W. Simmonds, Escuela de Agricultura de Edimburgo, Edimburgo, Escocia

Resumen

El objetivo de mejorar la resistencia a las enfermedades es proteger la biomasa y, por consiguiente, el rendimiento del cultivo. La resistencia es simplemente un estado de "menos enfermedad" y la ausencia de enfermedad (inmunidad) rara vez constituye un objetivo realista. Para fines prácticos, se pueden distinguir cuatro tipos de resistencia: 1) la resistencia vertical (RV) específica para el patógeno, producida por genes mayores; 2) la resistencia horizontal (RIH) poligénica, no específica para el patógeno; 3) la resistencia producida por genes mayores no específica para el patógeno (RN), y 4) la resistencia combinada o de interacción (RI). La RV a menudo es efectiva contra agentes patógenos inmóviles, pero en general no muestra efectos durables contra los agentes patógenos móviles, transmitidos por el aire; no obstante, a veces es posible el control continuo de las enfermedades mediante el empleo de genes múltiples (acumulados) de RV, incorporados con un estricto manejo genético y patológico. La RI es durable y, por lo general, en gran medida heredable; en muchos cultivos se controlan las enfermedades de este modo. La RN es valiosa pero poco frecuente. Se sabe poco acerca de la RI, causada esencialmente por elementos heterogéneos de RV, pero es probable que tenga más valor del que en general se le atribuye.

En cuanto a las royas del trigo, durante años se ha combatido con éxito la roya del tallo en todo el mundo empleando genes acumulados de RV, pero es preciso tener en cuenta que aún podrían presentarse brotes epidémicos de la enfermedad si se interrumpe el control genético y patológico estricto. La roya de la hoja es probablemente la más perjudicial de las tres royas en la actualidad; la base genética de la RV es estrecha y se pueden esperar más epífitas. En el caso de la roya amarilla, a diferencia de lo que sucede con las otras dos royas, la RV parece haber fracasado por completo y, en consecuencia, los fitogenetistas europeos han comenzado a abandonarla. Se reconoce la eficacia de la RI y se ha comenzado a explotarla.

La labor fitogenética del CIMMYT hasta ahora se ha centrado principalmente en acumular RV a las tres royas, pero el control genético no ha sido muy estricto. Se podría argüir que esta estrategia es peligrosa, ya que los pequeños agricultores de los países menos desarrollados no pueden afrontar epífitas que serían tolerables para los agricultores de los países ricos. Se señala que la investigación y explotación de la PH y la RI sería un cambio razonable y factible de estrategia: ambas resistencias han sido poco estudiadas en relación con las royas del tallo y de la hoja, pero es legítimo suponer que tienen un gran potencial. No se lograría trasladar con rapidez la atención dedicada a la RV a la RIH y la RI, pero ese cambio ofrece grandes posibilidades a largo plazo y es de enorme importancia práctica. Esto implica que la estrategia general del Centro cambiará, de modo paralelo, del mejoramiento genético de las variedades de trigo a la investigación estratégica (no básica), modificación que parece concordar con la política del Grupo Consultivo para Investigaciones Agrícolas Internacionales.
Chapitre 1

Le rôle de gènes spécifiques dans l’amélioration de variétés de blé et de triticale dotées de résistance durable à la rouille noire

R.A. McIntosh, Institut de phytogénétique, Castle Hill, Australie

Résumé
La sélection conduite pour obtenir une résistance à la rouille noire (ou rouille de la tige) a été couronnée de succès. Les types de résistance employés jusqu’alors en agriculture dépendaient d’un seul gène identifiable ou de combinaisons de gènes identifiables. Dans beaucoup de régions, le gène Sr2 a contribué à conférer une résistance durable à la plante adulte. Dans les zones céréalières du nord-ouest de l’Australie, cette résistance à la rouille a été basée sur l’usage de formes résistantes qui sont remplacées au fur et à mesure de la détection de pathotypes virulents. Dans ce sens, la sélection de gènes est étroitement liée aux études en matièrde pathogénicité, à la connaissance des gènes présents dans les cultivars de blé et à la coopération de l’industrie pour remplacer rapidement ces dernières. La vulnérabilité génétique à la rouille dans le programme triticaie du CIMMYT pourrait être réduite en mettant à profit l’information provenant d’Australie. L’étroite base génétique associée aux mécanismes de résistance pourrait être élargie grâce à l’emploi de variétés européennes de blé et de triticale et de seigle. Toutefois, il est bon de préserver la diversité génétique qui différencie le blé et le triticale.

Chapitre 2

Résistance du blé à la rouille brune des feuilles et de la tige

A.P. Roelfs, Cereal Rust Laboratory, Service de recherches du Département de l’agriculture des États-Unis et Université du Minnesota, St. Paul, Minnesota

Résumé
Bien qu’au cours des 80 dernières années les rouilles des céréales aient su affecter nombre de variétés supposées résistantes, bien d’autres variétés ont été cultivées avec succès sur de grandes étendues. La rouille de la tige a été contrôlée à la faveur de combinaisons de gènes résistants, dont Sr2 transmis
à Hope et H-44 de l'amidonnier par McFadden en 1923. Sr26 (en provenance de Agropyron elongatum), Sr31 (de Secale cereale) et Sr36 (de Triticum timopheevii) semblent être les gènes simples de résistance les plus efficaces dans le monde. La variété Thatcher (dont la résistance est due à T. durum) produite par Hayes et al. en 1934 offre une résistance satisfaisante dans la plupart des régions. La rouille des feuilles a été combattue avec succès au moyen d’une combinaison de Lr13 et 34. Ces gènes résistants ont été utilisés pour commencer dans les variétés Frontana (Brésil, 1934) et Americano 44D (Uruguay, 1918). Leur combinaison continue à être employée dans la culture de variétés offrant une résistance durable, telles que Chris, Era, Ciano 67, Pavon 76, etc. Les hypothèses touchant à la génétique et à la durabilité de certains types de résistance ont fait obstacle à la sélection et au développement de variétés résistantes.

Chapitre 3

Analyse de la pathogénicité de la rouille jaune (striée) du blé et son importance dans le contexte mondial

R.W. Stulbs, Institut sur la protection des plantes (IPO), Wageningen, Pays Bas

Résumé
La rouille jaune (striée) du blé (Puccinia striiformis Westend. f.sp. tritic) fait l’objet d’études au niveau international de la part de l’IPO. En conditions contrôlées, certaines races (virulences) sont identifiées dans des plantules d’un large ensemble de variétés différentielles “anciennes” et “nouvelles” portant des gènes de résistance dont quelques uns sont connus, mais en majorité inconnus. La virulence liée à la résistance des plantes adultes, spécifique pour chaque race, est étudiée en parcelles séparées. La relation qui existe entre la distribution de la virulence des agents pathogènes et la résistance des plantes hôtes est évidente, mais elle continue à faire l’objet d’études. Son donné ici les résultats d’une recherche sur la rouille jaune, laquelle affecte les triticales et les cultivars dotées de résistance dérivée du seigle, ainsi que la distribution des races de rouille jaune dans diverses zones d’Europe, d’Afrique, d’Asie et d’Amérique du sud. Il est très souhaitable que les études visant à connaître les modifications de la pathogénicité dans diverses populations de races se poursuivent activement afin d’élever le degré de résistance et mesurer la capacité de résistance des plantes-hôtes dans des conditions environnementales différentes.
Chapitre 4

Utilisation de la résistance polygénique pour accroître la résistance du blé à la rouille noire (rouille de la tige)

D.R. Knott, Département de sciences agricoles et phytoécologie, Université de Saskatchewan, Saskatoon, Canada

Résumé
Depuis plusieurs années déjà la résistance multigénique à ce type de maladie est connue. Mais bien que supposée, l'existence d'une résistance non spécifique est difficile à prouver. Une résistance partielle et une infection à développement très lent sont souvent déterminées par la présence de plusieurs gènes considérés parfois comme non spécifiques. Lors de recherches effectuées à Saskatoon, on a pu développer certains blés non résistants à la race 15B-1 au stade de plantule, mais qui possédaient une bonne résistance au champ. Il a été prouvé que cette résistance était déterminée par la présence de 3 à 5 gènes récessifs dont chacun joue un certain rôle. Ces gènes réduisaient la période latente ainsi que le nombre et la taille des pustules. La résistance due à la présence de plusieurs gènes ayant chacun de petits effets est sans doute relativement durable, que cette résistance soit ou non spécifique. Bien qu'il soit difficile d'avoir recours à la résistance polygénique dans les programmes d'amélioration du blé, elle pourrait être d'une grande utilité.

Chapitre 5

Stratégies pour l'utilisation de la résistance partielle dans la lutte contre les rouilles des céréales

J.E. Parlevliet, Département de phytogénétique, Université agricole, Wageningen, Pays Bas

Résumé
Toute résistance aux rouilles qui affectent les céréales est de type spécifique au regard de l'espèce, c'est-à-dire que la résistance n'est efficace qu'à l'égard d'un seul type de rouille. Contre chaque agent pathogène de la rouille deux types de résistance peuvent être identifiés: 1) un type de résistance hypersensible déterminée par des gènes majeurs et caractérisée par une infection limitée, la spécificité selon la race et une durabilité éphémère; 2) un type quantitatif de résistance (résistance partielle) caractérisée par un faible taux de propagation épidémique, en dépit du niveau élevé de l'infestation, par l'absence d'effets spécifiques importants pour la race (bien que certains petits effets puissent avoir lieu) et par sa durabilité. En l'absence de gènes majeurs, il est facile d'opérer une sélection pour obtenir une résistance partielle. Et même une sélection moyennement rigoureuse, mais systématiquement effectuée et visant à éliminer la sensibilité, est très efficace, accumulant des gènes propres à conférer à la variété une résistance...
partielle. Cette sélection douce permet à l'améliorateur de sélectionner en même temps d'autres caractéristiques. S'il s'agit d'accroître la résistance partielle en présence de gènes majeurs qui n'ont pas été totalement neutralisés par l'agent pathogène, l'efficacité de la sélection est considérablement diminuée. Dans toute la mesure du possible, l'améliorateur devra exposer la population-hôte à une seule race d'agent pathogène, capable de neutraliser un nombre maximum de gènes majeurs. De cette population-hôte, l'améliorateur devra éliminer les gènotypes les plus sensibles à chaque étape de la sélection de même que les gènotypes qui ne présentent qu'une infection peu importante. S'il s'avère difficile d'opérer cette dernière séparation, l'améliorateur devra éliminer les gènotypes les plus résistants en même temps que les plus sensibles, puisque les premiers sont supposés porter des gènes majeurs. Dans certains cas, la population pathogène à laquelle est exposée la population-hôte n'est guère contrôlable et constitue en fait un mélange de races. Il est alors très difficile d'opérer une sélection en vue d'obtenir une résistance partielle. L'élimination constante des lignées les plus sensibles en même temps que de celles les moins affectées favorisera l'apparition d'une résistance partielle, mais les progrès dans ce sens sont plus tâtonnements qu'on ne le souhaiterait.

Chapitre 6

Résistance durable du blé à la rouille jaune et ses répercussions en phytogénétique

R. Johnson, Institut de phytogénétique, Cambridge, Angleterre

Résumé
La rouille jaune (striée) causée par le parasite obligé Puccinia striiformis, affecte les cultures de blé dans toutes les régions de climat frais. Plusieurs gènes de résistance, spécifique à chaque race, et efficaces dans les plantules du blé ont été identifiés, mais il en reste encore beaucoup à identifier. Ces gènes de résistance peuvent être dominants ou récessifs et l'expression de certains d'entre eux est grandement influencée par le milieu ambiant et leur patrimoine génétique. La résistance acquise quand la plante a dépassé le stade de plantule est souvent aussi spécifique pour la race. La combinaison de gènes de résistance spécifique selon les races n’a pas eu de succès en Grande Bretagne dans la lutte contre la rouille jaune. Cependant, la résistance acquise ultérieurement à l’étape de plantule ne fait preuve d’aucune spécificité pour les races, même après des essais prolongés et très étendus. Cette résistance durable ne peut se distinguer des résistances spécifiques des plantes adultes qu'au moyen de tests prolongés. Bien qu'elle puisse être soumise à un contrôle génétique complexe, cette résistance peut être mise à profit dans des programmes d'amélioration tels que ceux décrits ici, mais la durabilité de la résistance obtenue dans ces programmes ne peut être garantie. Il faut donc suivre de très près l'évolution de toutes les nouvelles variétés résistantes, quelle que soit la méthode d'amélioration appliquée, pour détecter l'existence de races pathogènes de pathogénicité équivalente.
Chapitre 7

Idées actuelles sur le parti à tirer de la diversité pour protéger les céréales d'agents pathogènes foliaires épidémiques et variables: Problèmes et perspectives d'avenir

J.A. Browning, Département de phytogénétique et microbiologie. Station agricole expérimentale du Texas. Collège Station, Texas

Résumé
Les populations autochtones de progénitures de céréales et leurs parasites naturels, qui ont évolué parallèlement, sont stabilisées par la résistance dilatoire épidémio- logique produite génétiquement par la résistance générale qui implique la présence de gènes d'une grande efficacité pour la résistance spécifique dans des configurations génico-spatiales distinctes. Si la résistance générale protège la plante, la résistance spécifique protège la population en minimisant l'agressivité de l'agent pathogène par ajustement de la rétroaction parfaitement harmonisée des systèmes génétiques de l'hôte et de l'agent pathogène. Résistance générale et résistance spécifique devraient être mises à profit dans des systèmes agricoles cohérents avec leurs origines évolutives. Toutefois, l'agriculture a été souvent dépendante de gènes de résistance spécifique, non pour protéger la population, comme il en est dans la nature, mais pour défendre la plante d'agents pathogènes foliaires épidémiques. Ainsi en a-t-il été lors de l'emploi de gènes de résistance dans des variétés cultivées dans des populations homogènes sur de vastes étendues. Contrairement à cela, dans des programmes réalisés en Colombie, en Angleterre, aux Pays Bas, en Inde et aux États-Unis (Iowa et Washington), ont été développés des gènes de résistance spécifique dans des populations diverses et la protection de la population a été assurée avec une résistance d'un tiers à peine. Cette petite résistance a stabilisé les populations dans un écosystème naturel varié et le chiffre paraît donc réel. Des données concordantes obtenues dans des écosystèmes naturels et agricoles indiquent qu'un système global de manipulation des gènes qui inclut la diversité, comme il en est dans la nature, permettrait d'obtenir une résistance véritablement durable. En dépit des nombreux avantages qu'offre la diversité, elle n'a été que relativement peu mise à profit en raison: 1) d'un problème paradigmatique et 2) d'une erreur cryptique dans l'expérimentation où la valeur potentielle de la résistance a été sous-estimée, ce qui a découragé son emploi. Les combinaisons de trois variétés constituent un compromis adéquat entre la légitime nécessité d'uniformité agronomique et les avantages de la diversité.

Chapitre 8

Le mélange de variétés comme moyen de lutte contre les maladies et de stabilisation du rendement

M.S. Wolfe, Institut de phytogénétique. Cambridge, Angleterre

Résumé
Ce chapitre comporte une description sommaire des principaux facteurs conduisant à une perte d'efficacité de la résistance aux maladies et des fongicides dans l'agriculture européenne. L'emploi de mélanges de variétés
est exposé en détail comme étant l'un des moyens dont disposent les améliorateurs et les agriculteurs pour améliorer la situation. Nombre de données obtenues d'essais au champ révèlent les avantages d'un tel système pour lutter contre les maladies, augmenter et stabiliser les rendements; ce système peut être appliqué parallèlement à tout autre méthode de lutte contre les maladies.

Chapitre 9

Méthodes du CIMMYT pour améliorer la résistance du blé à la rouille

S. Rajaram, R.P. Singh et E. Torres, Programme blé, CIMMYT, Mexique

Résumé

Des variétés de blé provenant des ressources génétiques du CIMMYT sont cultivées sur plus de 50 millions d'hectares dans le monde et décrit développement. L'étendue même de ces cultures appelées à prendre encore plus d'extension à l'avenir a amené le CIMMYT à orienter sa politique d'amélioration vers la conservation et l'accroissement de la capacité de résistance aux rouilles du matériel génétique de blé. Les essais entrepris au niveau international dans de multiples sites ont largement contribué à confirmer cette diversité génétique. De plus, le CIMMYT dans sa stratégie amélioration a recours à des analyses génétiques. Les chercheurs du CIMMYT pensent que l'utilisation de la résistance verticale (RV) spécifique pour le pathotype et produite par des gènes majeurs comme la décrit Simmonds (chap. 10) pourrait conduire à des situations précaires. A titre d'alternative à la perpétuelle incorporation ou combinaison de gènes de résistance, le CIMMYT a essayé et recommande l'amélioration pour obtenir ce que Simmonds décrit (chap. 10) comme résistance horizontale (RH) non spécifique pour le pathotype et d'origine polygénique, laquelle favoriserait la durabilité de la résistance. Dans le contexte mondial, la résistance durable (ou stabilité) et la diversité génétique ont une énorme importance dans le programme d'amélioration du CIMMYT. L'idéal serait de pouvoir identifier un gène ou un ensemble de gènes dont on pourrait prouver qu'il confère une résistance durable, et ensuite combiner de façon continue d'autres gènes de résistance afin d'assurer la diversité génétique. La résistance à la rouille de la tige (complexe Sr2) dérivée de la variété Hope et la résistance à la rouille des feuilles (complexe Lr13) dérivée de la variété Frontana sont la base de la durabilité de la résistance à ces deux maladies dans le matériel génétique du CIMMYT. En ce qui concerne la rouille jaune (strie), selon des informations fournies à ce sujet, la variété Anza produite par le CIMMYT est dotée d'une résistance durable à cette maladie. Le CIMMYT procède ordinairement à l'identification sur place de lignées offrant une résistance partielle (avance lente de la maladie) et a, de la sorte, largement contribué à maintenir les rendements dans le monde. Le CIMMYT poursuit ses recherches sur la résistance basée sur de multiples gènes majeurs à titre de stratégie complémentaire et cherche activement à obtenir le développement et l'emploi de composés de plusieurs lignées et de mélanges de variétés.
Chapitre 10

Synthèse: Stratégie visant à accroître la résistance aux rouilles

N.W. Simmonds, Ecole d'agriculture d'Edimbourg, Edimbourg, Ecosse

Résumé

Accroître la résistance aux maladies équivaut à assurer la protection de la biomasse et, par suite, à élever le rendement des cultures. La résistance n'est autre qu'un état de "moindre maladie" et la suppression de la maladie (immunité) constitue rarement un objectif réaliste. On distingue quatre types de résistance: 1) la résistance verticale (RV) spécifique pour le pathotype, provenant de gènes majeurs; 2) la résistance horizontale (RH) non spécifique pour le pathotype et d'origine polygénique; 3) la résistance due à des gènes majeurs, non spécifique pour le pathotype (RN) et 4) la résistance d'interaction ou combinée (RI). La résistance verticale (RV) est souvent efficace quand il s'agit d'agents pathogènes immobiles, mais peu efficace dans le cas d'agents mobiles, c'est-à-dire transmis par l’air environnant. Toutefois, il est parfois possible de contrôler durablement les maladies à la faveur de gènes multiples (accumulés) de RV obtenus au moyen d'une rigoureuse manipulation génétique et pathologique. La RH est durable et en général en grande mesure héréditaire; nombre de maladies sont contrôlées de la sorte dans beaucoup de cultures. La RN est appréciable, mais peu fréquente. Mal connue, la RI est due essentiellement à des éléments hétérogènes de RV et a probablement plus de mérites qu'on ne lui en attribue généralement.

De toutes les rouilles qui affectent le blé, la rouille de la tige a été combattue avec succès dans le monde entier au moyen de gènes accumulés de RV, mais il ne faut pas oublier qu'il peut toujours y avoir des poussées épidermiques de la maladie en cas d'interruption du contrôle génétique et pathologique. La rouille des feuilles est probablement la plus préjudiciable de ces trois maladies; la base génétique de la RV est étroite et il y a lieu de craindre de nouvelles épidémies. Quant à la rouille jaune, contrairement à ce qu’il en est des deux autres, la RV semble avoir totalement échoué pour en venir à bout, et cet échec en a motivé l’abandon par les améliorateurs européens. L’efficacité désormais reconnue de la RH fait qu’elle commence à être mise à profit.
Le CIMMYT dans ses programmes d’amélioration s’est jusqu’à présent employé à accumuler RV pour lutter contre les trois rouilles, sans toutefois que le contrôle génétique ait été très rigoureux. Sans doute cette stratégie peut-elle être jugée dangereuse, car les petits agriculteurs des pays moins développés ne sont pas en mesure de faire face au risque d’épidémies que tolèrent plus facilement les agriculteurs de pays riches. Cette stratégie pourrait être modifiée au moyen de recherche et d’exploitation des résistances RH et RI qui, jusqu’à présent, n’ont pas fait l’objet de recherches très poussées en tant que moyen de lutte contre la rouille de la tige et la rouille des feuilles, sans cependant que leur potentiel ne puisse être légitimement mis en doute. Il ne faut pas espérer que l’attention jusqu’alors accordée à la RV soit rapidement détournée au profit de RH et de RI, bien que ce changement d’orientation offrirait à long terme de grands avantages. Parallèlement, le Centre devrait adjoindre à sa stratégie d’amélioration génétique des variétés de blé la recherche stratégique (non de base) coincidant avec la politique du Groupe consultatif pour la recherche agricole internationale.