Meeting the Challenge of Yellow Rust in Cereal Crops

Editors
Roy Johnson
Amor Yahyaoui
Colin Wellings
Abbas Saidi
Habib Ketata

International Center for Agricultural Research in the Dry Areas

About ICARDA and the CGIAR

Established in 1977, the International Center for Agricultural Research in the Dry Areas (ICARDA) is governed by an independent Board of Trustees. Based at Aleppo, Syria, it is one of 16 centers supported by the Consultative Group on International Agricultural Research (CGIAR).

ICARDA serves the entire developing world for the improvement of lentil, barley and faba bean; all dry-area developing countries for the improvement of on-farm water-use efficiency, rangeland, and small-ruminant production; and the Central and West Asia and North Africa region for the improvement of bread and durum wheats, chickpea, and farming systems. ICARDA's research provides global benefits of poverty alleviation through productivity improvements integrated with sustainable natural-resource management practices. ICARDA meets this challenge through research, training, and dissemination of information in partnership with the national agricultural research and development systems.

The results of research are transferred through ICARDA's cooperation with national and regional research institutions, with universities and ministries of agriculture, and through the technical assistance and training that the Center provides. A range of training programs is offered, from residential courses for groups to advanced research opportunities for individuals. These efforts are supported by seminars, publications, and specialized information services.

The CGIAR is an international group of representatives of donor agencies, eminent agricultural scientists, and institutional administrators from developed and developing countries who guide and support its work. The CGIAR receives support from many country and institutional members worldwide. Since its foundation in 1971, it has brought together many of the world's leading scientists and agricultural researchers in a unique South–North partnership to reduce poverty and hunger.

The mission of the CGIAR is to promote sustainable agriculture to alleviate poverty and hunger and achieve food security in developing countries. The CGIAR conducts strategic and applied research, with its products being international public goods, and focuses its research agenda on problem-solving through interdisciplinary programs implemented by one or more of its international centers, in collaboration with a full range of partners. Such programs concentrate on increasing productivity, protecting the environment, saving biodiversity, improving policies, and contributing to the strengthening of agricultural research in developing countries.

The World Bank, the Food and Agriculture Organization of the United Nations (FAO), the United Nations Development Programme (UNDP), and the International Fund for Agricultural Development (IFAD) are cosponsors of the CGIAR. The World Bank provides the CGIAR System with a Secretariat in Washington, DC. A Technical Advisory Committee, with its Secretariat at FAO in Rome, assists the System in the development of its research program.

Meeting the Challenge of Yellow Rust in Cereal Crops

Proceedings of the First Regional Conference on Yellow Rust in the Central and West Asia and North Africa Region, 8-14 May 2001, Karaj, Iran

Editors
Roy Johnson*
16 Coppice Avenue, Great Shelford, Cambridge CB 25 AQ, UK
*(*Deceased)*
Amor Yahyaoui
Senior Cereal Pathologist
ICARDA, Aleppo, Syria
Colin Wellings
University of Sydney, Plant Breeding Institute, Cobbitty, Australia
Abbas Saidi
Director General
Seed and Plant Improvement Institute, Karaj, Iran
Habib Ketata
Cereal Breeder and Coordinator
ICARDA/Iran Project, Tehran, Iran

Organized by:
International Center for Agricultural Research in the Dry Areas (ICARDA)
and
Seed and Plant Improvement Institute (SPII)
The Islamic Republic of Iran

International Center for Agricultural Research in the Dry Areas

Recommended Citation:
Johnson, Roy; Yahyaoui, Amor; Wellings, Colin; Saidi, Abbas; and Ketata, Habib (eds.). 2002. Meeting the Challenge of Yellow Rust in Cereal Crops. Proceedings of the First Regional Conference on Yellow Rust in the Central and West Asia and North Africa Region, 8-14 May 2001, Karaj, Iran. Organized by the International Center for Agricultural Research in the Dry Areas (ICARDA) and the Seed and Plant Improvement Institute (SPII), Karaj, the Islamic Republic of Iran. ICARDA, Aleppo, Syria. xiv + 280 pp.

ISBN: 92-9217-131-9

Printed at ICARDA.

ICARDA
P.O. Box 5466, Aleppo, Syria
Tel: (963-21) 2213433, 2213477, 2225112, 2225012
Fax: (693-21) 2213490, 2225105, 5744622
E-Mail: ICARDA@CGIAR.ORG
Web site: http://www.icarda.cgiar.org

TABLE OF CONTENTS

Pathogenic Variability

Use of Disease Nurseries

Biotechnology and Rust Resistance

Cytogenetics and Genetic Analysis

Epidemiology

BARLEY YELLOW RUST

FOREWORD

Yellow rust is an important disease of wheat, particularly in the Caucasus, Central and West Asia, the Nile Valley, and the Horn of Africa, where it has caused recurrent severe damage since time immemorial. Epidemics continue to cause severe losses. Recently, 30-40% yield losses have been recorded in major wheat producing areas in Azerbaijan, Kyrgyzstan, and southern Kazakhstan. In Uzbekistan, more than 60% of the wheat area is sprayed with fungicides during the rainy cool season to combat the disease. Scientists, breeders, policy advisors, and farmers must join to counter the yellow rust problem.

The apparent increased frequency and severity of yellow rust epidemics raises questions about the nature of the disease, the factors that contribute to epidemic development, and variation in disease response of wheat cultivars. Comprehensive information on pathogen virulence and variation, and epidemiological information on pathogen movements is critical. It could provide the basis for the (1) development of an early warning system that might reduce the frequency and magnitude of losses; (2) identification and subsequent management of susceptible cultivars; and (3) development of varieties with durable resistance.

Using resistant cultivars is the best strategy for disease control, as it carries no additional cost to farmers and is environmentally safe. Some improved cultivars, with resistance based on a single race-specific gene, or combinations of genes, are currently grown on large areas in various countries. However, the genes are vulnerable to pathogen plasticity, and their longevity can vary, from rapid vulnerability to relative durability. It is likely that most specific resistances, whether based on a single major gene or combination of major genes, will sooner or later succumb to new adaptive pathotypes if careful deployment is not practiced.

The diversity of the pathogen population poses a great challenge, but more information is emerging, slowly, about durable resistance to yellow rust of wheat. Evidence suggests that adequate levels of resistance could be obtained with a few additive genes, each having small to moderate effect. Despite our advances in knowledge, we need to improve the application of programs to achieve durable resistance. I hope that in collaboration with our national research partners we can find a 'durable' solution to the yellow rust problem.

Recognizing the importance of yellow rust in the region, the First Regional Yellow Rust Conference for Central and West Asia and North Africa (CWANA) was organized by the International Center for Agricultural Research in the Dry Areas (ICARDA) and the Seed and Plant Improvement Institute (SPII) of the Islamic Republic of Iran. Conference participants were able to discuss and share the latest information on wheat and barley yellow rust. This volume contains the text of scientific presentations made at the Conference. The topics covered are

important to many countries, not only in CWANA, but also in Europe, Central and South America, and Australia

Dr Roy Johnson played the lead role in editing this book but, unfortunately, did not live to see it in its present form. His death saddened us all, but this book will keep his memory and contributions fresh in the international scientific community.

I trust that those concerned with wheat improvement will find this volume useful.

Prof. Dr. Adel El-Beltagy
Director General
ICARDA

ACKNOWLEDGMENTS

The Editors are grateful for the financial support of the Islamic Republic of Iran for the organization of the First Regional Yellow Rust Conference and for publishing this volume of proceedings.

Dr. M. Torabi and his cereal pathology staff are to be congratulated on the logistic arrangements for all the participants and for the field tours and visits in Iran. Special thanks to Dr. S. Varma and his staff in the Communication, Documentation and Information Services Unit of ICARDA for the preparation and printing of the manuscript, and to the Germplasm Program for hosting the late Dr. Roy Johnson at ICARDA for the editing of the document.

Preface

This first regional conference was timely because yellow rust remains a serious challenge for the CWANA region, having developed as a much more important disease of wheat since the late 1980s than it was formerly. This increased importance has arisen because of the greater use of irrigation in the region and the widespread use of cultivars that became susceptible as the pathogen, *Puccinia striiformis* f. sp. *tritici*, evolved to new combinations of virulence, as is well-illustrated in the papers presented.

The meeting was organised by the combined efforts of the Seed and Plant Improvement Institute (SPII), Karaj, the Agricultural Research, Education and Extension Organization, Tehran, Iran, and ICARDA. The Iranian Ministry of Agriculture must be congratulated and thanked warmly as a major contributor to the financing of the meeting and for provision of the excellent modern facilities of the new Conference Center at SPII. The hospitality was excellent.

The meeting provided the opportunity for workers within the region to meet each other, and to meet international scientists from many other countries. It is a great tribute to the large majority of the participants whose native language is not English, that all papers were presented in English. Not surprisingly, the task of editing the papers for publication included considerable revision of the English. The editors hope, in doing this, they have not changed the meaning intended by authors. Much effort was also needed on other aspects of the editing. A feature in some papers was a lack of realisation of past experience with this disease and some presentation of results that were not discriminating enough, or apparently not possible. Perhaps exceptionally, the editors have inserted a few comments to indicate possible problems in interpretation of data and statements. The editors hope authors will forgive this unusual intrusion into their work, as it was done to try to improve clarity of the work.

In addition to the many papers on yellow rust of wheat there is also a set on the yellow rust of barley. *P. striiformis* f. sp. *hordei* reached South America for the first time in 1975, since when it has spread to cool barley-growing areas of the whole American continent, causing some severe epidemics and generating new research projects. These are described in several papers, all from the American continent, North and South.

It is to be hoped that much further co-operation on controlling yellow rust of wheat and barley will be stimulated by the papers in these proceedings and by the contacts established between the workers who attended the Conference. To strengthen these contacts and develop further co-operation, a further regional Conference in a few years would be highly beneficial and is under consideration.

WHEAT YELLOW RUST

Genetics and Breeding for Resistance

Looking Ahead in Wheat Breeding

S. Rajaram

International Maize and Wheat Improvement Center (CIMMYT). Mexico D.F. Mexico

In the 20th Century, plant breeding witnessed enormous advances in genetic improvement of many crops, including food, feed and forage species. Since the first application of Mendelian genetics in wheat breeding for stripe (yellow) rust resistance in England in the beginning of the 20" Century, there have been significant advances in understanding of polygenic systems and their deployment in breeding improved crops. The exploitation of heterosis in maize and sorghum and simply inherited dwarfing traits in wheat, rice, and barley are amongst the major contributions of 20th Century plant breeding.

While the discovery and limited deployment of durable disease resistance genes began in mid 20th Century, because of difficulties associated with such a conceptual hypothesis, its application has remained sporadic and is perhaps less appreciated than it should be. The stripe rust epidemic in East Africa and Middle East in 1990's serves as a constant reminder to concerned scientific communities at large, of how vulnerable the varieties are, particularly those protected through major gene resistance. I hope that this assembly finds a collaborative approach towards a durable solution to the stripe rust situation in Central and West Asia.

What about yield potential improvement? Perhaps attention to it is urgently warranted as well. Are there new wheat genetic resources whose sink potential is large enough to house a larger grain reserve? At CIMMYT we have successfully created a super plant type dubbed "AGROPOLITETRA Wheats" which were assembled from *Agrotriticum, polonicum, tetrastichon* and common wheats, which offer enormous potential to pack a large number of grains/spike. The tillering capacities of these types have been restored to normal and grain filling potential has been enhanced. We hope to release these types to our collaborators worldwide in a couple of years.

We also have to address the issue of marginal environments through the application of breeding and efficient water conservation methodology. It is becoming clear that genetically based drought enhancing capacity can apparently be truly combined into high yielding cultivars with responsive capability. The variety Baviacora 92 (Babax group) represents such exemplary combination.

Additive Genes for Durable Resistance to Yellow Rust in Wheat: Genetics, Molecular Mapping and Breeding at CIMMYT

R. P. Singh[1], J. Huerta-Espino[2] and M. William[1]

[1]CIMMYT, Apdo., Mexico, DF,
[2]Campo Experimental Valle de Mexico-INIFAP, Apdo. Chapingo, Edo. de Mexico, Mexico.

Introduction

Yellow (or stripe) rust, caused by *Puccinia striiformis* f. sp. *tritici,* is an important disease of wheat in most wheat growing regions including West Asia, Central Asia and North Africa (CWANA). Using resistant cultivar for disease control is the best strategy as it has no cost to the farmer and is environmentally safe. Historically, race-specific major genes have been used to breed resistant cultivars. At present 30 genes are catalogued (McIntosh *et al.* 1998). A majority of these are race-specific and virulence has been identified for most of them at least somewhere in the world. Some important cultivars where resistance is based on a single race-specific gene, or combinations of two of them, are currently grown on a large area in countries where yellow rust has caused major losses or threats in the past years. Resistances of Inquilab 92 based on *Yr27* and PBW343 based on the combination of *Yr9* and *Yr27* are highly vulnerable as they are the most important cultivars in northwestern Pakistan and India, respectively, and virulences for these genes and their combinations are known. These cultivars show unacceptable levels of adult plant resistance in Mexico when tested with a race virulent on the above genes. Similarly, a number of Kauz derived varieties, e.g. Bakhtawar 94 (Pakistan), WH542 (India), Memof (Syria), Basribey 95 and Seyhan 95 (Turkey) and Atrak (Iran), were released following the widespread epidemic in these countries on Veery#5 derived cultivars. Immunity of Kauz in these countries is due to the presence of the combination of *Yr9* and *Yr27.* Combination of virulences for these two genes in the yellow rust population do not exist at present in the above countries, however, is known to occur in Mexico and Ecuador. Slow rusting gene *Yr18,* also present in Kauz, does not confer enough protection under high disease pressure (Ma and Singh 1996) and hence Kauz shows unacceptable disease levels when tested in Mexico. This kind of information on the genetic basis of resistance could be extremely useful for a country to prepare for a forthcoming epidemic and take all measures to diversify the crop by promoting additional genetically diverse cultivars. One of the main objectives of CIMMYT's wheat genetics and breeding

programs is to generate diverse germplasm. We strive to achieve durable resistance by combing genes that have small to intermediate but additive effects. The progress of a decade of research at CIMMYT on durable resistance to yellow rust is discussed in this paper.

The terms used in this presentation are defined as follows:

Race-specific resistances are resistances that are easily detected with specific pathotypes or races of the pathogen and are controlled by genes having major effects. In wheat-rust pathosystems, these resistances are recognized by characteristic low infection types. Numerous genes are now known and have been catalogued by McIntosh *et al.* (1998). Most of these genes can be detected in seedling evaluations using specific pathotypes. However, detection of a few others requires testing at post-seedling growth stages. Major genes are implicitly vulnerable to pathogen plasticity, and their longevity can range from rapid vulnerability to relative (and often deceiving) durability. It is likely that most specific resistances, whether based on a single major gene or a combination of major genes, will sooner or later succumb to new adaptive pathotypes if careful deployment is not practiced. In the Australian continent, major genes for stem rust resistance, such as *Sr26*, in practice can be considered of a durable type because no pathotype with corresponding virulence has evolved until now, even after more than 20 years of this gene's deployment over a large area. [Editor's comment: Thus, although mentioned in this section on race-specific genes, it has not yet been shown to be race-specific, although its phenotype is similar in appearance to that of other genes known to be race-specific. R. Johnson].

Race-nonspecific resistances are resistances that operate against all pathotypes or races of a pathogen. The genetic nature of this type of rust resistance is usually complex and based on the additive interaction of a few or several genes having minor to intermediate effects.

Slow rusting and partial resistance are almost synonymous terms. As defined by Caldwell (1968), slow rusting is a type of resistance where disease progresses at a retarded rate, resulting in intermediate to low disease levels against all pathotypes of a pathogen. Partial resistance, as defined by Parlevliet (1975) referring to leaf rust resistance in barley, is a form of incomplete resistance characterized by a reduced rate of epidemic development despite a high or susceptible infection type. The components that cause slow rusting of a cultivar are longer latency period, low receptivity or infection frequency, as well as smaller uredial size and reduced duration and quantity of spore production. All these components can affect disease progress in the field.

Durable resistance (Johnson 1978) is resistance that has remained effective in a cultivar during its widespread cultivation for a long sequence of generations or period of time in an environment favorable to a disease or pest.

Diversity of race-specific genes for yellow rust resistance in CIMMYT's germplasm

Possible diversity for race-specific genes present in CIMMYT wheats distributed during recent years is shown in Table 1. Known genes, such as *Yr1*, *Yr15* and *Yr17* could be identified in some wheat lines. Noteworthy is line Milan, which carries *Yr17*. This gene is not yet deployed in the region and could be effective to the current population of *P. striiformis*. However, it was used extensively in Europe, Australia and New Zealand resulting in the identification of virulent races recent years in each of the above continents. We can indicate the presence of at least five additional unknown genes, probably of wheat origin, in the germplasm. A gene conferring seedling infection type 34 and high level of adult plant resistance is present in wheat lines Pastor, Tinamu, Ducula, etc. and may be derived from some selections of Bobwhite. This gene is effective in this region and all other sites reporting data on these lines. Its proportion in new CIMMYT lines is increasing due to the use of Pastor in many crosses. Pastor has high yield potential, wide adaptation, excellent industrial quality and resistance to *Septoria tritici* making it an attractive parent in the crosses.

The use of Chinese wheat germplasm to incorporate resistance to head scab has also introduced at least two major yellow rust genes in CIMMYT germplasm. Examples of these CIMMYT lines are some selections of Catbird and SW89.2089/Kauz that have low and moderate seedling resistance and high levels of adult plant resistance (Table 1). CIMMYT lines Weaver and Star appear to carry genes that confer intermediate seedling infection types but moderate and moderately high field reactions, respectively.

Some new genes are also entering in the germplasm from synthetics (Table 1). We recently identified and designated gene *Yr28* (Singh *et al.* 2000b) of *T. tauschii* origin. At least two, or more, additional genes are also present in synthetic derived lines.

In all it can be stated that CIMMYT germplasm contains a high degree of genetic diversity for race-specific genes that are currently effective in most developing countries. These genes will probably protect some important varieties in the future but eventually succumb to new virulences that will arise in time.

Table 1. Usual seedling infection type (IT) and adult plant responses (APR) observed in Mexico on race-specific genes present in CIMMYT germplasm.

Gene	Response		Line
	Seedling IT[a]	APR[b]	
Yr1	01	0	TJB368.251/Buc//Oci
Yr15	1	0	V763.2312/V879.C8.11.11.11(36)/Star/3/Star
Yr17	23	5MR	Milan
?[c]	34	5R-MR	Pastor, Bobwhite, Tinamu, Ducula
?	23	0	Catbird
?	45	5R-MR	SW89.2089/Kauz
?	45	20MR	Weaver
?	56	60M	Star
Yr28	45	30MR	Altar 84/Ae. tauschii//Opata
?	12	1MR	Opata//Sora/Ae. tauschii (323)
?	13	1MR	Croc1/Ae. tauschii (205)//Kauz/3/Sasia

[a] Seedling infection type follow a 0-9 scale as described in Roelfs *et al.* (1992)

[b] The APR has two components, % rust severity based on the modified Cobb Scale (Peterson *et al.* 1948) and response to infection as described by Roelfs *et al.* (1992)

[c] *Unknown, probably new genes for resistance*

Yr18 and other minor genes for durable resistance to stripe rust

In recent studies, Singh (1992) and McIntosh (1992) have indicated that the moderate level of durable adult plant resistance to stripe rust of the CIMMYT-derived US wheat cultivar Anza and winter wheats such as Bezostaja is controlled in part by the *Yr18* gene. This gene is completely linked to the *Lr34* gene. The level of resistance it confers is usually not adequate when present alone. However, combinations of *Yr18* and 2-4 additional slow rusting genes result in adequate resistance levels in most environments (Singh and Rajaram 1994). Cultivars carrying *Yr18* in such combinations are listed in Table 2. Genes *Lr34* and *Yr18* occur frequently in germplasm developed at CIMMYT and in various countries. Using Jupateco 73 near-isogenic reselections, studies at CIMMYT have shown that the gene *Yr18* also increases latent period while decreasing infection frequency and length of infection lesions (stripes) to stripe rust in greenhouse experiments (Table 3). The conclusion was that these components were under pleiotropic genetic control.

Table 2. Some seedling susceptible bread wheats that carry good adult plant resistance to stripe rust in field trials in Mexico and other countries.

Genotype(s)	Usual yellow rust response[1]	Additive for resistance genes[2]
Jupateco 73S	100MS	Susceptible
Jupateco 73R	50M	*Yr18*
Parula, Cook, Trap	15M	*Yr18* + 2 genes
Tonichi 81, Sonoita 81, Yaco	10M	*Yr18* + 2 or 3 genes
Chapio, Tukuru, Kukuna, Vivitsi	1M	*Yr18* + 3 or 4 genes
Amadina	30M	3 genes
Pavon 76, Attila	20M	3 genes

[1] Yellow rust response data from Mexico has two components, % severity based on modified Cobb scale (Peterson *et al.* 1948) and reaction based on Roelfs *et al.* (1992). The reactions are M = moderately resistant to moderately susceptible, sporulating stripes with necrosis and chlorosis; and S = sporulating stripes without chlorosis or necrosis.

[2] Minimum number estimated from genetic analysis.

Table 3. Comparison of the three components of slow rusting resistance to stripe rust in seedling and flag leaves of near-isogenic *Yr18* Jupateco 73 reselections tested at 15°C.

Genotype	Latent period (Days)	Infection frequency (stripes/cm2)	Length of stripes (mm)
Jupateco +*Yr18*	20.1	0.7	12.5
Jupateco -*Yr18*	15.9	7.1	47.7

Because stripe rust can develop systemically, it is different from the other two rusts, where every new pustule develops from a new infection. The epidemiology of stripe rust is also different from that of the other two rusts. Johnson (1988) presented examples of adult plant resistance genes that are race-specific in nature. It is difficult to distinguish such resistance from the resistance conferred by genes of race-nonspecific nature based on the adult plant infection type. Low disease severity to stripe rust is most often associated with at least some reduction in infection type.However, it was observed that in the case of potentially durable slow rusting resistance, the first uredinia to appear are moderately susceptible to susceptible. Subsequent growth of fungal mycelium causes some chlorosis and necrosis; therefore, the final infection type is usually rated as moderately resistant-moderately susceptible. Durability of such resistance can be expected if the cultivar's low disease severity is due to the additive interaction of several (4 to 5) partially effective genes.

Genetic linkage/pleiotropism of resistance genes involved in slow rusting to different rust pathogens

Genetic linkage between slow rusting genes *Lr34* and *Yr18* was described earlier. More recently it has been shown that durable stem rust resistance gene *Sr2* is closely linked to a minor gene, *Yr30*, conferring yellow rust resistance (Singh *et al.* 2000b). Quantitative trait locus (QTL) analysis of slow rusting resistance to leaf rust and yellow rust in two recombinant inbred populations at CIMMYT has shown that several QTLs confer resistance to both leaf and yellow rust (Table 4). Disease specific QTLs were also present for both leaf and yellow rusts, indicating that close genetic linkage or pleiotropism is not a rule. Slow rusting leaf rust resistance gene *Lr46* was linked to a gene for slow rusting yellow rust resistance, recently designated as *Yr29*. Functional aspects of slow rusting genes may be better understood once they are cloned. Because the same, or closely linked, minor slow rusting genes confer resistance to more than one rust disease, generating multiple rust resistance germplasm should be simpler than previously thought.

Table 4. QTLs for slow rusting, additive genes involved in resistance to leaf and yellow rust diseases of wheat mapped by evaluating RILs from crosses of susceptible wheat 'Avocet S' and resistant 'Pavon 76' and 'Parula' for three years at field sites in Mexico.

Cultivar	Location	Marker	Disease severity reduction (%) Leaf rust	Yellow rust	Named genes
Pavon 76	1BL	Wms259	35	27	*Lr46, Yr29*
	4B	Wms495	18	15	
	6A	Wms356	14	18	
	6B	PaggMcaa	-	18	
	3BS	PacgMcgt	-	11	*Sr2, Yr30*
Parula	7DS	Wms130, Ltn[1]	56	46	*Lr34, Yr18*
	7B or 7D	Pcr156	29	-	
	1BL	Wms259	15	16	*Lr46, Yr29*
	Unknown	PaagMcta	22	14	
	3BS	Glk2	-	12	*Sr2, Yr30*

[1] Leaf tip necrosis, a morphological marker linked to gene *Lr34*.

Crossing and selection procedures employed to achieve resistance based on additive interactions of slow rusting genes

It is often thought that selecting for resistance based on additive minor genes is difficult. However, at CIMMYT certain measures aimed at enhancing the accumulation of such genes are being taken. These measures are:

(i) Selecting parents that lack effective major genes and have moderate to good levels of slow rusting resistance to local rust pathotypes. Such parents are easily identified by testing them at the seedling stage in the greenhouse and as adult plants in the field using the same pathotype. Parents of interest should show susceptibility at the seedling stage and slow rusting in the field. Cultivars known to have durable resistance are also included.

(ii) Maintaining genetic diversity. Parents having different sets of additive genes based on available information are used in crossing. If such information is not available, parents of diverse origins or diverse pedigrees are selected for crosses.

(iii) Establishing high disease pressure in the breeding nursery with chosen rust pathotypes. Spreader rows are planted at optimum distance and artificially inoculated to ensure homogeneous disease spread of desired rust pathotypes in the plot. Susceptible and slow rusting checks are included to assess disease pressure.

(iv) Selecting plants with low to moderate terminal disease severity in F_2 and F_3, and from F_4 onwards, selection of plants or lines with low terminal severity. Because adequate resistance levels require the presence of 4 to 5 additive genes, the level of homozygosity from the F_4 generation onwards is usually sufficient to identify plants or lines that combine adequate resistance with good agronomic features. Moreover, selecting plants with low terminal disease severity under high disease pressure means that more additive genes may be present in those plants.

(v) Maintaining leaf tip necrosis or mild pseudo-black chaff phenotypes. Because leaf tip necrosis is linked to durable resistance gene *Yr18*, and pseudo black chaff is linked to *Yr30*, these traits are useful morphological markers.

(vi) Conducting multilocational testing. As discussed earlier, multilocational testing of useful advanced lines can indicate the effectiveness and stability of resistance across environments. Based on the results, new lines are identified for future crossing.

(vii) Genetic analyses of selected lines. To confirm the presence of resistance based on additive genes, important lines are genetically analysed.

Following the methodology described above, we have successfully combined high levels of resistance (comparable to near-immunity) to leaf and yellow rusts with high grain yield potential in wheat lines such as Chapio, Tukuru, Kukuna and Vivitsi (Table 2) (Singh *et al.* 2000a). Genetic analysis of such resistance has shown that at least 4 or 5 minor, additive genes conferred resistance to both leaf and yellow rusts. These wheat lines could be released directly for cultivation or be used in future breeding programs.

References

Ma H, Singh RP, 1996. Expression of adult resistance to stripe rust at different growth stages of wheat. *Plant Disease* **80**:375-379.

Caldwell RM, 1968. Breeding for general and/or specific plant disease resistance.In *Proceedings of the 3rd International Wheat Genetics Symposium,* 263-272. Canberra, Australia.

Johnson R, 1978. Practical breeding for durable resistance to rust diseases in self-pollinating cereals. *Euphytica* **27**: 529-540.

Johnson R, 1988. Durable resistance to yellow (stripe) rust in wheat and its implications in plant breeding. In: N.W. Simmonds and S. Rajaram (eds.) *Breeding Strategies for Resistance to the Rusts of Wheat,* p. 63-75. CIMMYT, Mexico.

McIntosh RA, 1992. Close genetic linkage of genes conferring adult-plant resistance to leaf rust and stripe rust in wheat. *Plant Pathology* **41**: 523-527.

McIntosh RA, Hart GE, Devos KM, Gale MD, Rogers WJ, 1998. Catalogue of gene symbols for wheat. A. E. Slinkard (ed.) *Proceedings of the 9th International Wheat Genetics Symposium,* 2-7 Aug. 1998, Saskatoon, Canada. Vol 5:1-235.

Parlevliet JE, 1975. Partial resistance of barley to leaf rust, *Puccinia hordei.* I. Effect of cultivar and development stage on latent period. *Euphytica* **24**:21-27.

Peterson RF, Campbell AB, Hannah AE, 1948. A diagrammatic scale for estimating rust intensity of leaves and stem of cereals. *Canadian Journal of Research Section C.* **26**: 496-500.

Roelfs AP, Singh RP, Saari EE, 1992. *Rust Diseases of Wheat: Concepts and Methods of Disease Management.* CIMMYT, Mexico, D.F. 81pp.

Singh RP, 1992. Genetic association of leaf rust resistance gene *Lr34* with adult plant resistance to stripe rust in bread wheat. *Phytopathology* **82**: 835-838.

Singh RP, Huerta-Espino J, Rajaram S, 2000a. Achieving near-immunity to leaf and stripe rusts in wheat by combining slow rusting resistance genes. *Acta Phytopathlogica Hungarica* **35**: 133-139.

Singh RP, Nelson JC, Sorrells ME, 2000b. Mapping *Yr28* and other genes for resistance to stripe rust in wheat. *Crop Science* **40**: 1148-1155.

Singh RP, Rajaram S, 1994. Genetics of adult plant resistance to stripe rust in ten spring bread wheats. *Euphytica,* **72**: 1-7.

Prospects for Achieving Durable Resistance to Yellow Rust in Wheat in the 21st Century

R. Johnson

16, Coppice Avenue, Great Shelford, Cambridge CB2 5AQ, UK.

Introduction

Durable resistance to a plant disease is resistance that remains effective during widespread and long-term cultivation of a cultivar in an environment favourable to the disease (Johnson and Law 1975). For yellow rust of wheat some cultivars fail to maintain their resistance, introduced by breeding, when they are deployed commercially. This has occurred with all known resistance genes that have been effective from the seedling stage and throughout the life of the plant when used in commercially grown wheat cultivars. It has also occurred with some genes that are not effective in seedlings but become more effective in adult plants, such as *Yr11, Yr12, Yr13* and *Yr14* (McIntosh *et al.* 1995). Such genes, whether they are effective from the seedling stage onwards, or only later in the life cycle, can be described as race-specific. Despite these failures of introduced resistance, other cultivars with adult plant resistance have remained adequately resistant during widespread and prolonged cultivation (Johnson 1988). Genes such as *Yr11* to *Yr14* show, however, that adult plant resistance is not diagnostic for durable resistance to yellow rust (see Table 1) (Johnson and Taylor 1980). The objective is to achieve positive breeding for durable resistance and avoidance of the creation of non-durable types of resistance.

Materials and Methods

Durable resistance test

The test requires the widespread commercial cultivation of cultivars for years in an environment favourable to yellow rust. Multi-location tests, tests with existing collections of pathogen isolates, and small-scale cultivation do not provide adequate tests.

Resistance tests

For understanding the expression of resistance and making hypotheses about the genetic basis of resistance, plants must be tested at both the seedling stage, in glasshouses or growth cabinets, and also at later growth stages, often in field nurseries, using controlled inoculations with pathogen isolates (Johnson and Taylor 1980).

Genetic studies

Standard genetic methods, intercrossing cultivars, and cytogenetic methods, using aneuploid plants and chromosome substitution lines, are useful for investigating the genetic basis of durable resistance (Law *et al.* 1978).

Genetic linkage and markers

Linkage between genes for resistance to yellow rust and for resistance to other diseases or other characters can be detected by standard genetic and cytogenetic studies. In addition, the techniques of molecular biology are being applied for the detection of linkage to yellow rust resistance genes (Chagué *et al.* 1999; Singh *et al.* 2000; Peng *et al.* 2000).

Results and Discussion

A number of wheat cultivars have been identified to posses durable resistance to yellow rust including Cappelle Desprez, Hybride de Bersée (Johnson 1988) and Bezostaja.

The former two French wheat cultivars were investigated cytogenetically and shown to possess part of their resistance under control of genes on a small translocated chromosome, 5BS-7BS (Law *et al.* 1978) (Table 1). Other chromosomes in them also contribute to the final expression of resistance.

Table 1. Percentage infection on cultivars with *Yr13* with avirulent *(Av13)* and virulent (*v13*) races of *P. striiformis* and percentage infection on euploids and nullisomics 5BS-7BS of French wheat cultivars.

	Races		French wheat test		
Cultivar	41E136(*Av13*)	41E136(*v13*)	Cultivar	Euploid	Nulli 5BS-7BS
C. Desprez (control)	42	43	C. Desprez*	12	49
Maris Huntsman (*Yr13*)	45	77	H. de Bersée	2	29
Maris Nimrod (*Yr13*)	20	40			

* different trial with less infection on euploid C.Desprez

Some cultivars, such as Bezostaja were shown to possess the gene *Yr18* on chromosome 7D, detected by genetic linkage with *Lr34* for resistance to leaf rust (McIntosh *et al.* 1995) and *Yr29* linked to *Lr46* are, so far, the only named genes believed to control components of durable resistance to yellow rust (R. P. Singh, *personal communication)*

The evidence indicates that the components providing durable resistance are expressed in adult plants, but, as noted, adult plant resistance is not necessarily of a durable type. Because durable resistance to yellow rust in wheat is of the type mainly expressed in adult plants, it can be hidden by race-specific genes giving higher levels of resistance, and by combinations of such genes. These can occur in many crossing programmes, sometimes without any intention of the breeder to generate them. The gene *Yr17* provides an example. It originated from crosses made with the objective of transferring resistance to the eyespot disease, from *Aegilops ventricosa*. Lines generated in this work were found to possess genes for resistance to the three rust diseases of wheat, of which the gene for yellow rust was designated *Yr17* (Bariana and McIntosh 1993). Use of these lines in breeding resulted in *Yr17* in commercial wheat cultivars in the UK, the first of which, Rendezvous, was not widely grown, reaching a maximum of 1.6% of the area of winter wheat (Table 2). Virulence for *Yr17* was not detected in this period. However, when later cultivars with the gene were introduced on an increasing area, virulence occurred rapidly and became widely distributed. At this time, the *Yr17*-virulent races lacked virulence for *Yr6* and so cultivars with *17* plus *6* were still resistant. When these entered commercial use, combined virulence for both genes rapidly arose. In the UK, virulence for *Yr17* is now combined with virulence for *Yr1, 2, 6, 9* and the resistance of Carsten's V. Cultivars with combinations of all six of these genes would not be resistant.

Table 2 Virulence of *P. striiformis* for wheat cultivars with *Yr17* and in the UK (NIAB data)

Years	86	87	88	89	90	91	92	93	94	95	96	97	98	99
Yr17 cultivars %	0.2	1.3	1.6	1.0	0.3			1	17	27	31	34	24	14
V17 races %								**1**	**4**	**28**	**81**	**98**	**93**	**80**
Yr6/17 cultivars %													3.8	11
V6/17 races %													**2**	**20**

Where effective combinations of race-specific resistance genes occurred, the breeders were not able to select for any less highly expressed resistance, including any possible durable components. When virulence occurred, some of these cultivars were highly susceptible. To avoid these negative outcomes, and to capture genetic components controlling durable resistance, it is necessary to include sources of durable resistance in crosses, and to avoid the creation of new combinations of race-specific genes. This is easier said than done.

The level of resistance provided by *Yr18* on its own may not be sufficient in highly conducive environments for yellow rust. Even so, it may give useful protection of yield (Ma and Singh 1996). The possible linked character of leaf tip necrosis may assist in the selection of plants possessing this gene. Combining *Yr18* with other genes, such as *Yr29* and the components of resistance found in such wheats as Cappelle Deprez, could produce adequate and perhaps durable resistance for most environments.

Of papers so far published on the use of molecular genetic markers for yellow rust resistance genes, most make no contribution towards the manipulation and exploitation of durable resistance to the disease. Most have looked mainly at genes of large effect, and also of alien origin in wheat, for which it is easier to produce markers. The genes so far marked are mainly of the type that have already displayed their non-durable and race-specific effects, or are of the type that are most likely to do so if deployed commercially. One such paper describes linkage to *Yr17* using RAPD and SCAR markers (Robert *et al.* 1999). The authors claimed that this gene was durable in France, but gave no data on the extent of its use in France, and seemed unaware that the cereal rust pathogens are no respecters of national boundaries. Virulence for *Yr17* was already known in the UK (Table 2) and Denmark at the time of publication. As the authors were aware of this virulence, they suggested that the markers could be used to assist in combining (or pyramiding) genes - but offered no suggestion as to which genes should be combined. The idea of pyramiding genes is not new and, as indicated above, combining known genes effective in seedlings has not produced durable resistance to yellow rust. This indicates that the authors were more familiar with the techniques of molecular marker technology than with the history of breeding for resistance to yellow rust. In a non-molecular paper, McIntosh and Lagudah (2000) suggested a possible use of molecular markers for assisting the back-crossing of a gene such as *Yr24* derived from durum wheat, into a cultivar with known durable resistance, to provide resistance at early growth stages. This is a more focussed suggestion, but even so, much would depend on whether the molecular technique was quicker and cheaper than the classical back-cross method with testing for resistance.

A molecular marker possibly linked to *Yr18* was indicated by Singh *et al.* (2000) but it appears that practical application of this marker to selecting for *Yr18* would be difficult.

At the beginning of the 21st Century, then, the options for breeding positively for durable resistance to yellow (stripe) rust are still almost entirely by standard breeding and cytogenetic procedures, not enhanced by molecular marker technology. It will thus only slowly become easier as more genetic information becomes

available and with much more relevant development of molecular markers, which will be more difficult than for alien genes and genes of large effect.

References

Bariana HS, McIntosh RA, 1993. Cytogenetic studies in wheat XIV. Location of rust resistance genes in VPM1 and their genetic linkage with other disease resistance genes located in chromosome 2A. Genome **36**, 476-482.

Chagué V, Fahima T, Dahan A, Sun GL, Korol Abm, Ronin YI, Grama A, Röder MS, Nevo E, 1999. Isolation of microsatellite and RAPD markers flanking the *Yr15* gene of wheat using NILS and bulk segregant analysis. *Genome* **42**, 1050-1056.

Johnson R, 1988. Durable resistance to yellow (stripe) rust in wheat and its implications in plant breeding. In: Simmonds NW, Rajaram S, (eds) Breeding Strategies for Resistance to the Rusts of Wheat. CIMMYT 1988, Mexico DF CIMMYT.

Johnson R, Law CN, 1975. Genetic control of durable resistance to yellow rust *(Puccinia striiformis)* in the wheat cultivar Hybride de Bersée. *Annals of Applied Biology* **81**, 385-391.

Johnson R, Taylor AJ, 1980. Pathogenic variation in *Puccinia striiformis* in relation to the durability of yellow rust resistance in wheat. *Annals of Applied Biology* **94**, 283-286.

Law CN, Gaines RG, Johnson R, Worland AJ, 1978. The application of aneuploid techniques to a study of stripe rust resistance in wheat. *Proceedings of the 5th International Wheat Genetics Symposium, New Delhi, India* pp427-436

Ma H, Singh RP, 1996. Contribution of adult plant resistance gene *Yr18* in protecting wheat from yellow rust. *Plant Disease* **80**, 66-69.

McIntosh RA, Lagudah ES, 2000. Cytogenetical studies in wheat. XVIII. Gene *Yr24* for resistance to stripe rust. Plant Breeding **119**, 81-83.

McIntosh RA, Wellings CR, Park RF, 1995. *Wheat Rusts. An Atlas of Resistance Genes.*CSIRO and Kluwer Academic Publishers, Dordrecht, The Netherlands.

Peng JH, Fahima T, Röder MS, Li YC, Grama A, Nevo E, 2000. Microsatellite high-density mapping of the stripe rust resistance gene *YrH52* region on chromosome 1B and evaluation of its marker-assisted selection in the F_2 generation in wild emmer wheat. New *Phytologist* **146**, 141-154.

Robert O, Abelard C, Dedryver F, 1999. Identification of molecular markers for the detection of yellow rust resistance gene *Yr17* in wheat. *Molecular Breeding* **5**, 167-175.

Singh RP, Nelson JC, Sorrells ME, 2000. Mapping *Yr28* and other genes for resistance to stripe rust in wheat. *Crop Science* **40**, 1148-1155.

Wheat Breeding for Resistance to Yellow Rust in Iran

Abbas, Saidi

Cereal Research Department, Seed and Plant Improvement Institute (SPIT), Karaj, Iran

Introduction

Diseases are recognized as a major wheat production constraint in Iran. Due to various climatic conditions prevailing in different parts of the country, wheat is subject to attack by diseases the most important of which are rusts, bunts, fusarium head blight, and septoria leaf blotch. The damage they cause varies with the nature of the pathogens, severity of disease and climatic conditions.

Yellow (stripe) rust, caused *by Puccinia striiformis* f.sp. *tritici,* is the most important rust disease in Iran and is present in all wheat growing areas, especially where favorable climatic conditions prevail. Its importance from area to area depends on climate and the predominant cultivars. Although the disease was first reported in 1947 (Esfandiari 1947), it never caused significant economic loss until 1993. In 1993 and 1995, yield losses due to yellow rust were estimated at 1.5 and 1 million tonnes, respectively (Torabi *et al.* 1995). During the epidemic of 1993, the most widely grown, high yielding cultivars that were highly affected by yellow rust were Falat, Quds, Navid and Bayat.

CIMMYT crosses involving "Veery" resulted in germplasm that gave rise to many desirable and high yielding cultivars. The Veery 5 selections, in particular, became popular and were cultivated widely. Pakistan released the first Veery variety, Pak 81, in 1981. This, and others such as Seri 82, Pirsabak 85, Falat, CPAN 3004 and WH 542 were released in different countries in West and South Asia. They all belong to the Veery 5 family, and possess the 1 B/1 R translocation that carries the linked resistance genes *Sr31, Lr26, Yr9,* and *Pm8.* These genes conferred good resistance to the three rusts at the time of their release in the mentioned varieties.

Yellow rust pathotypes with virulence for *Yr9* appeared suddenly and in high incidence. The initial reports of the *Yr9* virulence in West Asia were received from Syria and Yemen in 1991 (Louwers *et al.*, 1992). This was followed by reports of *Yr9* virulence on Veery 5 cultivars in Iran in 1992, with epidemics occurring in 1993 and 1994 (Torabi *et al.*, 1995). Reports of yellow rust on Veery lines were received from Afghanistan but the extent and incidence was not determined. By

1996, yellow rust was widespread in the northern areas of the Punjab and throughout the foot-hills of the Himalayas in India.

In Iran, one of the major factors contributing to the yellow rust epidemics was the widespread planting of widely adapted variety Falat carrying genes *Yr7* and *Yr9*. From 1994, efforts have concentrated on the release of more varieties (21 varieties) with different genetic backgrounds, for a given zone as well as for the whole country. Such deployment of varieties has effectively controlled the disease and not much chemical control has been implemented since 1996. Newly released varieties are more resistant/tolerant to the diseases than the previous varieties.

Establishment of the Cereal Pathology Unit

Due to the recent epidemics of yellow rust and the lack of information on the races of the pathogen and their virulence, and the genetics of resistance in wheat, an urgent need for close collaboration between the breeders and pathologists was recognized. Direct intervention of his Excellency Dr. I. Kalantari, former Minister of Agriculture, led to the establishment of the Cereal Pathology Unit within the Cereal Research Department at SPII in 1993. Every effort was made to hire and train qualified pathologists, especially those who could work and interact with breeders and other scientists.

Several greenhouse complexes equipped with appropriate equipment were developed to carry out the research required in this field. In addition, mist irrigation systems were set up in several hot spot regions to create, artificially, the proper environment for yellow rust screening. This national laboratory soon became a center of excellence for yellow rust, as well as for research on other major wheat diseases in the country. This center provides national pathotype surveys for yellow rust, undertakes ongoing searches for new sources of resistance, carries out genetic analyses of these sources, provides rust screening for breeders, and carries out epidemiological as well as gene postulation research. Some of the achievements of the Pathology Section include race identification of 60 isolates in the last three years. Many of these activities are jointly with breeders.

Breeding wheat for resistance to yellow rust

Although wheat research in Iran started in 1930, breeding wheat for biotic stresses such as yellow rust was initiated only in recent years. The first breeding efforts were initiated by the School of Agriculture in 1930. In 1942 the first wheat variety, Shahpasand, a selection from local populations, and in 1962, the first wheat variety derived from local crosses, were released.

Up to 1994, all breeding efforts centered around selection of good parents, which were used in crosses without screening (i.e., at least artificially) of the segregating populations. Thus, resistance occurred without breeders' selection for disease resistance. Nowadays SPIT, provides assistance in screening for disease resistance under artificial inoculation of all segregating and advanced lines of wheat in Iran. In addition to breeding for yellow rust resistance, other main objectives of the wheat breeding program are to increase yield potential, stability, adaptation, and resistance/tolerance to abiotic stresses such as drought, heat, cold, salinity, pre-harvest sprouting, and durable resistance to other diseases and pests, with medium to high flour quality for traditional flat bread baking. Navabi (1997) and Saidi *et al.* (1999) reported, respectively, that for the period 1942-95, an average annual increase in yield of 1.02% and 0.9% occurred. Yield potential increase in Iranian varieties was associated with decrease in plant height, and increases in harvest index, number of grains/m2, protein yield/m2, and grain weight/spike.

Important features of the Iranian wheat breeding program include: study of local landraces as sources of resistance to yellow rust for possible use in the crossing blocks, utilization of germplasm received from International centers such as CIM-MYT, ICARDA, and NARS as sources of resistance to diseases, high yield potential, quality and other desirable agronomic characteristics, use of spring x spring, spring x winter, and winter x winter crosses, shuttle breeding for spring materials and utilization of doubled-haploid lines. Wheat research activities are implemented in 33 research centers/stations representing the four agro-climatic zone in Iran. Breeding objectives are targeted according to major environmental stresses (i.e. for biotic and for abiotic) in each zone. Generally, yield in :ones 1 and 2 are limited by biotic and abiotic stresses, respectively.

Breeding methodology

The Pedigree method was the oldest method used by Iranian breeders since the breeding program was initiated in the Cereal Research Department. However, from 1990 a modified bulk or selected generally bulk method generally replaced the pedigree method. This method combines advantages of both b%u)k and pedigree methods and saves time and money in comparison with the traditional pedigree r method. Moreover, due to the different biotic and abiotic stresses in the agroclimatic zones, a new breeding method, namely Modified Pedigree-bulk, has been utilized from 1998. When compared with modified (selected) bulk, this method allows faster evaluation of the segregating material (except in F2) and thus a reduction in labor required to evaluate the material. In addition, in early generations (i.e., F2 and F3) the number of desired crosses making up the next

generation will be increased (Saidi 1998). Three-way crosses, single crosses and backcrosses make up, respectively, about 70%, 20%, and 10% of all crosses.

Breeding strategies for yellow rust resistance

Efforts have also been made to the production of newly released variety or release candidates having durable adult plant resistance. Breeding for adult plant resistance is not as easy as is thought. Often environmental conditions would not lead to adequate infestation and subsequent evaluation of segregating materials would be done at the seedling stage. If this happens, selection based on the plant reactions to yellow rust at the adult plant stage may be confounded with seedling resistance. Due to very dry environmental conditions during the last few years uniform disease infestation in the field trials has not been possible. Therefore, it is imperative that the promising lines be tested to confirm the presence of seedling and/or adult plant resistance.

To ensure resistance to different yellow rust virulence factors, advanced promising lines are tested in several locations, to test their yield stability as well as reaction to yellow rust and other diseases. For example, seedling and adult plant reactions to yellow rust of a promising line (N-75-16, a CIMMYT origin with pedigree Shanghai7 // Hahn "S"*" 2/Prl "S") to be released in Zone 1 (i.e., Caspian sea shore area) has shown susceptible reactions to several yellow rust races at the seedling stage but has shown resistance at the adult stage in tests at several locations.

Another method to achieve a more durable resistance in our breeding program is use of different *Yr* genes, in combination Pyramiding strategy has been applied, using several strong resistance sources with the objective of producing durable resistance to wheat yellow rust. Genetic analysis will be conducted to confirm the nature of this resistance.

During the last five years more than 50 Doubled haploid (DH) wheat lines resistant to yellow rust have been produced. These DH analogues have been evaluated in yield trials in several locations over two successive years. At present, they are being tested for yield and quality as well as reactions to yellow rust at several locations, before their possible release.

Research achievements and wheat varietal diversification

As noted above, the epidemics of wheat yellow rust caused significant yield losses in 1993 and 1994. It was found extremely risky to depend on varieties of similar genetic background. Ever since then, efforts have been made towards the release of more varieties with different genetic backgrounds for a given agroecological zone as well as for the whole country. This has effectively controlled the disease and not much hemical control has been implemented since 1996. Newly released varieties are more tolerant to foliar diseases such as yellow rust, leaf rust, fusarium head blight, and karnal bunt. The current strategy is to utilize durable resistance genes in the breeding program.

In order to increase the wheat yield, and thus production, specially designed breeding programs are needed, involving both indigenous and exotic germplasm, breeding for multiple disease resistance, with greater emphasis on durable and/or race non-specific resistance for diseases, breeding for more resistance to abiotic stresses, particularly more water-use efficient cultivars. In addition, adoption of agronomic technologies (i.e. raised-bed systems with minimum to zero tillage) which allow improvement of soil organic matter, higher moisture retention, better weed control, less soil compaction, and reduced farming operational costs must be seriously considered wherever they are suitable.

References

Esfandiari A. 1947. Iranian cereal rusts. *Journal of Plant Disease.* 4: 67-76.

Navabi A.R. 1997. Contribution of genetic improvement in yield potential increase and changes in yield related traits in Iranian released wheat cultivars. In: Proceedings of the International Group Meeting *Wheat Research Needs Beyond 2000 AD. pp.* 345-351.

Saidi A. 1998. Strategy and use of breeding methods in Cereal Research Dept.: Past, Present, and Future. In the *5 th Iranian Congress on Crop Production and Plant Breeding.* Karaj, Iran.

Saidi A. Najafi M. Naseri M. Amin G. Nikzad A. Radmeh M. Kalateh M. Tarinejad A. 1999. Contribution of genetic improvement in yield potential increase and changes in yield related traits in Iranian wheat cultivars released from 1956-1997. In: Proceedings of the *6th Iranian Congress of Crop Production and Plant Breeding. pp.* 44.

Torabi M. Mardoukhi V. Nazari K. Afshari F. Foroutan A.R. Ramai M.A. Golzar H. Kashani A.S. 1995. Effectiveness of wheat yellow rust resistance genes in different parts of Iran. *Cereal Rusts and Powdery Mildews Bulletin.* 23: 9-12.

Louwers J.M. Van Silfhout C.H. Stubbs R.W. 1992. Race analysis of yellow rust in wheat in developing countries. Report 1990-1992. *IPO-DLO Report* 92-11, 23p. DLO Research Institute for Plant Protection. (IPO-DLO), Wageningen, The Netherlands.

Variability of Wheat Land Races for Reaction to Yellow Rust

H. Ketata[1], D. Sadekzadeh[2], M. Jarrah[1], M. Roustai[2], A. Yahyaoui[1] and H. Hassanpour [2]

[1]International Center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, Syria

[2] Dry Land Agricultural Research Institute (DARI), Maragheh, Iran.

Introduction

Wheat (*Triticum aestivum* L. em Thell) land races generally are not suitable for intensive wheat cultivation and produce lower grain yields than modern cultivars when grown under favorable conditions. Yet, they are still cultivated over large areas in certain countries of Central and West Asia and North Africa, where they are generally confined to remote highlands, poor soils, or other marginal areas. Land races, such as 'Sardari' in Iran, 'Local White' in Pakistan, and 'Ashure' and 'Kirik' in Turkey, are tolerant to prevailing abiotic stresses (e.g. drought, cold, heat) through years of gradual genetic adaptation. But they are generally poorly adapted (or susceptible) to rare events in those environments, such as foliar diseases or high rainfall. Because farmers with little or no selection generally propagate them, land races tend to be heterogeneous for various traits, and may therefore offer valuable genetic resources (Harlan 1975, Carnide and Guedes-Pinto, 1998, Keser *et al.*1998, Ketata *et al.*1998, Khelifi *et al.*1998, Zanatta *et al.*1998). This study reports on intra-land race variability of two wheat land races for reaction to yellow rust (*Puccinia striiformis* f. sp. *tritici*).

Materials and Methods

Spikes were randomly picked from a farmer's field grown to cv. Sardari near Maragheh, in Northwestern Iran. They were increased as separate head-row families and subsequently tested for several traits, including reaction to yellow rust under natural disease infection at DARI research station, Maragheh, during the season of 1997-1998. The same 106 families were also field grown and tested at ICARDA research station, Tel Hadya, Syria, during the same season, under artificial epiphytotics, using a local yellow rust inoculum.

In another experiment, wheat spikes were randomly taken from 3 different farms (designated as Farm1, Farm2, and Farm3) grown to cv. Ashure in eastern Turkey. Spikes (about 100) from each farm were increased into head-row families, and

tested for reaction to yellow rust during the season of 1998-1999 at ICARDA, Tel Hadya, under artificial epiphytotics. The same testing was repeated the following season at the same site on representative samples of 50 families from each group.

Host plant reaction to yellow rust was evaluated, using the modified Cobb scale, and thereafter conveniently converted to a 3-class grouping: resistant (R), moderately susceptible (MS) and susceptible (S) corresponding to a disease severity of (0-5%), (10-35%), and (> 40%), respectively. Chi-square and correlation tests were performed as appropriate.

Results and Discussion

Sardari

The land race Sardari has been rated highly susceptible, with scores of up to 100S. However, the 106 Sardari-derived pure lines revealed the appearance of resistant families, both at Tel Hadya, Syria and Maragheh, Iran. In general, disease severity was higher at Tel Hadya where it reached 100% for a large number of lines, in contrast to Maragheh, where most susceptible families scored 40-60% only, and where the highest score of 80% was exceptional. This is mainly due to the environmental conditions which were less conducive to good disease development at Maragheh, as compared to Tel Hadya, and perhaps also to the difference in race spectrum between the two sites. The distribution of yellow rust score was therefore significantly different between the two sites (Table 1). At Maragheh, there were more families in the MS class and less in the S class, in comparison with Tel Hadya. However, the proportion of resistant families was similar in the two sites. Of 13 resistant families at Maragheh and 15 at Tel Hadya, 9 families were common, showing the value of those 9 families, as having resistance to a wider spectrum of yellow rust races.

Table 1. Distribution of 106 Sardari-derived pure lines for their reaction to yellow rust at sites in Iran and Syria, 1997-1998.

Class of yellow rust score	Sites	
	Maragheh, Iran	Tel Hadya, Syria
Resistant (R)	13	15
Moderately susceptible (MS)	71	7
Susceptible (S)	22	84
Total	106	106

These results indicate that, although the original Sardari is a typically susceptible land race, it did possess a sizeable amount of genetic variability, with respect to yellow rust, to allow the extraction of useful, resistant families.

Ashure

The original land race Ashure has been rated highly susceptible. Nevertheless, the pure lines derived from the three Ashure fields, comprised in each case, a number of resistant families (Table 2).

Table 2. Distribution of Ashure-derived pure lines, from each of 3 farms, for their reaction to yellow rust, Tel Hadya, Syria, 1998-1999.

Yellow rust class	Number of families in Ashure sample from		
	Farm1	Farm2	Farm3
Resistant (R)	6	7	10
Moderately susceptible (MS)	21	29	44
Susceptible (S)	92	54	55
Total	119	90	109

Although the distribution of the derived lines over yellow-rust classes was statistically different among the three farms, the proportion of resistant families did not differ across farms, with an average value of about 7%. The repeated testing in 1999-2000 of a sub sample of 50 lines from each group (farm) confirmed this result. The coefficients of correlation of scores across the 2 seasons were 0.85**, 0.82**, and 0.85**, for the 3 farms, respectively. These results again confirm the opportunity to extract from a susceptible land race, pure lines possessing a good level of resistance to yellow rust.

The fact that yellow rust-resistant pure lines could be derived from land races reputed for their high susceptibility to the disease points to the importance of land races as a valuable source of useful traits for breeding and other purposes, and therefore to the need for their preservation and maintenance for immediate and future use.

References

Harlan JR, 1975. Our vanishing genetic resources. *Science* **188**: 618-621.

Carnide V, Guedes-Pinto H, 1998. *In situ conservation of a Portuguese bread wheat landrace*: Barbela. In: Zincirci *et al.* (Eds.), *Proceedings of the International Symposium on In situ Conservation of Plant Genetic Diversity*, p. 227-230. CRIFC, Ankara, Turkey.

Keser M, Bolat N, Altay F, Bayramoglu HO, Colak N, Sever L, 1998. Performance of land races versus modern cultivars in durum wheat. In: Zincirci *et al.* (Eds.), *Proceedings of the International Symposium on In situ Conservation of Plant Genetic Diversity*, p. 109-111. CRIFC, Ankara, Turkey.

Ketata H, Sadekzadeh D, Tahir M, Amiri A, 1998. Variability of morphophysiolocal traits within a wheat (*Triticum eastivum* L. em Thell) land race. In: *Proceedings of the 9th International Wheat Genetics Symposium*, Saskatoon, Saskatchewan, p. 253-255. University Extension Press, University of Saskatchewan, Canada.

Khelifi D, Cherdouh A, Carillo JM, Nieto-Talidriz MT, 1998. Variability for glutenin proteins in Algerian durum wheat landraces. In: *Proceedings of the 9th International Wheat Genetics Symposium*, Saskatoon, Saskatchewan, p. 260-262. University Extension Press, University of Saskatchewan, Canada.

Zanatta ACA, Keser M, Kilinc N, Brush SB, Qualset CO, 1998. Competitive performance of wheat landraces from western Turkey: basis for locally based conservation of genetic resources. In: *Proceedings of the 9th International Wheat Genetics Symposium*, Saskatoon, Saskatchewan, p. 379-380. University Extension Press, University of Saskatchewan, Canada.

Breeding Winter Wheat for Yellow Rust Resistance in Tajikistan

Eshanova Z. Sh.[1], F. Kosimov [1], A. Yorov[2], E Khuseinov[3], S. Naimov[4], S. Oripov[5], B. Sanginov[6] and A. Morgounov[7]

[1] Tajik Research Institute of Farming, Sharora; [2] Vakhs Branch of the Tajik Research Institute of Farming, Kurgan-Tube; [3] Kulyab Agricultural Research Station, Dangara; [4] Tajik Institute of Genetics and Plant Physiology; [5] Tajik Variety Testing Commission, Dushanbe; [6] GTZ-CIMMYT Project, Dushanbe; [7]CIMMYT-Central Asia and Caucasus Program, Almaty.

Introduction

In Tajikistan, valleys suitable for cropping occupy only 7% of the total land area of 143,100 km^2. Wheat is important, occupying up to 350,000 ha. It was a minor crop in the past when cotton dominated but in the 90s the area increased because of concerns about food security. Most wheat is planted in the fall (November-December) and harvested in May-June. Around 40% is grown under irrigation in lowlands and the rest is under rainfed conditions in the mountain foothills or on top of the steep hills where application of machinery is difficult. Most cultivated varieties are facultative or even spring type and are rarely damaged by cold. Wheat yields can reach 6-8 t/ha under irrigation.

Wheat breeding started in Tajikistan in 1932 at Sharora, 20 km from Dushanbe where the Tajik Research Institute of Farming is now situated, using selection from locally grown land races and germplasm from the former USSR. Around forty varieties of bread and durum wheat were developed and released in the past. These included selections for rainfed environments, like Irody 1006 and Surkhak 5688. For irrigation, Siete Cerros was released in the 70s and is still cultivated in some valleys. In the 80s and early 90s new high-yielding Tajik varieties Ferrugineum 67, Tajikskaya 13, Novruz and Sharora were released. The latter two occupied main areas when the crop area expanded in mid 90s. Two durum wheat varieties Vatan and Bakht were also released by the institute and are grown on a small area.

Yellow rust was not a major problem until the expansion of irrigation and use of susceptible varieties, combined with high winter rainfall, resulted in severe epidemics in 1997-1999 when some fields produced hardly any grain. Almost all the varieties (Siete Cerros, Novruz, Sharora, Bezostaya 1, Progress, Zhetisu) cultivated in Tajikistan at that time were susceptible to various degrees.

Resistant varieties were imported from USA (Jagger), Turkey (Sultan, Atay, Kinaci) and Kazakhstan (Steklovidnaya 24, Karlygash). Although this improved the situation, it is still necessary to develop local varieties resistant to yellow rust and adapted to local conditions.

Table 1. Yield of winter wheat lines resistant to yellow rust in two locations in Tajikistan in 1998.

Variety/Line	Country of origin	Yield, t/ha			Rank
		Dangara	Vakhsh	Average	
NAVRUZ (Local check)	TAJ	2.63	3.64	3.14	38
SHARORA (Local check)	TAJ	3.50	2.86	3.18	35
ZANDER-12	TCI	4.70	4.80	4.75	1
ZCL/3/PGFN//CNO67/SON64 (ES86-8)/4/SERI/5/UA-2837	TCI	4.07	4.68	4.37	2
CHAM6//1D13.1/MLT	TCI	3.80	4.68	4.24	3
PTZ NISKA/UT1556-170	OSU-CIT	4.03	4.42	4.23	4
PTZ NISKA/UT1556-170	OSU-CIT	4.00	4.30	4.15	5
SULTAN95	MX-OR	3.80	4.16	3.98	6
KHARKOVSKAYA106	UKR	3.97	3.90	3.93	7
JUP/4/CLLF/3/I114.53/ODIN/ /CI13431/WA00477	MX-OR	3.23	4.55	3.89	8
ADMIZ	ROM	3.53	4.16	3.85	9
GENE	USA	4.03	3.64	3.84	10
VORONA/HD2402	MX-TCI	4.50	3.10	3.80	11
CO724377/NAC//SERI	MXORTCI	2.63	4.94	3.79	12
ORE F1 158/FDL//BLO/3 /SH14414/ CROW	SYR	3.57	3.90	3.73	13
PYN/BAU	TCI	3.57	3.90	3.73	14
MADSEN	USA	3.57	3.90	3.73	15

Since 1995 the wheat breeding program at Tajik Research Institute of Farming established close collaboration with CIMMYT and ICARDA.Germplasm was also obtained from Iran, China, Uzbekistan and Kazakhstan, Ukraine and Russia. In total, 2500 entries were introduced and screened at Sharora under natural epidemic conditions. A few hundred resistant lines were selected and went through several cycles of selection for adaptation to local conditions, and yield trials were conducted for selection of the most promising lines (Table 1). Two lines were identified that combined good resistance to yellow rust with high yield and other good agronomic traits and were submitted for official testing under the names Norman (to honor Dr. N. Borlaug) (5th FAWWON-37, ORE F1.158/FDL//BLO/3/SHI4414/ CROW) and Tacika (stands for Tajikistan-CIMMYT-ICARDA cooperation) (5th FAWWON-35, TAS/SPRW//ZAR) in 2000.

Till 1999 the work on breeding for yellow rust resistance was mainly based at Sharora but for 1999-2000, the breeding nurseries (preliminary yield trials and yield trials) and screening for resistance were expanded to the Kulyab region (Kulyab Agricultural Experimental Station in Dangara) and Vakhsh Valley (Vakhsh Branch of the Tajik Institute of Farming in Kurgan Tube) as part of the activities of GTZ-CIMMYT project. For 2000-2001 the yield trials were further expanded to the Northern Sugdt region in Khodjent (Khodjent Agricultural Research Station), Zeravshan Valley (Pendjikent Agricultural Research Station), Sovetskiy county (farm Chorubkul), mountainous Faizabad region (a station of the Tajik Institute of Genetics and Plant Physiology). This covers the major wheat growing environments assuring adequate testing for adaptation and yellow rust resistance. Advanced lines starting from preliminary yield trial were also sent to the Agricultural Research Institute in Otar, Kazakhstan for tests of yellow rust resistance in seedlings and to the Kazakh Research Institute of Crop Protection to test for common bunt resistance.

Table 2. The yield and yellow rust performance of the winter wheat varieties tested in Uniform Yield Trial in 2000 (data from the Tajik Variety Testing Commission).

Variety	Origin	YR	Yield (t/ha)				
			Sovetsk	Bokhtar	Nau	Average	Rank
Bezostaya 1 (LC)	Russia	20	3.38	3.05	2.80	3.08	8
7C (LC)	Mexico	30	2.24	3.50	3.10	2.95	14
Sharora (LC)	Tajikistan	40	2.49	2.60	2.30	2.46	34
Steklovidnaya 24	Kazakhstan	10	3.63	3.45	3.00	3.36	1
PYN/BAU	Mexico-Taj	0	3.66	3.25	3.02	3.31	2
Karlygash	Kazakhstan	5	3.54	3.55	2.60	3.23	3
Kauz	Mexico	0	3.64	3.07	2.92	3.21	4
Krasnovodopadsk-aya 25	Kazakhstan	0	3.49	3.30	2.70	3.16	5
Jagger	USA	5	3.40	3.20	2.85	3.15	6
Yanbash	Uzbekistan	5	3.51	3.10	2.77	3.13	7
Bogarnaya 56	Kazakhstan	20	3.34	3.37	2.25	2.99	9
Zhetisu	Kazakhstan	0	2.96	3.10	2.87	2.98	10

The Tajik Variety Testing Commission conducted a uniform trial of the best local and introduced varieties in three regions of the country in 1999-2000. The data (Table 2) assisted in identification of several new varieties combining resistance and high yield. The data may also guide aid agencies/projects for selecting the varieties that can be imported from outside for cultivation in Tajikistan.

Monitoring of the yellow rust population is important and started in 1999 based on CWAYRTN (Central and West Asia Yellow Rust Trap Nursery) distributed by ICARDA. Initially this was planted only in Sharora and later in 1999 was expanded to the Vakhsh Valley (Kurgan Tube - 100 km from Dushanbe over a mountain range) and Tursun-Zade regions (50 km from Dushanbe in the same valley). The results in 2000 showed that the population of yellow rust is similar in two regions and that resistance genes that are effective are *Yr 5, 8, 10, 12, 15, 17, 18 YrSp.* Unfortunately, the facilities in Tajikistan do not allow tests for resistance at the seedling stage for identification of the genes. It is intended to continue identification of the best diverse lines with yellow rust resistance and pyramiding of resistance genes through a crossing program.

Table 3. Central and West Asia Yellow Rust Trap Nursery (CWAYRTN-98) Tajikistan (Sharora) 1999.

Cultivar	Yr gene	Origin	Yr	Lr
Chinese 166 (W)	1	IPO-NL	90 S	
Lee (S)	7	"	80 S	
Heines Kolben (S)	6	"	60 MS	
Vilmorin 23 (W)	3V	"	90 S	
Moro (W)	10	"	90 S	
Strubes Dickkopf (W)	SD	"	20 MR	
Suwon 92xOmar (W)	SO	"	0	15 R
Clement (W) 9,2+	"	20 MR		
Hybrid 46 (W)	4+	"	15 R	20 MR
Reichersberg 42 (W)	7+	"	10 R	10 R
Heines Peko (S)	6,2+	"	5 R	15 R
Nord Desprez (W)	3N	"	10 R	20 MR
Compair (S) 8,18	"	10 R		
Carstens V (W)	?	"	10 R	
Spaldings Prolific (W)	SpP	"	10 R	
Heines VII (W)	2, 11 +	"	0	
Aroona*5/Yr1 (S)	1	PBI-AUS	5 R	
Aroona*6/Yr5 (S)	5	"	20 MR	5 R
Aroona*6/Yr8 (S)	8	"	90 S	
Aroona*3/Yr15 (S)	15	"	70 S	
Aroona*6/Yr 15 (S)	15	"	5 R	
Aroona*6/Yr 17 (S)	17	"	40 MR	
Avocet1R` (S)	A	"	0	5R
Avocet`S` (S)	-	"	30 MR	
Avocet S*6/Yr5 (S)	5	"	40 MR	
Avocet S*6/Yr8 (S)	8	"	90 MR	
AvocetS*6/Yr15 (S)	15	"	10 R	
M2435 (S) -	"	10R		
M2435*6/Yr5 (S)	5	"	0	

Contd.....

Contd (Table 3)

M2435*6/Yr10 (S)	10	"	90 S	
Federation (S)	-	"	15 MR	15 MR
Fed 4*/Kavkaz (S)	9	"	20 MR	
Jupateco `S` (S)	-	"	90 S	
Jupateco `R` (S)	18	"	90 S	
Kalyansona (S)	2	"	80 S	
Cranbook (S)	2	"	80 S	
Corella (S)	7	"	90 S	
Oxley (S)	6	"	80 S	
Cook (S)	APR	"	90 S	
Anza (S)	A,18	"	80 S	
Sonalika (S)	-	"	50 MR	
T.spelta (Inter) (S)	5	"	60 MS	
Gerek 79	-	Turkey	80 S	
Cham 1	-	ICARDA	0	
Seri 82	7,9	CIMMYT	80 S	
Morocco	Check	-	80 S	
Almout (W)	-	Iran	80 S	
Darab 2 (W)	-	"	100 S	
Nicknejab (S)	-	"	10 R	
Morocco	Check		90 S	
M-70-12 Mahdavi (S)	-	"	5 R	
W-70-15 Atrak (S)	-	"	40 MR	
C-70-16 Zarrin (F)	-	"	10 R	
C-70-20Alvand (W)	-	"	30 MR	
W-18 Bow`S`/NKT(Tajen) (S)	-	"	20 MR	
Almout/Ti71/3Maya//BB/Linia /4/Karaj2/5Anza (S)	-	"	80 S	
Erith 15236 (S)	-	Ukraina	50 MR	
Lut 17044.12 (S)		"	20 MR	
Lut 20133 (S)		"	80 S	
Morocco	Check		90 S	
Lut 20148 (S)	-	"	60 MS	
Lut 20161 (S)	-	"	100 S	
Lut 20191 (S)	-	"	70 MS	
Eritrspernum 5678/87 (S)	-	"	10 R	
Lut 9489 (F)	-	"	70 MS	
Krasunia Odesskay (S)	-	"	40 MR	
Ukrainka Odesskaya (S)	-	"	30 MR	
Vimpel Odesskiy (S)	-	"	70 MS	
Fantaziya Odesskaya (S)	-	"	80 S	
Morocco	Check		90 S	
Zabava Odesskaya (S)	-	"	80 S	
Nadiya (S)	-	"	100 S	
Darunok (S)	-	"	20 MR	
Porada (S)	-	"	80 S	
Strunok (S)	-	"	60 MS	

Contd.....

Contd (Table 3)

Polovchanka (F)	-	Russia	30 MR
Knayzjna (F)	-	"	60 MS
Demetra (F)	-	"	20 MR
Zolotaya (S)	-	Ukraina	80 S
Morocco	Check	-	90 S
Zimorodok (F)	-	Russia	90 S
Umanka (S)	-	"	100 S
Pobeda (F)	-	"	40 MR
Ekho (F)	-	"	20 MR
Ofeliya (S)	-	"	60 MS
Bezostaya (S)	-	"	60 MS
Spartanka (F)	-	"	90 S
Yuna (S)	-	"	50 MR
Skifiyanka (F)	-	"	60 MS
Morocco	Check	-	80 S
Dakha (S)	-	"	70 MS
Sphera (S)	-	"	100 S
Eika (S)	-	"	60 MS
C-1252 (S)	-	Turkey	80 S
Ikizce-96 (S)	-	"	70 MS
Kiziltan 91 (F)	-	"	40 MR
Cham 6 (S/F)	-	Syria/Lebanon	40 MR
Cham 3 (S/F)	-	"	40 MR
Cham 5 (S/F)	-	Syria	0
Morocco	Check	-	90 S

Improvement of Wheat Yellow Rust Resistance in Kazakhstan and Uzbekistan Through Sub-regional Co-operation

A. Absattarova[4], S. Baboyev[1], K. Bulatova[4], M. Karabayev[2], M. Koishibayev[3], A. Kokhmetova[4], V. Kuklacheva[1], A. Morgunov[2], S. Rsaliev[5], A. Sarbayev[4], R. A. Urazaliev [4], M. Yessimbekova[4] and C. R. Wellings[5].

[1] Institute of Genetics and Molecular biology, Tashkent, Uzbekistan; [2] CIMMYT - CAC, Kazakhstan; [3] Kazakh Institute of Crop Protection, Almaty, Kazakhstan; [4] Kazakh Research Institute of Farming, Almaty region, Kazakhstan; [5] Agricultural Research Institute, Otar, Djambyl region, Kazakhstan; [6] University of Sydney, Plant Breeding Institute Cobbitty, Australia.

Introduction

In Central Asia wheat is grown on 15 x 10^6 ha, including 5 x 10^6 ha of winter or facultative wheat and 10 x 10^6 ha of spring wheat. In recent years wheat stripe rust (*Puccinia striiformis* f. sp. *tritici*) was among the major factors that adversely affected yield and quality. Stripe rust is principally a disease of wheat in areas with cool and wet environmental conditions (2-15° C), which are mainly associated with northern latitudes or cooler years (Roelfs *et al.* 1992). Although the weather in spring and summer is usually unfavorable, stripe rust survived and spread in Southern Kazakhstan, Uzbekistan and Kyrgyzstan in 2000. It never caused serious economic damage before the epidemics that have occurred since 1998, when yield losses of commercial varieties were up to 20-35% on susceptible varieties. In Kazakhstan and Uzbekistan the area affected by yellow rust could be 1.5 x 10^6 ha. In 2000 this caused initiation of informal cooperation with the following objectives:

a) to evaluate the level and distribution of wheat stripe rust in Kazakhstan, Kyrgyzstan and Uzbekistan.
b) to identify the resistance genes in commercial varieties or new lines adapted to the region;
c) to develop germplasm combining resistance to yellow rust with high yield, broad adaptation and good end-use quality.

Support for the project was given by the CIMMYT-CAC office in Almaty.

Materials and Methods

A total of 250 samples of wheat were included in field tests, including 100 from the Central and West Asian Yellow Rust Trap Nursery (CWAYRTN), 84 from the CAC- Regional Winter Wheat Exchange Nursery and 66 local sources of stripe rust resistance from the Kazakh Gene Bank. Cultivar Morocco was used as the susceptible variety for multiplication of the pathogen in the greenhouse and as spreader in the field. A large collection of yellow rust samples of *Puccinia striiformis* from 34 locations over the region was used in this study. Disease severity and reaction types were recorded following McIntosh *et al.* (1995). Inoculum used in field tests was a mixture of identified isolates maintained on susceptible varieties. Thus material was screened to stripe rust races that predominated in the region. Seedling tests were carried out by Dr. C. R. Wellings in Australia to determine the probable presence of known major genes in seedlings of commercial varieties.

Results and Discussion

Effective and relevant surveys of variability of the pathogens are fundamental to the achievement of genetic control of the cereal rusts (McIntosh *et al.*, 1995). Such surveys were conducted in spring - summer 2000 over a route of 2000 km in Kazakhstan, Kyrgyzstan and Uzbekistan. During 1999 severe stripe rust occurred in most regions of Central Asia, but was less severe in 2000.

Previous analyses of the stripe rust population, including isolates from wheats of Southern Kazakhstan and West Siberia in 1986-1989, were conducted by the Kazakh Agricultural Research Institute (Otar). These identified 9 races on wheat and 6 on wild cereals. Four races (7E148, 7E156, 7E140 and 39E158) were predominant, as well as being the most virulent. In Kyrgyzstan, Tajikistan and Uzbekistan a survey by the Central Asian Institute of Phytopathology (CAIP) in Tashkent in 1977-89 reported races 6E20, 6E148, 7E148, 7E150, 15E150 (Koishibayev M., personal communication). More recent data are not available.

Surveys of wheat stripe rust for Djambyl, Almaty and Taldykorgan regions of Kazakhstan and Issyk-kool region of Kyrgyzstan, indicated that differences between the level of disease development depended on genotypes and locations. Reaction of the high yielding adapted and widely grown commercial variety Steklovidnaya 24 varied from susceptible (up to 75% leaf area infected) to resistant (5R and 10R) in a number of locations of the Jambul region. Zhetisy, grown in fewer locations, varied from 9.4 to 17.5%. Besostaya 1, Erithrospermum 350 and Kiyal were highly susceptible but Tilek was less susceptible. In Uzbekistan, data from the Institute of Genetics and Molecular Biology showed that Kupava, Kroshka and Umanka had up to 40% infection in foci while Sanzar 8 reached 70-

80%. Samples of yellow rust collected in these surveys were sent to CAIP for multiplication in growth chambers, thus providing an initial spore collection for tests in growth rooms and inoculation in the field.

A severe epidemic of stripe rust in the Western part of the Almaty region decreased in intensity from West to East indicating the probable spread of air born inoculum from the Western parts, including Shu valley, Uzbekistan and Tajikistan. The hill ranges of Zailyisky, Kyrgyzsky and Zhungarsky Alatau 50-100 km wide prevented further spread.

Winter wheat varieties from Kazakhstan and CIMMYT included 25 entries planted in Umbetaly farm demonstration plots, subjected to naturally occurring races of yellow rust (Table 1). Since there was no previous breeding work for stripe rust resistance, most of the released commercial varieties were susceptible. A few Kazakh varieties such as Almaly, Arap, Aksham and entries from CIMMYT, 245/99 (FAWWON-6 PTZ NIS KA/VT 1556-170), 2909 and 345/99 were resistant.

Table 1. Reaction of adult plants of a set of commercial winter wheat varieties to *P. striiformis*, Umbetaly, Almaty reg., 2000.

Varieties and lines	Severity, min, %	Severity, max, %	Average, %
Steklovidnaya 24	25	100	50-75
Sultan	5-10	100	50-75
Opaks	5-10	75	25-50
Erithrospermum 350	50	100	75-100
Besostaya 1	1-5	25	5-10
N°245-99	0-1	25	5-10
Phyrotrix	5-10	100	75-100
Arap	0-1	10	5
Almaly	0-1	10	5
Progress	10-25	100	50-75
N°241/99	25	75	25-50
Bayandy	50	100	75-100
Yuzhnaya 12	50	100	75-100
N°345-99	5-10	50	10-25
N°15116	10	75	50-75
Aksham	0-1	25	5-10
N°300/99	1-5	50	5-10
N°289/99	5	50	10-25
N°11115	5	50	10-25
N°15742	0	1	0
N°2897	50	100	50-75

The Yellow Rust Trap Nursery (CWAYRTN-2000) including standard set of differentials, commercial cultivars and lines of wheat was studied at several locations (Table 2). Virulence for *Yr5* (Avocet) and *Yr17* (Aroona) occurred at Kyrgyzstan, but not at two locations of Kazakhstan. Virulence for *Yr8* (Aroona) was high at two sites of Kazakhstan, but low in Kyrgyzstan, although virulence for Yr8 (Avocet) was observed at all three locations.

Table 2. Field responses to *Puccinia striiformis* f. sp. *tritici* among the PBI (Sydney) Isogenic Differentials and other differential varieties in different locations of Central Asia.

Isogenic lines, European Differentials	Testing Sites - Severity/field response		
	Almaty, hill-steppe zone, Kazakhstan 8.06. 2000	Almaty, hill zone, Kazakhstan 9.07.2000	Bishkek, hill Steppe zone Kyrgyzstan 20.05.2000
Aroona'S 70S	30MS	20MR	
Aroona*5/Yr1	20MR	25MR	35MS
Aroona*6/Yr5	20MR	5R	35MS
Aroona*6/Yr8	70S	50S	10MR
Aroona*3/Yr15	R	5R	R
Aroona*6/Yr17	R	5R	35MS
Avocet'R'(YrA)	R	5R	15MR
Avocet'S' 70S	5R	R	
Yr5/6*Avocet'S'	R	R	80S
Yr8/6*Avocet'S'	80S	50MS	85S
Yr9/6*Avocet'S'	60MS	20MR	20MR
Yr10/6*Avocet'S'	35MR	R	R
Yr18/3*Avocet'S'	10MR	R	10MR
Yr15/6*Avocet'S'	40MS	R	85S
M2435'S' 70S	10MR	20MR	
M2435*6/Yr5	20MR	R	R
M2435/Yr10	15MR	5R	40MS
Heines VII (Yr2, 11, 25?)	R	R	R
Hybrid 46 (Yr4+)	R	5R	R
Anza (Yr18+A)	40MR	5R	50MS

(**Editor's Note:** In columns two and three of this table Avocet S was recorded as resistant. Susceptibility in other Avocet lines in these columns is therefore not expected and the data indicate errors in the table. R. Johnson).

The data indicate that *Yr4+* (Hybrid 46) and Heines VII provided the highest resistance and would be potentially available for transfer to wheat cultivars in Central Asia. [Editorial comment. Although Heines VII is also highly resistant in the region, it possesses at least three genes, all of which are known to be race-specific (Table 2). When these are overcome it is highly susceptible and therefore

should not be used for breeding in the region (R. Johnson)]. The gene *Yr18* from Anza is interesting for breeding as a source of durable resistance; but it varied from 5R-40MR in Kazakhstan to 100S in Tajikistan indicating that further studies are needed to use this source in breeding programs.

Data from the 3rd Elite Yield Trial in 1999 (severe yellow rust year) distributed by International Winter Wheat Improvement program (IWWIP-Turkey) were used to compare results in Kazakhstan, Azerbaijan, Tajikistan, Turkey and Uzbekistan (Tables 3 and 4). Some wheat lines were susceptible (Katya 1, SN64//SKE/2* ANE/3/SX/4/BEZ/5/JUN) or resistant (BWD, VORONA/HD2402) across all locations. However, most of them showed variable degrees of resistance suggesting that either the environment or the pathogen population or, most likely both, differ between locations. The closest correlations were found between Almaty, Ankara (Turkey) and Tashkent (Uzbekistan). Since Ankara is routinely used by IWWIP for yellow rust screening, it is likely that the germplasm selected in Turkey will be resistant in Almaty and Tashkent. The variation in the yellow rust performance across the region strongly suggests the necessity of closer regional collaboration.

Table 3. Yellow rust severity in the 3rd Elite Yield Trial across Central Asia and Turkey in 1999 (data from International Winter Wheat Improvement Program).

Pedigree	Baku, AZ	Almaty, KZ	Red Fall KZ	Dushanbe TJ	Adana TK	Ankara TK	Tashkent UZ
BEZOSTAYA1	42	40	5	43	20	50	10
KATIA1	53	10	55	60	40	70	30
SULTAN95	9	20	28	15	0	0	0
CHAM4/TAM200 /FDL 483	23	20	10	18	0	20	0
DRUM-1	25	15	8	25	10	60	30
SHARK-1	1	40	10	3	0	0	0
338K1.1//ANB/ BUC OR	1	40	38	3	0	15	0
F1.158/FDL//BLO/3/ SHI4414/CROW	1	15	15	12	0	0	10
CHAM6//1D13.1/MLT	7	10	20	10	0	0	10
BLUEGIL-3	45	40	25	87	10	20	30
BWD	1	0	5	5	0	0	10
AGRI/NAC//MLT	27	0	15	30	5	5	0
VORONA/HD2402	1	5	5	7	0	0	0
VORONA-8	7	40	15	87	20	70	0

Contd....

Contd (*Table 3*)

SN64//SKE/2*ANE/3/ SX/4/BEZ/5/JUN	28	20	23	57	40	45	30
HATUSHA-7	15	40	10	8	0	20	0
NVSR3/5/BEZ/TVR/5/ CFN/BEZ//SU92/CI13645 /3/NAI60/4/EMU/7/ KATYA A1	9	20	10	5	5	20	10
NEELY/SPN//SPN/3/SPN/ /63.189.66.7/BEZ	28	40	60	43	0	50	10
TEMU39.76/CHAT//CUPE /3/M1223.3D.1D/ALD	30	20	13	43	20	60	0
Average	19	23	19	30	9	27	9

It is known that gene *Yr9*, on the 1BL/1RS chromosome, was effective ten years ago in many varieties in the republics of the former Soviet Union. Wellings *et al.* (2000) indicated that *Yr9* remained effective in India, China, South Africa and Australia. However, this source became ineffective in Central Asia. Gliadin (Gli-B3) markers have been used for identification of the rye translocation in a set of regional varieties. It was found that most carriers of 1BL/1RS (Ekinchi, Nairi 131, Ani 352, Ani 591, Jalvar, Bermet, Mtskhetskaya 1, Ulugbek 600, Sharora, Ozoda) were highly or moderately susceptible to yellow rust.

In tests by Dr. C.R. Wellings on the CAC Winter Wheat Exchange Nursery, consisting of entries from all eight countries of the region, with four isolates of *P. striiformis* with known virulence, several were resistant to all four isolates. Barakatly, Karakylchyk 2, Alyndza 84, Turan, Shiraslan 23 (all durum wheats from Azerbaijan),HYS/7C//KRC(ES84-16)/3/SERI (Guncha) (Turkmenistan), Armyanka 60, Ani 326 (Armenia), Erithrospermum 760 (Kyrgyzstan), Altyn Masak (Kazakhstan), Yanbash.

Ulugbek 600 (Uzbekistan) and Sharora (Tajiikistan) were all resistant. Known genes were postulated such as *Yr1* in Krasnovodopadskaya 25, Yuzhnaya 12, Pamyat 47 (Kazakhstan), Adyr (Kyrgyzstan) and Ozoda (Tajikistan). *Yr9* could be present in Skiphyanka (Turkmenistan) and probably *Yr6* and *Yr7* or combinations of these in Taragi (Azerbaijan), SN64//SKE/2*ANE//3/SX/4/BEZ/5/SERI (7H) (Bitarap) (Turkmenistan), Norman (5th FAWWON-37, ORE F1.158/FDL//BLO/3/SHI4414/CROW) and Tacika (5th FAWWON-35, TAS/SPRW//ZAR) (Tajikistan).

Table 4. Coefficients of correlation between yellow rust infection of 18 winter wheat genotypes from 3rd EYT in different locations of Central Asia and Turkey, 1999.

	Baku AZ	Almaty KZ	Red Fall KZ	Dushanbe, TJ	Adana TK	Ankara TK	Tashkent UZ
Baku AZ	X	0,102	0,198	0,324	0,68	0,738	0,407
Almaty KZ		X	0,379	0,303	0,251	0,689	0,587
Red Fall KZ			X	0,671	-0,012	0,319	0,47
Dushanbe, TJ				X	0,651	0,278	0,295
Adana TK					X	0,623	-0,103
Ankara TK						X	0,595
Tashkent UZ							X

From this Nursery 18 wheats were chosen that were adapted to most locations and demonstrated good resistance across several sites. These included Yuzhnaya 12, Sapaly, Naz, Oktyabrina 70, Almaly, Arap, Taza (Kazakhstan), Kupava, Knyazhna, Umanka (Russia), BDME-9, Norman (5th FAWWON-37, ORE F1.158/FDL//BLO/3/SHI4414/CROW), Sultan 95 (Turkey), Ani 591, Nairi 149 (Armenia), Tilek, Adyr (Kyrgyzstan) and Ulugbek 600 (Uzbekistan). The second group included high yielding susceptible varieties such as Skiphynka, Zhetisu, Steeklovidnaya 24, Karlygash, Bermet, Sanzar 8 and Sharora. As possible sources of durable resistance Anza *(Yr18),* BWKLDN-95, BWKLDN-9 and BWKLDN-33 as well as entries with stable resistance to yellow rust based on global observations in international nurseries (FAWWON) were selected and included in crosses.

There were two types of crosses: a) for development of rust resistant high yielding germplasm adapted to target regions. Commercial wheat cultivars with adaptability and resistance were intercrossed (100 crosses); b) for genetic analysis, crosses were made of resistant varieties to susceptible varieties (Morocco and Avocet) and to several isogenic lines shown to confer resistance to stripe rust in this region (*Yr10*, *Yr2+* (Heines VII) and *Yr4+* (Hybrid 46). This type included 40 crosses made in spring 2000 and planted for multiplication in the field near Almaty and in a greenhouse in Tashkent The segregating populations will be used to estimate the number of genes for resistance in the varieties.

Plans for 2001 include observations on F1 hybrids, additional crosses, further screening of germplasm to identify adapted and resistant genotypes and establishment of a reliable protocol for screening seedlings and adult plants.

References

Roelfs AP, Singh RP, Saari EE, 1992. *Rust Diseases of Wheat: Concepts and Methods of Disease Management.* Mexico, D.F.:CIMMYT. 81 p.

McIntosh RA, Wellings CR, Park RF, 1995. *Wheat Rusts: An Atlas of Resistance Genes.* CSIRO. Australia 1995.

Wellings CR, Singh RP, McIntosh RA, Yayhaoui A, 2000. The assessment and significance of pathogenic variability in *Puccinia striiformis* in breeding for resistance to stripe (yellow) rust: Australian and international studies. Proceedings of the 11th Regional Wheat Workshop for Eastern, Central and Southern Africa. Addis Ababa, Ethiopia, 18-22 Sept., 2000.

Winter Wheat Breeding for Resistance to Rust Diseases under Irrigation in Uzbekistan

A. Amanov and A. I. Nurbekov

Galla-Aral Branch of Andijan Research Institute of Grain, Uzbekistan

Introduction

In Uzbekistan the most serious diseases of wheat are the three rusts, stem rust *(Puccinia graminis)*, brown rust *(Puccinia recondita)* and yellow rust *(Puccinia striiformis)*. Of these, brown rust and yellow rust are the most damaging under irrigation and yellow rust under rainfed conditions. There was an epidemic of yellow rust in southern Uzbekistan in 1999, which caused significant yield losses.

Damage caused by the rusts results from destruction of plant tissues and increased transpiration and, depending on timing of infection, yield losses can be severe (Heyne 1987, Nelson *et al.* 1972, Roelfs 1978). Grain quality and weight can be reduced and losses of 30-40% were reported from yellow rust epidemics in USA in 1960 and 1961 (Quisenberry 1967).

The most effective and ecologically sound method of control of the rust diseases is the development and release of wheat varieties with resistance, which is the objective of the work described below.

Materials and Methods

To select wheat with resistance to rust diseases in Uzbekistan, 350 wheat lines from the world genebank were tested for resistance under irrigation from 1998 to 2000. Trials were sown in rows by machine and control varieties were sown every 10 rows. The controls were Yanbosh, moderately resistant to yellow rust and stem rust, Sanzar-8, resistant to leaf rust and Itensivnay, susceptible to all three rusts. The trials were inoculated in spring with isolates of the rusts collected in Uzbekistan. They were scored for percentage leaf area infected by each rust. In another trial, with somewhat different levels of disease, tillering and grain weight and number per ear were recorded to identify productive lines.

Crosses were made between selected cultivars and near-isogenic lines with genes *Lr9* and *Lr19* for brown rust and *Yr5, Yr8* and *Yr10* for resistance to yellow rust. The F2 generations were tested for resistance.

Results and Discussion

From 350 tested lines, 18 were resistant to one or more of the rusts (Table 1). Full resistance to all three rusts was shown by Ramoga, Uz-666785, Ulugbek-600, Uz-000863, and No:99/10. Among lines with low rust incidence were Uz-666785, Kupava, No:98/20 and No:98/18, all of which had a maximum of 10% infection to any rust. Bacanora was resistant to yellow and stem rust but rather susceptible to brown rust in 1999. Some lines were resistant or moderately resistant to yellow rust, but susceptible to brown or stem rust (Table 1). There was variation between years, with higher levels of infection with yellow and brown rust in 1999 than in the other two years, following a warm winter from 1998 to 1999.

Table 1. Bread wheat selections and their resistance to the rust diseases in 1998-2000.

No.	Name of varieties or line	Country	Yellow rust %			Brown rust %			Stem rust %		
			1998	1999	2000	1998	1999	2000	1998	1999	2000
1	Zanzar-8	Uzbekistan	10	100	25	0	0	0	0	0	0
2	Yanbosh	-	0	10	0	20	20	20	20	10	20
3	Bacanora	Mexico	0	0	0	20	60	0	0	0	0
4	Lov26	-	0	20	10	30	40	20	0	0	0
5	Ramo a	-	0	0	0	0	0	0	0	0	0
6	Beabor	France	0	10	0	0	10	20	0	20	30
7	Uz-000728	-	0	20	0	20	100	20	100	60	40
8	Dostlik	Gal/CIT	0	15	5	0	0	20	10	0	10
9	K-56864	Norway	0	0	0	0	0	0	0	0	0
10	Uz-666771	Germany	0	0	0	0	0	0	0	0	0
11	Uz-666785	-	10	0	0	0	0	0	0	0	0
12	Polavchanka	Russia	0	5	0	0	0	0	0	0	0
13	Ku ava	-	0	10	5	0	10	0	0	0	0
14	Steklovidnay	Kazakhstan	0	15	5	10	10	10	0	0	0
15	Ulu bek-600	Uzbekistan	0	0	0	0	0	0	0	10	0
16	Selyanka	Ukraine	0	10	0	0	10	20	20	10	0
17	Uz-000863	Turkey	0	0	0	0	0	0	0	0	0
18	No: 98/20	Uzbekistan	0	10	5	10	5	0	0	0	0
19	No: 98/18	-	0	0	0	10	0	0	0	0	0
20	No: 99/100	-	0	0	0	0	0	0	0	0	0

In the trial to assess productivity (Table 2), there was wide variation in tillering capacity, ranging from 3.6 for Yanbosh to 9.3 for Uz-000863. Three cultivars from Uzbekistan had the highest grain weight per spike and grain yield per plant, Ulugbek-600, No:98/20 and No:98/18. These may be useful for breeding for rust resistance combined with good agronomic characters for Uzbekistan.

In the F2 generations from the crosses (Table 3) there were susceptible plants from all crosses, indicating that any resistance in the lines selected for study was not provided by *Lr9, Lr19, Yr5, Yr8 orYr10*. Various ratios were tested by O^2 and several fitted two gene ratios, but further work would be necessary for accurate diagnosis of the number of genes segregating.

Table 2. Characteristics of bread wheat selected lines, resistance to rusts, tillering and grain yields (thousand grain weight (TKW), grain weight per spike and per plant 1998-2000 Galla-Aral.

No.	Name of varieties or lines	Country	Disease reaction in %			Tiller production	Grain weight in gram		
			Yellow	Brown	Stem		TKW	Per spike	Per plant
1	Intensivany	Kyrgyzstan	60	60	0	4.2	37	1.3	5.4
2	Yanbosh	Uzbekistan	0	20	0	3.6	38	1.2	4.5
3	Bacanora	Mexico	0	10	0	7.8	39	1.2	8.5
4	Luma/II	China	10	20	10	9.0	38	1.1	9.1
5	Ramo a	Mexico	0	0	0	5.3	42	1.5	8.2
6	Beabor	France	5	10	25	6.0	43	1.8	9.3
7	Uz-000728	-	10	60	70	7.6	43	1.08	8.3
8	Dostlik	Gal/CIT	15	10	10	6.3	40	1.5	8.5
9	K-56864	Norway	0	0	0	6.5	39	1.1	6.1
10	Uz-666771	Germany	0	0	0	4.6	50	1.8	8.2
11	Sanzar-8	Uzbekistan	70	0	0	6.3	38	1.5	4.3
12	Polovchanka	Russia	0	0	0	5.2	41	1.7	8.2
13	Ku ava	-	10	10	0	5.1	40	1.6	7.5
14	Steklovidnay	Kazakhstan	20	10	0	6.3	43	1.7	8.1
15	Ulu bek-600	Uzbekistan	0	0	10	5.5	44	2.0	9.9
16	Sel anka	Ukraine	10	20	10	8.2	41	1.09	9.3
17	Uz-000863	Turkey	0	0	0	9.3	43	1.2	9.0
18	No: 98/20	Uzbekistan	10	20	0	4.5	46	2.3	9.6
19	No: 98/18	-	0	0	0	4.6	46	2.4	9.5
20	No: 99/10	-	0	0	0	5.2	42	1.8	7.3

Table 3. Segration of resistant:susceptible F2 seedlings from crossing bread wheat varieties with near-isogenic lines for *Lr9, Lr19, Yr5* and *Yr10* to brown and yellow rust.

Cross	Crossing combination	Ratio observed	Ratio tested	X^2
1	UZ-000528xLr9	152:12	15:1	0.03
2	UZ-000529xLr19	210:11	15:1	0.06
3	UZ-00529xYr5	103:7	15:1	0.02
4	UZ-0066671xY8	307:18	15:1	0.45
5	Sanzar-8x Yr10	207:8	635:1	0.05
6	Ulu bek-600x Lr9	122:3	63:1	2.30
7	Polovchancax Yr9	260:25	13:1	1.32
8	K-5686xYr-10	289:16	15:1	0.10
9	Sanzar-8x Yr5	179:10	15:1	0.06
10	Yanboshx Lr8	347:23	15:1	0.07

References

Heyne EG, 1987. *Wheat and Wheat Improvement,* USA American Society of Agronomy, Madison, USA. pp 549.

Nelson LR, Morey DD, Brown AR, 1972. Severe leaf rust on wheat in Georgia. *Plant Disease Reporter* **58**:21-23.

Quisenberry KS, 1967. *Wheat and Wheat Improvement,* USA American Society of Agronomy, Madison,USA. pp 549.

Roelfs AP, 1978. Estimated losses caused by rust in small grain cereals in the United States 1918-76. USA Miscellaneous Publication 1363 US Printing Office, Washington DC.

Pathogenic Variability

Current International Awareness of Pathogenic Variability in *Puccinia striiformis*

C.R. Wellings [1], R.P. Singh [2] and A. Yahyaoui [3]

[1] The University of Sydney, Plant Breeding Institute Cobbitty, Australia (seconded from NSW Agriculture);
[2] CIMMYT, Mexico DF, Mexico;
[3] ICARDA, Aleppo, Syria

Introduction

Cereal rust epidemics have been of significant historical importance since the earliest recorded histories of civilisation. In Australia, a series of wheat rust epidemics in the late 19th Century lead to the formation of government departments of agriculture in two states to address this and other issues of concern to primary industries. A widespread stem rust epidemic in southern Australia in 1973 resulted in the establishment of the National Cereal Rust Control Program located at The University of Sydney, Plant Breeding Institute (McIntosh *et al.* 1995). Wheat leaf rust *(Puccinia triticina)* epidemics in certain regions of Australia have caused crop losses over several seasons in the 1990s, while the detection of virulence for *Yr17* in 1999 resulted in isolated crop losses due to wheat yellow (stripe) rust *(Puccinia striiformis* f.sp. *tritici, = Pst)* in southern Australia (Wellings, unpublished).

Similarly, yellow rust epidemics in countries of East Africa, the Middle East, China and Central and West Asia have caused severe crop losses in wheat during certain seasons over the past decade. The apparent increasing frequency and severity of yellow rust epidemics in recent times raises questions concerning the nature and contributing factors in epidemic development, and the need to respond as a community of pathologists, breeders, policy advisors and farmers.

Three broad areas are of concern in understanding yellow rust epidemics and the variation in disease response of cultivars. Firstly, environmental effects such as mild winters and extended cool, moist spring seasons will provide ideal opportunities for pathogen development. Hovmöller (this volume) described extinction phenomena in *Pst* populations in Denmark during severe winter conditions, which appeared to eliminate the pathogen. In contrast, the yellow rust epidemic in the Great Plains of the USA in 2001 was exacerbated by extended cool wet spring weather in regions that normally experience rapid temperature increase in this

period (Hughes, 2001). In these conditions of severe inoculum pressure, cultivar response to *Pst* has been unexpectedly variable (R. Bowden, pers. comm.). Other environmental effects have included the development of irrigation facilities, such as in Syria (El Nami, this volume), and the expansion of wheat cultivation into fertile regions such as the Fergana Valley in Tajikistan and Uzbekistan.

The second factor is the nature of the cultivars deployed in agriculture. The expression of disease resistance varies between cultivars, with resistance onset occurring later in those displaying adult plant resistance (APR). The interaction between the timing of disease onset, climate and the nature of APR can lead to unexpected disease responses. For example, high temperature adult plant resistance (HTAPR) has been described and selected in the USA as an effective APR when temperatures characteristically rise in spring. However, the extended cool spring in the Great Plains of the USA in 2001 caused great fluctuations in cultivar response, in part due to the ineffectiveness of HTAPR under these climatic conditions (R. Bowden, pers. comm.).

The third factor underlying change in cultivar disease response is pathogenic evolution. The most common mechanisms driving evolution in *Pst* are mutation for increased virulence and selection on cultivars with the matching gene for resistance. The evolution of *Pst* in Australasia has followed a series of step-wise mutation events, which have included occasional reversions to avirulence (Wellings and McIntosh 1990). The latter studies have been based on disease collections during annual surveys of farmers' fields and the determination of pathotypes using seedling tests of differential cultivars under controlled conditions in the greenhouse.

Pathogenic change has also been a significant factor in the recurrent *Pst* epidemics in the Middle East during the 1990s. Virulence for *Yr9*, which was widely deployed in cultivars selected from the Veery group of materials, apparently spread rapidly throughout the region beginning in Yemen-Ethiopia in the late 1980s (Wellings *et al.* 2000). However, the opportunity to monitor pathogenic change throughout the region has been limited by the availability of facilities and expertise. Hence an alternative method using materials developed for trap plot nurseries has been evaluated over the past several seasons. This paper describes the method and the results from regional trap nurseries.

Materials and Methods

Approximately 30 genes for resistance to *Pst* have been described, although not all of these are of importance in commercial wheat germplasm or are represented in current differential sets assembled for studies of pathogenic variability.

A selected group of these genes were backcrossed to the Australian cultivar Avocet. This cultivar was earlier shown to be heterogeneous for an undescribed seedling gene (Wellings *et al.* 1988), and thus the susceptible selection Avocet S was used as the recurrent parent for the development of a set of near isogenic lines (NILs). This parent combines several useful characters including the *Sr26* resistance to stem rust (caused by *P. graminis tritici*) and the *Lr13* resistance to leaf rust (caused by *P. triticina*), with moderate vernalisation and neutral daylength requirements.

Homozygous resistant lines were selected after the sixth backcross, although certain NILs were selected at backcross three for preliminary studies prior to final completion. Several NIL sets comprising different entries were assembled and distributed to international co-operators over several seasons. Details of the NILs2000 set is presented in Table 1.

Table 1. Characteristics of lines designated as components of the NILs 2000 set.

Identification	Pedigree	Plant Selection	Donor Source
2000.1	Yr1 / 6* Avocet S	Cx93.51.3.3	Chinese 166
2000.2	Yr5 / 6* Avocet S	Cx86.6.1.20	T. spelta f. sp. album
2000.3	Yr6 / 6* Avocet S	Cx94.2.2.25	Oxley
2000.4	Yr7 / 6* Avocet S	Cx93.21.3.1	Lee
2000.5	Yr8 / 6* Avocet S	Cx86.18.1.8	Compair
2000.6	Yr9 / 6* Avocet S	Cx93.24.1.22	Clement
2000.7	Yr10 / 6* Avocet S	Cx93.53.3.1	Moro
2000.8	Yr11 / 3* Avocet S	Cx94.3.1.11	Joss Cambier
2000.9	Yr12 / 3* Avocet S	Cx94.6.1.15	Mega
2000.10	Yr15 / 6* Avocet S	Cx89.1.1.27 V763-251-wb	T. dicoccoides derivative
2000.11	Yr17 / 6* Avocet S	Cx94.8.1.25	Shortim/VPM derivative
2000.12	Yr18 / 3* Avocet S	Cx94.10.1.7	Jupateco R
2000.13	Yr24 / 3* Avocet S	Cx96.1.3.12 T.tauschii (CPI 18911)	*Meering2*//K733/*
2000.14	Yr26 / 3* Avocet S	Cx96.72.1 bulk	Triticum durum
2000.15	YrSp / 6* Avocet S	Cx94.14.1.15	Spaldings Prolific
2000.16	YrSk / 3* Avocet S	Cx94.19.1.1	Opata 85
2000.17	*Jupateco R (Yr18)*		
2000.18	Jupateco S		
2000.19	Avocet R (YrA)		
2000.20	Avocet S		

The NIL sets were planted in short rows in a range of locations, and assessed for response to *Pst*. In general, the pathogen occurred naturally in each location, although infection was also encouraged by using susceptible material adjacent to

the nursery. Comparisons with the recurrent parent were made in order to determine the presence of virulence for any particular gene in the pathogen population. Avocet S was the main recurrent parent for these comparisons, although Jupateco S was the contrast for assessing the effectiveness of *Yr18* in Jupateco R.

Results

A sample of data obtained from several nurseries is presented in Table 2.

Table 2. Disease responses[1] in NIL nurseries exposed to *Puccinia striiformis tritici* at various locations.

	Location					
NIL Entry	Tuwaitha Iraq	Khorasan Iran	Almaty Kazakhstan	Greytown South Africa	Kalengyere Uganda Ecuador	Santa Catalina
Yr1 / 6* Avocet S	R	60S	80	R	R	100S
Yr5 / 6* Avocet S	R	R	R	R	R	R
Yr6 / 6* Avocet S	100S	90S	80	80S	80S	100S
Yr7 / 6* Avocet S	100S	80S	80	90S	100S	100S
Yr8 / 6* Avocet S	5R	R	40	10R	tMR	R
Yr9 / 6* Avocet S	100S	50S	40	30MR	80S	100S
Yr10 / 6* Avocet S	R	R	R	R	R	R
Yr11 / 3* Avocet S	65S	80S	60	10S	50S	100S
Yr12 / 3* Avocet S	5R	70S	40	10S	5MS	10MR
Yr15 / 6* Avocet S	R	R	R	R	R	R
Yr17 / 6* Avocet S	5R	80S	60	40MR-MS	5MS	MR
Yr18 / 3* Avocet S	65S	-	60	15MR-MS	40S	100S
Yr24 / 3* Avocet S	-	-	-	40MR	50MS	90S
Yr26 / 3* Avocet S	-	-	-	5R	50MS	40M
YrSp / 6* Avocet S	R	R	R	R	R	10M
YrSk / 3* Avocet S	25MR	25S	40	5R-10S	40S	80S
Jupateco R (Yr18)	-	R	-	tR	10MR	90S
Jupateco S	-	60S	-	60MR-MS	60S	90S
Avocet R (YrA)	100S	70S	80S	5MR-MS	60S	100S
Avocet S	100S	80S	80S	90S	80S	100S

[1] Disease response based on combinations of: Infection Type (IT) including R (resistant), MR (moderately resistant), MS (moderately susceptible), S (susceptible); Percent leaf area affected by the disease (modified Cobb scale).

A number of features are evident from these and other data gathered by co-operators:

1. Virulence for *Yr6* and *Yr7* is common.
2. Avirulence for *Yr5, Yr10, YrSP* and *Yr18* was observed at all sites. However,

the conclusion of a virulence in respect to *Yr18* remains controversial. The Ecuador site appeared to show very high responses for both the Yr18NIL and Jupateco R in comparison with their recurrent parents, Avocet S and Jupateco S. However, this data set was collected late in the epidemic season and probably reflected severe terminal disease reactions in a very conducive environment.

3. Several resistance genes showed variation between locations. Virulence for *Yr1* was common in Central Asia and China, but rare in the Middle East and absent in southern Africa. The occurrence of virulence for *Yr9* has become widespread over the past decade, although the gene has remained effective in certain areas such as South Africa. Virulence for *Yr24* and *Yr26* was evident at some locations although these genes have not, to our knowledge, been deployed in commercial cultivars. In contrast, *Yr17* has been deployed and remains effective in some areas such as Iraq, Uganda and Ecuador.

Conclusions

The data arising from these nurseries is assisting in the global understanding of variation in *Puccinia striiformis* f. sp. *tritici*. The regional effectiveness of certain resistance genes, such as *Yr9, Yr17 and Yr18,* will need to be monitored continually in order to detect the occurrence of virulence. While these field nurseries offer advantages in this regard, it is clear that this approach will be associated with several problems and potential dangers:

1. Errors will occur during transfer of certain genes. This has already become evident with the Yr12NIL which was shown to posses *Yr17*, and the Yr11NIL which has been shown not to carry this particular gene (R. Johnson, personal communication).
2. The identical phenotypic features of the NIL set will mean that mixtures of the lines may occur form time to time and will be difficult to recognise and rectify.
3. Certain NIL lines appear to show evidence of carrying additional adult plant resistance, presumably *Yr18,* which improves the level of resistance and so makes it difficult to draw conclusions in regard to the presence/absence of virulence for certain genes.
4. It is likely that certain NIL materials might be considered as sources of resistance in breeding programs. This should not be encouraged, since these genes are largely those that have failed in agriculture. Among the group which have remained effective, the use of the NIL stock as a resistance source is associated with the danger of transferring single genes from a highly susceptible

genotype such as Avocet S with no associated genetic background effects which may contribute to resistance complexity in a breeding program.

In summary, relevant and effective pathogenicity surveys are of fundamental importance to breeding programs aimed at incorporating resistance to obligate plant pathogens. Surveys monitor the distribution of current pathotypes, are directed at the early detection of new avirulence/virulence combinations of importance to agriculture, and, if results can be related genetically to cultivar genotypes, contribute to decisions on cultivar recommendation. Surveys also allow the selection of pathogen isolates of known pathogenic profile for use in germplasm screening, and the accumulated historical data provides valuable insights into pathogen epidemiology and disease management. Thus an efficient and relevant means of monitoring pathogenicity facilitates cultivar development and deployment.

References

Hughes ME, 2001. Cereal Rust Laboratory, seasonal disease survey reports. http://www.cdl.umn.edu/

McIntosh RA, Wellings CR, Park RF, 1995. *Wheat Rusts - An Atlas of Resistance Genes.* CSIRO Publications Australia, 200 pp.

Wellings R, McIntosh RA, 1990. *Puccinia striiformis* f.sp. *tritici* in Australasia: pathogenic changes during the first ten years. *Plant Pathology* **39**:316-325.

Wellings CR, McIntosh RA, Hussain M, 1988. A new source of resistance to *Puccinia striiformis* f.sp. *tritici* in spring wheats *(Triticum aestivum). Plant Breeding* **100**:88-96.

Wellings CR, Singh RP, McIntosh RA, Yahyaoui A, 2000. The assessment and significance of pathogenic variability in *Puccinia striiformis* in breeding for resistance to stripe (yellow) rust: Australian and international studies. *Proceedings 11th Regional Workshop for Eastern, Central and Southern Africa.* Addis Ababa, September 2000, pp 134-143.

Wheat Yellow Rust Pathotypes in Western Asia

M.S. Hakim[1], A. Yahyaoui[2], M. El-Naimi[2], I. Maaz[2]

[1]Aleppo University, Faculty of Agriculture, Aleppo, Syria;
[2]ICARDA, International Center for Agriculture Research in the Dry Areas, Aleppo, Syria

Introduction

Yellow rust of wheat (*Puccinia striiformis* West.f.sp.*tritici*) is the most important wheat disease in cool and humid wheat growing areas (Stubbs 1988, Johnson 1988, Danial 1994) and has become a major wheat disease in Western Asia since the late 1980 's (Mamluk *et al.* 1996). Several yellow rust epidemics have occurred in Western Asia during the last decade, causing severe losses in wheat production. In Pakistan significant losses were recorded in the 1991/92 crop season (Ahmed *et al.* 1992). In Iran three yellow rust epidemics were recorded during 1993-1995 (Torabi *et al.* 1995). In Turkey severe losses were recorded in 1991 (Braun and Saari 1992). In Ethiopia and Yemen early yellow rust epidemics were recorded since 1988 (Mamluk *et al.* 1996). In Syria, annual occurrence of yellow rust were observed since 1987 (El-Naimi and Mamluk 1995) and the disease spread to all wheat growing areas (Mamluk *et al.* 1990, Mamluk and El-Naimi 1992). In Lebanon severe losses were recorded in 1994 (Mamluk 1995).

A wide range of virulent pathotypes is evolving in this region causing failures of resistance of widely-grown wheat cultivars. Therefore, surveys of *P. striiformis* pathotypes and the gentic variation within the pathotypes are important and provide valuable imformation to the breeding programs.

Surveys of occurrence and frequency of virulence factors in the rust populations have been carried out for many years at the IPO*, but the rust populations in Western Asia are highly diverse and new pathotypes of *P.striiformis* f.sp.*tritici* have been found in Syria annually (Hakim and Mamluk 1998, Hakim and El-Ahmed, 1998). This study, reports on the prevailing pathotypes of wheat yellow rust pathogen in the region.

IPO*: (Institut voor Planteziekenkunding Onderzoek, Wageningen)

Materials and Methods

A set of the World and European wheat yellow rust differentials as proposed by Johnson *et al.* (1972), was used in this study, in addition to five supplementary cultivars/lines sonalika, Anza, Federation 4 /Kavkaz, Gereck79, and Cham1, making a total of 22 cultivars/lines (Table1).

Table 1. Yellow rust differential cultivars, used for identification of wheat yellow rust pathotypes (races).

Differential *cultivars	Decanery value	*Yr* resistance gene
World set		
Chinese 166	1 (=2^0)	*1*
Lee	2 (=2^1)	*7*
Heines Kolben	4 (=2^2)	*6* (1)
Vilmorin 23	8 (=2^3)	*3V*
Moro	16 (=2^4)	*10*
Strubes Dickkopf	32 (=2^5)	*SD*
Suwon92xOmar	64 (=2^6)	*SU*
Clement	128 (=2^7)	*9, 2 +*
Triticum spelta f.sp. *album*	256 (=2^8)	*5*
European set		
Hybrid 46	1 (=2^0)	*4 +*
Reichersberg 42	2 (=2^1)	*7 +*
Heines peko	4 (=2^2)	*6,2 +*
Nord Desprez	8 (=2^3)	*3N*
Compare	16 (=2^4)	*8*
Carstens V	32 (=2^5)	*CV*
Spaldings Prolific	64 (=2^6)	*SP*
Heines VII	128 (=2^7)	*2, 11 +*
Supplemental set		
Sonalika		*2,A*
Anza		*18,A*
Federation 4/Kavkaz		*9*
Gerek 79		-
Cham 1		-

Source of seeds : R.W. Stubbs, IPO. The Netherlands

The wheat yellow rust differentials were planted as a trap nursery at different sites in Syria in addition to Tel Hadya (ICARDA) main station during the cropping seasons 1993/1994-1998/1999 and in Lebanon at one site, Terbol for the same period.

Each differential cultivar/line was planted in two rows, 1-m long and 30cm apart. At Tel Hadya, the plants were inoculated at tillering growth stage (growth stage

22-29 Zadoks *et al.*1974) with a mixture of urediospores collected from farmers' fields in different locations of Syria in the previous season and at Terbol, with a mixture of urediospores collected from Lebanon in the previous season. Infected leaves were obtained from yellow rust trap nurseries and from farmers fields, throughout Syria and Lebanon for each growing season. The collections were increased on seedlings of the susceptible cultivar Morocco. Urediospores of a single pustule from the infected leaf were isolated and multiplied on seedlings of Morocco. 14 days after inoculation urediospores were developed and spores were collected every 2 days.

Each isolate was then tested on a set of wheat yellow rust differentials. Seedlings of the differential set were inoculated with each isolate and placed for 48 hours in a dew chamber at 11°C. They were then transferred to a growth chamber at 17 °C with 16/8 hour day/night. Infection types were assessd on a 0-9 scale (Mc Neal *et al.* 1971), 17 days after inoculation. Infection types equal or higher than '7' are considered as virulent and infection types less than '7' as avirulent (Johnson *et al.* 1972).

Results and Discussion

Genetic variation in the yellow rust pathogen is continuously evolving in the region. In the 1990/1991 crop season a number of pathotypes were recorded in the region, eight in Ethiopia, three in Yemen, three in Turkey and six in Syria (Table 2). In 1993/1994 crop season 15 yellow rust pathotypes were identified in Iran (Table 2).

Table 2. Yellow rust pathotypes (races) identified in Ethiopia, Yemen, Syria and Iran.

Season	Ethiopia	Yemen	Syria	Turkey	Iran [2]
Country		1990/91			1993/94
Pathotypes	6E16	6E16	6E16	6E16	6E16
	6E18		6E18		6E6
	70E16				6E0
	82E0				6E20
	82E16		82E16		84E16
	134E146	134E146	134E146		134E22
	134E150	134E150	134E150		
			166E150		
			2E0	2E0	22E0
			2E16	2E16	16E0
					2E18
					4E20
					18E0
					34E0
					64E0

Sources: IPO (Louwers *et al.* 1992)
S.P.I.I. (Seed and Plant Improvement Institute, Karaj, Iran)

In Syria during the seasons 1993/1994 to 1998/1999 twenty five yellow rust pathotypes were identified and twelve in Lebanon for the same period (Tables 3,4). In 1993/94 crop season, six yellow rust pathotype (6E0, 6E134, 6E148, 6E150, 20E148, 38E150) were identified in Syria and three in Lebanon (6E0, 38E134, 166E150). Only one pathotype, 6E0, was found in both countries. In 1994/95 nine yellow rust pathotypes were identified in Syria (4E0, 6E0, 6E18, 6E144, 38E128, 38E134, 38E150, 134E146, 82E16) and four in Lebanon (6E0, 38E134, 38E16, 166E150).Two pathotypes (6E0, 38E134) were found in Syria and Lebanon. In 1995/96 crop season thirteen pathotypes were identified in Syria and six in Lebanon. Three pathotypes (6E0, 38E134, 166E150) were recorded in both countries.

Table 3: Yellow rust pathotypes (races) identified in Syria during 1993/94 and 1998/99 cropping seasons.

Seasons					
Pathotypes					
1993/1994	1994/1995	1995/1996	1996/1997	1997/1998	1998/1999
			2E0	2E0	
	4E0	4E0			
6E0	6E0	6E0	E60	6E0	6E0
		6E16			
	6E18	6E18		6E18	6E18
		6E20	6E20		
6E134		6E134	6E134		
	6E144				
6E148					
6E150		6E150			
			18E0		
20E148		20E148	20E148		
	38E128	38E128	38E128		
	38E134	38E134	38E134		38E134
38E150	38E150	38E150	38E150	38E150	
				38E6	38E6
					38E0
	82E16			82E16	
	134E146	134E146		134E146	
			134E146		
			134E16		
				134E134	
		166E150			
					68E130
					230E134
					230E150

However, during the survey period seven pathotypes (6E0, 20E148, 38E134, 166E150, 6E20, 134E150 and 230E150) were found in both countries. The

pathotypes 6E0, 38E150 and 134E146 were recorded in six, five and four consecutive growing seasons. Two pathotypes (38E134 and 6E18) occurred during four seasons. Four new pathotypes (230E134, 230E150, 68E130, 38E0) were recorded in 1998/1999 crop season. The most virulent pathotype was 230E150, and was recorded in Syria, Lebanon, Iraq and Yemen in the same season.

The pathotypes 6E18, 134E150, 134E146, 6E16 were recorded since 1990/1991 in Ethiopia, Yemen and Syria, the pathotype 82E16 was recorded in Syria and Ethiopia in 1990/1991 crop season (Table 2) while the pathotype 166E150 was first recorded in Ethiopia in 1990.

However, the yellow rust population in the region consists of a number of pathotypes that differ in their pathogenicity toward the host plant some pathotypes such as 2E0, 6E0 can attack only two resistance genes in the host plant and some pathotypes such as 198E150, 230E150 can attack 11 identified genes in the host plants. The Iranian yellow rust pathotypes do not differ in their pathogenicity from those found in Syria and Lebanon.

Table 4: Yellow rust pathotypes (races) identified in Lebanon during 1993/94 and 1998/99 cropping seasons.

Seasons					
Pathotypes					
1993/1994	1994/1995	1995/1996	1996/1997	1997/1998	1998/1999
6E0	6E0	6E0	6E0	N.S	
	38E16	38E16			
					38E22
38E134	38E134	38E134	38E134		
166E150	166E150	166E150	166E150		
	172E146	172E146			
	182E150	182E150			
					70E148
					134E150
					198E150
					230E150
					6E134

N.S : No samples

Virulence on Yr (6, 7, 6+, 7+, 8, 2, 9 and A) were found in the all countries of Western Asia. Of these virulences, races possessing the combination of virulence for *Yr7* and *Yr9* were particularly implicated in the epidemics described above because this combination overcame the resistance of Seri 82 and the many derivatives of this that were widely grown in the region. Virulence on the resistance

factors *YrSD* and *YrSU* were found in Syria and Lebanon in some pathotypes. The pathotypes 230E134 and 230E150 can overcome the two resistant factor *YrSD* and *YrSU,* while virulence on the resistance factor *Yr3V* was found in Lebanon only in the pathotype 172E146.

Conclusion

The composition of the yellow rust population changes through the time and this can be an important consideration for breeding programs in the region. Therefore, the pathogen population should be monitored regularly to determine whether new virulent genotypes have been introduced or developed in the region.

References

Ahmed S, Rodriguez A, Farid Sabir G, Roidar Khan B, Panah M, 1992. Economic losses of wheat crops Infested with yellow rust in highland Balochistan. *MART/AZR Project Research, Report* # **67**.ICARDA Quetta.15pp.

Braun HJ, Saari EE, 1992. An assessment of the potential of *Puccinia striiformis* f.sp. *tritici* to cause yield losses in wheat on the Anatolian plateau of Turkey. Pages 121-123 In: *proceedings, 8th European and Mediterranean Cereal Rusts and Mildews Conference* (Eds. Zeller F J and Fischbeck G), 8-11 September 1992. Weihenstephan, Germany.

Danial DL, 1994. Aspects of durable resistance in wheat to yellow rust. *Ph.D. thesis,* Wageningen Agricultural University, The Netherlands, 143 pp.

El-Naimi M, Mamluk OF, 1995. Occurrence and virulence of wheat rusts in Syria. *Arab Journal of Plant Protection,* **13**: 76-82.

Hakim MS, El-Ahmed A, 1998. The physiological races of wheat yellow rust *Puccinia striiformis* f.sp.*tritici* in Syria during the period 1994-1996. *Arab Journal Plant Protection.* **16** (1):7-11.

Hakim MS, Mamluk OF, 1998. Monitoring the pathotypes and virulences of *Puccinia striiformis* f. sp. *tritici* in Syria. *Phytopathologica Mediterranea.* **37**:106-110.

Johnson R, Stubbs RW, Fuchs E, Chamberlain NH, 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. *Transactions of the British Mycological Society* **58**: 475-480.

Johnson R, 1988. Durable resistance to yellow (stripe) rust in wheat and its implication in plant breeding. In: *Breeding Strategies for Resistance to Rusts of Wheat* (Eds. Simmonds N W and Rajaram S), CIMMYT, Mexico, 63-75.

Louwers JM, Silfhout CH, Stubbs RW, 1992. Race analysis of yellow rust of wheat in developing countries. Report 1990-1992, IPO-DLO report **92**-11.23 pp.

Mamluk OF, Al-Ahmed M, Makki MA, 1990. Current status of wheat diseases in Syria. *Phytopathologia Mediterranea* **29**: 143-150.

Mamluk OF, El-Naimi M, 1992. Occurrence and virulence of wheat yellow rust in Syria. pp.115-117. In: *Proceedings of the 8th European and Mediterranean Cereal Rusts and Mildews Conference* (Eds. Zeller F J and Fishbeck G). 8-11 September. 1992. Wheihenstephan, Germany.

Mamluk OF, 1995. Our race with the yellow rust disease. Agrotica **4**:18-19.

Mamluk OF, El-Naimi M, Hakim MS, 1996. Host preference in *Puccinia striiformis* f. sp. *tritici*. In: *Proceedings of the 9th European and Mediterranean Cereal Rusts and Powdery Mildews Conference,* September 2-6, 1996, Lunteren, The Netherlands, pp:86-88.

McNeal EH, Konzak CF, Smith EP, Tate WS, Russell TS, 1971. A uniform system for recording and processing cereal research data. *Agriculture Research Service,* pp: 34-121 Washington, D.C. USA.

Stubbs RW, 1988. Pathogenicity analysis of yellow (stripe) rust of wheat and its significance in a global context. In: *Breeding Strategies for Resistance to the Rusts of Wheat.* (Eds. Simmonds N W and Rajaram S), CIMMYT, Mexico 23-38.

Torabi M, Mardoukhi V, Nazari K, Afshari F, Foroutan A R, Romai MA, Golzar H, Kashani AS, 1995. Effectiveness of wheat yellow rust resistance genes in different parts of Iran. *Cereal Rusts and Powdery Mildews Bulletin.* **23**:9-12.

Zadoks JC, Chang TT, Konzak CF, 1974. A decimal code for the growth stages of Cereals. *Weed Research,* **14:** 415-421.

Virulence of Wheat Stripe Rust Pathotypes Identified in Egypt During 1999/2000 and Sources of Resistance

S.A. Abu El-Naga, M.M. Khalifa, S. Sherif, W.A. Youssef, Y.H. El-Daoudi and Ikhlas Shafik

Plant Pathology Research Institute, Cereal Disease Research Department, (ARC), Egypt.

Introduction

Wheat stripe rust is one of the recent problems of wheat production in Egypt, where it was formerly known as sporadic, but has been recorded annually since the early 1990's. The first yellow rust epidemic was recorded in 1967; however during the1990's, three major epidemics were recorded in 1995, 1997, and 1999. Sporadic infections were observed during the other years (Abdel-Hak *et al.* 1972, El-Daoudi *et al.* 1977, Abu El-Naga *et al.* 1999). The most important stripe rust epidemic occurred in 1995 particularly in the Northern and Southern Delta areas, and slight infections were also recorded in Middle and Upper Egypt. Yields of most of the popular cultivars (Gemmeiza 1, Giza 163, 157, 164 and 166, Sakha 8, 69 and 92) were significantly reduced by yellow rust. The objectives of this study were to assess the virulence of the stripe rust pathogen and the effectiveness of the resistance genes, and to study the reactions of commonly grown cultivars and elite breeding lines to yellow rust.

Materials and Methods

Screening for resistance to yellow rust was usually conducted under natural infection. However, new facilities at Sakha experimental station also allowed screening under artificial inoculation in the greenhouse. Annual surveys were carried out within the Northern Governorates (districts) of the Delta region and at Quina in the South, where the susceptible wheat cultivars Sakha 8, Sakha 69, Giza 160 and Giza 164 continue to be grown. Infected leaves and spikes were collected and yellow rust spores were multiplied on the susceptible cultivars: Little Club, Baart, Giza 160, Triticum spelta f. sp. saharense.

World and European wheat yellow rust differentials were used (table 2). The differentials were planted in a growth chamber under 16/8 hours of light. Light intensity was about 7500 lux and the temperature was maintained between 15-18 °C. Plants were inoculated 7-10 days after planting. Prior to inoculation, seedlings were spayed with water containing a few drops of Tween 20 or Triton. Spores were then rubbed by finger onto the moist leaves. Following inoculation, seedlings were kept in an incubating chamber for 48hr at 90°C, then transferred to the growth chambers where humidity was maintained at 95% for 18-20 days.

Twelve commercial varieties and 163 entries from the Sakha Crossing Block 2000/2001, and wheat germplasm of various research experiment stations of Egypt. (*i.e.* Sakha, Gemmeiza, Sids), entries from the D-Trial that includes durum and bread wheat lines were also tested under controlled conditions. The final D-Yield trials are annually performed in about 33 locations throughout the region by the Wheat Research Section, Field Crops Research Institute of the Agricultural Research Center at North Delta (5), South Delta(10), Middle Delta (4), Upper Egypt (9) and Out Valley (5). The inoculation was done by mixing yellow rust spores with talcum powder (Tervet and Cassel, 1951) and dusted on to moist adult plants grown in the green house The cultivars tested for resistance at the adult growth stage represented a collection of different sets of Egyptian breeding lines. At the seedling stage disease records were performed using the 0-9 scale of McNeal *et al.* (1971) (Table 1).

Table 1: The infection types of stripe rust of wheat and barley and code symbol or index value of the scale proposed by McNeal *et al.* (1971).

Basic scale Descriptions of signs *and symptoms*	Code symbol	Index value	Expanded scale Descriptions of infection type	Code symbol	index value
No visible infection	0	0	No visible infection	0	0
Necrotic and/or chlorotic areas, no sporulation or trace sporulation	R	2	Necrotic or chlorotic flecks, no sporulation	VR	1
			Necrotic and/or chlorotic stripes,no sporulation	R	2
			Necrotic and/or chlorotic stripes, trace sporulation	MR	3
Necrotic and/or chlorotic stripes, light to moderate sporulation	M	5	Necrotic and/or chlorotic stripes, light sporulation	LM	4
			Necrotic and/or chlorotic stripes,intermediate sporulation	M	5
			Necrotic and/or chlorotic stripes, moderate sporulation	HM	6
Necrosis and chlorosis may or may not be present, abundant sporulation	S	8	Necrotic and/or chlorotic stripes, abundant sporulation	MS	7
			chlorosis behind abundant sporulating area	S	8
			No chlorosis or necrosis, abundant sporulation	VS	9

The Nile Valley and Red Sea project (NVRSP) tested a wheat rust trap nursery (NVRSWRN) 2000/2001 in Egypt, Ethiopia, Sudan and Yemen under natural infection. The trap nursery included the rust differentials and selected cultivars

from the participating countries. One set was also tested under artificial inoculation in Egypt. The trap nursery included 206 entries distributed as follows:

Differential cultivars
High yielding breeding lines
Commercial cultivars and susceptible checks
Wild relatives

Results and Discussion

Pathotypes

The analysis of yellow rust populations (Table 2), on the European and World differentials, indicated the presence of eight pathotypes of *Puccinia striiformis* f.sp *tritici.* These were identified as: 242E100, 128E61, 0E0, 194E101, 458E45, 234E109, 450E109 and 192E109.

Table 2. Seedling reaction of differential genotypes to yellow rust identified in Egypt (1999-2000).

Cultivar	Yr Gene	242E100	128E61	0E0	194E101	458E45	234E109	450E109	192E109
				World Differential Set					
Triticum spelta	Yr5	R	R	R	R	S	R	S	R
Clement	Yr9, Yr2 +	S	S	R	S	S	S	S	S
Suwon 92xOmar	SU	S	R	R	S	S	S	S	S
Strubes Dickkopf	SD	S	R	R	R	R	S	R	R
Moro	Yr10	S	R	R	R	R	R	R	R
Vilmorin 23	Yr3V	R	R	R	R	S	S	R	R
Heines Kolben	Yr6	R	R	R	R	R	R	R	R
Lee	Yr7	S	R	R	S	S	S	S	R
Chinese 166	Yr1	R	R	R	R	R	R	R	R
				European Differential Set					
Heines VII	Yr2 +	R	R	R	R	R	R	R	R
Spaldings Prolific	SP	S	R	R	S	R	S	S	S
Carstens V	CV	S	R	R	S	S	S	S	S
Compair	Yr8, APR	R	R	R	R	R	R	R	R
Nord Desprez	3N	R	R	R	R	S	S	S	S
Heines Peko	Yr6, Yr2 +	S	R	R	S	S	S	S	S
Reichersberg 42	Yr7 +	R	R	R	R	R	R	R	R
Hybrid 46	Yr4 +	R	S	R	S	S	S	S	S

Race 242E100 possessed virulence for Clement, Suwon92 x Omar, Moro, Lee, Spaldings Prolific, Carstens V and Heines Peko. This race therefore has virulence for the resistance conferred by *Yr9, Yr SU, Yr10, YrSP, YrCV, Yr2* and *Yr6.* Race 0E0 was avirulent to all European and World differential cultivars. Race 194E69

was virulent on the differential cultivars Clement, Suwon92 x Omar, Lee, Spaldings Prolific, Heines Peko and Hybrid 46 and consequently has virulence for *Yr9, YrSu, YrSP, Yr2, Yr6,* and *Yr4* resistance genes. Race 458E45 was virulent on *Triticum spelta* f. sp. album, Clement, Vilmorin 23, Suwon92 x Omar, Carstens V, Nord Desprez, Heines Peko, and Hybrid 46 and therefore has virulence for *Yr5, Yr9, YrSu, Yr3, YrCV, Yr2, Yr6,* and *Yr4.*

Race 234 E109 was virulent on Clement, Suwon x Omar, Strubes Dickkopf, Carstens V, Nord Desprez, Heines Peko and Hybrid 46, whereas Race 450E109 was virulent on *Triticum spelta* f. sp. *album,* Clement, Suwon92 x Omar, Lee, Spaldings Prolific, Carstens V, Nord Desprez, Heines Peko, and Hybrid 46. Race 192E109 had virulence for Clement, Suwon92 x Omar, Strubes Dickkopf, Lee, Spaldings Prolific, Carstens V, Nord Desprez, Heines Peko and Hybrid 46. Resistance genes possessed by the respective cultivars were considered as ineffective against the designated races that possessed virulence for them.

The relative frequency of yellow rust race distribution (Table 3) was determined from the survey samples of the 1999-2000 crop season in Northern Egypt. The data reveal that race 0E0 was the most frequent (44.9%) followed by 128E61 (15.3%), 458E45 (9.18%), and both 242E100 and 234E109 (8.16%). Race 450E109 and 192E109 each accounted for about 3% and were restricted to Quina in Southern Egypt. Some of the races exhibited high levels of virulence and there was great variation in virulence. The frequent occurrence of race 0E0 among the samples would suggest that supplementary differentials are needed to determine the full variation among the yellow rust pathotypes in Egypt.

Table 3: Pathotypes of wheat stripe rust (*Puccinia striiformis* West), their frequency (%) and distribution in Egypt during 1999/2000.

Pathotypes	Number of samples	Freqency (%)	Distribution
242 E 100	40	8.16	Sakha
128 E 65	75	15.31	Belquas, Damyatta
0 E 0	220	44.9	Sakha, Belquas, Damyatta El-Mahalla, Quina
194 E64	39	7.69	Sakha, Belquas
458 E 45	45	9.18	Sakha, Belquas, Damyatta
234 E 109	40	8.16	Damyatta El-Mahalla
450 E 109	16	3.27	Quina
192 E 109	15	3.06	Quina

References

Abdel-Hak TM, Stewart DM, Kamel AH, 1972. The current stripe rust situation in the Near East countries. *Regional Wheat Workshop.* Beirut, Lebanon, 1-29.

Abu El-Naga SA, Khalifa MM, Bassiouni AA, Youssef WA, Shehab TM, Abdel-Latif AH, 1999. Revised evaluation for Egyptian wheat germplasm against physiologic pathotypes of stripe rust (*Puccinia striiformis* West.) In *Agriculture Science Mansoura University* **24:** 477-488.

El-Daoudi YH, 1977. Studies on stripe rust of wheat in Egypt. *Master of Science Thesis.* Faculty of Agriculture, Cairo University. 78 pp.

Hakim MS, El-Ahmed A, 1998. The physiologic races of wheat stripe rust *Puccinia striiformis* West. in Syria during the period 1994-1996. *Arab Journal of Plant Protection* **16:** 7-11.

Hovmöller MS, 1993. *Puccinia striiformis* on wheat, biology and varietal resistance. Tenth Danish Plant Protection Conference. *Pests and Diseases,* Lyngby. Denmark, Landburgs, Ministeriet Statenns Planteaulsforsog p94-60.

Mc Neal FH, Konzak CF, Smith EP, Tate WS, Russe l, 1971. A uniform system for recording and processing cereal research data. *US Department of Agriculture, Agriculture Research Service,* ARS 34-121.

Tervet I, Cassel RC, 1951. The use of cyclone collectors in race identification of microscopic particles. *Phytopathology* **41**:282-285.

Zwer PK, Qualset CO, 1991. Genes for resistance to stripe rust in four spring wheats: seedling responses. *Euphytica,* **58**: 171-181.

Yellow (Stripe) Rust (*Puccinia striiformis* f.sp. *tritici*) in Central and Western Asia

A. Yahyaoui [1], M.S. Hakim[2], K. Nazari[3] , M. Torabi[3] and C. R. Wellings[4]

[1] *International Center for Agricultural Research in the Dry Areas, Aleppo, Syria;*
[2] *Aleppo University, Aleppo, Syria;*
[3] *Seed and Plant Improvement Institute, Karaj, Iran;*
[4] *Plant Breeding Institute, Cobbitty, Sydney, Australia.*

Introduction

Comprehensive information on pathogen virulence and variation, and epidemiological information on pathogen movements, provide a basis for the development of an early warning system. It allows the identification and subsequent management of susceptible cultivars and the development of varieties with resistance to yellow rust some of which might be durable. This monitoring system should lead to a reduction in the frequency and magnitude of devastating losses experienced in the region from yellow rust epidemics and therefore will improve the stability of wheat production and enhance food security in the region.

Detection of pathogen variation has traditionally relied upon the identification of virulence variation (races) in the pathogen populations by inoculating a sample of pathogen isolates on a series of host differentials only some of which have defined resistance genes, and observing the resulting compatible or incompatible disease phenotype. This approach to monitoring pathogen populations has been tremendously valuable in the development and deployment of host resistance, and has provided important insights into the evolution of pathogen populations in response to selection by host resistance genes. Seedling resistance is usually race specific and can be recognized by its characteristic resistance type at all plant stages (Hong and Singh 1996). Adult resistance can be either race specific or race non-specific and is usually better recognized after the seedling stage (Johnson 1988). Singh (1992) reported that that adult plant resistance gene *Yr18* is involved in durable resistance of several bread wheat cultivars, including the North American cultivar Anza. Genes conferring adult plant resistance are known to occur in wheat (*Triticum aestivum* L.). Several high yielding spring wheat varieties possess adult plant resistance (Singh 1992, Singh *et al.* 1994).

Materials and Methods

Virulence surveys of cereal rust fungi have traditionally used differential host lines that express resistance in the primary leaves of seedling plants (Kolmer 1997). In this study, yellow rust populations were characterized using two types of method: 1) use of a trap nursery (Central Western Asia yellow Rust Trap Nursery-CWAYRTN) planted at yellow rust hot spots in Azerbaijan, Uzbekistan, Kyrgyzstan, Tajikistan, and Kazakhstan since 1999, and 2) artificial inoculation at experimental stations in Syria, Lebanon, and Turkey. Surveys of yellow rust populations and evaluation of trap nurseries indicated the presence of many virulence types in Central and Western Asia.

The host. Forty eight bread wheat cultivars in the yellow rust trap nursery (Table 1) selected for the study comprise three sets of cultivars (Table 1) grouped as follows:

- Group I comprises a world differential set, 9 accessions
- Group II comprises a European differential set, 8 accessions
- Group III comprises a supplementary differential set, 4 accessions
- Group IV includes Avocet Near Isogenic lines,16 accessions
- Group V includes common varieties with know resistance factors, 11 accesions.

The pathogen. Artificial inoculation at Tel Hadya (Syria), Terbol (Lebanon), and Hymana (Turkey) was achieved using a mixture of yellow rust isolates collected the previous year from wheat fields in the respective countries. At ICARDA (Tel Hadya) rust spores were multiplied on seedlings of a mixture of susceptible varieties then dusted on spreader rows in the field. At the other testing sites spreader rows and susceptible checks provided natural spread of the rust pathogen.

Field evaluation. Each cultivar's field response to yellow (stripe) rust was evaluated at the respective experimental stations. Environmental conditions at the testing sites in Syria, Turkey, Azerbaijan, and Kyrgyzstan were favorable for the development of yellow rust during the 1999 and 2000 crop seasons.

The disease reaction was recoded at heading stage using a modified Cobb scale (Peterson *et al.* 1948) which is the most commonly used by national research programs. The cultivars Morocco and Avocet S are highly susceptible and were used as susceptible controls. The remaining cultivars were found to have different degrees of adult plant resistance to natural populations of *Puccinia striiformis* f.sp. *tritici* in the respective testing sites.

Table 1. Differential cultivars used in the Central Western Asia Yellow Rust Trap Nursery (CWAYRTN).

Differential Cultivars	Resistance Gene/Genes	Differential Cultivars	Resistance Gene/Genes
GI. World Differential Set[1]		**GIV. Avocet Near Isogenic Lines[2]**	
Chinese 166	*Yr1*	Avocet S	Check
Lee	*Yr7*	Avocet R	YrA
Heines Kolben	*Yr6, Yr2*	Yr1/6* Avocet S	Yr1
Vilmorin 23	*Yr3V*	Yr5/6* Avocet S	Yr5
Moro	*Yr10*	Yr6/6* Avocet S	Yr6
Stubes Dickkopf	*YrSD*	Yr7/6* Avocet S	Yr7
Suwon92xOmar	*YrSu*	Yr8/6* Avocet S	Yr8
Clement	*Yr9, Yr2* +	Yr9/6* Avocet S	Yr9
T. spelta album	*Yr5*	Yr10/6* Avocet S	Yr10
GII. European Differential Set[1]	YrSk/3* Avocet S	Yr29	
Hybrid 46	*Yr4* +	YrSP/6* Avocet S	YrSP
Reichersberg 42	*Yr7* +	Yr11/3* Avocet S	Yr11
Heines Peko	*Yr6, Yr2* +	Yr12/3* Avocet S	Yr12
Nord Deprez	*Yr3N*	Yr15/6* Avocet S	Yr15
Compare	*Yr8, APR*	Yr17/4* Avocet S	Yr17
CarstensV	*YrCV*	Yr18/3* Avocet S	Yr18
Spaldings Prolific	*YrSP*		
HeinesVII	*Yr2, Yr11* +		
GIII. Supplemental Differential Set[1]		**GV. Common Cultivars**	
Sonalika	*Yr2, YrA*	Seri 82	Yr9, Yr7
Anza	*Yr 18, YrA*	Cook	APR
Fed./Kavkaz	*Yr9*	Corella	Yr6, Yr7
Gerek	-	Oxley	Yr6, APR
Cham 1	-	Kalyansona	Yr2
		Federation	
Morocco	Check	Crankbrook	
Seri 82	*Yr9, Yr7*	Jupateco'R'	Yr18 +
		Jupateco'S'	-

Source: [1] R.W.Stubb (IPO); [2] C.R. Wellings (Cobbity PBI)

Results and Discussions

The surveys indicate that rust populations are often variable for genes determining virulence, although the extent of variability varies with time, with locations, and between crop species (Yahyaoui 2000). The Syrian pathogen population was more diverse for virulence than that in Iran. The Syrian population also produced a significantly higher ACI compared to the Iranian population in controlled inoculation studies (Yahyaoui 2000). It is important to expose resistant plants to all potential

variation in the pathogen population. This may involve using a much larger number of pathogen genotypes than those currently used in breeding programs in the region.

Yellow rust populations in Central and Western Asia (CWA) were genetically diverse and differences in pathogenicity have been identified; however the precise identification of physiological races in CWA has not been fully determined (Yahyaoui 2000). At all the testing sites reported in this study, Morocco and Avocet S were in fact the most susceptible cultivars in the field, followed by other known susceptible cultivars such as Gerek, Federation, Sonalika, Kalyansona, and Seri 82 (Table 2). Disease reactions of common cultivars tested in CWA allow comparison with the behavior of known varieties to yellow rust isolates in different agro-ecological zones (Table 3). Seri 82, Federation, and Sonalika were susceptible to yellow rust at all sites for three consecutive years but were rated as resistant in Turkey and Kazakhstan during the 2000 crop season. This may indicate a change in virulence or a misreading of the lines and would have to be followed up closely. This information is essential for wheat breeders to understand the behavior of the varieties in a broader agro-ecological region. Levels of virulence to differential lines (group I) with *Yr1* and *Yr3V* followed similar trends in Syria, Lebanon, Turkey, Tajikistan, and Kyrgyzstan. In both regions no virulence to *YrSp, YrCV,* and Yr8 was detected or was consistently low.

In CWA, virulence on yellow rust resistance genes was variable. For some genes virulence was detected only at one site such as the case of *Yr4+* and *Yr3N* in Tajikistan during the 2000 crop season. Virulence on *Yr5* was reported in Tajikistan in the 2000 field nurseries. Virulence on *Yr10* was noted in Syria in 1998 but not found in the following seasons, likewise in Tajikistan in 1999.

Table 2. Reaction of differential genotypes to yellow rust in Western and Central Asia over three cropping seasons (1998-2000).

		Testing sites[1]											
Cultivar	***Yr Gene***	**SYR00**	**SYR99**	**SYR98**	**LEB00**	**LEB99**	**LEB98**	**TUR00**	**TUR99**	**TUR98**	**TAJ00**	**TAJ99**	**KYR99**
Chinese 166	*1*	1R	1R	1R	5MS	1R	5MR	1R	1R	1R		90S	5
Lee	*7*	60S	60S	80S	5MR	60S	70S	80S	60S	S	0	80S	10
Heines Kolben	*6*	80S	90S	85S	15MS	80S	70S	60MS	70S	S	0	60S	60
Vilmorin 23	*3V*	5R	10R	1R	15MS	1R	10MR	R	1R	R	R	90S	0
Moro	*10*	10MR	10R	10S	5S	1R	5MR	R	20MS	R	0	90S	0
Strubes Dickkopf	*SD*	10MR	30S	30MS	TMS	5S	5MS	20MR	1R	R	0	20MR	0
Suwon 92xOmar	*SU*	20MR	30MS	30MS	5MR	5MS	30MS	20MS	20MS	R	4MR	1R	0
Clement	*9, 2 +*	30MS	25MS	60S	TR	5S	40MS	10MS	1R	R	4MR	20MR	0
Triticum spelta	*5*	1R	5R	1R	TR	1R	1R	10S	1R	MR	50S	50MS	Na
Hybrid 46	*4 +*	1R	1R	1R	5MS	1R	5MR	R	1R	R	S	50MR	0
Reichersberg 42	*7 +*	10MR	60MS	60MS	5MS	5MS	15MS	R	10MR	R	MS	10MR	0
Heines Peko	*6, 2 +*	10R	60MS	60MS	TMR	5S	25MS	R	20MR	R	MS	5R	1
Nord Desprez	*3N*	1R	5R	1R	TMR	5S	15MR	R	1R	R	S	10MR	0
Compair	*8, APR*	5R	5MR	10MR	TMR	1R	5MR	20MS	10MR	R	MR	10MR	1
Carstens V	*CV*	1R	5R	1R	TMR	1R	5MR	R	1R	R	MR	10MR	0
Spaldings Prolific	*SP*	1R	1R	1R	TR	1R	5MR	R	1R	R	MR	10MR	0
Heines VII	*2, 11 +*	10R	30MS	50S	TR	5MS	30MS	10MR	5MR	MR	5R	1R	0
Anza	*18, A*	65MS	70S	75S	60S	30S	70S	R	80S	S	0	90S	5
Fed.4/Kavkaz	*9*	90S	80S	85S	40S	65S	90S	60S	80S	S	60S	80MS	20
Gerek		75MS	40S	90S	70S	20S	50S	R	80S			80S	
Cham1		5R	5R	1R	5MS	1R	1R	1R	1R			1R	

[1]SYR:Syria, LEB:Lebanon, TUR:Turkey, TAJ:Tajikistan, KYR: Kyrgyzstan

Table 3. Reaction of common cultivars to yellow rust in Western and Central Asia over three cropping seasons (1998-2000).

		Testing sites[1]											
Cultivar	**Yr Gene**	**SYR00**	**SYR99**	**SYR98**	**LEB00**	**LEB99**	**LEB98**	**TUR00**	**TUR99**	**TUR98**	**TAJ00**	**TAJ99**	**KYR99**
Morocco		95S	99S	99S	70S	90S	95S	50S	95S			80S	
Jupateco R	18	50MS	60MS	85S	10MS	25S	60S	30MS	40MS		R	40MS	10
Jupateco S	-	85S	90S	95S	40S	70S	70S	40MS	80S		R	80S	20
Kalyansona	2	80S	90S	95S	40S	80S	80S		80S	S	4MR	80S	60
Federation S	9	90S	90S	95S	70S	90S	90S	R	70S		40S	90S	60
Cranbrook	7	85S	90S	95S	30S	40S	80S		70S		4MR	80S	na
Corella	6, 7	60S	80S	90S	10MS	25S	80S	40MS	80S		MR	90S	0
Oxley	6, APR	60S	70S	80S	25S	10S	60MS	10S			MR	80S	na
Cook	APR	30MR	70MS	20MS	TR	5S	25MS	R	1R		40S	80S	0
Sonalika	2, A	70MS	80S	90S	40S	70S	70S	R	80S	S	50S	80S	na
Seri 82	9, 7	70S	90S	40S	25S	80S	80S	R	50S			80S	

SYR: Syria, LEB: Lebanon, TUR: Turkey, TAJ: Tajikistan, KAZ: Kazakhstan, KYR: Kyrgyzstan..

The information from the set of the Avocet near isogenic lines (NIL) was analyzed during 2000 crop season (Table 4). In this differential set (Group IV) the assessment of the reaction of the genotypes to yellow rust was relatively easier compared with rating of the differentials listed in groups I, II, and III where several cultivars carry more than one resistance gene. Virulence for *Yr* genes in the Avocet NIL's was evaluated at major experimental stations in Syria, Lebanon, Turkey, Kyrgyzstan, Azerbaijan, and Tajikistan. Virulence for *Yr1* (Table 4) was only found in Kyrgyzstan (Bishkek station). Virulence for *Yr11* and *Yr12* reported in this study do not reflect the true reactions types that would be associated with these genes R. Johnson, (*personal communication*); these lines will be discarded from the NIL's in future studies. Virulence for *Yr1* and *Yr10* was observed in Tajikistan in 1999 but was not found in 2000 on the NIL's or the world differentials (Tables 2,4). Avirulence for *Yr9* (Table 4) was observed in Turkey and at one site in Azerbaijan, even though good conditions for yellow rust development prevailed in both countries during the 2000 crop season. Virulence for *Yr9* was found at all the other sites. The virulence for *Yr5* observed in Turkey (Table 4) should be confirmed, otherwise, *Yr5* remains as an effective resistance gene in this region. The effectiveness of *Yr5* could be attributed to its limited exposure as it is not present in commercial varieties.

Adult plant resistance in the Avocet *Yr18* line was better than that in Avocet S even though it gave high responses compared with several other genes. Its performance was not adequate at most testing sites (Table 4), hence the resistance conferred by *Yr18* should be associated with other genes in order to be effective in CWA (Table2) (See R. P. Singh, this volume). Higher infection was observed on the cultivar Anza than on Jupateco R, even though both cultivars have *Yr18*. The latter variety must have a gene or genes in addition to *Yr18* and hence less infection with yellow rust. R. Johnson (*personal communication*) suggested that, in addition to *Yr8*, the cultivar Compair also carries *Yr18* derived from its parent, Chinese Spring; hence the adult plant resistance reported in this study could be attributed to *Yr18* (see *Lr34* in McIntosh *et al.* 1995).

A wide range of virulent pathotypes is evolving in this region (Hakim *et al.* in this publication) causing the break down of widely utilized sources of resistance in wheat (Yahyaoui *et al.* this volume). The dynamics of yellow rust in this region are now better understood, but the pathways through which the pathogen is spreading are still unknown. Hence the knowledge of the yellow rust pathway is essential for the proper exploitation and management of available sources of resistance. In CWA, surveys of pathogen populations and the genetic characterization of resistance continue to provide valuable information used to design breeding strategies.

Table 4. Reaction of Avocet Near Isogenic lines to yellow rust in Western and Central Asia during 2000 cropping season.

		Testing site[1]						
Cultivar	***Yr Gene***	**SYR00 (Th)**	**LEB00 (Te)**	**TUR00 (Hym)**	**KYR00 (Bish)**	**AZE00 (AbS)**	**AZE00 (Tar)**	**TAJ00**
Yr1/ 6* Avocet S	*1*	1R	5MS	R	30S	0	0	6MR
Yr 5/ 6* Avocet S	*5*	10R	TMS	80S	1R	0	0	50R
Yr 6/ 6* Avocet S	*6*	90S	TMS	R	40S	80S	20S	S
Yr 7/ 6* Avocet S	*7*	95S	70S	70S	60S	90S	20S	S
Yr 8/ 6* Avocet S	*8*	10MR	70S	R	70S	10MR	1R	3MR
Yr 9/ 6* Avocet S	*9*	95S	40S	R	10MS	60S	40S	5MR
Yr 10/ 6* Avocet S	*10*	5MS	70S	R	1R	10MR	1R	MR
Yr 11/ 3* Avocet S	*11*	90S	5MR	30S	50S	30S	20S	4MR
Yr 12/ 3* Avocet S	*12*	80S	50S	80S	40S	50S	20S	0
Yr 15/ 6* Avocet S	*15*	1R	40S	30S	5R	1R	20S	0
Yr 17/ 4* Avocet S	*17*	50S	5MR	40MS	30S	20MS	1R	0
Yr 18/ 3* Avocet S	*18*	65MS	10S	70S	20S	20MS	40S	R
Yr Sp / 6* Avocet S	*Sp*	1R	15S	60MS	1R	1R	1R	R
YrSk / 3* Avocet S	*27*	60S	15S	40MS	20S	10MS	1R	4MR
Avocet S	-	99S	70S	75S	80S	70S	70S	70S
Avocet R	*A*	90S	40S	20S	70S	60S	70S	50S

SYR: Syria, LEB: Lebanon, TUR: Turkey, TAJ: Tajikistan, KAZ: Kazakhstan, KYR: Kyrgyzstan, AZE: Azerbaijan

References

Hong Ma, Singh RP, 1996. Expression of adult Resistance to Stripe Rust at Different Growth Stages of Wheat. *Plant Disease* **80** 4: 375-375

Johnson R, 1988. Durable resistance to yellow (stripe) rust in wheat and its implications in plant breeding. Pages 63-75 In: *Breeding Strategies for Resistance to the Rusts of Wheat.* N.W. Simmonds and S. Rajaram, Eds. CIMMYT, Mexico, D.F., Mexico.

McIntosh RA, Wellings CR, Park RF, 1995. *Wheat Rusts: An Atlas of Resistance Genes.* CSIRO, Melbourne, Australia pp 200.

Peterson RF, Cambell AB, Hannah AE, 1994. A diagrammatic scale for estimating rust intensity of leaves and stem of cereals. *Canadian Journal of Research Section* C **26:** 496-500.

Singh RP, 1992. Genetic association of leaf rust resistance gene *Lr34* with adult plant resistance to stripe rust in bread wheat. *Phytopathology* **82:** 835-838.

Kolmer JA, 1997. Virulence in *Puccinia recondita* f.sp. *tritici* isolates from Canada to genes for adult-plant resistance to wheat leaf rust. *Plant Disease* **81** 3: 267-271.

Singh RP, Rajaram S, 1994. Genetics of adult plant resistance to stripe rust in ten spring bread wheat. *Euphytica* **72:** 1-7.

Yahyaoui A, 2000. Emergence of new races of yellow rust of wheat in West and Central Asia. *Germplasm Program Cereal Annual Report for 2000.* ICARDA 2001.

Virulence of Wheat Yellow Rust on Field Grown Yellow Rust Differentials, Turkish and Regional Wheat Varieties in Ankara

L. Çetin[1], F. Dusunceli[1], S. Albustan[1], Z. Mert[1], C. Wellings[2], M. Torabi[3] and A. Yahyaoui[4]

[1] The Central Research Institute for Field Crops (CRIFC), Ulus, Ankara, Turkey,
[2] The University of Sydney, Plant Breeding Institute, Cobbity, Australia,
[3] The Seed and Plant Improvement Institute, Karaj, Iran,
[4] The International Center for Agricultural Research in the Dry Areas, Aleppo, Syria

Introduction

Wheat is the most important cereal crop in Turkey, with most production from Central Anatolia. The mean yield has been increased to over 2000 kg/ha in recent years, but the yield and grain quality are frequently reduced by rust epidemics. Of the three wheat rusts, yellow (stripe) rust caused by *P. striiformis* f.sp. *tritici* is the most prevalent in Central Anatolia favored by prolonged cool and moist springs (Cetin *et al.* 1996). However, yellow rust (YR) may also occur in other parts of the country in some years with favorable conditions. In the last decade yellow rust epidemics caused significant yield losses in Central Anatolia in 1991 (Braun and Saari 1992), in Cukurova in 1995 (Dusunceli *et al.* 1996) and again in Central Anatolia in 1998 (Dusunceli *et al.* 1999).

The disease becomes more destructive following formation or arrival of new pathotypes carrying virulence on resistance genes that may be widely used in a region. In this respect, the severity of the 1995 epidemic in Cukurova has been attributed to the arrival of virulence for *Yr9* from the south. Thus, monitoring virulences in the population is vital in order to take necessary precautions. In this study, the change in virulence pattern of the YR populations around Ankara was monitored through observations on field grown trap nurseries, consisting of entries with known resistance genes and some varieties from Turkey and also from around the region.

Materials and Methods

A set of YR differentials was sown as part of the Cereal Diseases Trap Nursery (CDTN) in 1991 and continued throughout this study. This nursery included World differentials, European differentials and a supplemental set (kindly provided by R. Johnson of the John Innes Centre, UK and G. H. J. Kema of the Research Institute for Plant Protection (IPO), The Netherlands, and some Turkish wheat varieties (Table 1). This nursery was supported by addition of two other nurseries of international significance. The Near Isogenic Lines (NILs) carrying *different Yr genes in the background of the susceptible cultivar Avocet S* (Table 2) and Central and West Asian Yellow Rust Trap Nursery (CWAYRTN) pepared *by ICARDA (Syria) and SPII (Iran) were used between* 1998 - 2001. The CWAYRTN contained *varieties from countries of the region* as well as other differential genotypes including World differentials, European differentials, supplemental set and some entries of NILs. Resistant and susceptible checks were also included in the nurseries.

The nurseries were grown at two research sites of the Central Research Institute for Field Crops-Ankara (CRIFC). The Yenimahalle location is in the city of Ankara (840 m altitude) and the Haymana location is 45 km south-west of Ankara (1150 m altitude). Entries of the nurseries were hand planted in single rows of 1-2 m among other wheat nurseries sown for screening for resistance to YR. The nurseries were inoculated with uredospores collected from around Ankara in the previous year. Scoring was done using the modified Cobb scale. The disease development was monitored through the season and three scores were taken.

Results and Discussion

Good disease development was observed in most years, but in some, rust development was low owing to higher temperatures, such as in 1992 and 1993. In general, the Yenimahalle location warms up more quickly than the Haymana location and therefore the disease severity was lower there than in Haymana. The resistant check Pastiche was resistant while the susceptible checks Michigan Amber and Taichung gave susceptible readings of over 70S in both locations, in all years the study was undertaken.

The traditional World differentials, European differentials and supplemental set were included both in Cereal Diseases Trap Nursery (CDTN) planted between 1991 - 2001 and in the Central and West Asian Yellow Rust Trap Nursery (CWAYRTN) planted in the period 1998 - 2001. Of these differentials, Chinese

166, Vilmorin 23, Hybrid 46, Nord Desprez, Compair, Carstens V, Spaldings Prolific and *Triticum spelta* f. sp. *album*, carrying respective Yr resistance factors *Yr1, Yr3a+, Yr4+, Yr3a+, Yr8, CV, Sp* and *Yr5* were resistant all through the study between 1991-2001 in both locations (Table 1). The differentials Lee, Heines Kolben and Kalyansona, carrying *Yr7, Yr6* and *Yr2* respectively, were susceptible in all seasons. Similarly, the lines Anza, Sonalika and Gaby were also susceptible in general, although the scores were variable between TS - 70S between 1995 -2001 in which they were included in the study. The susceptible check Taichung scored over 80S between 1995 - 1999 and was removed from the nursery in following years. In contrast to these genotypes some others gave variable YR readings. For example Clement (*Yr9+*) gave maximum readings of TS, 5MS, 5-40MS, T-30MS in years 1996, 1997, 1998 and 2001 in Haymana and 5-40S in 2001 in Yenimahalle, while in other years it showed no infection.

Similarly, Heines Peko gave readings of between TMS - 30 MRMS in Haymana in years 1995 - 1999 and produced no infection in 2000, but in 2001 it scored 5-30 MSS and 20MS in Haymana and Yenimahalle respectively. Heines VII showed no infection except in 1998 (20MS) in Haymana and in 2001 in Haymana (5MSS) and in Yenimahalle (10MSS).

The greatest change was recorded in the virulence for Federation 4 / Kavkaz carrying *YR 9*, which was overcome (80S in Haymana and 40S in Yenimahalle) in 1995. This coincided with the loss of resistance of Seri 82 in the Cukurova region on the eastern Mediterranean coast resulting in losses of more than half a million tonnes of wheat (Dusunceli *et al.* 1996) in the same year. Susceptible readings with varying degrees of severity (20S - 90S) were recorded on Federation 4 / Kavkaz in the following years.

More recently, signs of virulence for *YR10* were observed first in year 1998 on the cross of M2435 * 6/*Yr10* (Table 2), which gave maximum readings of 70S in 1998, 5-40MSMR in 1999 and 20-60MSS in 2000. However, in these years the genotype Moro *(Yr10)* produced no infection at all, suggesting that Moro may carry effective additional adult plant resistance the or that the M2435 * 6/*Yr10* cross may not carry *Yr10*. Thus this entry was excluded from the nurseries in 2001. The near isogenic lines of Aroona crosses were also excluded from the CWAYRTN in the 3rd and 4th year as they were giving false readings.

Significant changes were observed in the virulence pattern of YR populations in 2001. The virulence for the genotype Moro *(Yr10)* was recorded for the first time in Haymana - Ankara in this year. The Moro entries were scored 10-50S and

Table 1. Stripe (Yellow) rust (*Puccinia striiformis* f.sp. *tritici*) scores (1) on World, European and supplemental yellow rust differential set entries in Haymana and Yenimahalle locations in Ankara between 1991 and 2001.

		Years										
		1991	1992	1993	1994	1995		1996		1997	1998	
Genotypes	*Yr Gene*											
						Locations 1						
		Hay.	Y.M.	Hay.	Y.M.	Hay.	Y.M.	Hay.	Y.M.	Hay.	Hay.	Hay.
							Nurseries 2					
		CDTN	CDTN	CDTN	CDTN	CDTN	CDTN	CDTN	CDTN	CDTN	CDTN	CWA
Chinese 166	*1*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*
Lee	*7*	60S	5S	5S	50S	60S	20S	80s	50S	70S	50S	90S
Heines Kolben	*6*	40MS	5S	40SMS	TS	60S	50S	30S	20S	70S	60S	90S
Vilmorin 23	*3V*	0	0	0	0	0	0	0	0	0	0	0
Moro	*10*	0	0	0	0	0	0	TMR	0	0	0	TMR
Strubes Dickkopf	*SD*	0	0	0	0	0	0	0	0	0	0	0
Suwon 92 x Omar	*SO*	0	0	5MS	0	0	0	0	0	0	0	5-30MR
Clement	*9,2 +*	0	0	0	0	0	0	TS	0	5MS	10MS	5-40MS
Hybrid 46	*4 +*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*	*0*
Reicherberg 42	*7 +*	0	0	0	0	0	0	0	0	0	TMS	5-20MS
Heines Peko	*6, 2 +*	NT	0	NT	NT	20MR	5SMS	TS	0	20MS	30MS	5-20MS
Nord Desprez	*3N*	0	0	0	0	0	0	0	0	0	0	0
Compare	*8, 18*	0	0	0	0	0	0	TMS	0	0	0	TMS
Carstens V	*CV*	0	0	0	0	0	0	0	0	0	0	0
Spaldings Prolific	*SpP*	0	0	0	0	0	0	0	0	0	0	0
Heines VII	*2, 11 +*	0	0	0	0	0	0	0	0	0	20MS	T-10MS
Feder 4 x Kavkaz	*9*	0	0	0	0	80S	40S	20S	5S	70S	70S	70S
Kalyansona	*2*	60S	50S	60S	50S	80S	30S	30S	20S	70S	70S	70S
Anza	*A*	NT	NT	NT	NT	70S	5S	5S	TS	50S	70S	5MS
Sonalika	*2, A*	NT	NT	NT	NT	70S	5S	10S	TS	70S	70S	10MS
Gaby	*Gaby*	NT	NT	NT	NT	10S	0	5S	TS	10MS	70S	NT
T. spelta f.sp. album	*Yr-5*	0	0	0	0	0	0	0	0	0	0	0
Taichung	*Susc.*	NT	NT	NT	NT	90S	80S	90S	90S	90S	90S	NT
Pastiche	*Res.*	0	0	0	0	0	0		0	0	0	NT
Michigan amber	*Susc.*	100S	50S	90S	70S	90S	50S	90S	60S	90S	90S	NT

1: Based on Modified Cobb scale, NT: Not tested either because of exclusion from the nursery or because of poor stand; *2: Hay.: Haymana, Y.M.: Yenimahalle - Ankara; 3: CDTN: Cereal Disease Trap Nursery, CWA: Central and West Asia Yellow Rust Trap Nursery (CWAYRTN).

(Contd.....)

Contd Table 1

Genotypes	*Yr Gene*	Years									
		1999			2000			2001			
		Locations 1									
		Hay.	Y.M.	Hay.	Hay.	Y.M.	Hay.	Hay.	Hay.	Y.M.	Y.M.
		Nurseries 2									
		CDTN	CDTN	CWA	CDTN	CDTN	CWA	CDTN	CWA	CDTN	CWA
Chinese 166	*1*	0	0	0	0	0	0	0	0	0	0
Lee	*7*	50S	50S	10-50S	70S	50S	80S	50S	10-70S	60S	70S
Heines Kolben	*6*	60S	60S	20-50S	60S	50S	60MS	40S	20-70S	70S	70S
Vilmorin 23	*3V*	0	0	0	0	0	0	0	0	0	0
Moro	*10*	0	0	0	0	0	0	0	5-50S	TMR	0
Strubes Dickkopf	*SD*	0	0	0	0	0	0	0	5-30MS	5-20S	20MR
Suwon 92 x Omar	*SO*	0	0	0	0	0	0	0	0	5-20MS	10MR
Clement	*9,2 +*	0	0	0	0	0	0	0	T-30MS	5-20MS	5-30S
Hybrid 46	*4 +*	0	0	0	0	0	0	0	0	5-40S	0
Reicherberg 42	*7 +*	0	0	TMS	0	0	0	0	5-20MS	0	20MR
Heines Peko	*6, 2 +*	TMS	TMS	TMS	0	0	0	0	5-30MS	20MS	20MR
Nord Desprez	*3N*	0	0	0	0	0	0	0	0	20MS	0
Compare	*8, 18*	0	0	0	0	0	5-30MS	0	0	0	0
Carstens V	*CV*	0	0	0	0	0	0	0	TS	0	0
Spaldings Prolific	*SpP*	0	0	0	0	0	0	0	0	0	0
Heines VII	*2, 11 +*	0	0	0	0	0	0	0	0	5MRMS	10MS
Feder 4 x Kavkaz	*9*	40S	5-40S	5-40MS	70S	5MS	20-70S	70S	10-50S	70S	20-60S
Kalyansona	*2*	40S	5-40S	5-20S	30MSS	-	70S	30MSS	70S	70S	10-50S
Anza	*A*	40S	40S	60SMS	50S	20-50MS	60S	50S	70S	70S	20-60S
Sonalika	*2, A*	50SMS	50SMS	20-60S	5-90S	30MS	TMR	5-90S	NT	5-20S	NT
Gaby	*Gaby*	5S	5S	NT	NT	NT	NT	NT	NT	NT	NT
T. spelta f.sp. album	*Yr-5*	0	0	0	0	0	0	O	0	0	0
Taichung	*Susc.*	90S	90S	NT	NT	NT	NT	NT	NT	NT	NT
Pastiche	*Res.*	0	0	NT	NT	NT	NT	NT	NT	TN	NT
Michigan amber	*Susc.*	70S	70S	NT	90S	90S	NT	60S	NT	80S	NT

1: Based on Modified Cobb scale, NT: Not tested either because of exclusion from the nursery or because of poor stand; *2: Hay.: Haymana, Y.M.: Yenimahalle - Ankara; 3: CDTN: Cereal Disease Trap Nursery, CWA: Central and West Asia Yellow Rust Trap Nursery (CWAYRTN).

5-50S in the nurseries of CDTN and CWAYRTN. However, two entries of *Yr10*/6* Avocet in the NILS and CWAYRTN nurseries were resistant in Yenimahalle and Haymana in the seasons in which it was included (1998 to 2001, Table 2). This contradicting result may be either the result of a low frequency of the pathotype carrying virulence for *Yr10* in the field or to additional resistance in the NIL.

In 2001 the highest YR scores were recorded for Strubes Dickkopf (5-30MS and 5-20S in Haymana and Yenimahalle respectively), for Suwon x Omar (5-20 MS in Yenimahalle), Clement (T-30MS and 5-40S in Haymana and Yenimahalle) and Reichersberg 42 (20 MS in Yenimahalle). Also the previously resistant bread wheat varieties Sultan 95 and Mizrak 98 giving maximum scores of TMR were recorded as susceptible 70 SMS and 5-60SMS for the first time in Haymana in 2001 (Table 3). The coincidence of such levels of YR infection on these genotypes and on Moro might indicate that these two cultivars may carry *Yr10* or that these cultivars and the susceptible differentials may carry other common genes such as the resistance symbolised in Clement as the + sign and probably common in wheat from Western Europe, such as Strubes Dickkopf, Clement and Reichersberg 42 (R. Johnson, personal communication).

An apparently similar contradictory observation was also made for *Yr8*. While the genotype Compair showed no infection, the *Yr8*/6* Avocet S entry of NILs gave gave maximum scores of 60MS in 1999, 40MS in 2000 and 50S in 2001. The same entry gave 80S and 90S score in CWAYRTN in Yenimahalle and Haymana. These differences can probably be explained by the presence of adult plant resistance in Compair controlled by the gene *Yr18*, derived from Chinese Spring during its development. These results demonstrate the value of near-isogenic lines over cultivars in the interpretation of pathogen virulence characteristics in trap nurseries.

Climatic conditions such as temperature, light and humidity may have been playing a role in expression of some genes in different backgrounds and occurrence of various complications. These matters remain unresolved and seedling tests and further gene postulation studies are required under controlled conditions to clarify these issues.

Of the other entries in the NILs nursery, which was included in the study after 1998, The *Yr1*, *Yr5* and *YrSp* were resistant while check entries Jupateco S, Avocet R *(YrA)* and Avocet S were susceptible scoring over 70S in all 4 years. The genotypes carrying *Yr6*, *Yr7*, *Yr9*, *Yr18*, *Yr24* and *Yr26* gave susceptible

readings in all years, though in some years severity was low. Although high infections were noted for *Yr18* (70S), *Yr24* (40MSS) and *Yr26* (20-60S) in 2001, these levels were lower than that in relevant control line Avocet S (80s). Similarly Jupateco R, which carries *Yr18* did not exceed that of its control genotype Jupateco S. Hence it was concluded that these genes have remained effective. Virulence for *Yr17* was first observed at both locations in 2001. Also, the *YrSkI* line *(Yr27)* was recorded as resistant in 1999 and 2000 but it was susceptible in 2001 scoring 5-30S in Yenimahalle and 80S in Haymana.

Registered Varieties and Candidate Lines

Most of the older Turkish wheat varieties were susceptible all through the study (Tables 3 and 4). The previously resistant Lancer and Yektay maintained their resistance through the study. The previously resistant varieties Sultan 95 and Mizrak 98 became susceptible in 2001 as noted above. The bread wheat varieties Bayraktar 2000, Aksel 2000, Yakar 2000 and Demir 2000, which were recently released by the Central Research Institute for Field Crops, maintained their resistance all through the study. Of the 45 regional varieties included in the CWAYRTN 11 gave clear susceptible readings and the others showed varying degrees of resistance.

Table 2. Stripe (Yellow) rust *(Puccinia striiformis* f.sp. *tritici)* scores (1) on Near Isogenic Lines (NIL) and additional differentials of the Central and West Asia Yellow Rust Trap Nursery (CWAYRTN) in Haymana and Yenimahalle locations in Ankara between 1998 and 2001.

		1998		1999				2000			2001		
								Locations 2					
		Hay.		Y.M.		Hay.		Hay.	Y.M.		Y.M.		Hay.
								Nurseries 3					
Pedigree	Gene	NIL	CWA.	NIL	CWA.	NIL	CWA.	NIL	NIL	CWA.	NIL	CWA.	CWA.
Yr1 / 6* Avocet S	*Yr1*	0	0	0	NT	0	0	0	0	NT	0	0	0
Yr5 / 6* Avocet S	*Yr5*	0	0	0	TMR	0	TMR	0	0	0	0	0	0
Yr6 / 6* Avocet S	*Yr6*	-	NT	80S	NT		NT	90S	NT	NT	80S	90S	90S
Yr7 / 6* Avocet S	*Yr7*	80S	NT	70S	NT	60S	NT	90S	5S	NT	90S	90S	90S
Yr8 / 6* Avocet S	*Yr8*	TR	5MS	5-30MS	10-50MSS	20MSMR	TMR	5-20MRMS	20-70S	5-30MS	0	10-30MRMS	50S ?
Yr9 / 6* Avocet S	*Yr9*	70S	NT	10-40MSS	NT	-	NT	60MSS	-	NT	80S	80S	90S
Yr10 / 6* Avocet S	*Yr10*	0	NT	0	NT	0	NT	0	0	NT	0	0	0
Yr11 / 6* Avocet S	*Yr11*	-	NT	10-40MSS	NT		NT	-	-	NT	70S	70S	90S
Yr12 / 6* Avocet S	*Yr12*	-	NT	5-20MSS	NT		NT	50SMS	-	NT	5-30MS	70S	70S
Yr15 / 6* Avocet S	*Yr15*	0	0	0	0	0	0	0	0	0	0	0	0
Yr17 / 6* Avocet S	*Yr17*	0	NT	0	NT	0	NT	0	0	NT	30S	1-20MS	0 ?
Yr18 / 6* Avocet S	*Yr18*	-	NT	5MRMS	NT		NT	60S	-	NT	10-60S	5-50S	70S
Yr24 / 6* Avocet S	*Yr24*	-	NT		NT		NT	-	-	NT	5-30MS	5-40MSS	40MSS
Yr26 / 6* Avocet S	*Yr26*	-	NT		NT		NT	-	-	NT	TMS	5-40MSS	20-60S
YrSp / 6* Avocet S	*YrSp*	-	NT	0	NT		NT	0	-	NT	0	0/10S ?	0
YrSk / 3* Avocet S	*YrSk*	-	NT	1-20MS	NT	70S	NT	TMS	20-60 SMS	NT	5-30S	60S	80S
Jupateco R (Yr18)	*Yr18*	30-50MS	20-50MSS		20-60S	70S	0		5-30S	70S	1-20MSS	5-40S	50S
Jupateco S	*Check*	70S	70S		70S		5MSS			20-60S	5-40S	20-60S	80S

1: Based on Modified Cobb scale, '-': Not tested either because of exclusion in the nursery or because of poor stand; 2: Hay.:Haymana Y.M.:Yenimahalle - Ankara;
3:CDTN:Cereal Disease Trap Nursery, CWA: Central and West Asia Yellow Rust Trap Nursery (CWAYRTN).

(Contd.....)

Contd Table 2

		1998				1999			2000			2001	
		Locations 2											
		Hay.		Y.M.		Hay.		Hay.		Y.M.	Y.M.		Hay.
		Nurseries 3											
Pedigree	*Gene*	NIL	CWA.	NIL	CWA.	NIL	CWA.	NIL	NIL	CWA.	NIL	CWA.	CWA.
Avocet R (YrA)	*YrA*	90S	NT	70S	NT	70S	NT	-	-	90S	90S	90S	90S
Avocet S	*Check*	90S	90S	70S	80S	70S	50S	90S	NT	90S	80S	90S	NT
AROONA (S)	*Check*		90S				5/50MSS			70S		70S	80S
AROONA*5 / YR1 (S)	*Yr1*		0				0			-		0	0
AROONA*6 / YR5 (S)	*Yr5*		TMS				TMS			TMS		-	-
AROONA*6 / YR8 (S)	*Yr8*		1-10MSS			-			60S		10MSS	TS	
AROONA*3 / YR15 (S)	*Yr15*		0				0			0		0	0
AROONA*6 / YR17 (S)	*Yr17*		0				0			0		-	-
M2435			70S				1MS			0/50MS		-	-
M2435*6 / YR5 (S)	*Yr5*		0				R			0		-	-
M2435*6 / YR10 (S)	*Yr10*		70S				1MR			5-40MS		-	-
FEDERATION (S)	*Yr9+*		80S				70S			70S		70S	80S
CRANBROOK (S)	*Yr7*		70S				5-20S			70S		20-70S	60S
CORELLA (S)	*Yr6+Yr7*		-				5-40S			70S		5-30S	50S
OXLEY (S)	*Yr6+APR*	-				1MS			-		5-40S	10MSS	
COOK (S)	*APR*		0				0			5-20MS		5MSS	5S
MOROCCO	*CHECK*		90S				100S			90S		90S	8S

[1]: Based on Modified Cobb scale, '-': Not tested either because of exclusion in the nursery or because of poor stand; [2]: Hay.:Haymana Y.M.:Yenimahalle - Ankara; [3]:CDTN:Cereal Disease Trap Nursery, CWA: Central and West Asia Yellow Rust Trap Nursery (CWAYRTN).

Table 3. Maximum yellow rust (*Puccinia striiformis* f.sp. *tritici)* scores of some well known Turkish Bread and Durum wheat varieties under artificial epidemics in Haymana and Yenimahalle locations in Ankara between 1991 and 2001.

Varieties	Type	Max. YR Score (1991-2000)	YR Score (2001)	Varieties	Type	Max. YR Score (1991-2000)	YR Score (2001)
SURAK1593-51	Bread wheat	100S	100S	GUN 9	Bread wheat	70 S	80S
HAYMANA 79	,,	40 MSMR	1-10-50s	IKIZCE 96	,,	5 MS	1-10MS
CUMHURIYET 75	,,	80 S	NT	SULTAN	,,	5 MR	70S
YAYLA 305	,,	90 S	10-60$_S$	Mizrak 98	,,	5MR	5-60S
KIRKPINAR	,,	100 S	80S	Demir 2000	,,	5MS	TMR
YEKTAY 406	,,	30 SMS	5-50MSMR	Aksel 2000	,,	5MS	TMS
GEREK 79	,,	90 S	70S	Bayraktar 2000	,,	5MS	TMS
ATAY 85	,,	70 S	10-40MSS	Yakar 2000	,,	5MS	TMS
BEZOSTAYA I	,,	40 MSS	5-40MSS	BERKMEN	Durum Wheat	70 S	NT
BOLAL 2973	,,	70 S	20-60S	TUNCA	,,	70 S	70S
KIRAC 66	,,	40 MSS	10-60S	CAKMAK 79	,,	5-60 SMS	5MS
SIVAS 111/33	,,	80 S	70S	KUNDURU 1149	,,	50 MS	5-30MS
KöSE 220/39	,,	90 S	20-60S	DICLE 74	,,	70 S	NT
Seri 82	,,	20-80S	20-70S	KIZILTAN 91	,,	50 MSS	5-40S
Kinaci 97	,,	50MSS	20-70S	CESIT 1252	,,	40 MS	60MSS
Lancer	,,	5-40MSMR	TMS				

Table 4. Maximum yellow rust (*Puccinia striiformis* f.sp. *tritici*) scores of wheat varieties from the Central and West Asian countries under artificial epidemics in Haymana and Yenimahalle locations in Ankara between 1998 and 2001.

Variety	Origin	Years Tested	Max YR score	Variety	Origin	Years Tested	Max YR score
MOROCCO	CHECK	1998-2001	90S	UMANKA (S)	RUS	1998-2000	5MS
ALMOUT (W)	Iran	1998-2001	TMS	POBEDA (F)	RUS	1998-2000	60MSS
DARAB 2	Iran	1998-2001	0	AKHO (7F)	RUS	1998-2000	40SMS
NICKNEJAD (S)	Iran	1998-2001	0	OPHELIA (S)	RUS	1998-2000	80S
M-70-12 MAHDAVI (S)	Iran	1998-2000	0	BEZOSTAYA (W)	RUS	1998-2000	50MS
W-70-15 ATRAK (S)	Iran	1998-2000	0	SPARTANKA (F)	RUS	1998-2001	50MSS
C-70-16 ZARRIN (F)	Iran	1998-2000	TMS	YUNA (S)	RUS	1998-2001	80S
C-70-20 ALVAND (W)	Iran	1998-2000	0	SKIPHYANKA (F)	RUS	1998-2001	70S
W-18 BOW 'S' /NKT (TAJEN) (S)	Iran	1998-2000	0	DAKHA (S)	RUS	1998-2000	70S
ALMOUT / T171 /3/ MAYA //							
BB INIA /4/ KARDJ2 /5/ ANZA (S)	Iran	1998-2000	0	SPHERA (S)	RUS	1998-2000	60S
Sardari	Iran	,,	60S	EIKA (S)	RUS	1998-2000	60S
ERITH. 15236	UKR	1998-2000	80S	CHAM-6	SYR	1998-2001	0
LUT 17044.12 (S)	UKR	1998-2000	20-70S	CHAM 3	SYR	1998-2000	60MSS
LUT 20133 (S)	UKR	1998-2000	20-70S	CHAM 5	SYR	1998-2000	20MSMR
LUT 20148 (S)	UKR	1998-2000	20-60MSS	Cham 1	Syr/Lebanon	2001	0
LUT 20161 (S)	UKR	1998-2000	0	Cham 4	Syr/Lebanon	,,	0
LUT 20191 (S)	UKR	1998-2000	5-70S	Ulugbek 600	Uzbekistan	,,	0
ERYTHROSPERMUM 5678 / 87 (S)	UKR	1998-2000	5-20MSS	Sanzar 4	Uzbekistan	,,	80S
LUTESCENSN 9489 (F)	*UKR*	*1998-2000*	*5-50SMS*	*Sanzar 8*	*Uzbekistan*	,,	*70S*
KRASUNYA ODESSKAYA (S)	UKR	1998-2000	20-70S	Bakht	Tajikistan	,,	10-50S
UKRAINKA ODESSKAYA (S)	UKR	1998-2000	20-70S	5th FAWWON 35	Tajikistan	,,	0
VYMPEL ODESSKIY (S)	UKR	1998-2000	70S	Krasnonodopadsaya 25	Turkmenistan	,,	0
FANATSIA ODESSKAYA (S)	UKR	1998-2000	5-40MSS	Ak bugday	,,	,,	20-70S
ZABADA ODESSKAYA (S)	UKR	1998-2000	5-60SMS	Adyr	Kyrgyzistan	,,	0/60S
NADIA (S)	UKR	1998-2000	20-60S	Kyial	,,	,,	70S
ZOLOTAVA (S)	UKR	1998-2000	5-60SMS	Tilek	,,	,,	20-60S
DARUNOK (S)	UKR	1998-2000	5MS	Opaks 26	Kazakhistan	,,	20-70S
PORADA (S)	UKR	1998-2000	20-70S	Zhetysu	,,	,,	50S
STRUMOK (S)	*UKR*	*1998-2000*	*50S*	*Karakylchyk 2*	Azerbaijan	,,	*0*
POLOVCHANKA (F)	RUS	1998-2000	5MSS	Mirbashir 128	,,	,,	20-70S
KNYIGZNA (F)	RUS	1998-2000	TMS	Ani 591	Armenia	,,	0
DEMETRA (F)	RUS	1998-2000	70S	Lutescens 9489 (F)	UKR	,,	10-50S
ZIMORODOK (F)	RUS	1998-2000	50MSS	Aisi	Georgia	,,	50S

References

Braun HJ, Saari EE, 1992. An assessment of the potential of *Puccinia striiformis* f.sp. *tritici* to cause yield losses in wheat on the Anatolian Plateau of Turkey. *Vortrage für Pflanzenzüchtung.* **24**:121-123.

Cetin L, Dusunceli F, Albustan S, 1996. Evaluation of the Turkish winter and Facultative wheat germplasm for rust resistance in Central Anatolia. Cereal rusts and Powdery Mildews Bulletin. **24:** 240, Supplement, *Proceedings of the 9th Cereal Rusts and Powdery Mildews Conference,* 2-6 September 1996, Lunteren, The Netherlands.

Dusunceli F, Cetin L., Albustan S,. 1996. Occurrence and Impact of wheat stripe rust *(Puccinia striiformis)* in Turkey in 1994/95 crop season. Cereal rusts and Powdery Mildews Bulletin, **24**: 309, *Proceedings of the 9th Cereal Rust and Powdery Mildews Conference* 2-6 September 1996, Lunteren, The Netherlands.

Dusunceli F, Cetin L., Albustan S, 1999. Occurrence and importance of rust diseases *(Puccinia spp.)* in wheat fields of Central Anatolia and precautions against them. In Proceedings of the Conference on *Constraints to Cereal Production in Central Anatolia,* 8-11 June, 1999, Konya-Turkey, pp: 693-696 (In Turkish).

Use of Disease Nurseries

Effective Resistance Genes to Yellow (Stripe) Rust of Wheat in Central and Western Asia

A. Yahyaoui[1], C.R. Wellings[2], M. Torabi[3], K. Nazari[3], H. Ketata[1] and L. Çetin[4]

[1]International Center for Agricultural Research in the Dry Areas, Aleppo, Syria
[2]Plant Breeding Institute, Cobbitty, Sydney, Australia
[3]Seed and Plant Improvement Institute, Karaj, Iran
[4]CRFCI, Ankara, Turkey

Introduction

Yellow (stripe) rust (*Puccinia striiformis* f.sp. *tritici*) presents a constant threat to wheat production to many countries in Central and Western Asia (CWA) that include Lebanon, Iran, Iraq. Syria and Turkey in Western Asia, and Azerbaijan, Tajikistan, Uzbekistan, Kyrgyzstan, and Kazakhstan in Central Asia. A wide range of virulent yellow rust pathotypes are evolving in this region causing the break down of widely utilized sources of resistance in wheat (Hakim *et al.* this volume). Knowledge of effective resistance genes in the region will enable breeders to target those useful genes in their breeding programs with the hope of avoiding rust epidemics and subsequent crop losses in the future.

Wheat cultivars grown in CWA were resistant to the prevalent yellow rust populations when initially released. Within a few years of cultivation, the corresponding virulence of *P. striiformis* f.sp. *tritici* emerged and the resistance genes became ineffective. Currently, most wheat cultivars grown in CWA are susceptible to yellow rust. Earlier epiphytotics were recorded in the CWA region (Ahmad *et al.* 1991). The wide cultivation of susceptible cultivars in Syria, Lebanon and Turkey allowed development of a large population of *P. striiformis*, which allowed the development and spread of virulence changes and consequent epidemics. In Turkey, the wheat cultivar 'Gerek 79' was grown on more than one million hectares, and incurred losses of around 26.5% due to the yellow rust epidemic of 1991 (Braun and Saari, 1992. In Syria, annual occurrence of yellow rust has been observed since 1987 (El-Naimi and Mamluk 1995), and the disease spread to all wheat-growing areas (Mamluk *et al.* 1990). Under irrigated conditions and in high rainfall areas in northern Syria, the severity of infection reached up to 80 % on susceptible cultivars. Estimated yield losses on the cultivar Mexipak were 29% (Mamluk *et al.* 1989). Epidemics in Lebanon resulted in an approximate loss of 30% in national grain production in 1994 due to the widespread cultivation of Seri

82 and Mexipak (Mamluk 1995). During the 1993 and 1995 crop seasons, yellow rust epidemics caused significant yield losses in Iran (Torabi *et al.*, 1995). Severe damage was caused by yellow rust in 1998 in Azerbaijan (Yahyaoui 2000). In some cultivars such as Bezostaya, resistance remained effective over many years of widespread cultivation; such cultivars could have durable resistance (Johnson and Law, 1975; Johnson, 1978). Durable resistance (Johnson 1981, 1988) is resistance that has remained effective in a cultivar during its widespread cultivation for a long sequence of generations or period of time in an environment favorable to disease or pest. The objective of this study is to assess the resistance levels in cultivated wheat varieties deployed in CWA and the effectiveness of resistance genes to yellow rust.

Materials and Methods

Wheat cultivars. The wheat varieties used in this investigation were grouped in three categories:

a) 22 Wheat varieties or isogenic lines that have know resistance genes to yellow rust. Varieties/lines used in this study were those carrying genes of major importance to CWA region. Some of the resistance genes, such as*Yr9, Yr18*, were widely exposed to yellow rust pathotypes over the years, other like *Yr1, Yr5* have not been used in cultivated wheat and hence have had limited exposure to yellow rust in CWA.
b) 12 Wheat varieties that are grown over large areas in one or more countries in CWA. Varieties such as Bezostaya, Gereck, Yuna, Cham6, and Mirbachir 80 were cultivated over large areas and some have been in cultivation for over 20 years.
c) 14 Selected facultative winter wheat genotypes. The International Winter Wheat Project (IWWP) in Turkey developed facultative winter wheat varieties, targeted to highland regions and areas characterized by cool environments, in collaboration with CIMMYT and ICARDA.

Testing sites. The 48 wheat accessions, as described above, were incorporated in the yellow rust trap nursery (Yahyaoui, this volume) and planted at different sites in CWA. The testing sites covered experimental stations in Syria, Lebanon, and Turkey in Western Asia, and Azerbaijan, Kyrgyzstan, and Tajikistan in Central Asia. Collaborators in the respective countries carried out the evaluations. Some testing sites were not listed due to the inconsistency of the data reported, or the data have been presented in conferences by collaborators.

Yellow rust inoculation and evaluation. The wheat accessions were tested under artificial inoculation at Terbol (Lebanon), Tel Hadya (Syria), and Haymana

(Turkey) experimental stations, and natural infection at Selection-Bishkek (Kyrgyzstan), Gobustan and TarTar, Absheron (Azerbaijan), experimental stations, as well as the main experimental station in Tajikistan. Inoculation and evaluation methods were described by Yahyaoui (this volume). Reaction to yellow rust (Tables 1-3) is based on the highest reading observed during the study period at the respective testing sites. Conflicting reaction types of the resistance genes (Table1) that were recorded at two different stations within a testing site were also reported. Additional information on resistance genes (Table 1) associated with Seri 82 and Compair was proposed by Johnson (personal communication).

Results and Discussion

Resistant cultivars ought to be deployed against populations of pathogens rather than against a particular race (Martens *et al.* 1970, Schafer and Long 1988, Watson 1970). Specific resistance could lead to the development of virulent pathotypes as soon as it is deployed over large areas of cultivation. The development of durable and effective resistance to yellow rust pathogen would be the most appropriate control measure to adopt in the CWA region. Candidate genes for this type of resistance are those that confer adult plant resistance and when in combination with other resistance genes could allow the development of low levels of infection on the host cultivar. In the case of yellow rust, reduction of inoculum production would limit the multiplication and spread of new rust pathotypes.

At all the testing sites reported in this study the susceptible cultivars Morocco, Jupateco S and Avocet S were in fact the most susceptible cultivars in the field, followed by other known susceptible cultivars such as Gerek, Federation, Yuna, Sardari, and Seri 82 (Tables 1, 2). The virulence on the susceptible cultivars confirms the presence of yellow rust disease at the testing site; the differential reaction of the resistance genes indicates the presence of different yellow rust pathotypes (Yahyaoui, Hakim, this volume). The effectiveness of specific resistance genes such as *Yr1, Yr15, Yr17, Yr5, Yr9, Yr3N* and *YrA* varied among and between sites. The resistance gene *Yr9* is present in a wide array of winter and spring wheats (McIntosh *et al.* 1995) and is among the genes that have been exposed the most to yellow rust in Western Asia.

The reaction *Yr9* to yellow rust (Table 1) differed with the site and the genotypes in which it occurs. *Yr9* in Clement shows a resistance type that varied from highly resistant (R) to moderately susceptible (MS). The resistant reactions of *Yr9* in Avocet S in Turkey and Kyrgyzstan ought to be verified, because in both countries the Federation/Kavkas line with *Yr9* was susceptible, indicating the presence

of virulence for *Yr9* in the trials. Combinations of specific resistance genes do not appear to be effective, at least in the case of association of *Yr9* with *Yr7* in Seri82 and *Yr2* with *YrA* in Sonalika. The specific resistance gene, *Yr5,* shows high level of resistance at most sites and could be considered as an effective resistance gene to exploit in the breeding program. However, precautions should be taken in using such genes that have not been exposed in nature to a wide array of yellow rust pathotypes and environmental conditions. Adult plant resistance associated with *Yr18/* Avocet S, Jupateco R, and Anza respectively (Table 1) reduced infection when compared with the corresponding susceptible lines Avocet S and Jupateco S and Avocet S at the six testing sites over the past two crop seasons (1999-2000) during which yellow rust development was also adequate to discriminate between cultivars (Yahyaoui, this volume).

Apparently *Yr18* by itself does not confer enough protection against yellow rust in CWA. Association of *Yr18* and *YrA* in the cultivar Anza gave an intermediate resistant reaction (MR) in Kyrgyzstan, an intermediate to susceptible reaction (S-MS) in Syria and Azerbaijan, and a susceptible reaction (S) in Lebanon, Turkey, and Tajikistan. Jupateco R *(Yr18 +)* showed a susceptible reaction in Lebanon and Kyrgyzstan respectively, and a relatively high intermediate susceptible reaction at the other sites. Johnson (Personal communication) suggested that the cultivar Compair must have an APR gene (adult plant resistant gene) in addition to *Yr8* and this is likely to include *Yr18.* This confirms the resistant (R) to intermediate resistant (MR) reaction of Compair to yellow rust at the six sites (Table 1). As reported by Ma and Singh (1996), the slow rusting *Yr18* gene present in Kauz that did not confer adequate resistance under high disease pressure, likewise, *Yr8* alone (in Avocet) did not confer adequate resistance.

Hence adult plant resistance to be considered effective ought to be tested under different agro-ecological climates and should associate genes that alone or in combination have been exposed in association with commonly grown cultivars in the area of utilization. Bezostaya could be considered as one of the wheat cultivars that have been in cultivation in the CWA region for a long period of time. The reaction of Bezostaya to yellow rust (Table 2) during 2000 crop season gives an indication that this cultivar has adult resistance to yellow rust that is probably at least partly dependent on *Yr18* (McIntosh *et al.* 1995). The rough division into the categories of resistance used in the table, unfortunately, does not provide adequate discrimination to indicate possibly important differences between cultivars given the susceptible assessment 'S' (R. Johnson, Personal communication).

Table 1. Field reaction of wheat genotype with known resistance genes to yellow rust at six sites in Western and Central Asia (1999-2000).

Cultivar	Resistance Genes	Testing Sites (Countries)[1] and Reaction2 to Yellow Rust SYR	LEB	TUR	KYR	TAJ	AZE
Avocet S	-	S	S	S	S	S	S
Yr1/ 6* Avocet S	Yr1	R	MR	R	S	S	R
Chinese 166	Yr1	R	R-MS	R	MR-S	S	R
Yr15/ 6* Avocet S	Yr15	R	R	S	R	MS	R-S
Yr17/ 4* Avocet S	Yr17	S	MR	MS	S	R	R-MS
Yr 5/ 6* Avocet S	Yr5	R	R	R	R	MS	R
Triticum spelta	Yr5	R	R	R	-	MS	R
Nord Desprez	Yr3 N	R	MR	R	R	MR	R
Clement	Yr 9	MS-S	MS	R	R	MR	MR-MS
Yr 9/ 6* Avocet S	Yr 9	S	S	R	R	MR	S
Federation/4* Kavkas	Yr 9	S	S	MS-S	S	S	S
SERI 82	Yr 9, Yr 7	S	S	S	-	S	S
Avocet R	Yr A	S	S	R	S	R	S
Kalyansona)	Yr 2	S	S	S	S	S	S
Sonalika	Yr 2, Yr A	S	S	MS	-	S	S
Yr8/ 6* Avocet S	Yr 8	MS	R	R	S	S	R-S
Compare	Yr 8, APR	R	MR	MR	R	MR	R
Yr18/ 3* Avocet S	Yr 18	MS	S	S	S	S	S
Jupateco R	Yr 18, +	MS	S	MS	S	MS	MS-S
Anza	Yr 18, Yr A	MS-S	S	S	MR	S	MS-S
Jupateco S -	S	S	MS	S	S	S	
MOROCCO	-	S	S	S	S	S	S

[1]SYRr: Syria, LEB: Lebanon, TUR: Turkey, KYR: Kyrgyzstan, TAJ: Tajikistan, AZE: Azerbaijan.
[2]Field reaction to yellow rust at adult growth stage over 2 years (1999-2000)

The reaction of commonly grown cultivars in CWA (Table 2) suggests that some cultivars such as Cham 6 and Polovchanka could have effective adult plant resistance that may be controlled by more than one gene. The susceptible varieties being cultivated over large areas in CWA such as Gerek in Turkey, Sardari in Iran, Yuna in Uzbekistan, Mirbachir 80 in Azerbaijan, Seri 82 in Lebanon, and Skifyanka in Kyrgyzstan, to mention only a few, should be avoided as much as possible.

The introduction of new resistance cultivars in many countries in CWA is an absolute necessity in order to reduce adverse effects of yellow rust on wheat production in the region. The introduction of Polovchanka in Uzbekistan and reduction of area occupied by Yuna caused a big change in the development of yellow rust over the three previous cropping season. Low levels of yellow rust were observed in Uzbekistan in 2000 where most of the fields surveyed were grown to

the variety Polovchanka compared to 1999 and 1998 where the variety Yuna occupied over 60% of the wheat area (Yahyaoui, 2000).

Table 2. Field reaction of wheat cultivars commonly grown in Central and Western Asia to yellow rust at six location.

	Testing Sites (Countries)1 and Reaction2 to Yellow Rust					
Cultivar	SYR	LEB	TUR	KYR	TAJ	AZE
Gerek 79	S	S	R	MR	R	S
Seri 82	S	S	R	MR	MR	S
Sardari	S	MS	R	-	S	S
Bezostaya (S)	MS	S	MS	MS	S	MR
Polovchanka (F)	MR	S	R	R	MS	-
Yuna (S)	S	S	S	S	S	S
Skiphyanka (F)	S	S	S	S	S	S
Cham 6 (S/F)	MS	MS	R	-	MS	R
Skifyanka S	S	S	S	MS	S	
Opaks 26 S	S	S	S	MS	S	
Mirbachir 80				-	-	S

[1]SYRr: Syria, LEB: Lebanon, TUR: Turkey, KYR: Kyrgyzstan, TAJ: Tajikistan, AZE: Azerbaijan.
[2]Field reaction to yellow rust at adult growth stage during 1999-2000 crop seasons

New resistance sources to yellow rust are being developed by CIMMYT/ICARDA project in Turkey. A set of lines that was proposed and assembled by Ketata and Braun was tested at the same sites and evaluated over two crop seasons at some sites. The results obtained permitted classification of the fourteen accessions tested (Table 3) into five distinct groups (GR).

GR.1: Highly resistant at all sites but Turkey, entry Nos. 3, 4, 5, 9, 10, and 11.
GR.2: Highly resistant at all sites but Azerbaijan, entry No. 1.
GR.3: Highly resistant in Central but not in Western Asia, entry Nos. 2, 6.
GR.4: Mixed reaction over sites, entry Nos. 7, 8.
GR.5: Moderate infection types, entry Nos. 12, 13, 14.

The data suggest existence of specific resistance genes in the genotypes listed in groups 1, 2, and 3, and possibly in GR 4. Genotypes in GR5, showed a wide range of reaction types that varied from resistant (R) to intermediately susceptible (MS), particularly entries 12 and 13. These most likely have adult plant resistance conferred by at least two genes and should be further investigated.

In Central and Western Asia, none of the specific resistance genes gave complete resistance at all sites. Genes that have not been used in breeding programs in the region such as *Yr1* and *Yr5* showed better levels of resistance, which may due to the absence of virulence for these genes. Nonetheless virulence on *Yr1* has been

observed overt the past crop seasons in Tajikistan, Kyrgyzstan (Yahyaoui, this volume), and in Syria (H.K. Hakim, Personal communication.). The use of adult plant resistance in the breeding programs would allow enhancement of wheat productivity in CWA. The knowledge of effective resistance genes in the region will enable breeders to incorporate and accumulate/pyramid these genes in wheat germplasm, thus contributing to the development of resistant cultivars that sustain resistance over a longer period and may prove to be durable.

Table 3. Selected facultative winter wheat genotypes with resistant to yellow rust in CWA region.

Facultative winter wheat genotype	Ent. No	Testing Sites (Countries)[1] and Reaction[2] to Yellow Rust SYR	LEB	TUR	KYR	TAJ	AZE
AGRI/NAC//ATTILA CMSW92WM002325-0SE-0YC-12YE-0YC	GRP1 3	R	R	S	R	R	R
JI5418/HATUSHA CIT 930124-0SE-0YC-9YE-0YC	GRP1 4	R	R	S	R	R	R
KARL/ARIESAN CIT 930082-0SE-0YC-2YE-0YC	GRP1 5	R	R	MS	R	R	R
DYBR1982.83/842ABVDC50//KAUZ/3/PLK70/ LIRA CIT 89061T-0SE-0YC-3YC-0YC-2YC-0YC-1YC-0YC	GR1 9	R	R	MS	R	R	R
RSK/CA8055//CHAM6 CIT922189-0SE-0YC-1YC-0YC-2YC-0YC	GRP1 10	R	R	MS	R	R	R
AIZA0781/6/LOV11/SON64/4/PJ/GB55//093/ 3/44/ STW597947/5/TRK13/7/ORF1.158/FDL// BLO/3/CA8055 CIT937064-0SE-2YE-1YC-0YC	GRP1 11	R	R	S	R	R	R
TAM200/JI5419 CIT 930099-0SE-0YC-1YE-0YC	GRP2 1	R	R	R	R	R	S

Facultative winter wheat genotype	Ent. No	Testing Sites (Countries)[1] and Reaction[2] to Yellow Rust SYR	LEB	TUR	KYR	TAJ	AZE
MNCH/5/BLL/F72.23/4/TLLA//2*FR/KAD/ 3/2*GB CIT932082-0SE-0YC-1YE-0YC	GRP3 2	MS	R	S	R	R	R
LFN/VOGAF//LIRA/5/K134(60)/4/TOB/							

contd...

contd (Table 3)

BMAN//BB/3/CAL /6/F339P1.2 CIT935039-0SE-0YC-5YE-0YC	GRP3	6	MS	S	S	R	R	R
OK 84306//CN079/PRL/3/BRUL/TRAKIA CIT935016-0SE-0YC-2YE-0YC	GRP4	7	MR	S	S	MS	R	S
WA476/391/3/NUM/W22//ANA/4/TAM200/ 5/852HONG56/6/KS8ZW409/SPN CIT935324-0SE-0YC-3YE-0YC	GRP4	8	R	MR	S	S	R	MS
HATUSHA/TX81 V6614//TAM105RESEL CMWW90M202-0YC-0YC-0YC-2YC-0YC-1YC-0Y	GRP5	12	MR	R	MS	MR	MR	R
362K2.111/6/NKT/5/TOB/CN067//TOB/8156/ 3/CAL// BB/CN067/4/TRM CMSW90M476 -0YC -0YC-0YC-3YC-0YC-1YC-0YC	GRP5	13	MR	MS	R	R	R	R
ID800994.W/VEE//P10P10/3/CTY/CSM CIT935113-0SE-0YC-8YC-0YC	GRP5	14	MS	MS	MS	R	R	MS

[1]SYRr: Syria, LEB: Lebanon, TUR: Turkey, KYR: Kyrgyzstan, TAJ: Tajikistan, AZE: Azerbaijan.

[2]Field reaction to yellow rust at adult growth stage during 1999 and 2000 crop seasons

References

Ahmad S, Rodriguez A, Farid Sabir G, Roidar Khan B, Panah M, 1991. Economic losses of wheat crop Infested with yellow rust in highland Balochistan. MART/AZR *Project Research Report* **67**. ICARDA - Quetta.15pp.

Bayles RA, Priesly RH, 1983. Yellow rust of wheat. United Kingdom Cereal Pathogen Virulence Survey. *Annual Report* for 1982. pp: 27-36

Braun HJ, Saari EE, 1992. An assessment of the potential of *Puccinia striiformis* f.sp.*tritici* to cause yield losses in wheat on the Anatolian plateau of Turkey. Pages 121-123 In: *Proceedings of the 8th European and Mediterranean Cereal Rusts and Mildews Conference* (F.J. Zeller and G. Fischbeck Eds.), 8-11 September 1992. Weihenstephan, Germany.

El-Naimi M, Mamluk OF, 1995. Occurrence and virulence of wheat rusts in Syria. *Arab Journal of Plant Protection* **13**: 76-82.

Johnson R, 1978. Practical breeding for durable resistance to rust diseases in self-pollinating cereals. *Euphytica* **27**: 529-540.

Johnson R, 1981. Durable resistance: definition of, genetic control and attainment in plant breeding. *Phytopathology* **71**: 567-568.

Johnson R, 1988. Durable resistance to yellow (stripe) rust in wheat and its implications in plant breeding. In N.W. Simmonds and S.Rajaram, eds. *Breeding Strategies for Resistance to Rusts of Wheat,* p.63-75. CIMMYT, Mexico.

Johnson R, Law CN, 1975. Genetic control of durable resistance to yellow rust *(Puccinia striiformis)* in the wheat cultivar Hybride de Bersée. *Annals of Applied Biology* **81**, 385-391.

Ma H, Singh RP, 1996. Expression of adult resistance to stripe rust at different growth stages of wheat. *Plant Disease* **80**:375-379

Mamluk 0F, Haware MP, Makkouk KM, Hanounik SB, 1989. Occurrence, losses and control of important cereal and food legume diseases in West Asia and North Africa, pp: 131-140. In: Crop Losses Due to Disease Outbreaks in the Tropics and Counter Measures. *Proceeding of the 22nd International Symposium on Tropical Agriculture Research.* Kyoto, Japan, August 25-27, 1988.

Mamluk OF, AI-Ahmed M, Makki MA, 1990. Current status of wheat diseases in Syria. *Phytopatologia Mediterranea* **29**:143-150.

Mamluk OF, 1995. Our race with the yellow rust disease. *Agrotica* **4**: 18-19.

Martens JW, McKenzie RIH, Green GJ, 1970. Gene-for Gene relationships in the *Avena-Puccinia graminis* host-parasite system in Canada. *Canadian Journal of Botany* **48**: 969-975.

McIntosh RA, Hart GE, Devos KM, Gale MD, Rogers WJ, 1998. Catalogue of gene symbols for wheat. A.E.Slinkard (Ed.) *Proceedings of the 9th International Wheat Genetics Symposium,* 2-7 Aug. 1998, Saskatoon, Canada. Vol 5:1-235.

McIntosh RA, Wellings CR, park RP, 1995. Wheat Rusts. *An Atlas of Resistance Genes.* CSIRO and Kluwer Publishers, Dordrecht, The Netherlands. pp.149-177.

Schafer JF, Long Dl, 1988. Relations of races and virulences of *Puccinia recondita* f. sp. *tritici* to wheat cultivars and areas. *Plant Disease* **72**: 25-27

Torabi M, Mardoukhi V, Nazari K, Afshari F, Forouotan AR, Romai MA, Golzar H, Kashani AS, 1995. Effectiveness of wheat yellow rust resistance genes in different parts of Iran. *Cereal Rusts and Powdery Mildews Bulletin* **23:** 9-12.

Yahyaoui A, 2000. Emergence of new races of yellow rust of Wheat in West and Central Asia. *Gerplasm Program Cereal Annual Report for 2000* . ICARDA 2001.

Watson IA, 1970. Changes in virulence and population shifts in plant pathogens. *Annual Review of Phytopathology* VIII. (J. G. Horsfall and K. F. Baker Eds.) Annual Reviews. Palo Alto, pp. 209-230.

Identification of Yellow (Stripe) Rust Resistance Genes in a Group of International Wheat Nurseries

F. Afshari [1], R.A. McIntosh [2] and C.R. Wellings [2]

[1]Seed and Plant Improvement Institute, Karaj, Iran.
[2] Plant Breeding Institute, Australia

Introduction

Wheat cultivars derived from germplasm produced and distributed by the International Maize and Wheat Improvement Centre (CIMMYT) in Mexico are planted on more than fifty-one million hectares throughout the world (Dalrymple, 1986). Multilocational disease testing is used to obtain data to support breeding strategies aimed at broadening the genetic base of resistance in CIMMYT germplasm. The material selected in these tests should also serve as useful germplasm for local breeding programs.

The use of actual infection type data and pedigree information can contribute to the resistance gene postulation process. Dubin *et al.* (1989) used multipathotype tests to postulate genes for yellow rust resistance in selected CIMMYT lines. They detected genes *Yr3, Yr6, Yr7* and *Yr9,* either alone or in combination, in 12 wheats. From seedling tests of 26 wheat cultivars from Pakistan, a number of which were derived from CIMMYT germplasm, with 18 British *Puccinia striiformis* f. sp. *tritici* (Pst) pathotypes, Perwaiz and Johnson (1986) postulated the presence of genes *Yr6, Yr7, Yr9* and perhaps *Yr2* along with some unknown genes for resistance. Wellings (1992) reported a study of the 22nd International Bread Wheat Screening Nursery in which approximately 35% of entries carried *Yr27* (YrSk) alone, or in combination with other yellow rust resistance gene(s). Apart from *Yr2, Yr3* and *Yr4,* which are not effective in Australia, the distribution of seedling resistance genes in Australian wheats generally reflects that of CIMMYT materials.

In general, resistance genes expressed in seedling tests have not provided long-lasting, or durable, resistance to the cereal rusts. The objective of this study was to postulate genes for rust resistance in a group of nurseries distributed from CIMMYT and to interpret those postulations in regard to field assessments from PBI Cobbitty field nurseries in Australia.

Materials and Methods

The materials studied in detail were from the 30th IBWSN (International Bread Wheat Screening Nursery with 61 entries), 8th HRWSN (High Rain Fall Wheat Screening Nursery with 16 entries), 5th HRWYT (High Rain Fall Wheat Yield Trial with 10 entries), 5th HTWYT (High Temperature Wheat Yield Trial with 15 entries) and 5th SAWYT (Semi Arid Wheat Yield Trial with 21 entries).

Tests were performed on 10-15 seedlings of each line. The selected pathotypes were 106 E139 A-,Sk-;106 E139 A-,Sk+; 110 E143 A+; 111 E143 A- and 108E141 A-. The designations A+, A-, Sk+, Sk- indicate virulence (+) or avirulence (-) for genes *YrA* and *YrSk (Yr27)* that are not present in the standard differential set. Postulation of resistance genes was based on comparisons of infection types, and infection type arrays of the tested lines with selected controls and differential lines. Infection types 0 to 3 were interpreted as low or resistant whereas ITs 3+ or higher as susceptible (McIntosh *et al.* 1995). Adult plant tests were based on a 1 m row (30 seeds per row) of each line in the field. Adult plants were scored on the basis of leaf area affected (modified Cobb scale as illustrated in Peterson *et al.* 1948) and disease response. The pathotype used in the field was 110 E143 A+.

Results

Routine seedling tests at PBI Cobbitty based on a single avirulent pathotype of each rust pathogen (*Pst, P. recondita* f. sp. *tritici* and *P. graminis* f. sp. *tritici)* enable reliable identification of lines with the 1BL.1RS translocation containing genes *Yr9, Lr26* and *Sr31*. Lines with the translocation display combined infection type (IT) responses of 0; to ; with *Pst* pt. 110 E143 A+, IT $2^{=}$ to 2^{-} with Pgt pt. 34-1,2,3,4,5,6,7 or 98-1,2,3,5,6 and IT 0; with *Prt* pt. 104-1,2,3,(6),(7),11. Among the nurseries examined, 372 lines (75.15%) carried the translocation. As there is no virulence for *Yr9* in the current *P. striiformis* population in Australia the present studies concentrated on the remaining 123 (24.85%) lines from five nurseries. The results for these lines tested as seedlings are summarised in Table 1. Lines considered to have *Yr27* displayed necrotic seedling low infection types (LIT, 12CN to 23-CN) with frequent pustule blackening due to development of telial like structures, and susceptible responses (IT3+) with pt. 106 E139 A+,Sk+. *Yr7* was postulated on the basis of a ;N to 1N with pt. 108 E141 A- and a high infection type (HIT) 3+ with pt. 106 E139 A-. Lines with *Yr6* gave LIT (;N to ;1 N) with pt. 106 E139 A- and HIT 3+ with the other three pathotypes. *Yr1* was postulated in two entries on the basis of LIT 0; with pt. 110 E143 A+ and a HIT 3+

with pt. 111 E143 A+. The remaining lines were divided into two groups; firstly, those with LITs (ranging from 0; to 23C) for all pathotypes and, secondly, those susceptible with all five pathotypes. In each category there was a range of adult plant responses.

Among the 61 lines from the 30th IBWSN, 14 possessed *Yr27*, 9 carried *Yr7*, 1 carried *Yr6*, 19 had unknown seedling resistance gene(s) and 18 lines carried no seedling resistance (Table 1). The frequency of lines with *Yr27* (22.95% of tested entries) was higher than any other known gene except *Yr9*. All lines with *Yr27* were resistant in the field, indicating the likely effectiveness of this gene in conferring adult plant resistance in Australia. Of 16 lines from the 8th HRWSN, 3 possessed *Yr27*, 5 had *Yr7*, 2 had *Yr1*, and 6 lines had no seedling resistance (Table 1). Lines with *Yr1* were highly resistant in the field. Of 10 lines from the 5th HRWYT, 1 carried *Yr27*, 4 had *Yr6* alone, 1 had *Yr6+Yr7* and 4 possessed unknown gene(s). Some lines with *Yr6* were susceptible in the field, others with *Yr6* and the line with *Yr6+Yr7* were resistant indicating the presence of APR genes in these lines.

Among fifteen lines from the Stn HTWYT, 1 possessed *Yr27*, 2 had *Yr7*, 6 had *Yr6*, 2 carried *Yr6+Yr7* and 4 lines did not carry an effective seedling resistance gene. All lines, except one with *Yr6*, showed acceptable resistance in the field (Table 1). Among 21 lines of the 5th SAWYT, 4 possessed *Yr7*, 4 had *Yr6*, 3 had *Yr6+Yr7*, 5 carried an unknown gene(s) and 5 lacked seedling resistance. This nursery included a higher frequency of lines with Yr6, *Yr7* or *Yr6+Yr7*. Overall 107 of the 123 lines tested were resistant in the field. The 16 susceptible lines either had *Yr6*, *Yr7*, *Yr6+Yr7* or possessed no seedling resistance. Some entries clearly had no seedling resistance but carried APR. The 28 lines postulated to have unknown gene(s) for resistance to stripe rust (Table 1) appeared to give three main response arrays. Firstly, a group of lines that gave IT ; to ;1 with all pathotypes and second group gave IT ;1 to ;1+; and a large group that gave IT ;CN to 2+CN with all pathotypes. The response of this last group was similar to that conferred by *Yr27* but a low infection type was also given with pt. 106 E139 A-, Sk+ indicating that *Yr27* alone could not be responsible.

Table 1 Distribution of seedling resistance genes and adult plant responses to stripe rust among 123 non-1 BL.1 RS entries in five CIMMYT nurseries.

Nursery	Total	*Yr27*	*Yr7*	*Yr6*	*Yr6+Yr7*	*Yr1*	Unknown susceptible	Seedling Sus.	APR	Field
30th IBWSN	61	14	9	1	-	-	19	18	56	5
8th HRWSN	16	3	5	-	-	2	-	6	14	2
5th HRWYT	10	1	-	4	1	-	4	-	7	3
5th HTWYT	15	1	2	6	2	-	-	4	14	1
5th SAWYT	21	-	4	4	3	-	5	5	16	5
Total	123	19	20	15	6	2	28	33	107	16

Discussion

Before discussion of the present results two points must be made. Firstly, the postulation that a line possesses a recognised gene or genes does not constitute a claim that the full resistance genotype of the cultivar has been described. Secondly, the postulations that were made are preliminary and, where the information is important, would require further confirmation. It was postulated that 19 lines in the five nurseries have *Yr27*. Pathotypes avirulent to this gene gave LIT (2-CN to 23-CN) and only pt. 106 E139 A+, Sk+ gave a clear HIT (3+). The *Yr27* (Selkirk) resistance gene was first noted by Zadoks (1961) but it may be present in Webster, an entry in the original German differential set. Further experiments conducted at the Institute for Phytopathological Research (IPO), The Netherlands, by Wellings (1992) indicated that *Yr27* was present in a range of international germplasm. The entries with *Yr1* were selected from crosses involving U.K. wheats. Perwaiz and Johnson (1986) found that some CIMMYT lines possessed *Yr6* and *Yr7*. While the current project did not directly address the question of the extent of stripe rust resistance in wheat generated and distributed by CIMMYT and carrying *Yr9*, the finding of widespread resistance among non1BL.1RS germplasm was encouraging. It seems reasonable to argue that the distribution of resistance genes including those for APR should be similar in both 1BL.1RS and 1B genotypes.

The present work identified low numbers of lines that carried potentially new genes for seedling resistance to yellow rust. These lines could be examined more closely as potential sources of new variability for resistance breeding. Initial studies however should focus on the possibility that they are genes that are currently known and designated but occur only at very low frequencies in CIMMYT material. Details of which resistance genes were postulated for each cultivar are available in Afshari (2000).

References

Afshari F, 2000. *Studies on Rust Resistance in Wheat with Particular Emphasis on Stripe Rust.* Ph.D. Thesis, University of Sydney, Australia.

Dalrymple DG, 1986. Development and spread of high-yielding wheat varieties in developing countries. USAID. Washington, D.C. No. 86/71247.

Dubin HJ, Johnson R, Stubbs RW, 1989. Postulated genes for resistance to stripe rust in selected CIMMYT and related wheats. *Plant Disease* **73:** 472475.

McIntosh RA, Wellings CR, Park RF, 1995. *"Wheat Rusts: An Atlas of Resistance Genes".* CSIRO publications, Victoria, Australia, pp. 200.

Perwaiz. MS, Johnson R, 1986. Genes for resistance to yellow rust in seedlings of wheat cultivars from Pakistan tested with British isolates of *Puccinia striiformis.* Plant Breeding **97:** 289-296.

Peterson RF, Campbell AB, Hannah AE, 1948. A diagrammatic scale for estimating rust intensity of leaves and stems of cereals. *Canadian Journal of Research* **26:** 496-500.

Wellings CR, 1992. Resistance to stripe (yellow) rust in selected spring wheats. *Vortrage für Pflanzenzüchtung* **24:** 273-275.

Zadoks JC, 1961. Yellow rust on wheat. Studies in epidemiology and physiologic specialisation. *Tijdschrift over Planteziekten* **67:** 69-256.

Evaluation of International Wheat Nurseries for Stripe Rust (*Puccinia striiformis* f.sp. *tritici*) Resistance in Ankara - Turkey in the Period 1995-2000

F. Dusunceli [1], L. Çetin [1], S. Albustan [1], H.J.Braun [2], H. Ketata [3], Z. Mert [1], S.P.S.Beniwal [4]

[1] *The Central Research Institute for Field Crops (CRIFC), Ulus, Ankara, Turkey;*
[2] *CIMMYT - Turkey*
[3] *ICARDA - Iran*
[4] *ICARDA - Uzbekistan*

Introduction

Wheat is the major crop in agriculture of Turkey. Most of the total of 18-20 million tonnes annual wheat production comes from Central Anatolia. The wheat yield averages around 2000 tonnes/ha and it is heavily affected by abiotic and biotic stresses. In addition to drought and sunn pest, the rust diseases *(Puccinia spp.)* are among the major constraints to wheat production. Leaf rust *(Puccinia recondita)* is more prevalent in coastal areas and transitional zones, while stem rust *(P. graminis f.sp. tritici)* occurs in some years at higher altitude areas and where late maturing wheats are grown. In contrast, yellow rust (YR) *(P. striiformis* f.sp. *tritici)* can occur in almost all parts of the country if cool and wet conditions prevail in the spring. With favourable conditions, the Central Anatolian Plateau experiences serious epidemics and losses due to YR more frequently than other regions (Cetin *et al.* 1996). In the last decade a number of YR epidemics have occurred in Central Anatolia in 1991 (Braun *et al.* 1992), in Cukurova in 1995 (Dusunceli *et al.* 1996) and again in Central Anatolia in 1998 (Dusunceli *et al.* 1999).

In order to reduce the vulnerability of wheats of Central Anatolia to YR, various efforts are made. Among these, breeding for resistance has been the major activity, mainly at the agricultural research Institutes in Ankara, Eskisehir and Konya. The International Winter Wheat Improvement Programme (IWWIP) run by Turkey-CIMMYT-ICARDA has also generated valuable genetic materials for the region as well as for the winter wheat sown areas in other parts of the world. The objective of this work was to evaluate international germplasm originating from IWWIP and ICARDA in order to identify YR resistant genotypes for utilization in breeding programmes.

Materials and Methods

In total 83 nurseries consisting of 22 813 entries, most of which were generated for the winter sown wheat areas by IWWIP, were used in the study. These included entries from crossing blocks, preliminary yield trials, advanced yield trials, observation nurseries, international candidates, candidates from Turkey, introductions and some other specific nurseries, such as some Key Location and YR Germplasm Pool nurseries developed by ICARDA.

The nurseries were evaluated at the experimental research sites of the Central Research Institute for Field Crops (CRIFC) mostly in Haymana (1150 m), 50 km south west of Ankara and in Yenimahalle (850 m) in Ankara. Each entry was hand-sown in October in 1-2 m rows or as hill plots spaced at 35 cm. Michigan Amber was used as a susceptible check and as a spreader row after every 10 test entries. Six rows of susceptible varieties Sürak, Michigan Amber and Little Club were sown with a drill around the nursery as spreader plots.

For inoculation, uredospores were collected in the previous year from the nurseries and farmers' fields around Ankara. Inoculum was stored in glass tubes in liquid nitrogen and multiplied in the greenhouse. Inoculation was carried out by spraying in light-weight mineral oil (Soltrol 170) or in talc powder over the whole nursery and was performed towards the evening especially on cloudy and moist days and after application of mist irrigation. This was repeated 4-5 times. After the establishment of YR, the nurseries were also inoculated with a mixture of uredospores of leaf rust and stem rust.

The nurseries were irrigated with mist and flood irrigation to promote YR development. The YR on each entry was scored twice using the modified Cobb scale, starting when the susceptible check Michigan Amber reached 80S infection severity. For comparison, the Coefficient of Infection (CI) was calculated with the following equation:

CI=% severity x CRT (Coefficient of Reaction Type)

where the scale for CRT was S:1, MS:0.8, MSMR-MRMS:0.6, MR:0.4: R:0.2.

Then the entries were classified according to their CI values: Resistant:0-10, Moderately resistant: 10-30, Moderately susceptible: 30-50, Susceptible: 50-100. The entries in the resistant and moderately resistant categories were selected as candidates for the long term resistance sources nursery (LTRSN). They were screened in the following 1-2 years again and for the final selection they were also evaluated for their response to LR, SR and general stand in the field.

Results and Discussion

Good artificial epidemic development was established in all 6 seasons. The average infection severity (AIS) on susceptible Michigan Amber was over 80S in all the seasons. The uniform YR development allowed successful evaluation of the nurseries for resistance. The CI for YR on test entries varied between 0 and 100.

The nurseries and % of entries selected for YR resistance in each year are shown in table 1. The % selection was 24.9 % in the first year (1995) and this figure increased to 56.7%, 24%, 51, 45 and 56 % in the years 1996, 1997, 1998, 1999 and 2000 respectively. As an average 44.6 % of the 22 813 entries were identified as having high and moderate levels of resistance to YR.

The entries with good levels of resistance to YR were also evaluated for their leaf rust resistance, stem rust resistance and field stand. Finally, entries with different pedigrees were selected for inclusion in the Yellow Rust Resistance Sources Nursery (YRRSN). As a result of this 6-year study 482 genotypes were selected. The contribution (as number of genotypes and their share of the total) of different nurseries to the YRRSN is indicated in Table 2. The number of entries each nursery contributed to the YRRSN was 3, 5, 58, 51, 18, 3, 18, 45, 26, 5, 8, 31, 5, 84 and 122 for the nurseries of CanInt 94, YT 94, CanInt 95, WYR 95, CBWF 96, CIS 96, MI 96, YT 96, CanInt 97, EYT-RF 97, FAWWON 97, AYT 97, WCBYRGP 97, WKL 97 and YT 97, respectively. The study facilitated 1) selection of more resistant genotypes for further studies, 2) identification of resistance sources for yellow rust resistance for use in breeding programmes in future and 3) realization of importance of strict screening for resistance in developing YR resistant cultivars.

Table 1. The international nurseries evaluated for stripe rust resistance and % of entries showing high or moderate level of stripe rust resistance from 1995 to 2000 *[1]

	1995 [1]		1996 [1]		1997 [1]		1998 [1]		1999 [1]		2000 [1]	
Nursery Type	Nursery	S.%	Nursery	S.%	Nursery	S.%	Nursery	S.%	Nursery	S.%	Nursery	S.%
Elite Yield Trials- Rainfed			EYT-RF	45	EYT-Rf	28		EYT-RF		60	4.YET-RF	52
Elite Yield Trials- Irrigated			EYT-IR	33	EYT-Ir	28			3,EYT-IR	44	4.YET-IR	68
Facultative & Winter Wheat	FAWWON	8	FAWWON	21	6th FAWWON	5.5	FAWWON	50	8. FAWWON	27	9.FAWWON	49
Observation Nurseries											10.FAWWON	29
Various Observation nurseries							IWWON-RF	59	2WWON-IR	66	3.WON-SA	50
							IWWON-IR	78		55	3.WON-IRR	71
							SW ON	49		74		
Advanced Yield Trials	AYT95	19	AYT	35	AYT- Ir & rf	14.6	AYT-RF	56	AYT-RF	46		
							AYT-IR	60	AYT-IR	44		
Yield Trials	YT	52	YT	41	YT- Ir & rf	10.8	YT-RF	56	YT-RF	53	YT-RF	55
							YT-IR	63	YT-IR	66	YT-IR	63
Preliminary Yield Trials					PYT *	49.2	PYT-RF	53	PYT-RF	37	PYT-RF(ESK)	75
							PYT-IR	62	PYT-IR	51	PYT-IR (ESK)	86
Other Yield Trials									Grain Size YT		2.WW EERYT	34
									SW YT		CDW EERYT	31
Crossing Blocks			CB	38.4	CB WF	44	CB WF	62			CB WF	57
					CB S	41	CB S	40			CB S	88
International candidates	CANInt	28			CAN -nt	12	CAN. int	36	CAN.Int	19		
Introduced Materials			MI	19.8	MI	31	MI	39	MI	36	MI LARGE	30
			CIS	8.6	KAZUZVAR	27	LATE MI	23			MI SMALL	32
					Iran-landrace	17						
Disease Resistance N.			DRN	61.7	DRN	75	DRN	83	YRMEX	88		
Wheat Key Location N.			W KL	92	W KL	61	W KL	84				
			WYR 95	100	W CBYR GP	71						
Segregating Populations					F2	10.3					F3 (440)	
Other Nurseries							KRASS...	10				
							YR STUDY 1	52				
							YR STUDY 2	73				
Total entries (22,813 entries in 85 *nurseries*)	878	24.9	1506	56.7	4672	24.0	6019	51.0	4247	45.0	5481	56.0

[1]: S.%: Selection percentage; RF:Rainfed; IR:Irrigated; SW:Spring Wheat; ON:Observation Nursery; WF:Winter and Facultative; EERYT:East European Regional Yield Trial; CBYR:Common Bunt Yellow Rust; GP:Germplasm Pool

Table 2. Number of entries selected from nurseries for the Yellow Rust Resistance Sources Nursery (YRRSN) and their share in total

Nursery Contributing to YRRSN	Contribution to YRRSN Number of Genotypes	% in Total
CanInt 94 3	0.6	
YT 94	5	1.0
CanInt 95 58	12.0	
WYR 95	**51**	**10.6**
CBWF 96 18	3.7	
CIS 96	3	0.6
MI 96	18	3.7
YT 96	45	9.3
CanInt 97 26	5.4	
EYT-RF 97	5	1.0
FAWWON 97	8	1.7
AYT 97	31	6.4
WCBYRGP 97	**5**	**1.0**
WKL 97	84	17.4
YT 97	122	25.3
TOTAL	**482**	**100.0**

References

Braun H-J, Saari EE, 1992. An assessment of the potential of *Puccinia striiformis* f.sp. *tritici* to cause yield losses in wheat on the Anatolian Plateau of Turkey. *Vortrage für Pflanzenzüchtung,* **24,** 121-123.

Cetin L, Dusunceli F, Albustan S, 1996. Evaluation of the Turkish winter and facultative wheat germplasm for rust resistance in Central Anatolia. *Cereal rusts and Powdery Mildews Bulletin,* **24,** page 240, Supplement, *Proceedings of the 9th Cereal Rusts and Powdery Mildews Conference,* 2-6 September 1996, Lunteren, The Netherlands.

Dusunceli F, Cetin L, Albustan S, Beniwal SPS, 1996. Occurrence and impact of wheat stripe rust *(Puccinia striiformis)* in Turkey in the 1994/95 crop season. *Cereal Rusts and Powdery Mildews Bulletin,* **24**, page 309 Supplement. *Proceedings of the 9th Cereal Rusts and Powdery Mildews Conference,* 2-6 September 1996, Lunteren, The Netherlands.

Dusunceli F, Cetin L, Albustan S, 1999. Occurrence and importance of rust diseases *(Puccinia* spp.) in wheat fields of Central Anatolia and precautions against them. In proceedings of the conference on *'Constraints to Cereal Production in Central Anatolia'*, 8-11 June, 1999, Konya-Turkey, pages 693-696 (In Turkish).

New Sources of Resistance to Yellow Rust in Bread Wheat and Winter Durum Wheat in Uzbekistan

N. Umirov, A. Haitboev and A. Amanov

Galla-Aral Branch of the Andijan Research Institute of Grain, Uzbekistan

Introduction

There are two centres of infection for yellow rust in Uzbekistan, Trans-Caucasus and Asia. The first includes central and foot-hill zones of Azerbaijan, Armenia, and Eastern Georgia. The second includes Bishkek, Issik-kul regions of Kyrgyzstan and foot-hills of Southern Kazakhstan, Tajikistan and Uzbekistan. The fungus migrates across vertical zoning of the climate and can survive on crops of winter wheat and on wild cereals, especially *Aegilops.* In Uzbekistan the disease development starts at the end of March through to the beginning of April. The weather conditions were favourable for a yellow rust epiphytotic in 1999, when varieties Una, Unumli-Bugdoy and Sanzar-8 were infected from 40 to 80% and yields were decreased by 20 to 40%. It was therefore considered necessary to study variation in the pathogen, *Puccinia striiformis* f. sp. *tritici,* and assess the resistance of available varieties.

Materials and Methods.

Pathogen collection

Spore samples were collected from infected wheat crops in Djizakh and Chimkent and also in Kyrgyzstan and Tajikistan.

Tests of varieties

During the last three years 4500 entries from ICARDA, CIMMYT, and the Vavilov Institute, Russia (VIR) were evaluated under natural and artificial infection. Two types of trials were used:

1) Natural infection on fertile soils
2) Artificial infection on irrigated trials, with three irrigations during the growing period.

Inoculations were carried out in the evening and the plots were covered with polythene overnight to maintain high humidity and encourage infection. Assessment of infection was carried out every 14 days after inoculation. Scoring the trials was by Infection type on a 0 to 5 scale, as used at the Vavilov Institute, and percentage leaf area infected . The analysis of data was performed according to the methods of the State Commission Variety Trials.

Results and Discussion

The data identified 66 immune sources (score 0), 118 resistant (score less than 10% infection) and 287 moderately susceptible (score less than 20% infection) to yellow rust (Table 1). These were not uniformly spread between the countries of origin. There was a high proportion of resistant accessions from North and South America and also among accessions from South Eastern Europe, particularly from Bulgaria and Yugoslavia, probably because of intensive selection for resistance to the pathogen in these countries. Also resistant lines were found among wheat accessions from Ethiopia and Kenya. (Voronkova 1974, Grigoreiva 1975, Berliand-Kodjevnikov *et al.* 1975).

Table 1. Disease reaction of bread wheat accessions to yellow rust in Galla-Aral, 1987-2000.

Country of origin	Accessions	Disease reaction %			
		0	<10%	<20%	>20%
CIS (USSR)	139	4	8	5	122
West and central Europe (Great Britain, France, San-Marino, Germany, Poland)	60	3	19	7	31
Slovakia, Hungary, Romania	80	2	-	25	53
Balkan (Bulgaria, Yugoslavia)	48	6	4	14	24
South Western Asia Pakistan, India	10		-	-	10
Eastern Mediterranean Sea Iraq	7		-	1	6
Eastern Asia (China, Japan)	9	-	-		9
North and South America (USA, Argentina, Mexico)	128	15	22	16	75
Ethiopia, Kenya	232	6	5	19	202
CIMMYT and ICARDA	3787	30	60	200	3497
Total	4500	66	118	287	4029

The wheat lines with the highest rust resistance detected in the trials are listed in Table 2 with their Vavilov Institute accession numbers. These wheat lines were also resistant to lodging.

Table 2. Accessions with the highest resistance to rust diseases in bread and durum wheat from trials in Galla-Aral, 1988-2000.

Catalog number of VIR	Country of origin	Rust reaction (0-5)/ % leaf area infected		
		Yellow	Leaf	Stem
		Bread wheat		
K-57669	Odessa	0/0	0/0	2/5
K-57479	Mironovka	0/0	2/5	0/0
K-56380	Slovakia	0/0	3/5	0/0
K-57661	Nemchinovka	0/0	0/0	2/10
K-57250	Ukraine	3/5	0/0	0/0
K-55366	France	0/0	0/0	0/0
K-56222	Bulgaria	0/0	0/0	0/0
K-489553	USA	0/0	0/0	0/0
K-489396	USA	0/0	0/0	0/0
K-489416	USA	0/0	0/0	0/0
K-429696	Ethiopia	0/0	0/0	0/0
K-429289	Ethiopia	3/10	0/0	0/0
K-429285	Ethiopia	0/0	0/0	0/0
K-429554	Ethiopia	0/0	0/0	0/0
		Durum wheat		
K-54460	Moldavia	0/0	0/0	0/0
K-56625	Moldavia	0/0	0/0	0/0
K-43903	Ukraine	0/0	0/0	0/0
K-45337	Ukraine	0/0	0/0	0/0
K-48102	Krasnodar	0/0	0/0	0/0
K-54382	Krasnodar	0/0	0/0	0/0
K-54450	Rumania	0/0	0/0	0/0
K-54460	Rumania	0/0	0/0	0/0
K-54470	Rumania	0/0	0/0	0/0
K-56667	Bulgaria	0/0	0/0	0/0
K-56671	Bulgaria	0/0	0/0	0/0

From many years of observation, the most effective genes in Galla-Aral for resistance to yellow rust are: *Yr3c, Yr5* and *Yr4b,* for leaf rust: *Lr 9, Lrl9, Lr23, Lr24,* and *LrZ.* The last one is a new gene developed by crossing a local variety Sanzar-85 with entry K-17146 (Syria). The gene was officially registered by the laboratory of immunity of VIR.

Many researches (Anpilogova 1982, Voronkova 1974) indicated that it is easier to select for resistance with genes that give resistance at all stages of plant growth than for those that give resistance at one or other stage of growth. Often, resistance in adult plants was found to be controlled by genes different from those effective throughout the growth of the plants (Voronkova 1974, Mustafaev and Ibragimva 1977). Many varieties that were susceptible at the seedling stage showed high resistance to the rust as adult plants in different regions of study (Voronkova 1977).

Conclusions

In Central Asia yellow rust is more harmful than other diseases on irrigated and semi-arid areas. Effective against the local yellow rust population, resistance genes *Yr3c, Yr5* and *Yr4b* have been identified. Their introduction by back-crossing permitted the production of resistant lines that possess also the many useful traits of the recurrent variety and they are being used in the breeding program of the Galla-Aral branch of the Andijan Research Institute of Grain. This has resulted in creation of new bread wheat varieties Yanbash and Maijon, highly resistant to yellow rust and other diseases. Also a durum wheat variety, Melionopus 170, has been sent to the state testing committee.

Editorial Comment (R. Johnson). The genes selected in this paper as providing high levels of resistance (*Yr3c, Yr5* and *Yr4b,* and *Lr 9, Lrl9, Lr23* and *Lr24)* are all known, from the literature, to be race-specific. They are most unlikely to provide long term resistance to either yellow rust or leaf rust if used in commercial wheat varieties. Careful watch should be kept on any new varieties possessing these genes, to look for evidence of virulence in the pathogen populations.

References

Anpilogova LK, 1982. Genetic basis of resistance to yellow rust *(Puccinia striformis* West.) for testing wheat varieties of standard selections. *Genetica* **12:** 477-484.

Berliand-Kodjevnikov VM, Mikhilova LA, Boradenenko NK, 1975. Genetics of resistance to brown (leaf) rust in the VIR collection of wheat with races 77, 151, and 202 of the pathogen. *Mycology and Pathology.* **9**: 109-113.

Voronkova AA, 1974. Parental lines for breeding stable resistance to brown and black rust diseases. In: *Genetics and Selection of Crop Varieties against Diseases.* p.41-65.

Voronkova AA, 1977. Use of back-crossing methods for analysis of resistance genes and use of different methods of selection for immunity. *Genetic Basis of Resistance against Diseases.* p.49-166.

Grigoreiva OJ, 1975. Wheat resistance against brown (leaf) rust in relation to ecological and geographical provenances. In: *All-Russian Panel Meeting for Immunity of Crop Varieties against Diseases and Pests.* p.174-175.

Mustafaev ED, Ibragimva AE, 1977. Investigation of new resources of resistant wheat against fungi in the Azerbaijan collection of botanic and genetic crops. *Vavilov Institute Russia* **58, 3**: 37-40.

Screening for Yellow Rust Resistance in Bread and Durum Wheat

M. El-Naimi[1], A. Yahyaoui[1], H. Ketata[1], O. Abdalah[2], M. Nachit[2] and M. S. Hakim[3]

[1]ICARDA, [2]CIMMYT. Aleppo, Syria
[3] Aleppo University, Syria

Introduction

Wheat is the most important food crop in Central and West Asia and North Africa (CWANA) countries and occupies a third of the arable area in the region. However, the average productivity of wheat in CWANA countries is still low due to biotic and abiotic stresses with an average yield around 1500 Kg/ha, which is less than two-thirds of the world average.

Yellow (stripe) rust of wheat caused by *puccinia striiformis* west. f.sp. *tritici* is the most widespread wheat disease and is of major importance in the cool and humid areas in the high lands of Turkey, Iran, Azerbaijan, Uzbekistan, Tajikistan, Pakistan and Kyrgzystan, where epidemics are frequent and are a continuous threat to wheat production (Yahyaoui, in this publication). Yellow rust continues to spread in CWANA due to changes in cropping systems where monoculture of limited numbers of varieties prevails and also to changes in climate. Cultural practices, such as the introduction of irrigation in rainfed areas contributed to the increase of production but also to the development and spread of yellow rust. In Syria yellow rust occurrence has become more frequent and its multiplication on cultivars such as Seri 82, Mexipak was extended due to longer green vegetation periods of the susceptible cultivars when grown under irrigation. This promotes moisture, which favors the spread of the disease the early spring, when air temperatures are relatively cool (Mamluk and El-Naimi 1992). During the last few years yellow rust epidemics occurred in most countries in the region (Mamluk *et al.* 1996), and resulted in severe yield losses that varied from 10-40% (Yahyaoui 2000).

Several control methods have been practiced in many countries in CWANA. Chemical foliar sprays have given good results but their use remains limited due to the extra cost incurred by farmers and their effect on the environment. Cultural practices that contribute to the development of the rust disease could be modified. Appropriate use of fertilizers, irrigation, planting date, and choice of varieties

could contribute to the control of the rust diseases. The applicability of these control measures could not be generalized; it is essential to use control measures that are applicable, economical and durable, and undoubtedly, host resistance will remain the major control method for rust diseases (Roelfs *et al.* 1992), particularly in CWANA, where the farmers could not afford chemical control and do not have much option to vary cultural practices in their agro-ecological regions. The objective of this study was to develop appropriate screening techniques to allow the identification of resistant genotype in breeding nurseries

Materials and Methods

Screening for host resistance is a major activity undertaken by ICARDA and CIMMYT/ICARDA wheat breeding programs. All the breeding nurseries are screened under artificial inoculation of yellow rust under field conditions. Breeders further select resistant material and then send them to national research programs in different agro-ecological regions of CWANA.

At Tel Hadya, the main ICARDA research station, wheat breeding nurseries are tested for resistance to yellow rust. Segregating populations, new introductions, and yield trial nurseries are evaluated in the breeding plots using spreader rows for disease spread. Selected nurseries are planted in hill plots in separate fields and all the entries are artificially inoculated by dusting yellow rust spores onto leaves at the early tillering growth stage. Inoculation may be repeated two to three times depending on climatic conditions. The material tested includes:

Crossing block nurseries
Disease monitoring nurseries
Advanced lines
Introductions or selected material from international nurseries
Wild relatives
Commercial cultivars and new releases

Rust inoculum, collected the previous year, is multiplied on seedlings of a mixture of susceptible cultivars, for use in field inoculations. Spreader rows, that encompass a mixture of susceptible cultivars, are planted at regular intervals, and as entries in hill plots. The use of a mixture of cultivars for spreader rows is essential to maintain the virulence spectrum of the rust pathogen in the population. Susceptible cultivars from different sources are added every year to the mixture for the spreader. Inoculum is collected from spreader rows and re-used to re-inoculate hill plots during the same season, and to store for use during the next season. Inoculum is vacuum dried and stored in small ampoules in the refrigerator. Bulks

of rust spores are analyzed in the laboratory and on the differential sets to ensure the presence of predominant virulence types in the rust population used for field artificial inoculation. The inoculum is dusted on the plants at a rate of 5gr of rust spores/hectare (Stubbs *et al.* 1986). The spores are mixed with talc powder. The ratio spores/talc powder is adjusted according to the area to be sprayed and the availability of inoculum.

The first artificial inoculation is often carried out in late afternoon, at least five hours after mist irrigation, or after rain showers, during periods where night temperatures are relatively low. Infection usually appears 10-14 days after inoculation. Mist irrigation is used once or twice a day for a period of one hour depending on the temperatures. Irrigation is often applied between 12:00 and 13:00 hrs. Artificial inoculation may be repeated if symptoms on the susceptible checks and spreader rows remain low.

During the 1999-2000 growing season, over 14 000 wheat accessions were screened. The accessions were distributed as follows:

Durum wheat fixed lines 1122
Durum wheat segregating populations 4000
Bread wheat fixed lines 1320
Bread wheat segregating populations 8000

Field evaluation for resistance to yellow rust was carried out at dough stage using the scale described by Peterson *et al.* (1948).

Results and Discussion

The screening techniques used at ICARDA permitted screening of large numbers of wheat populations with discrimination for different levels of resistance in the material. During this season it was found that 74% of durum wheat lines and 70% of bread wheat were resistant to moderately resistant (R-MR) while 26% of durum and 30% of bread wheat accessions were moderately susceptible to susceptible (MS-S) as shown in figure1.

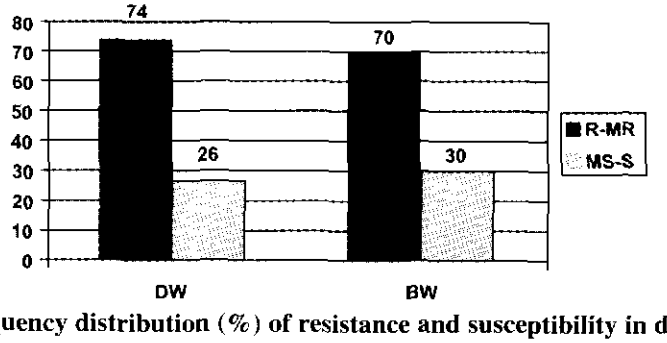


Figure 1. Frequency distribution (%) of resistance and susceptibility in durum (DW) and bread wheat (BW) germplasm.

Breeding lines that showed moderately resistant reactions were recommended to be retained for further screening and selection for agronomic traits. These lines could have durable resistance or more than one resistance gene. Lines that showed high resistance levels could have dominant resistance genes and are not recommended for further screening but may be used in crossing programs to combine with other resistance factors. Among the breeding lines, those that maintained moderate resistance for thee consecutive three years are recommended as potentially useful germplasm and could be exploited by national programs in CWANA. Table 1 shows the number of lines distributed to collaborators in NARS as sources of resistance to yellow rust.

Table 1. Germplasm pools for sources of resistance to yellow rust (1988-2000).

Crop	Pools	Lines	Sets Distributed
Durum Wheat	5	77	240
Bread Wheat	6	98	365
Total	11	175	605

Screening for resistance to yellow rust is conducted annually at ICARDA for all wheat accessions. Resistance sources are added every year to the germplasm pools and dispatched to national research programs in CWANA.

References

Mamluk OF, El-Naimi M, 1992. Occurrence and virulence of wheat yellow rust in Syria, pages 115-117. In: *Proceedings of the 8th European and Mediterranean Cereal Rusts and Mildews Conference* (Eds. F.J.Zeller and G.Fischbeck), 8-11 September 1992, Weihenstephan, Germany.

Mamluk OF, El-Naimi M, Hakim M, 1996. Host-Preference in *Puccinia striiformis* f.sp.*tritici* pages 86-88 In: *Proceedings of the 9th European and Mediterranean Cereal Rusts and Powdery Mildews* Conference (Eds. G.H.J. Kema, R.E. Niks & R.A. Daamen) 2-6 September 1996. Lunteren, The Netherlands.

Peterson RF, Campbell AB, Hannah E, 1948. A diagrammatic scale for estimating rust intensity of leaves and stem of cereals. *Canadian Journal of Research Section C* **26:** 469-500.

Roelfs AP, Singh RP, Saari EE, 1992: *Rust Disease of Wheat: Concepts and Methods of Disease Management,* Mexico, D.F. CIMMYT, 81 pages

Stubbs RW, Prescott JM, Saari EE, Dubin HJ, 1986: *Cereal Disease Methodology Manual.* Centro international de Mejoramiento de Maize y Trigo (CIM-MYT), Mexico 1986.

Yahyaoui A, Hakim S, El-Naimi M, Nachit M, 2000. Multiple disease resistance in durum wheat, *Option Mediterranean's.* Durum wheat improvement in the Mediterranean region: New Challenges. Serie A: Seminaries Mediterranean's Numéro 40: 387-392.

Seedling and Adult Plant Resistance to Yellow Rust in Genotypes of the Preliminary Wheat Screening Nursery (PWSN) of Iran in the 1999-2000 Cropping Season

A. Malihipour[1], M. Torabi[1], R. Houshyar[2], A. Tarinejad[3] and M. Ahmadian-Moghaddam[4]

[1]Seed and Plant Improvement Institute, Karaj, Iran;
[2]Miandoab Agricultural Research Station, Miandoab, Iran;
[3]Moghan Agricultural Research Center, Pars Abad, Iran;
[4]Mazandaran Agricultural Research Center, Sari, Iran.

Introduction

Yellow rust caused by *Puccinia striiformis* f. sp. *tritici*, is one of the most important diseases of wheat in the world. In Iran, the disease is most destructive in various parts of the country. It has been present for many years but due to favorable climatic conditions combined with cultivation of some susceptible cultivars for its development, the disease reached epidemic levels during the early 1990s with the most severe epidemics occurring in 1993 and 1995. Yield losses of 30% were reported for these years (Torabi *et al.* 1995).

The use of resistant cultivars is the most effective, economic, and environmentally safe method to control the disease. It is possible to recognize two types of specific resistance to yellow rust (Zadoks, 1961). The first type is evident at the seedling stage and lasts for the life of the host plant. This type of resistance is race-specific. The second type of resistance to yellow rust is apparent only during the adult plant stage, and its genetic control is currently less well understood. This type of resistance can also be race-specific (Johnson 1981, McIntosh *et al.* 1995).

Seedling resistance to *P. striiformis* in wheat can be detected in greenhouse tests, whereas adult plant resistance, which can be detected in glasshouse, is easier to observe in field tests (Wellings 1998).

The objective of this study was to determine the seedling and adult-plant resistance of 415 genotypes of Preliminary Wheat Screening Nursery (PWSN) of Iran in 1999-2000 cropping year in greenhouse and field conditions.

Materials and Methods

The host

415 bread wheat genotypes of the Preliminary Wheat Screening Nursery (PWSN) of Iran in 1999-2000 cropping year were tested at the seedling stage under controlled conditions and at the adult plant stage in field. A susceptible check cultivar (Bolani) was used in both tests.

The pathogen

The seedling tests were done with three pathotypes of *Puccinia striiformis* f.sp. *tritici:* 230E62A+ (from Miandoad area), 134E134A+ (from Moghan), and pathotype 38E2A+ (from Gharakhil). Adult plant tests were carried out at three different stations (Miandoab, Moghan, and Gharakhil), where the mentioned pathotypes of the pathogen were prevailing. In addition, artificial inoculation of the nurseries was done at the tillering stage using the relevant pathotype of each location.

Seedling tests

The seeds of 415 accessions were planted in 5cm diameter pots (4-6 seeds per pot) in the greenhouse. The seedlings were inoculated with fresh harvested urediniospores of *Puccinia striiformis* f.sp. *tritici* 9 days after sowing, when the first seedling leaves were fully expanded. Inoculated plants were placed inside a small plastic covered cage and kept at 10 °C and 90% RH in the darkness for 48 hrs and then transferred into a greenhouse at 15 °C. The reaction (infection type) of each line or cultivar was evaluated using McNeal *et al.*, (1971) 0-9 scale 15-17 days after inoculation.

Adult plant tests

Seeds of each entry were sown in December 1999. The seeds were planted in 1-meter long rows in two replications, spaced 30cm from each other. The susceptible check (Bolani) was planted every ten entries and also as borders of the nurseries. During stem elongation, an atomizer sprayer was used for inoculation of each nursery with a mixture of urediniospores and talcum powder. The reaction (infection type) of each line or cultivar was evaluated at the end of the cropping season. The percentage leaf area affected was scored using Cobb's modified scale at the same time. The two scores were then converted to a coefficient of infection (CI). This CI was obtained by multiplying the constant value for infection types (0=0; R=0.2; MR=0.4; I=0.6; MS=0.8; and S=1) and the leaf area affected.

Identification of resistance types

In seedling (greenhouse) tests, entries with an infection type (IT) of 0 to 3 were considered to be resistant or nearly so (R). Values of 4 to 6 were classified as intermediate infection types (I), and 7 to 9 to as susceptible infection types (S). At adult plant (field) tests entries with a coefficient of infection (CI) of 0 to 2 were

considered to be resistant (R). Values of 3 to 4 were classified as moderately resistant (MR), values of 5 to 12 as moderately susceptible (MS), and values of more than 12 were classed as susceptible reactions (S).

Results and Discussion

The results of seedling tests (Table 1) showed that most of the genotypes were susceptible to individual or combinations of the pathotypes of *Puccinia striiformis* f.sp. *tritici*. Some of the experimental materials were intermediate, and others were resistant. A large numbers of genotypes (71.3% of the entries, 296 accessions) were susceptible to pathotype 230E62A+. This pathotype can therefore be used as an effective tool for selection of wheat materials at the seedling stage. Frequencies of infection types 7-9 (susceptible) of genotypes to pathotypes 134E134A+ and 38E2A+ were 68.2% (283 accessions) and 48% (199 accessions) respectively. Many genotypes (35.9%, 149 accessions) consistently displayed susceptible reactions regardless of the pathotype used. This suggests that any one of the pathotyes used can overcome any seedling resistance genes present in these lines. Frequency of infection types 0-3 (resistant) were 21.9% (91 accessions), 11.8% (49 accessions), and 14.5% (60 accessions) of the entries to pathotypes 38E2A+, 134E134A+, and 230E62A+ respectively. This indicates the presence of seedling resistance genes effective against the mentioned pathotypes. Some of the entries (8.7%, 36 accessions) gave a resistance reaction when using pathotypes 230E62A+ and 134E134A+. Also 8.4% of the entries (35 accessions) gave the same reaction to pathotypes 230E62A+ and 38E2A+. Another materials (7.7%, 32 accessions) gave a resistance reaction when using pathotypes 134E134A+ and 38E2A+. A few genotypes (28 accessions) were resistant to all three pathotypes. A high level of seedling resistance for these lines indicates that one or more seedling resistance genes are involved in conferring resistance.

Table 1. Frequency of seedling infection types of genotypes to three individual pathotypes of *Puccinia striiformis* f.sp. *tritici* or their combinations in greenhouse conditions

Infection Types (IT)	230E62A+	134E134A+	38E2A+	230E62A+ & 134E134A+	230E62A+ & 38E2A+	134E134A+ & 32E2A+	230E62A+, 134E134A+, &32E2A+
0-3 (R)	60 (14.5%)	49 (11.8%)	91 (21.9%)	36 (8.7%)	35 (8.4%)	32 (7.7%)	28 (6.7%)
4-6 (I)	57 (13.7%)	80 (19.3%)	120 (28.9%)	32 (7.7%)	31 (7.5%)	31 (7.5%)	15 (3.6%)
7-9 (S)	296 (71.3%)	283 (68.2%)	199 (48%)	243 (58.6%)	172 (41.4%)	165 (39.8%)	149 (35.9%)

In field conditions, a severe epidemic of the disease was established on the susceptible check (Bolani) and other susceptible materials at each nursery. The results of adult plant tests (Table 2) showed that most of the genotypes were resistant to yellow rust in one or more locations. Only a few genotypes were moderately resistant, moderately susceptible, or susceptible. A large numbers of genotypes (65.8% of the entries, 273 accessions) were resistant to the disease at all three locations.

Table 2. Frequency of coefficient of infections of genotypes to yellow rust in three individual locations or their combinations at adult plant stage

Coefficient of Infection (CI)	*Miandoab*	*Moghan*	*Gharakhil*	Miandoab & Moghan	Miandoab & Gharakhil	Moghan & Gharakhil	Miandoab, Moghan, & Gharakhil
0-2 (R)	323 (77.8%)	334 (80.7%)	402 (96.9%)	276 (66.5%)	315 (75.9%)	330 (79.5%)	273 (65.8%)
3-4 (MR)	3 (0.7%)	31 (7.5%)	5 (1.2%)	0 (0.0%)	0 (0.0%)	2 (0.5%)	0 (0.0%)
5-12 (MS)	19 (4.6%)	19 (4.6%)	4 (1%)	1 (0.2%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
>12 (S)	70 (16.9%)	30 (7.2%)	4 (1%)	19 (4.6%)	4 (1%)	4 (1%)	4 (1%)

Comparison of the data of seedling and adult plant studies (Table 3) indicated that about 13% of the entries (55 accessions) with infection types of 0-3 (resistant) as seedlings were resistant or moderately resistant at the adult plant stage to yellow rust isolate of Miandoab (pathotype 230E62A+). There were about 12% (49 accessions) and 22% (90 accessions) for Moghan (pathotype 134E134A+) and Gharakhil (pathotype 38E2A+) isolates respectively. Some of the entries having infection types of 4-6 (intermediate) at seedling stage were resistant or moderately resistant at the adult plant stage. This includes 11.8% of the genotypes (49 accessions) for Miandoab, 16.7% (71 accessions) for Moghan, and 28.4% (118 accessions) for Gharakhil isolates. Moreover a large number of genotypes having infection types of 7-9 (susceptible) at the seedling stage were resistant or moderately resistant at the adult plant stage. This includes about 52% of the entries (215 accessions) for Miandoab, 59% (243 accessions) for Moghan, and 47% for Gharakhil isolates.

Table 3. Frequency of seedling infection types and coefficients of infection of genotypes to three pathotypes of yellow rust at seedling stage and in three locations at adult plant stage.

Infection Types (IT) and *Coefficient of Infection (CI)*	230E62A+ *(Miandoab)*	134E134A+ (Moghan)	38E2A+ (Gharakhil)
IT=0-3 (R), CI=0-2 (R)	54 (13%)	48 (11.6%)	89 (21.4%)
IT=0-3 (R) & CI=3-4 (MR)	1 (0.2%)	1 (0.2%)	1 (0.2%)
IT=4-6 (I) & CI=0-2 (R)	49 (11.8%)	68 (16.4%)	117 (28.2%)
IT=4-6 (I) & CI=3-4 (MR)	0 (0.0%)	3 (0.7%)	1 (0.2%)
IT=7-9 (S) & CI=0-2 (R)	214 (51.6%)	215 (51.8%)	190 (45.8%)
IT=7-9 (S) & CI=3-4 (MR)	1 (0.2%)	28 (6.7%)	3 (7.2%)

Finally a few genotypes (27 accessions) were resistant to all three pathotypes at the seedling stage, and were also resistant at adult plant stage at all three locations (Table 4).

Table 4. Genotypes resistant to all of three pathotypes of yellow rust at the seedling stage that were also resistant at adult plant stage at all three locations.

Entry No.	Plot No. 2000	Cross No.	Parentage
38	4046	1-13878	Flt/Attila
95	4115	1-13958	Bow"S"/Nkt"S"//Bez
184	4222	1-14099	M-70- /Ures/3/Gov/Az//Mus/4/Sara
185	4223	1-14099	M-70-/Ures/3/Gov/Az//Mus/4/Sara
186	4224	1-14100	M-70-4/Tui"S'
233	4281	1-14187	Alvd/MV 17
234	4282	1-14187	Alvd/MV 17
269	4323	ICW94-0038	Vee#7/kauz"S"
271	4327	ICW94-0038	Vee#7/kauz"S"
289	4347	ICW94-0150	Vee"S"/Nac//Shi#4414/Crow"S"
290	4348	ICW94-0150	Vee"S"/Nac//Shi#4414/Crow"S"
295	4355	ICW94-0183	Laj 2965//Shi#4414/Crow"S"
306	4368	ICW94-0234	Shi#4414/Crow"S"//Pgo/Seri
307	4369	ICW94-0234	Shi#4414/Crow"S"//Pgo/Seri
308	4370	ICW94-0234	Shi#4414/Crow"S"//Pgo/Seri
311	4375	ICW94-0240	PRL"S"/PEW"S"//Shi#4414/Crow"S"
312	4376	ICW94-0240	PRL"S"/PEW"S"//Shi#4414/Crow"S"
313	4377	ICW94-0240	PRL"S"/PEW"S"//Shi#4414/Crow"S"
314	4378	ICW94-0240	PRL"S"/PEW"S"//Shi#4414/Crow"S"
316	4380	ICW94-0246	VEE"S"/LIRA"S"//Shi#4414/Crow"S"
317	4381	ICW94-0246	VEE"S"/LIRA"S"//Shi#4414/Crow"S"
319	4383	ICW94-0247	VEE"S"/LIRA"S"//Shi#4414/Crow"S"
320	4384	ICW94-0255	TOW"S"/PEW"S"//Shi#4414/Crow"S"
321	4387	ICW94-0255	TOW"S"/PEW"S"//Shi#4414/Crow"S"
322	4388	ICW94-0255	TOW"S"/PEW"S"//Shi#4414/Crow"S"
324	4390	ICW94-0255	TOW"S"/PEW"S"//Shi#4414/Crow"S"
336	4404	ICW94-0322	Bow"S"/CROW"S"//GRU90-204781

References

Johnson R, 1981. Durable disease resistance. Pages 55-63 in J. F. Jenkyn and R. T. Plumb, eds. *Strategies for Control of Cereal Diseases*, Blackwell, Oxford.

Johnson R, 1993. Durability of Resistance in Crops: Some closing remarks about the topic of the symposium. Pages 283-300 in: T. Jacobs and J.E. Parlevliet, eds. *Durability of Disease Resistance.* Kluwer Academic Publisher, The Netherlands.

McNeal FH, Konzak CF, Smith EP, Tate WS, Russell TS, 1971. A uniform system for recording and processing cereal research data. *United States Department of Agricultural Research Service,* ARS 34-121. 42pp.

Roelfs A P, Singh RP, Saari EE, 1992. *Rust Diseases of Wheat: Concepts and Methods of Disease Management.* Mexico, D. F.: CIMMYT. 81pp.

Torabi M, Mardoukhi V, Nazari K, Afshari F, Forootan AR, Ramai MA, Golzar H, Kashani AS, 1995. Effectiveness of wheat yellow rust resistance gees in different parts of Iran. *Cereal Rusts and Powdery Mildews.* Bulletin **23:** 9-12.

Parlevliet JE, 1978. Further evidence of polygenic inheritance of partial resistance in barley to leaf rust. *Euphytica.* **27:** 369-379.

Mamluk OF, van Slageren MV, 1994. Sources of resistance to wheat diseases in *Aegilops and Amblyopyrum* spp. *Proceedings of 9th Congress of the Mediterranean Phytopathology,* Union, Kusadasi - Aydin - Turkiye, 1994. Pages 269-270.

Zadoks JC, 1961. Yellow rust on wheat: studies in epidemiology and physiologic specialisation. *Tijdschrift over Planteziekten* **67:** 69-256

Seedling and Adult Plant Reactions to Different Pathotypes of *Puccinia Striiformis* Westend. in Newly Released Wheat Varieties for Rainfed Areas of Iran

M. Hassanpour Hosni[1], M. Torabi[2] and V. Mardoukhi[2]

[1]Dryland Agricultural Research Institute (DARI), Maragheh, Iran;
[2]Seed and Plant Improvement Institute (SPII), Karaj, Iran

Introduction

Yellow or stripe rust of wheat (caused by *Puccinia striiformis* f.sp. *tritici* Westend.), is an important disease in many wheat growing regions of the world, especially in areas with cool and wet environmental conditions (Roelfs *et al.* 1992). Losses caused by the disease could be up to 75% (Roelfs, 1978).

Genetic resistance is the most economical and environmentally safe approach for disease control. Genes conferring seedling resistance are usually race-specific and can be recognized by their characteristic low infection types at all plant growth stages. Adult plant resistance can be either race-specific or race non-specific and is usually better recognized after the seedling stage (Johnson, 1980).

Some known adult plant resistance that have remained durable appear to involve slow rusting mechanisms conferred by minor additive genes (Singh and Rajaram 1994). Utilization of durable resistance for the global control of stripe rust is a major objective of the CIMMYT bread wheat breeding program (Rajaram *et al.* 1988).The objectives of this study were to evaluate the resistance of some rainfed wheat advanced lines/cultivars at the seedling and adult plant stages with some pathotypes of yellow rust.

Materials and Methods

A total of twelve advanced, promising and current commercial bread wheat cultivars (Sardari and Sabalan) plus the susceptible check Bolani were tested at the seedling stage in the greenhouse and the first 10 also at the adult plant stage in the field. The names of lines/cultivars were:

1- Omrabi 5,	2 - Omrabi 6,	3 - F134-71/Crow "S",
4 - Sxl/Vee"S"(15145),	5 - Fognchan/3/Trt,	6 - Nd/Vg 9144/Kal/,
7 - Sxl/Glennson Tx 84V1821,	8 - Tan"S"/Vee"S"/	9 - Kv2/PAK 20,
10 - Bb/Nor//Cal/7C,	11 - Sardari,	12 - Sabalan,
13 - Bolani		

Field studies were carried out at Maragheh, Sanandaj, Kermanshash and Hamadan under mist irrigation. The lines were inoculated artificially in the field to test for adult plant resistance. The infection types were recorded at the flag leaf stage and the incidence of disease was based on the modified Cobb's scale for percentage leaf area infected (Peterson *et al.* 1948).

At the seedling stage, five pathotypes of yellow rust including (134E148A+, 102E210A+, 66E2, 2E14 and 6E64A-) were selected from Hamadan, Kermanshah, Sanandaj, Uromieh, and Maragheh respectively and used. Pathotype identification procedures were conducted according to Johnson *et al.* (1972). Urediniospores of each pathotype were multiplied on the susceptible check. The lines/cultivars were inoculated at second leaf stage with rust pathotypes. Infection types were recorded on day 14 and 17 after inoculation using the 0-9 scale (McNeal *et al.* 1971) on the first and tip of second leaves.

Results and Discussion

Greenhouse Studies

Infection types at the seedling stage for each line/cultivar to pathotypes of stripe rust used in the greenhouse, were very varied due to virulence patterns of the pathogen (Table 1).

Table 1. The reactions of some rainfed wheat lines/cultivars to different pathotypes of yellow rust (*Puccinia striiformis* f. sp. *tritici* Westend.) at the seedling stage in greenhouse conditions

Lines	Yellow Rust Pathotypes / Infection Types				
No.	134E148A+	102E210A+	66E2	2E14	6E64A-
1	7	1	8	7	6
2	2	0	2	2	2
3	6	7	8	7	8
4	9	0	8	6	9
5	7	0	8	8	1
6	8	0	7	6	8
7	8	9	8	7	9
8	8	6	8	8	8
9	6	7	8	9	2
10	8	7	7	8	7
11	9	7	9	9	8
12	0	1	7	0	1
13	9	8	9	9	9

Field Observations

The results of adult plant stage studies showed that many of the lines had resistant reactions to yellow rust (Table 2). Highest levels of infection were observed at Hamadan and, in this trial, line 9 (Kv2/PAK 20) reached 70MS, where the race with wide virulence, 134E148+ was used.

The lines **1**(Omrabi 5), **3**(F134-71/Crow"S"), **6**(Nd/Vg 9144//Kal...) and **8**(Tan"S"/Vee"S"/...) were released by the Dryland Agricultural Research Institute (DARI) as Seimareh, Nik-Nejad, Gahar and Zagros respectively.

Table 2. The reactions of some rainfed wheat lines/cultivars at different locations to yellow rust (*Puccinia striiformis* f. sp *tritici* Westend.) at the adult plant stage in the field.

Line No.	Infection Type and Severity at Different Locations			
	Maragheh	Hamadan	Sanandaj	Kermanshah
1	15MR	10MR	0	20MR
2	0	40R	0	10MR
3	0	50R	0	0
4	15MS	30MR	5R	30MS
5	10MS	30MR	0	R
6	0	30R	VR	10R
7	5MS	30R	VR	10MR
8	15MS	5MR	VR	5R
9	15MS	70MS	0	30MS
10	10R	30MR	0	20MR

References

Johnson R, 1980. Genetics of adult-plant resistance to yellow rust in winter wheat cultivars. Pages 59-63 In: *Proceedings of the 5th European and Mediterranean Cereal Rusts Conference,* Italy.

Johnson R, Stubbs RW, Fuchs E, Chamberlain NH, 1972. Nomenclature for physiologic races of *Puccinia striiformis infecting* wheat. *Transactions of the British Mycological Society* **58:** 475-480.

McNeal FH, Konzak CF, Smith EP, Tate WS, Russell TS, 1971. A uniform system for recording and processing cereal research data. *United States Department of Agriculture Research Service,* ARS. 34-121. 42 pp.

Peterson RF, Campbell AB, Hannah E, 1948. A diagrammatic scale for estimating rust intensity of leaves and stem of cereals. *Canadian Journal of Research Section* C **26:** 469-500.

Rajaram S, Singh RP, Torres E, 1988. Current CIMMYT approaches in breeding wheat for rust resistance. Pages 101-118 in: Breeding Strategies for Resistance to the Rust of Wheat. N. W. Simmonds and S. Rajaram, Eds. CIMMYT, Mexico. D. F., Mexico.

Roelfs AP, 1978. Estimated losses caused by rust in small grain cereals in the United States-1918-1976. *Miscellaneous Publications of the United States Department of Agriculture*. **1363**: 1-85.

Singh RP, Rajaram S, 1994. Genetics of adult-plant resistance to stripe rust in ten spring bread wheats. *Euphytica* **72**:1-7.

Evaluation of Resistance to Yellow Rust in Wheat Landraces of the National Plant Genebank of Iran

Y. Arshad[1], F. Hemmati[1], A.R. Alitabar[2] and Raeiat-Panah[2]

[1] Department of Genetics and Plant Genetic Resources, SPII, Karaj, Iran;
[2] Mazandaran Agricultural Research Center, Sari, Iran.

Introduction

Yellow rust (Stripe rust) is an important diseases of wheat caused by *Puccinia striiformis* West. f. sp. *tritici*. Disease epidemics occur when susceptible cultivars and favorable climatic conditions are present, causing severe damage in wheat fields. Yellow rust is important in West Asia, Far East, South Africa, South America and Northern Europe (Saari and Prescott 1985). There are 6 million hectares of wheat in Iran and this disease is important in many of the major wheat cultivation regions, especially in the region adjacent to the Caspian Sea where epidemics occur each 3 to 4 years. (Niemann,*et al.* 1968, Khazra and Bamdadian 1974).

Several studies were conducted with the objective of producing cultivars with resistance to yellow rust for Iran. Dijk *et al.* (1988) tested 16 races of *P. striiformis* against various wheat cultivars and found 29 with resistance at the adult plant stage. Nazari *et al.* (1995) reported 85 advanced lines of bread wheat with resistance in the greenhouse and in the field. Torabi *et al.* (1995) described 19 advanced lines and commercial bread wheat cultivars which were resistant or moderately resistant to 3 races of *P. striiformis*.

The aim of this study was to find genetic stocks with resistance to yellow rust among the collection of wheat accessions in the National Plant Gene Bank of Iran (NPGBI), for use in the wheat improvement programs, to produce cultivars combining favorable agronomic characters and resistance to yellow rust.

Materials and Methods

A total of 1024 wheat accessions from the NPGBI collection were planted in Gharakhil Agricultural Research Station in Sari city where there was an epidemic of yellow rust during the growing season in 1997-98. The accessions were evalu-

ated for resistance to disease by observation under natural infection. Each accession was planted in two 2.5 m rows with 50 cm between rows. Bolani. a cultivar susceptible to yellow rust, was planted at the border of the field as a disease spreader. The rate and type of the infestation was documented using the Modified Cobb's Scale (Peterson, *et al.* 1948).

Infection was recorded at the flag leaf stage according to the following scale: 0 = Immune, no infection; R= resistant, appearance of chlorotic and necrotic lesions without spores or with very few spores; MR= moderately resistant, chlorotic and necrotic lesions with small pustules; MS= moderately susceptible, chlorotic lesions with rather large pustules; S= susceptible, without or with very few chlorotic lesions and with very large pustules. The amount of infection on the plants was recorded as the percentage of leaf surface covered by yellow rust pustules (0-100%).

Coefficient of Infection: To calculate the average coefficient of infection a common method from CIMMYT was used (Stubbs *et al.* 1986). The average of infection rate in each accession was multiplied by the coefficient of infection. The latter was determined as follows:

Constant coefficient of infection	Type of infection
0	0
0.2	R
0.4	MR
0.6	M (Intermediate)
0.8	MS
1	S

To evaluate the morphological, agronomic and phenologic characters, the descriptor of IPGRI (International Plant Genetic Resources Institute) was used.

The following statistical calculations were carried out to analyze the data:
- calculation of mean, standard deviation, minimum and maximum value, standard error, variance and coefficient of variation.
- correlation coefficient and factor analysis of characters

Results and Discussion

Among the evaluated wheats, 230 accessions showed no symptoms (0), 27 were resistant (R), and 34 were moderately resistant (MR). Mean, variance, minimum and maximum value, standard deviation, standard error, and coefficient of variation of the characters are shown in Table 1. Infection coefficient of yellow rust had an average of 37.76±1.18, minimum of 0 (no disease) and the maximum 100 (completely susceptible, 100S). These accessions will be tested again using artificial inoculation in the greenhouse and their resistance will be evaluated at

seedling and adult stages. In order to use them in the breeding program the resistant accessions will be tested for uniformity and investigated genetically, including heritablity and determination of resistance genes.

Simple correlation between evaluated characters in the Gharakhil station is shown in Table 2. Yellow rust correlated positively at 1% with Glume colour (r =0.182), Glume hairness (r =0.116), stem thickness (r =0.122), tiller number (r =0.127), and negatively with days of flowering (r = -0.363), number of seeds/spike (r = -0.134), number of nodes (r = -0.179), and plant height (r = -0.140). Yellow rust also correlated negatively at 5% with number of spikelets/spike (r = -0.067) and number of flowers/spikelet (r = -0.071).

Yang and Zeng (1980) concluded that there is direct relation between disease development and loss of yield. Heitage *et al.* (1988) suggested that the flag leaf is the most sensitive stage of growth to yellow rust for yield loss and the most important growth stage for grain formation. Ash and Brown (1990) also reported that disease infection at the early period of growth causes reduction in the weight of 1000 seeds and the number of seeds/spike, which consequently lead to yield loss. They showed that yellow rust had the highest negative correlation coefficient with days of flowering and the lowest negative correlation coefficient with the number of spikelets/spike.

Factor analysis of the evaluated characters in Gharakhil station

The results of factor analysis for 15 evaluated characters are shown in Table 3. Five major factors are presented, which have also relations with other analyzed characters and accounted for 57.1% of the total variance of the population. Factor 1, which in turn has relation with other analyzed characters, accounted for 17.9% of the total variance of the population. The characters of number of spikelets/spike, number of flowers/spike and number of seeds/spike correlated positively with this factor. Factor 2 also accounted for 13.9% of the total variance. Amount of yellow rust and type of growth correlated positively and days of flowering correlated negatively with this factor. Although transforming 15 characters to five factors reduced the total data to about one third and reduced the variance by about 42.9%, it helped to reveal the major effective characters and to group them according to their relationships. Yildrim *et al.* (1996) reduced 19 morphological, phenological and agronomic characters to five major factors, which accounted for 82.3% of the total variance of the population. Factor 1 itself consisted 26.6% of the total variance of the population and included the characters of days of flowering, days of spike formation, days of growing and days of harvesting.

Table 1. Statistical results of evaluated characters of 1024 wheat accessions from NPGBI at Gharakhil station in 1996-97.

Characteristics	Mean _(X)	Standard Error(SE)	Min	Max	Variance(S2)	Coefficient Variance(CV%)	Standard Deviation(S)
Growth Type	1.91	0.03	1.00	3.00	0.83	47.64	0.91
Length of Spike	8.98	0.05	4.50	13.60	2.29	16.82	1.51
Density of Spike	4.83	0.07	1.00	9.00	4.88	45.76	2.21
Awn	6.02	0.07	0.00	7.00	4.45	35.05	2.11
Glume Colour	1.37	0.02	1.00	3.00	0.43	48.18	0.66
Glume Hairness	2.55	0.09	0.00	7.00	7.34	106.27	2.71
Day of Flowering	154.80	0.23	136.00	177.00	52.80	4.69	7.27
Tiller Number	6.28	0.05	3.00	12.67	2.04	23.77	1.43
Plant Height	1.24	0.01	75.00	184.00	0.02	12.14	0.15
Stem Thickness	3.49	0.02	1.80	5.40	0.33	16.33	0.57
Number of Flowers/Spike	2.75	0.01	2.00	4.00	0.197	16.00	0.44
Number of Seeds/Spike	15.02	0.21	3.00	44.00	44.49	44.41	6.67
Number of Spikelet/Spike	18.70	0.10	7.96	28.00	9.40	16.42	3.07
Number of Nodes	4.39	0.02	3.00	6.00	0.28	12.07	0.53
Yellow Rust	37.76	1.18	0.00	100.00	1413.09	99.58	37.60

Table 2. Correlation coefficients of the evaluated characters of 1024 wheat accessions from NPGBI at Gharakhil station in 1996-97.

	Day of Flowering	Glume Colour	Glume Hairness	Number of Spikelet/Spike	Number of Flowers/Spike	Number of Seeds/Spike	Number of Nodes	Plant Height	Stem Thickness	Density of Spike	Tiller Number	Awn	Yellow Rust
Glume Colour	-0.21**	-											
Glume Hairness	-0.173**	0.484**	-										
Number of Spikelet/Spike	-0.115**	0.131**	0.138**	-									
Number of Flowers/Spike	-0.116**	-0.079*	-0.111**	0.141**	-								
Number of Seeds/Spike	-0.122**	0.078*	0.053	0.498**	0.305**	-							
Number of Nodes	0.119**	-0.019	0.012	0.061	0.09**	0.12**	-						
Plant Height	-0.135**	-0.036	-0.026	0.278**	0.14**	0.301**	0.239**	-					
Stem Thickness	-0.326**	0.189**	0.191**	0.228**	0.146**	0.257**	0.049	0.43**	-				
Density of Spike	-0.153**	0.171**	0.109**	0.113**	0.088**	0.215**	-0.057	0.103**	0.209**	-			
Tiller Number	-0.006	0.011	-0.049	-0.003	-0.051	-0.106**	-0.06	0.102**	0.056	-0.066*	-		
Awn	0.186**	-0.131**	-0.196**	-0.033	-0.024	0.024	0.038	0.037	-0.108**	-0.092*	0.02	-	
Yellow Rust	-0.363	0.182**	0.116**	-0.067*	-0.071*	-0.134**	-0.179**	-0.140**	0.122**	0.031	0.127**	-0.053	-
Length of Spike	-0.038	0.048	0.108**	0.448**	0.149**	0.227**	0.079*	0.133**	0.087**	-0.268**	0.027	-0.031	-0.051

□ and ** significant at 5% and 1% levels respectively.

Table 3. Factor analysis of the evaluated characters of 1024 wheat accessions from NPGBI in Gharakhil station in 1996-97.

Factor 1	Factor 2	Factor 3	Factor 4	Factor 5
Number of Spikelet/Spike (0.65)	Yellow Rust (0.67)	Glume Colour (0.78)	Plant Height (0.80)	Spike Density (-0.73)
Number of Flowers/Spike (0.62)	Days of Flowering (-0.72)	Glume Hairness (0.84)	Stem Thickness (0.60)	
Number of Seeds/Spike (0.75)	Type of Growth (0.64)			

References

Ash G, Brown JF, 1990. Yield losses in wheat caused by stripe rust *Puccinia striiformis* West. f.sp. tritici in North New South wales. *Australian Journal of Experimental Agriculture,* **30:** 103-108.

Dijk PVan, Parlevliet JE, Kema GHJ, Zeven AC, Stubbs RW, 1988. Characterization of durable resistance to yellow rust in old winter wheat cultivars in Netherlands. *Euphytica,* **38:** 149-158.

Heitage AO, Stapper M, Smith RGG, Correll RL, 1988. Model of stripe rust infection and control on irrigated wheat in the Murrum-Bigee irrigation area in New South wales. *Australian Journal of Botany* **19:** 51-61.

Khazra H, Bamdadian A, 1974. The wheat diseases situation in Iran. *Proceedings of the FAO Rockefeller Foundation Wheat Seminar 1973*, PP. 292-299.

Nazari K, Mamlouk A, Naimi M, 1995. Study on the resistance of bread wheat advanced lines to wheat yellow rust, *Puccinia striiformis* West f.sp. *tritici* at seedling and adult plant stages, under greenhouse and field conditions in ICARDA. *12th Congress of Plant Protection of Iran.* p.61.

Niemann E, Scharif G, Bamdadian A, 1968. Die Getreide roste in IRAN. Wirts, Bereich, Untercheidung, Bedevtung, Bekampfung. *Entomologie et Phytopathologie Appliquees,* **27:**25-41.

Peterson RF, Campbell AB, Hannah AE, 1948. A diagramatic scale for estimation rust intensity on leaves and stems of cereal. *Canadian Journal of Research,* **26:** 496-500.

Saari EE, Prescott JM, 1985. World distribution in relation to economic losses. PP.259-298. In: A.P.Roelfs and R.W. Bushnell (eds.) *The Cereal Rusts, Vol II* Academic Press, Orlando.

Stubbs RW, Prescott JM, Saari EE, Dubin HJ, 1986. Disease Methodology Manual. CIMMYT, Mexico D.F. 46 pp.

Torabi M, Nazari K, Mardoukhi V, Houmand NC, 1995. Study on the resistance of the few advanced lines and commercial cultivars of wheat to 3 races of yellow rust. *12th Congress of Plant Protection of Iran.* p.35.

Yang XB, Zeng SM, 1989. Effect of yellow rust on yield components of winter wheat in China. *Plant Pathology* **38:** 1- 8.

Yildirim MB, Budak N, Arshad Y, 1996. Factor Analysis of yield and related traits In bread wheat. *Turkish Journal of Field Crops* **1**: 11-15.

Evaluation of Some Advanced Lines and Cultivars of Wheat to Yellow Rust in Mazandaran

A. Foroutan and M.S. Ahmadian Moghaddam

Agricultural Research Center of Mazandaran, Sari, Iran.

Introduction

Wheat is the major food crop in Iran and is the third crop (about 50000 hectares), after rice and citrus, in Mazandaran province. Yellow rust, caused by *Puccinia striiformis* f.sp. *tritici,* is one of the most important diseases of wheat in Iran, specially in Mazandaran. In 1993 estimated yield losses were more than 30%, with an average national loss of 15% or 150000 tonnes (Torabi *et al.* 1995). Control of wheat yellow rust by identifying sources of resistance is the best method in Iran, as in most wheat growing countries. Breeding for resistance has been carried out for many years and must be continued. Because of virulence changes and development of new races of the yellow rust pathogen, cultivars with resistance sometimes become susceptible. This study was undertaken to confirm the resistant reaction of some advanced wheat lines and cultivars that have previously shown good performance.

Materials and Methods

One hundred and thirty-five resistant lines and cultivars, selected from multi-location tests in 1996-97 and 1997-98, were re-evaluated in the 1998-99 cropping season. Selected materials were planted in late November 1999. Each line or cultivar was planted separately in two rows of one meter at the Bayehkola Research Station under misted irrigation. Seeds of Bolani, as a susceptible cultivar, were sown between every ten lines and also round the nursery to spread the disease. One hundred kg/h ammonium phosphate (46%P_2O_5, 18%N), fifty kg/h potassium sulphate (50% K_2O) and one hundred kg/h urea (46%N) were applied to the nursery before planting. Also, fifty kg/h additional urea was applied as top dressing in early February. Artificial inoculation was carried out twice, at the seedling and tillering stages. Reactions of the tested materials i.e. infection type and severity, were recorded by modified Cobb's scale as resistant (R), moderately resistant (MR), intermediate (M), moderately susceptible (MS) and susceptible (S).

Results and Discussion

Based on the results, thirty-three lines and cultivars showed moderate resistance to yellow rust (Table 1). Similar resistant reactions of some lines have also been reported from other locations in the country. Two other diseases, leaf rust and powdery mildew, were also included in the selection program and some lines were

resistant either to leaf rust or powder mildew (Table 1). It is suggested that the lines with good resistance in this study can be used as good sources of resistance in breeding programs.

Table 1:Reaction of thirty-three lines and cultivars to yellow rust[1], leaf rust[1] and powdery mildew[2] in field conditions in the 1998-99 cropping season at Bayehkola Research Station of Mazandaran.

Pediree	Seed Source	Yellow rust	Leaf rust	Powdery Mildew
Rsh/Pewee	1-75-7	10R	10S	5
1-67-13//Sanine	1-75-11	5R	15S	5
Fertillo/Vee	1-75-15	10R	10MS	7
1-61-40/30/Sbn	1-75-18	10R	10M	5
Omid/H7/4P	1-75-30	10R	15MS	3
Falat/5/Gds/	1-75-32	10R	5S	3
Hys/7c//	1-75-69	10R	25S	7
Gods/4/Anza	1-75-84	5R	30S	5
Atay85/3/Rsh	1-75-90	10R	35S	7
ICWHA81	1-75-98	5R	20MS	5
MV-92-2854	1-75-109	5R	15M	5
Prl"S"/Koek	1-75-113	5R	20S	5
1-66-94/1-66-75	1-75-119	5R	20MS	5
Yr/Sprw	1-75-126	10R	10MR	5
1-66-31/5/Anza	1-75-128	10R	5R	7
Ald"S"/3/Cc//	1-75-133	10R	60S	7
Wa476/3/391	1-75-139	5R	7R	0
Yr/Sprw"S"//	1-75-155	5R	0	7
Tui"S"//Bank	1-76-2	10R	5R	3
1-63-40/3/Hork	1-76-27	7R	30S	5
Vee'S'/Snb	1-76-51	10	5R	5
Kauz"S"/-66-65	1-76-65	5R	30S	5
Kal/bb/Cj	1-76-9	10R	15MR	3
Seri/Buc	1-76-115	5R	20	3
Snb"S"/Kauz'S"	1-76-144	5R	5S	6
Rsh	1-76-164	10R	10S	3
1-67-78/1	1-76-180	5R	30S	0
Alvd//5/Gds	1-76-19	5R	40S	0
ICW-HA81	1-76-232	10R	10MR	3
4777//FKN	1-76-278	20M	10R	3
Bow"S"/	1-76-312	TR	40S	0
Navid/1-27	1-76-346	10R	30S	5
Bow"S"/Vee	1-76-350	TR	40S	3

[1] Modified Cobb's scale (R, MR, M, MS and S)

[2] Saari and Prescott (1975), scale (0-9)

References

Saari EE, Prescott JM, 1975. A scale for appraising the foliar intensity of wheat diseases. *Plant Disease Reporter* **59,** 377-380.

Torabi M, Mardoukhi V, Nazari K, Afshari F, Foroutan A, Rramai MA, Golzar H, Kashani AS, 1995. Effectiveness of wheat yellow rust resistance genes in different part of Iran. *Cereal Rusts and Powdery Mildews Bulletin* **23:** 9-12.

Yellow Rust Research in Pakistan: An Overview

Iftikhar Ahmad

Crop Diseases Research Institute, National Agricultural Research Centre, PARC, Islamabad, Pakistan.

Introduction

In Pakistan, as in other cereal producing areas of the world, wheat rusts are the major threatening diseases and continue to receive attention. At a very conservative estimate the average annual losses due to rusts is estimated at Rs. 1500 million at the present price level (Hussain, M; CDRI, NARC, Islamabad, Pakistan, Personal communication). Additional losses may occur in years when rusts appear in epidemic proportions as had happened in 1978. The average national loss due to leaf rust epidemics in wheat was estimated at 10 percent or 830,000 tons of wheat grain with a value of US$ 86 millions Hussain *et al.* 1999). The recent epidemic of stripe rust in northern Punjab and NWFP has caused a loss of Rs. 2 billion during 1994-95 and a close to that during 1995-96.

Historical

Research work on identification and distribution of physiological races of *Puccinia* species causing rusts in wheat in the then British India was started under the auspices of the Indian Agricultural Research Institute by K.C. Mehta and his group (Mehta, 1946). History of cereal rust research in Pakistan is intertwined with the history of Crop Diseases Research Institute (CDRI) as this is the premier Institute where work on cereal rusts is concentrated. Having been conceived in 1953 as a research scheme on rusts of wheat and barley, it has come to its present status in 1983 through a number of up-gradations (Table 1). In the early period up to 1971, work concentrated on occurrence, distribution and incidence of and losses due to rusts, role of collateral and alternate hosts, identification of physiologic races, and screening of wheat germplasm for resistance to rusts. From 1971 onward, studies on physiologic race and virulence analysis were intensified, and major emphasis was given to postulating genes in the current cultivars and advanced lines for rust resistance (Table 2) (Ahmad 1997, 1999).

Table 1. Crop Diseases Research Institute: Historical

1953	Conceived as research scheme "investigations on rust of wheat and barleyin Pakistan" Department of Plant Protection, Food and Agricultural Council of Pakistan
1955	Scheme Initiated
1958	Cereal Research Station
1977	Cereal Research Institute
1978	Taken over by Pakistan Agricultural Research Council (PARC)
1983	Crop Diseases Research Institute

Table 2. Rust Research: Historical

Early Period up-to 1971	*	Distribution and Incidence
	*	Alternate Hosts
	*	Race Analysis
1972-83	*	Race Analysis
	*	Virulence Analysis
	*	Resistance
1983 - onward	*	Race Analysis
	*	Virulence Analysis
	*	Genetic analysis
	*	Resistance

Yellow Rust Research

Yellow rust was reported to be high in sub-mountainous areas of Pakistan and remained a disease of little importance until 80's when a mini epidemic occurred in northern Pakistan (Figure 1, 2). There is a shift in the geographical distribution of the yellow rust and it has expanded from north towards the south of Pakistan. Until 1983 14 races of stripe rust were identified (Hafiz 1983). Yellow rust race groups reported in Pakistan include 64E0, 66E0, 66E(16), 6E16, 7E16, 38E16, 6(38)E16, 2(6)E16, 7E(15)150, 134E150 (Table 3). Race group 7E(15)150 reported in 1983-84 is virulent on *Yr1, Yr2, Yr6, Yr7, Yr* mex and *Yr A*. Race group 134E150 reported in 1994 can attack genotypes with *Yr9* (Ahmad 1999). The numbers in brackets indicate alternative classifications arising from variable responses of the differential cultivars to some isolates.

Table 3. Prevalence of Races of *Puccinia striiformis* (Stripe Rust) During Different Periods from 1969 onward in Pakistan

Period	Races
1969-1973	Race Groups 64E0, 66E0, 67E0, 66E(16) Predominant.
1973-1976	Race Groups 6E16, 66E16, 67E0, and 7E16 were identified. The Pathogen Carried Virulence for *Yr1, Yr6, Yr7, Yr8* and Suwon 92.
1977-1979	Race Groups 38E16, 6E16, 6(38)E16, 2(6)E16, 2E16 and 7E16 Isolated during this period.
1983-1984	New Race Group Reported: 7E(15)150 This race group has virulence for *Yr1, Yr2, Yr6, Yr7, Yr* mex and *Yr A*
1994	Identified Race 134E150, which can attack cvs. possessing host gene *Yr9*

Commercial wheat varieties which are either in cultivation or were in cultivation during the past as well as advanced lines which were to become varieties in the future were genetically analyzed (Hussain *et al.* 1988). Yellow rust resistance genes *YrA Yr6, Yr7,* and *Yr9* were found to be predominant in Pakistani wheat varieties (Table 4; Perwaiz and Johnson 1986, Dubin *et al.* 1989). However, the more recent germplasm has not been analysed and therefore little is known about the genes for yellow rust resistance in new cultivars.

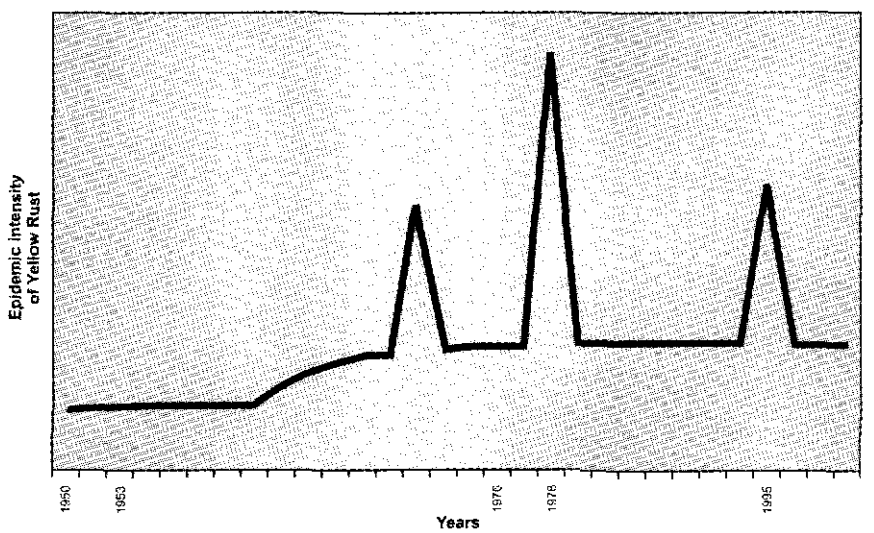


Figure 1. Temporal cereal rust situation in Pakistan

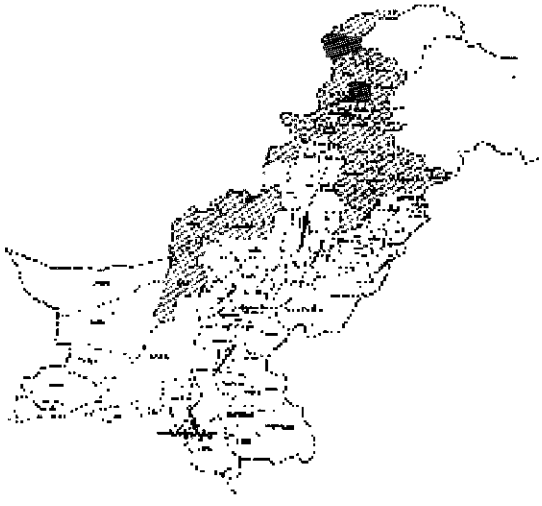


Figure 2. High intensity areas of yellow rust in Pakistan (shaded areas).

Table 4. Postulated seedling resistance genes for stripe rust in Pakistani wheats

Cultivar	Yr genes	Cultivar	Yr genes
Arz	*A*	Pirsabak	*9, 7*
Bahawalpur	*A*	Pothowar	-
Barani 83	*7, 2*	Punjab 76	*A*
Blue Silver	*A, 2*	Punjab 81	*A*
Chakwal	*9*	Punjab 85	*9*
Chenab 70	-	Rawal 87	*9*
Chenab 79	*A**	Sandal 73	*A, 6*
Faisalabad 83	*7, Mex*	Sarhad 82	*9, 7*
Faisalabad 85	*9, 4*	Sarsabz	-
Indus 79	*7*	Sindh 81	-
Jauhar 78	-	Sindh 83	*6?*
Khushal	-	Sonalika	*A*
Khyber 87	*9*	Sutlej	*9*
Khyber 79	-	WL711	*2*
Kohinoor 81	*9, 2*	Yecora 70	*A, 6*
Local White	-	ZA 77	-
LU 26	*6*	Zaminder 80	*A, 6*
Lyallpur 73	*A, 6*	Zarghoon	*6*
Mexipak 65	*2*		
Nuri 70	*A*		
Pakistan 81	*9, 7*		
Pari 73	*A, 6*		
Pavon 76	*6, 7*		

* = Heterogeneous - = No gene detected

? = Preliminary results requiring confirmation

Current Programme of Wheat Rusts Research in Pakistan

Crop Diseases Research Institute continues to be the main centre focussing on wheat rust research in Pakistan. As a partner in the National Agricultural Research System the Institute provides support to National Wheat Breeding Programme by collaborating with the National Wheat Coordinated Programme, NARC, Islamabad and the Provincial Wheat Research Institutes through its own basic research on host-parasite genetics, geographical and temporal rust monitoring, virulence analysis and by providing following services to the National Breeding programme such as National Wheat Diseases Screening Nursery (NWDSN), Wheat Rust Trap Nursery (WRTN), and National Uniform Yield Trials (NUYT) which make the backbone of national varietal release programme (Ahmad *et al.* 2000a, 2000b; Akhtar and Ahmad 1999, Hussain and Ahmad 1998, Hussain *et al.* 1999).

National Wheat Disease Screening Nursery

As part of its programme, the Institute prepares and distributes a National Wheat Diseases Screening Nursery (NWDSN) to other wheat research organizations. The NWDSN comprises 300 to 400 entries, including widely grown commercial varieties and advanced lines from national breeders. The nursery is planted at a dozen or so sites throughout the country, where artificial inoculations are performed to place have disease pressure on the breeding materials. This nursery help to identify new sources of resistance to the prevalent and new races of rusts and to evaluate breeding materials for their disease reaction.

Wheat Rust Trap Nursery

The Institute prepares and distributes the Wheat Rust Trap Nursery (WRTN) each year. This nursery includes commercial varieties and lines with single genes for resistance to leaf and stripe rust. It is planted at 30 to 40 sites and is used to monitor and trap the natural virulence of each race of rust prevalent in the country (Anon. 1989).

National Uniform Yield Trials

Data from these trials are vital for the National Wheat Breeding Programme and National Variety Release System. Disease Reactions of NUWYT are used by the Variety Evaluation Committee for releasing candidate lines as commercial varieties.

Rust Inoculum

CDRI provides rust inoculum consisting of prevalent virulences in the particular province to provincial and federal breeders for their screening programmes at provincial and federal level.

Outlook

Wheat continues to be the staple food in Pakistan and as such wheat rusts will continue to receive attention to have a stable production of the wheat crop within the context of food security of the Country. Manpower constraints at other phytopathological research institutions do not allow them to take up research on wheat rusts very extensively. Therefore, CDRI is likely to continue to a major programme of cereal rust research. The Institute needs to strengthen its activities on the epidemiology of rusts, broadening the genetic base for rust resistance and take up research on other newer approaches within the context of Integrated Disease Management Strategy. As yellow rust is now becoming a major disease likely to affect wheat production, it is imperative to expand research on it. There is need to identify newer sources of resistance to yellow rust, broaden the genetic base of Pakistani wheat cultivars and understand the changing patterns of virulences to shift the breeding programme accordingly. In this regard it is necessary to collaborate with the countries in the region on its epidemiology and exchanging germplasm.

References

Ahmad I, 1997. An overview of phytopathological research with particular emphasis on cereal rusts at the Crops Diseases Research Institute, Islamabad, Pakistan. *International Conference on Integrated Plant Disease Management for Sustainable Agriculture,* November 10-15, 1997, New Delhi, India. S9C-003. pp.122.

Ahmad I, 1999. Cereal rust research in Pakistan. *Second National Conference of Plant Pathology,* 27-29 September, 1999. University of Agriculture, Faisalabad. Pakistan Phytopathological Society (Abstract No. 75). pp.22.

Ahmad I, Akhtar M.A, Mirza J.I, Rattu A.R, 2000a. Evaluation of candidate lines against stripe and leaf rusts under National Uniform Wheat and Barley Yield Trial, 1999-2000. CDRI, PARC, Islamabad, Pakistan (mimeographed). p.23.

Ahmad I, Rattu A.R, Mirza J.I, and Akhtar, M.A, 2000b. Report on National Wheat Diseases Screening Nursery, 1999-2000. CDRI, PARC, Islamabad, Pakistan (mimeographed). p.42.

Akhtar M.A, and Ahmad I, 1999. Evaluation of candidate lines against stripe and leaf rusts under National Uniform Wheat and Barley Yield Trials, 1998-99. CDRI, PARC, Islamabad, Pakistan (mimeographed). p.28.

Anonymous, 1989. Wheat research and development in Pakistan. Mexico. D.G. CIMMYT, pp. 65-66.

Dubin HJ, Johnson R, Stubbs RW, 1988. Postulated genes for resistance to stripe rust in selected CIMMYT and related wheats. *Plant Disease* **73:** 472-475

Hafiz, A. 1983. Plant pathology research activities in Pakistan. PARC, Islamabad, Pakistan. pp. 154.

Hussain M, and Ahmad I, 1998. Evaluation of candidate lines against stripe and leaf rusts under National Uniform Wheat, Barley and Triticale Yield Trials, 1997-98. CDRI, PARC, Islamabad, Pakistan (mimeographed). p.23.

Hussain M, Ahmad I, Haque M I, Kirmani M A S, Hamid S J, Haque E U, Akhtar M A, Rattu A R, Mirza J I, Khan S A.J, Hakro A A, and Jaffery A H, 1999. Evaluation of candidate lines against stripe and leaf rusts under National Uniform Wheat, Barley and Triticale Yield Trial, 1997-98. *Proceedings of 2nd National Conference of Plant Pathology,* September 27-29, 1999. University of Agriculture, Faisalabad. Pp.112-119.

Hussain M, Hakro AA, Aslam M, Gordon-Werner E 1988. Postulated genes for rust resistance in Pakistani wheats. *Proceedings of Seventh European and Mediterranean Cereal Rust Conference,* Vienna, Austria, pp. 33-35.

Mehta KC, 1946. Furthers studies on cereal rusts in India Scientific Monograph No. 14 *Indian Committee on Agricultural Research,* pp. 1-240.

Perwaiz MS, Johnson R, 1986. Genes for resistance to yellow rust in seedlings of wheat cultivars from Pakistan tested with British isolates of *Puccinia striiformis. Plant Breeding* **97:** 289-296.

Biotechnology and Rust Resistance

Marker-Assisted Selection for Rust Resistance in Wheat in Australia

H. S. Bariana

The University of Sydney Plant Breeding Institute, Cobbitty, Australia.

Introduction

Availability of robust selection technology is essential to achieve desired levels and durability of disease resistance in commercial cultivars. Field evaluation either under natural infection or artificially created epidemic conditions has been used around the world to select genotypes with acceptable levels of disease resistance. The alternative to phenotypic selection is the use of low rust response linked markers. These include morphological markers, protein markers, DNA markers and linked disease resistance genes. Recently molecular marker linked to rust diseases have been developed. Different marker development technologies include, restriction fragment length polymorphism (RFLP), amplified fragment length polymorphism (AFLP), microsatellite/simple sequence repeat polymorphism (SSR) and sequence characterised amplified regions (SCARs). This paper reports the current status of the use of markers in breeding programs.

Available markers and their application

Morphological markers

The morphological markers associated with rust resistance genes are listed in Table 1. Of these, genetic association of *Yr10* with brown chaff and *Sr2* with pseudo-black chaff and seedling chlorosis have been used for backcrossing of these resistance genes in Australia. Linkage of leaf tip necrosis together with pedigree information is also used for the detection of *Lr34/Yr18* in advanced breeding materials undergoing rust screening in the National Cereal Rust Control Program, University of Sydney.

Protein markers

Certain resistance genes located in the short arms of homoeologous goup 1 chromosomes are genetically linked with protein encoding loci (Table 1).
Sec-1 specific ELISA has been used to select for the presence or absence of rust resistance genes *Sr31, Lr26,* and *Yr9* in the Northern Wheat Improvement Program in Australia. Other protein/resistance associations listed in Table 1 could also be used for selection, but have not yet been used in Australia.

Table 1. List of rust resistance linked morphological, protein and disease resistance markers.

Type of marker/Disease Morphological	Resistance gene	Marker	Reference
Stripe rust	*Yr10*	*Rg1*	Metzger and Silbaugh (1970), Payne *et al.* (1986)
Leaf rust	*Lr34*	*Ltn*	Singh 1992a
Stem rust	*Sr2*	*Pbc, Sc*	Hare and McIntosh (1979), Brown (1997)
Protein			
Stripe rust *Yr9*	*Sec-1*		Gupta *et al.* (1997)
	Yr10	*Gli-B1*	Payne *et al.* (1986)
	Yr10vav	*Gli-B1*	Bariana *et al.* 1999
Leaf rust	*Lr26*	*Sec-1*	Gupta *et al.* (1997)
Stem rust *Sr31*	*Sec-1*		Gupta *et al.* (1997)
Linked resistance gene			
		Yr9	Lr26/Sr31 Hu and Roelfs (1986)
	Yr17	Lr37/Sr38	Bariana and McIntosh (1993)
	Yr18	*Lr34*	McIntosh (1992), Singh (1992b)
	Yr29	*Lr46*	Singh (pers. comm.)
	Lr24	*Sr24*	McIntosh *et al.* (1977)
	Lr20	*Sr15/Pm1*	McIntosh (1977)
	Lr34	*Yr18/Bdv1*	Singh (pers. comm.)
	Lr35	*Sr39*	Kerber and Dyck (1990)
	Lr19	*Bdv1*	Singh (pers. comm.)
	Lr19	*Sr25**,	McIntosh *et al.* (1977)

* Sr25 is linked with *Lr19* in some accessions of reduced yellow flour pigment produced by Dr D. Knott.

Linked disease resistance genes

Genetically linked sources of resistance to more than one diseases are listed in Table 1. One such source of resistance, *Sr24/L24,* has been extensively used in Australia for over fifteen years prior to the detection of a pathotype virulent on *Lr24* in 2000. Low infection type produced by lines carrying *Lr24,* when infected with avirulent pathotypes, is diagnostic for the presence of *Sr24,* which produces infection type similar to that produced by several other stem rust resistance genes. Another example of use of linked rust resistance genes is VPM1-derived rust resistance genes *Sr38, Lr37* and *Yr17.* Although virulence was detected in 1999 for stripe rust resistance gene *Yr17,* genes *Sr38* and *Lr37* are effective against all current pathotypes of *Puccinia graminis tritici* (causal organism of stem rust of wheat) and *Puccinia triticina* (previously *Puccinia recondita* f.sp *tritici;* causal agent of wheat leaf rust), respectively. For example, cultivar Sunbri originally selected for stem rust resistance *(Sr38)* also carried leaf rust *(Lr37)* and stripe rust *(Yr17)* resistance. Similarly North American cultivars Hyak and Madsen, selected for stripe rust resistance, also carried stem rust and leaf rust resistance. Recently CIMMYT workers combined leaf rust resistance gene *Lr19* with barley yellow dwarf gene *Bdv1* (R.P. Singh pers. comm.). Use of this *Lr19* source would ensure simultaneous selection for barley yellow dwarf virus resistance. Lines selected by using linked resistance loci should be evaluated against the target pathogen at some stages of the breeding program.

Table 2. Current status of rust resistance linked PCR based markers.

Disease	Resistance gene	Marker	Reference
Wheat stripe rust			
	Yr10	STS-PCR	Frick *et al.* (1998)
	Yr10var	SSR	Bariana *et al.* (1999)
	Yr9/Lr26/Sr31	PCR	Rogowsky *et al.* (1992)
	Yr17/Lr37/Sr38	PCR	Seah *et al.* (2001)
	Yr15	SSR	Zakeri (pers. comm.)
	Yr24	SSR	Zakeri (pers. comm.)
	Yr26	SSR	Ma *et al.* (2001)
Leaf rust			
	Lr24/Sr24	PCR	Dedryvar *et al.* (1996)
	Lr35/Sr39	PCR	Gold *et al.* (1999)
	Lr47	PCR	Helguera *et al.* (2000)
Stem rust			
	Sr2	PCR	Johnston *et al.* (1998)
	Sr36	SSR	Bariana *et al.* (2001)

DNA markers

Identification of DNA markers linked to rust resistance loci has taken place recently (Table 2). The successful implementation of DNA markers in breeding programs depends on simple, user-friendly and high throughput protocols. Genetic associations of many RFLP markers with rust resistance genes have been reported. Only some markers have been converted to PCR based assays. On the other hand several instances of associations of SSRs with rust resistance genes have been demonstrated. These markers could be used for pyramiding of rust resistance genes. However, because of the emphasis on achieving more durable resistance using sources of adult plant resistance such as cultivar Cook, the currently available markers are not suitable for marker assisted selection for stripe rust resistance in Australian breeding programs. Development and validation of markers linked to adult plant resistance loci would provide opportunities for marker assisted selection for stripe rust resistance.

Concluding remarks

The application of markers in breeding programs is in the validation phase and has been limited to germplasm development. The majority of markers are for alien sources of resistance because of high levels of polymorphism detected in distant crosses. Recently efforts have been directed at the identification of markers linked to resistance genes that are important for practical plant breeding. It is important to remember that markers serve as alternate selection tool and are unlikely to replace final phenotypic evaluation. Therefore, materials derived from marker assisted selection programs must undergo phenotypic evaluation to ensure the presence of acceptable levels of resistance in breeding populations. The other constraint to the use of markers is the limited resources in public plant breeding programs. Development of markers linked to other agronomic traits would make marker assisted selection a more cost-effective strategy.

Acknowledgements

GRDC Australia is thanked for continuous financial support for rust research.

References

Bariana HS, and McIntosh RA, 1993. Cytogenetic studies in wheat XV. Location of rust resistance genes in VPM1 and their genetic linkage with other resistance genes in chromosome 2A. *Genome* **36,** 476-482.

Bariana HS, Ahmed NU, Hayden MJ, Bell JA, Sharp PJ, and McIntosh RA, 2001. Mapping of durable adult plant and seedling resistances to stripe rust and stem rust diseases in wheat. *Australian Journal of Agricultural Research* (in press).

Bariana HS, Brown, GN, Ahmed, NU, Khatkar, S, Wellings, CR. Conner, RL, Haley, S, Sharp, PJ and Laroche, A (2001) Characterisation of *Triticum vavilovii*-derived stripe rust resistance using genetic, cytogenetic and molecular analyses. *Theoretical & Applied Genetics* (in press).

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

Dedryvaer F, Jubier MF, Thouvenin J, and Goyeau H, 1996. Molecular markers linked to the leaf rust resistance gene *Lr24* in different wheat cultivars. *Genome* **39:** 830-835.

Frick MM, Huel R, Nykiforuk CL, Conner RL, Kuzyk A, and Laroche A, 1998. Molecular characterisation of a wheat stripe rust resistance gene in Moro wheat. In *Proceedings of the 9th International Wheat Genetics Symposium, Saskatoon, Canada,* Vol 3. (Ed: A.E. Slinkard) p181-182. (University Extension Press, University of Saskatchewan).

Gold J, Harder D, Townley-Smith F, Aung T, and Procunier J, 1999. Development of a molecular marker for rust rsistance genes Sr39 and Lr35 in wheat breeding lines. *Electronic Journal of Biotechnology* **2:**1-6.

Gupta RB, Mukai Y, Bariana HS, and Yamamoto M, 1997. Isolation and characterization of novel 1AL.1RS and 1BL.1RS wheat-rye translocation lines carrying Sec-1 genes from Petkus and Insave rye. *Journal of Genetics &. Breeding* **51:** 217-223.

Hare RA, and McIntosh RA, 1979. Genetic and cytogenetic studies of durable adult-plant resistances in 'Hope' and related cultivars to wheat rusts. *Zeitschrift für Pflanzenzüchtung* **83:** 350-367.

Helguera M,. Khan IA, Dubcovsky J, 2000. Development of PCR markers for the wheat leaf rust resistance gene Lr47. *Theoretical & Applied Genetics* **100:** 1137-1143.

Hu CC, and Roelfs AP, 1986. Postulation of genes for stem rust in 13 Chinese wheat cultivars. *Cereal Rusts Bulletin* **14:** 68-74.

Johnston SJ, Sharp PJ, McIntosh RA, Guillen-Andrade H, Singh RP and Khairallah M, 1998. Molecular markers for the Sr2 stem rust resistance gene. In *Proceedings of the 9th International Wheat Genetics Symposium, Saskatoon, Canada,* Vol 3. (Ed: A.E. Slinkard) p181-182. (University Extension Press, University of Saskatchewan).

Kerber ER, and Dyck PL, 1990. Transfer to hexaploid wheat of linked genes for adult-plant leaf rust and seedling stem rust resistance from an amphiploid of Aegilops speltoides X *Triticum monococcum*. *Genome* **33:** 530-537.

Ma J, Zhou R, Dong Y, Wang L, Wang X, and Jia J, 2001. Molecular mapping and detection of the yellow rust resistance gene *Yr26* in wheat transferred from *Triticum turgidum* L. using microsatellite markers. *Euphytica* **120:** 219-226.

McIntosh RA, 1977. Nature of induced mutations affecting disease resistance in wheat. *Induced Mutations against Plant Disease I.A.E.A., Vienna.* 551-565.

McIntosh RA, 1992. Close genetic linkage of genes conferring adult-plant resistance to leaf rust and stripe rust in wheat. Plant *Pathology* **41:** 523-527.

McIntosh RA, Dyck PL, and Green GJ, 1977. Inheritance of leaf rust and stem rust resistance in wheat cultivars Agent and Agatha. *Australian Journal of Agricultural Research* **28:** 37-45.

Metzger RJ, and Silbaugh BA, 1970. Inheritance of resistance to stripe rust and its association with glume colour in *Triticum aestivum* L. 'P.I. 178383'. *Crop Science* **10:** 567-568.

Payne PI, Holt LM, Johnson R, and Snape JW, 1986. Linkage mapping of four gene loci, *Glu-B1, Gli-B1, Rg1 and Yr10* on chromosome 1B of bread wheat. *Genetica Agraria* **40:** 231-242.

Robert O, Abelard C, and Dedryver F, 1999. Identification of molecular markers for the detection of the yellow rust resistance gene *Yr17* in wheat. *Molecular Breeding* **5:** 167-175.

Rogowsky PM, Shepherd KW, and Langridge P, 1992. Polymerase chain reaction based mapping of rye involving repeated DNA sequences. *Genome.* **35:** 4, 621-626.

Seah S, Bariana HS, Jahier J, Sivasithamparam K, and Lagudah ES, 2001. The introgressed segment carrying rust resistance genes *Yr17, Lr37* and *Sr38* in wheat can be assayed by a cloned disease resistance gene-like sequence. *Theoretical & Applied Genetics* **102:** 600-605.

Singh RP, 1992a. Association between gene *Lr34* for leaf rust resistance and leaf tip necrosis in wheat. *Crop Science* **32:** 874-878.

Singh RP, 1992b. Genetic association of leaf rust resistance gene *Lr34* with adult plant resistance to stripe rust in bread wheat. *Phytopathology* **82:** 835.

Conventional Genetic and QTL Analyses of Durable Adult Plant Stripe Rust Resistance in Wheat

H.S. Bariana, N. Ahmed, J.A. Bell, G.E. Standen, K. Kaur

The University of Sydney Plant Breeding Institute, Cobbitty, Australia.

Introduction

Durability of resistance has been reported to be due to combinations of resistance genes with additive effects. It has been argued that non-durable resistances could also provide long-term protection, provided the pathogen population is kept low through the release of resistant cultivars. Such strategy decreases the rate of pathotypic evolution. Many studies on genetics of adult plant stripe rust resistance demonstrated that acceptable levels of adult plant resistance to stripe rust is controlled by at least two or more genes (Wallwork and Johnson 1984, Bariana and McIntosh 1995, Afshari 2000, Gosal 2000, Singh *et al.* 2000).

In the present study doubled haploid (DH) populations CD87/Katepwa (CD/K) and Sunco/Tasman (S/T), produced by the National Wheat Molecular Marker Program co-operators to study a number of agronomically important traits, were screened for resistance to stripe rust. Genetic association analysis of phenotypic responses and DNA markers was performed to locate the adult plant resistance in CD/K and a temperature sensitive stripe rust resistance gene in S/T populations.

Materials and Methods

Pathogen

Puccinia striiformis f.sp. *tritici* pathotype 110 E143A+ was used to screen CD/K and S/T populations.

Host

176 DH lines derived from CD87/K.
171 DH lines derived from S/T.

Field plots

The CD/K population was planted as 60cm rows. Parents and the susceptible genotype Avocet S were used as controls in each experiment. Infector rows of the susceptible cultivar were planted after two sets of experimental rows.
Normal recommended rates of fertilizer were applied and plots were irrigated as required.

Creation of epidemic and disease assessments

An artificial epidemic of stripe rust was created using pathotype 110 E143A+. Inoculation was made at the tillering stage. Inoculum from early sown infector rows also facilitated the disease development. Disease severity (percent leaf area infected) assessments were recorded starting from boot stage onwards. The scores were made three times with final scoring after anthesis.

Seedling tests

Sowing and management

The S/T population was sown as four clumps of 10 seeds each in 9cm pots containing mixture of sand and pine bark mulch. Water soluble fertilizer Aquasol was applied before sowing and a single application of Nitram at a recommended rate was administered when the seedlings were 7 days old.

Inoculation and scoring

Seedlings were inoculated at the two-leaf stage by spraying an immersion of pathotype 110 E141A+ spores in light mineral oil. They were incubated at 9-12°C for twenty four hours under high humidity according to Bariana and McIntosh (1993). The inoculated material was moved into temperature-controlled greenhouse. Because Sunco carries a temperature sensitive gene, the temperature was set at 19±2°C instead of normal temperature (17±2°C) for stripe rust incubation. Infection type assessments were made 14 days after inoculation on a 0 - 4 scale.

Table 1. List of observed like lihood ration statistics values along with values for significant and highly significant associations.

Population/Trait-Chromosome	LRS value (analysed)	LRS value (significant)	LRS value (Highly significant)
CD/K *Yr18* -7D	27.4	13.2	24.1
CD/K *Yr29* -1BL	14.3	13.2	24.1
CD/K *YrKat* - 2DS	12.9	13.2	24.1
S/T *YrCK* - 2D	15.8	11.9	17.0

Statistical analyses

Chi-square analyses were performed to test the goodness of fit of the observed segregation to the expected genetic ratios. Interval mapping was used to detect association of phenotypic responses and DNA markers (Manly 1998a, 1998b). Permutation tests (Doerge and Churchill 1996) were used to define significance levels of particular regression statistics for an association of interest. Table 1 lists

the values for observed likelihood ratio statistics (LRS) and the threshold values for significance.

Results and Discussion

CD/K

Genetic analysis

Both parental lines, CD87 and Katepwa showed resistance to stripe rust. CD87 exhibited higher levels of resistance (10R) than Katepwa, which displayed moderate levels of resistance (40MR). Both lines were susceptible to 110 E143A+ in the seedling stage.

Tests on the CD/K population showed segregation for adult plant stripe rust response (Table 2). The presence of susceptible segregates indicated the genetic independence of resistance in CD87 and Katepwa. Chi-square analysis of pooled stripe rust response data for non-susceptible categories (5-80) versus susceptible (90-100) showed an excellent fit for segregation (167 resistant: 9 susceptible, $\chi^2_{15:1} = 0.39$, non significant at 1d.f. and 5% level of significance) at four independent loci. The segregation was also a good fit for 5 genes ($\chi^2_{31:1} = 2.3$, non significant at 1d.f. and 5% level of significance). Based on parental responses it appeared that CD87 contributed two or three genes and Katepwa contributed at least a single gene or alternatively genes from CD87 imparted larger effects.

Table 2. Distribution of scores for % leaf area affected by stripe rust in 176 adult DH lines from CD87/Katepwa.

Response level (%)	Frequency (No. of lines)
5	13
10	47
20	34
30	21
40	15
50	13
60	9
70	9
80	6
90	5
100	4

QTL analysis

Molecular marker data on the CD/K population generated by the NWMMP co-operators (Chalmers *et al.* 2001) were used to determine genetic associations of genomic regions contributing towards low stripe rust response. Three QTL regions were detected. Chromosome arm 7DS of CD87 contributed a highly

significant stripe rust severity reducing genomic region with likelihood ratio statistics (LRS) of 27.5 and this region was flanked by *P36/M41-2* and *Xwmc405b loci*. This QTL explained 15% of the phenotypic variation for adult plant stripe rust response.

Another significant genomic region responsible for reducing stripe rust severity (9%) was located on chromosome arm 1BL of CD87. Closely flanking 1BL loci included *Xpsr305*, *P39/M38-2*, *P36/M36-1* and *P32/M32-1*. Chromosome arm 2DS of Katepwa showed association with low stripe rust response and contributed 12% towards reducing stripe rust severity. The LRS value of 12.9 was slightly less than the significant value (13.2), presumably due to location of the QTL in a poorly defined chromosomal region. This QTL was flanked by microsatellite markers *wmc11* and *wmc025a*.

Table 3. Infection types produced by cultivars Sunco and Tasman and susceptible control Morocco when incubated at different temperatures.

Genotype	Infection type (17 C)	Infection type (19C)
Sunco	3+	;N to ;N12
Tasman	3+	3+
Morocco (susceptible)	3+	3+

Mapping of a temperature sensitive stripe rust resistance gene in the S/T population

A DH population derived from Sunco/Tasman cross was screened for a temperature sensitive stripe rust resistance gene. This gene does not express at normal temperature for stripe rust incubation. Table 3 represent infection types produced by parents Sunco and Tasman when incubated at two temperature regimes. Stripe rust tests on seedlings of DH population showed a monogenic segregation (86 resistant : 87 susceptible, $\chi^2_{1:1} = 0.01$, non significant at 1 d.f and 5% level of significance) for stripe rust resistance. Comparative analysis of stripe rust phenotype and molecular marker data located the temperature sensitive resistance carried by cultivar Sunco in chromosome 2D.

Discussion

Estimation of the number of genes controlling adult plant resistance to stripe rust in the CD87/Katepwa cross was higher (4-5 genes) in conventional genetic analysis based on phenotypic segregation than those indicated by QTL mapping (3 genes). This is due to the fact that conventional genetic analysis considered total phenotypic variation, whereas QTL mapping was dependent on the extent of available molecular marker information. In the present study the marker

information for certain chromosomes was very scarce. The genomic regions explaining phenotypic variation for adult plant stripe rust response, contributed by CD87 on chromosome 1BL and 7D, could be due to stripe rust resistance genes *Yr29* and *Yr18*, respectively, located on these chromosomes (Singh 1992a, Singh 1992b). The derivation of CD87 from a Pavon *(Yr29)*/Condor *(Yr18)* cross confirms above conclusion.

Chromosome 2DS of Katepwa also contributed in reducing stripe rust severity. The resistance locus was tentatively designated as *YrKat*. Similarly the temperature sensitive resistance from cultivar Sunco, tentative designated *YrCK*, was located in chromosome 2D near the centromere. The precise location of *YrCK* could not be determined because of limited marker information for chromosome 2D. Worland and Law (1986) located adult plant stripe rust resistance gene *Yr16* in chromosome 2DL. No clear pedigree information is known to explain relationship among these three sources of stripe rust resistance. Sunco derived *YrCK* from cultivar Cook which in turn was derived from WW15 based CIMMYT material introduced to Australia early in the last century. It is likely that one of the genes for adult plant stripe rust resistance in CIMMYT wheats may be *YrCK*. Durably resistant cultivar Cook carries at least 3 genes for adult plant resistance to stripe rust and two of those are *Yr18* and *YrCK*. Stripe rust resistance of cultivar Cook has remained effective in Australia for over 20 years. It appears that *YrCK* is a component of durable resistance to stripe rust displayed by cultivar Cook and its derivatives.

Acknowledgements

The National Cereal Rust Control Program and National Wheat Molecular Marker Program are funded by GRDC, Australia. We thank Dr R. Appels for statistical analyses.

References

Afshari F, 2000. Studies on rust resistance in wheat with particular emphasis on stripe rust. *Ph.D. Thesis*, University of Sydney.

Bariana HS and McIntosh RA, 1993. Cytogenetic studies in wheat XV. Location of rust resistance genes in VPM1 and their genetic linkage with other resistance genes in chromosome 2A. *Genome* **36**:476-482.

Bariana HS, and McIntosh RA, 1995. Genetics of adult-plant stripe rust resistance in four Australian wheats and the French cultivar 'Hybride-de-Bersée'. *Plant Breeding* **114:** 485-491.

Chalmers KJ, Campbell AW, Kretschmer J, Karakousis A, Henschke PH, Pierens S, Harker N, Pallotta M, Cornish GB, Shariflou MR, Rampling L, McLauchlan A, Daggard G, Sharp P, Holton T, Sutherland MW, Appels R, and Langridge P, 2001. Construction of three linkage maps in bread wheat *Triticum aestivum. Australian Journal of Agricultural Research* (in press).

Doerge RW, and Churchill GA, 1996. Permutation tests for multiple loci affecting a quantitative character. *Genetics* **142:** 285-294.

Gosal KS, 2000. Aspects of stripe rust resistance in wheat. *Ph.D. Thesis,* University of Sydney, Australia.

Manly KF, 1998a. User's manual for Map Manager Classic and Map Manager QT. http://mcbio.med.buffalo.edu/MMM/MMM.HTML.

Manly KF, 1998b. User's manual for Map Manager XT. http://mcbio.med.buffalo.edu/MMM/MMM.HTML.

Park RF, Ash GJ, Rees RG, 1992. Effects of temperature on the response of some Australian wheat cultivars to *Puccinia striiformis* f.sp *tritici. Mycological Research* **96:** 166-170.

Singh RP, 1992a. Association between gene *Lr34* for leaf rust resistance and leaf tip necrosis in wheat. *Crop Science* **32:** 874-878.

Singh RP, 1992b. Genetic association of leaf rust resistance gene *Lr34* with adult plant resistance to stripe rust in bread wheat. *Phytopathology* **82:** 835-838.

Singh RP, Huerta-Espino J, Rajaram S, 2000. Achieving near-immunity to leaf and stripe rusts in wheat by combining slow rusting resistance genes. *Acta Phytopathologica et Entomologica Hungarica* **35** (1-4), 133-139.

Wallwork H, and Johnson R, 1984. Transgressive segregation for resistance to yellow rust in wheat. *Euphytica* **33:** 123-132.

Worland AJ, and Law CN, 1986. Genetic analysis of chromosome 2D of wheat I. The location of genes affecting height, day-length insensitivity, hybrid dwarfism and yellow-rust resistance. *Zeitschrift für Pflanzenzüchtung* **96:** 331-345.

Future Approaches in Biotechnology to Wheat Rust Breeding at ICARDA

M. Baum, I. Eujayl, A. Yahyaoui and A. Osman

International Center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, Syria

Introduction

The objectives of the joint Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT)/ International Center for Agricultural Research in the Dry Areas (ICARDA) spring bread wheat is to increase yield and yield stability under variable arid and semi-arid conditions. The Programs focus their research on breeding and identifying high yielding and stable parental material, which is combined with biotic and abiotic stress resistance to produce desirable genotypes.

Yellow rust caused by the fungus *Puccinia striiformis* f.sp. *tritici* is among the most serious diseases of wheat in the cool winter areas in the West Asia and North Africa (WANA) region. The pathogen exists in a range of pathotypes that are capable of overcoming current resistance in cultivars. A number of epidemics have occurred since the late 1980's in WANA, and large virulence shifts in the yellow rust populations were observed in the region. In order to address the serious threats to wheat enhancement programs in WANA, the following biotechnological tools are to be used at ICARDA in collaboration with the regional programs to provide solutions to Yellow rust breeding:

a) Doubled haploid breeding to incorporate new sources of yellow rust resistance
b) Applications of DNA markers to map host-plant resistance genes
c) Genetic engineering to provide alternative host plant resistance genes and biosafety

Doubled Haploid Breeding

In the agricultural systems in WANA, usually only one generation per year is grown in the winter rainfed production programs. The value of doubled haploid (DH) line production for breeders is the reduced time for the development of homozygous lines. If an efficient doubled-haploid technique can be included into the breeding program, the time to obtain homozygous populations can be drastically reduced; two to three years in the variety development program can be

saved. Regenerated haploid plants of hybrids after colchicine doubling, comprise a completely homozygous population. Additionally, recessive traits are immediately fixed in doubled haploid lines that otherwise might be seen only after selfing generations. Furthermore, with the introduction of DNA-marker technology in plant breeding for gene-tagging and genome-mapping, DH lines represent the ideal plant material for the application of this technology.

In recent years, ICARDA has adopted an anther-/isolated microspore culture system in wheat for the production of doubled haploid lines. The plant material for anther-culture is planted in greenhouses. Spikes are collected when microspores are in early- or mid-uniculate stage. Spikes are cold treated at 4°C in darkness for four days to induce androgenesis. For anther culture, anthers are cultured in petri dishes and are incubated at 28°C in the dark. After about four weeks, developing calli are collected and transferred to regeneration media. Regular transfer of calli to fresh regeneration medium maintains the possibility of green-shoot induction up to three months after first incubation. Plantlets are grown on plantlet-regeneration medium until 4 to 5 tillers developed. Tillers are cloned *in vitro* to *multiply* the initial shoot. Subsequently, plantlets are transferred to peat moss and maintained in a growth chamber. When plantlets tiller again, three to five tillers are removed from the pots and treated with colchicine.

The induction and regeneration media used for the anther culture system have been extensively tested elsewhere (Picard *et al.* 1990; Lashermes *et al.* 1991; Trottier *et al.* 1993) and represent some of the best media used for anther culture in wide range of genotypes of bread wheat. Changes in the media composition might increase the frequency of green plant production for some genotypes; however, these media should provide a good standard for a wide range of genotypes. Frequencies of 10% or more green-plant production are sufficient to generate populations of DH lines from a single cross, if sufficient donor plants are available.

The DH technique was applied to crosses introducing yellow rust resistance into adapted breeding lines. Resistant lines originate from germplasm pools that are being developed at ICARDA with resistance to rusts (stem, leave, and yellow rust) and other plant pathogens (A. Yahyaoui, personal communication.). Although these germplasm pools provide resistance against current virulent races of yellow rust, proper gene postulation studies have not been carried out so far and the identity of the resistance genes in the pools and their chromosomal location is largely unknown (A. Yahyaoui, personal communication.).

Three crosses were subjected to anther-/isolated microspore culture system: for the cross Giza 164/Wyr1-96 14 F1, for Sids 6/Wyr1-96 nine and for the cross Mexipak /Wyr-4 three F1 plants were utilised. F1 plants were grown in a plastic-house with temperature/light cycles regimes of about 20°C during the day for 16 h, 10°C during night for 8 h. Detailed description of experimental procedures can be found elsewhere. The three crosses yielded 500, 200 and 237 green plantlets respectively leading to 145, 85 and 135 fertile DH plants. With further improvement of an infrastructure for doubled haploid line production at ICARDA, an increasing number of DH lines in relation to the number of green plantlets produced and an increased efficiency of green plantlet production per individual cross subjected to the anther-/iolated microspore culture can be expected.

Table 1. Response of spring bread wheat crosses to anther-culture.

Crosses	F1- plants	Calli	Green plants	DH Lines
Giza 164/Wyr1-96*	14	9977	500	145
Sids 6 /Wyr1-96	9	4483	200	85
Mexipak /Wyr-4**	3	662	237	135

*Wyr-1: Bow'S'/Vee'S'//Bow'S'/Tsi
**Wyr-4: Gv/Ald'S'/5/Ald's'/4/Bb/glI//Cno67/7C/3/Kvz/Ti

Applications of DNA markers to map host-plant resistance genes

Numerous DNA marker systems are available for genetic mapping in wheat such as Restriction Fragment Length Polymorphism (RFLP), Amplified Fragment Length Polymorphism (AFLP) and Simple Sequence Repeat (SSR) markers and numerous genetic maps have been developed for wheat (Devos *et al.* 1993, Röder *et al.* 1998). SSR markers are ideal DNA markers for population studies and genetic mapping because of their abundance, high level of polymorphism, dispersion throughout genomes, ease of an assay by polymerase chain reaction (PCR), and ease of dissemination of the technology among laboratories.

Among the SSR markers, genomic SSR marker have been developed for wheat from enriched genomic libraries (Röder *et al.* 1995, Röder *et al.* 1998) but recently also EST-derived SSR markers are becoming available through internationally coordinated efforts (WMC, Agrogene). EST-derived microsatellites are physically associated with the coding regions of the genome and might therefore potentially be usefull for linkage to agronomic traits.

In a recent study comparing genomic SSR markers (XGWM Gatersleben Wheat Microsatellite Markers, Röder *et al.* (1998), and WMC, wheat microsatellite club, Agrogene) and EST-derived microsatellite markers (Eujayl *et al.* (2001), Du Pont microsatellite collection) for the usefullness of discriminating 64 durum wheat

genotypes the following observations could be made: Based on the screening results of 245 pairs of primers amplification of the Du Pont microsatellite collection for fragment, fragment quality and polymorphism, 42 markers were selected for fingerprinting of the 64 durum genotypes. Approximately 25% of the EST derived SSRs were polymorphic compared to over 50% for the genomic SSR markers. However, both the XGWM and WMC primers had been previously screened for informativeness in hexaploid wheat. An important feature of the EST-derived SSR markers is the high quality fragment patterns obtained which are devoid of stuttering. The clarity of the amplification profiles will certainly ease the application of EST-derived SSR markers in cultivar identification and genetic mapping. The 22 selected EST clone sequences were subjected to BLAST (Altschul *et al.* 1990), 20 showed homology with gene sequences for which the putative gene functions are known, particularly in wheat and barley. A total of 189 alleles were detected, 89, 61 and 39 alleles being detected with the EST-SSR, XGWM and WMC SSR markers respectively (Table 2). The most informative microsatellite marker was XGWM 169, which detected 14 alleles in the 64 durum genotypes studied. Null allele has been observed in three EST-SSR (DuPw123, 165 and 207) and only in one genomic SSR XGWM 149.

Table 2. Number of tested SSR loci, polymorphism percentage and the average number of alleles detected by the selected loci used for Durum genotypying.

Source of SSR	Primers tested	No amplification	% Poly-morphism	Loci selected	Alleles	Average number of alleles
EST-SSR	137	32	24.8	22	89	4.05
XGWM[1]	68	05	52.9	11	61	5.54
WMC[2]	40	01	52.5	09	39	4.33
Total	245	38	35.9	42	189	

[1] = primers from Röder *et al.* (1998)

[2] = primers from wheat microsatellite consortium (Agrogene).

At ICARDA use will be made of the recent published EST-derived SSR markers of wheat (Eujayl *et al.* 2001) as well as further EST-derived SSRs from the Du Pont microsatellite collection for genetic mapping studies especially for the marker development of yellow rust resistance genes. Near isogenic lines (NILs) are available for most of the known resistance genes. However, there is a need to develop markers for the unknown resistance genes in the resistant germplasm pools as well as for the ineffective resistance genes in WANA wheat lines in order to allow an effective management of yellow rust resistance genes.

Doubled haploid populations segregating for yellow rust resistance will provide the suitable genetic material for this approach.

Genetic engineering to provide alternative host plant resistance genes and biosafety

Prior to engaging in the development of genetic engineering strategies to develop alternative host-plant resistance in wheat, ICARDA is committed to support the development of biosafety regulatory frameworks in the countries of WANA. For that purpose ICARDA, the Agricultural Genetic Engineering Research Institute (AGERI) in Cairo, Egypt, the Food and Agricultural Organizations of the United Nations (FAO), and the Syrian National Biosafety Committee (SNBC) have organised a workshop "Development and Harmonisation of Biosafety Regulations" in September 2000, in Aleppo, Syria to promote and harmonise the development of biosafety regulations in the WANA countries. The objectives of the workshop were to assess the current situation related to biosafety in WANA countries and to formulate national action plans for the development of biosafety regulations for the region. Representative from Algeria, Egypt, Jordan, Iraq, Lebanon, Morocco, Palestine, Sudan, Syria, Tunisia, and Turkey, attended the workshop and reported on the current situation of biotechnology and biosafety in their respective countries.

As for genetic engineering, strategies will be sought to engineer fungal resistance in wheat as an alternative source of resistance to control yellow rust. A number of genes and constructs inhibiting fungal growth in wheat plants such glucanase, chitinase will be tested for that purpose. Efficient transformation technologies such as the use of biolistic bombardment (Becker *et al.* 1994) are available for bread wheat. At ICARDA, there is interest in utilizing the isolated microspore culture system for wheat and barley transformation. In wheat, considerable progress has been made utilizing the isolated microspore culture system (Hu and Kasha 1997, De Buyser *et al.* 1998) where the frequency of embryo like structures is one for 2-3 % of the cultivated microspores combined with a high regeneration of green plantlets. With these efficiencies the system will also increase the chances of a successful transformation system for wheat microspores.

Acknowledgments

This research was financially supported by BMZ grant no.98.7860.4-001.00 and Arab Fund for Economic and Social Development, Kuwait. We are grateful to Dr. W. Powell, Du Pont for providing wheat microsatellite primer sequences. We would like to acknowledge the work of Ms Sawsan Tawkaz in developing

doubled haploid lines and Dr E. Picard, University Paris-Sud for his continuous support to develop the doubled haploid program at ICARDA. Special thanks to Dr. K. Wolpers, Dr. W. Kasten GTZ for their continous support.

References

Altschul SF, Gish W, Miller W, Myers EW, Lipman D, 1990 Basic local alignment search tool. Journal of *Molecular Biology*. **215:** 403-410.

Becker D, Brettschneider R, Lörz H 1994. Fertile transgenic wheat from microprojectile bombardment of scutellar tissue. *The Plant Journal* **5**: 299-307.

De Buyser J, Touraine P, Ambroise A, Picard E 1998 Induction of androgenetic embryos ans chlorophyllian plants of *Triticum aestivum* from isolated microspore culture. *Poster 9th International wheat Genetics Symposium.* Saskatoon, 2-7 August 1998.

Devos K, Gale M 1993. The genetic maps of wheat and their potential in plant breeding. *Outlook on Agriculture* **22**: 93-99

Hu T., Kasha KJ. 1997. Improvement of isolated microspore culture of wheat (*Triticum aestivum* L.) through ovary co-culture. *Plant Cell Reports* **16** : 520-525.

Eujayl I, Sorrells M, Baum M, Wolters P, Powell W. 2001. Assessment of genotypic variation among cultivated durum wheat based on EST-SSRS and genomic SSRS. *Euphytica* 119: 39-43.

Lashermes P, Engin G, Ferrara GOrtiz 1991. Anther culture of wheat (*Triticum aestivum)* adapted to dry wereas of West Asia and North Africa. *Journal of Genetics and Breeding.* **45**: 33-38.

Picard E., Röde A, Doussinault G, Rousset M, Rives M 1990. *In vitro* production and utilization of doubled haploid of wheat *(Triticum aestivum* L.). Ed. Y.P.S. Bajaj. *Biotechnology in Agriculture and Forestry.* **12**. 101-124.

Röder MS, Plaschke J, König SU, Börner A, Sorrells ME, Tanksley SD, Ganal MW 1995 Abundance, variability and chromosomal location of microsatellites in wheat. *Molecular General Genetics* **246**: 327-333.

Röder MS, Korzun V, Wendehake K, Plaschke J, Tixier M, Leroy P, Ganal M 1998 A microsatellite map of wheat. *Genetics* **149**: 2007-2023.

Trottier MC, Collin J, Comeau A 1993. Comparison of media for their aptitude in wheat anther culture. *Plant Cell, Tissue and Organ Culture* **35**: 59-67.L

Breeding and Genetic Studies of Yellow Rust Resistance in Wheat Using Doubled Haploid Lines.

A. Arzani

Department of Agronomy, College of Agriculture,
Isfahan University of Technology,
Isfahan, Iran

Introduction and Discussion

Future plant breeding must be even more efficient than in the past few decades to meet the needs of an ever-growing population. Genetic resistance is an ecologically and environmentally sound approach to disease control in crops and is a common and important objective of wheat (*Triticum aestivum* L.) breeding. Breeding for durable resistance to pathogens is a major task for modern plant breeders and in the case of yellow rust (*Puccinia striiformis* f. sp. *tritici)* in wheat, adult plant resistance and pyramiding different resistance genes into a genotype are ways of achieving this. Longer-term breeding strategies are aimed at achieving durability of resistance combined with the deployment of genotypic diversity to buffer potential losses. It has often been suggested that race-non-specific resistance to fungal pathogens such as *P. s.* f. sp. *tritici* and other diseases exists in wheat. Various workers have stressed the need to recognize and exploit such resistance for achieving durable resistance to yellow rust in wheat and associated such resistance with minor genes, polygenic inheritance, adult plant resistance and partial or incomplete resistance that reduces the infection rates (Johnson 1980, Milus and Line 1986, Qayoum and Line 1985, Sharp *et al.* 1976, Singh and Rajaram 1994, Kumar *et al.* 1999).

Information on the inheritance of resistance to yellow rust in wheats differing in levels of quantitative resistance would be useful for improving the resistance status of wheat cultivars. Doubled haploid (DH) lines represent very useful genetic materials since they are homozygous and homogeneous, and genetic effects are restricted to additive and additive epistatic variation without dominance and dominance-related epistatic effects being present. Indeed, in addition to their usefulness for conventional quantitative genetic studies, DHs provide a means of obtaining quantitative data that can be used to map resistance loci relative to molecular markers in plant genomes. Hence, quantitative trait locus (QTL) analysis may be used to detect chromosome regions that contribute to disease resistance. Although

single genes controlling yellow rust adult plant resistance (APR) have been identified (McIntosh *et al.* 1995), many sources of APR are controlled by more than one gene (Bariana and McIntosh 1995). Such sources of multigenic or quantitative resistance (quantitative trait loci - QTLs) are difficult to transfer to new cultivars in their entirety. To ensure that all the genes contributing to the APR are transferred to a new cultivar, molecular markers, closely linked to each gene, could be used to screen for the gene's presence. A DNA mapping program was initiated to find molecular markers linked to the genes contributing to the yellow rust APR in cultivar Kariega at the John Innes Centre, Norwich (Boyd 2001). A DH population from a cross between Kariega and the yellow rust susceptible cultivar Avocet S was developed. A DNA marker map of this population is being developed, using microsatellites to provide chromosome anchors, and Amplified Fragment Length Polymorphisms (AFLPs) to permit fine mapping. The disease phenotype of each DH line, obtained from the field trial, will be superimposed on the DNA molecular map using suitable statistical software for QTL analysis. This will identify DNA markers linked to the genes/QTLs contributing to the yellow rust APR seen in Kariega and locate their chromosomal position. These DNA markers provide the raw materials from which simple marker selection systems to identify the resistance genes during a wheat breeding program can be developed (Boyd 2001). These tools will allow them to transfer the complete APR found in Kariega to new wheat cultivars.

Backcrossing is used for introgressing target loci, such as stripe rust resistance QTLs, into adapted backgrounds. Molecular markers can increase the efficiency of the process in several ways. Flanking markers can be used to identify the backcross lines that are heterozygous for target genome regions. Advancing only these selected lines will also have an effect of reducing linkage drag (Tanksley and Nelson 1996). Single-copy, or low-copy, markers with defined map locations such as restricted fragment length polymorphisms (RFLPs) and simple-sequence repeats (SSR), are ideal for this step. Molecular markers could also increase the efficiency of backcrossing by allowing for the selection of genotypes with maximum percentage of the recurrent parent genome. Markers with higher information content per reaction, such as AFLPs, are ideal for this step (Waugh *et al.* 1997). DHs could be used for precise manipulation of QTLs by confirming the target phenotype in the selected progeny, since the phenotyping on a plot rather than an individual basis will be conducted (Toojinda *et al.* 1998).

Winzeler *et al.* (1987) suggested that disease resistance is more accurately and more easily assessed in DH lines as they are completely homozygous. They demonstrated that in comparison with lines selected by the pedigree method, the

DH lines were more resistant to yellow rust. Similarly, Arzani and Darvey (2001) indicated that DH lines were far more reliable materials for use of genetic host resistance. In triticale, DH lines were assessed for resistance to prevalent races of stem rust (caused by *P. graminis* f. sp. *tritici),* yellow rust (due to *P.* s. f. sp. *tritici)* and leaf rust (due to *P. recondita* f. sp. *tritici).* A DH line (DH19-4) derived from Polony Q/TW 179 (F_2) population with good performance, which was also resistant to prevalent races of leaf rust, stem rust and yellow rust pathogens (Arzani and Darvey, 1998), will be released in Australia the current year (Arzani and Darvey 2001).

DHs enable breeders to develop completely homozygous genotypes from heterozygous parents in a single generation. No other technique allows this facility and in essence, DHs are a method of fixing recombinant gametes directly as fertile homozygous lines. Indeed, in addition to their use for varietal production, these systems provide a means of producing unique experimental genotypes that are useful in a range of plant sciences impinging on plant breeding.

References

Arzani A, Darvey NL, 1998. Assessment of doubled haploids and mid-generation populations of forage and dual purpose triticale for rust resistance under Australian conditions In: P. Juskiw (Ed.) *Proceedings of 4th International Triticale Symposium.*, July 26-31, 1998, Red Deer, Alberta, Canada. pp. 164-173.

Arzani A, Darvey NL, 2001. The effect of colchicine on triticale anther-derived plants: Microspore pre-treatment and haploid-plant treatment using a hydroponic recovery system. *Euphytica* **118** (in press).

Bariana HS, McIntosh RA,1995. Genetics of adult plant stripe rust resistance in four Australian wheats and the French cultivars 'Hybride-de-Bersee'. *Plant Breeding* **114:** 485-491.

Boyd LA, 2001. Wheat Resistance to South African *Puccinia striiformis* f.sp. *tritici* Pathotypes. The Newsletter of the British Society for Plant Pathology 38: online edition (http://www.bspp.org.uk/bsppnews/bspp-news38/bsppnews38-2.html)

Johnson R, 1980. Genetics of adult plant resistance to yellow rust in winter wheat cultivars. *Proceedings of the 5th European and Mediterranean Cereal Rusts Conference* pp. 59-63.

Kumar J, Singh RP, Nagarajan S, Sharma AK, 1999. Further evidences on the usefulness of *Lr34/Yr18* gene in developing adult plant resistant wheat genotypes. *Wheat Information Service* **89**: 23-29.

Milus EA, Line RF,1986. Gene action for inheritance of durable, high temperature adult plant resistance to stripe rust in wheat. *Phytopathology* **76**: 435-441.

McIntosh, R.A., C.R. Wellings and R.F. Park (1995) Wheat rusts: an atlas of resistance genes. CSIRO, Sydney, Australia. pp.200.

Qayoum A, Line RF, 1985. High temperature, adult plant resistance to stripe rust of wheat. *Phytopathology* **75**: 1121-1125.

Sharp EL, Sally BK, Taylor GA, 1976. Incorporation of additive genes for stripe rust resistance in winter wheat. *Phytopathology* **66:** 794-797.

Singh RP, Rajaram S, 1994. Genetics of adult plant resistance to stripe rust in ten spring bread wheats. *Euphytica* **72**: 1-7.

Tanksley, S.D. and J.C. Nelson (1996) Advanced backcross QTL analysis: a method for the simultaneous discovery and transfer of valuable QTLs from adapted germplasm into elite breeding lines. *Theoretical and Applied Genetics* **92**: 191-203.

Toojinda T, Baird E, Booth A, Broers LHM, Hayes P, Powell W, Thomas W, Vivar HE, Young G, 1998. Introgression of quantitative trait loci (QTLs) determining stripe rust resistance in barley: An example of marker-assisted line development. *Theoretical and Applied Genetics* **96** : 123-131.

Waugh R, Bonar N, Baird E, Hayes P, Graner A, Thomas WTB, Powell W, 1997. Homology of AFLP products in three mapping populations of barley. *Molecular General Genetics* **225:** 311-321.

Molecular Markers in Wheat Yellow Rust: I. Genetic Diversity and Discussion of Their Usefulness in Survey Studies

M. S. Hovmöller and A. F. Justesen

Research Centre Flakkebjerg, Danish Institute of Agricultural Sciences, Denmark

Introduction

The amount of molecular- and isozyme-variation in *Puccinia striiformis* f.sp. *tritici*, which causes yellow rust on wheat, is low compared with that of other pathogenic fungi on agricultural crops. A British study in the 1980s of 19 isozymes did not reveal any polymorphism, even for isolates of very different geographical origin and virulence pattern (Newton and Caten 1985), and only minor differences were subsequently observed for dsRNA (Jennings *et al.* 1997). A Chinese RFLP-study using a single moderately repetitive probe (Shan et al. 1998), and a North American RAPD-study revealed molecular variation at a higher level (Chen *et al.* 1993); however, in both cases, isolates of very different origin were analysed. A recent Australian study did not reveal any RAPD or AFLP variation among isolates of Australian origin (Steele *et al.* 2001).

In 1996 a study was begun in which Amplified Fragment Length Polymorphism (AFLP) was applied to yellow rust isolates of Danish and NW-European origin. The first aim was to implement the AFLP-technique for the small quantities of spore samples that were at our disposal and improve the yield and quality of the DNA as compared with previous studies. The second aim was to detect sufficient molecular variation at the scale of isolates ranging from within single fields to within NW-European countries, and collected within recent years, *i.e.* the level of relevance for a population study. Today, these goals have been achieved to the extent that a set of AFLP markers is now available that permit investigation of yellow rust pathways (migration) and recombination (or lack of) in the NW-European area (Justesen *et al.* 2001, Hovmöller *et al.* 2001). In these two studies, more than 15,000 AFLP fragments were screened for polymorphism, resulting in 28 useful AFLP markers. Based on more than 100 isolates of recent origin in the area, it was observed that at maximum 0.5% of the AFLP fragments revealed polymorphism between any pair of isolates. When comparing isolates of very different origin (in time and space) considerably higher levels of polymorphism may be obtained.

Molecular markers in virulence survey studies

The usefulness of molecular markers in survey studies depends on the following issues: 1) survey aims; 2) association or linkage between molecular markers and specific phenotypic traits, e.g. individual virulence characters or specific combinations of these (pathotypes); 3) utility of markers in relation to survey aims; 4) technology (facilities and education) and costs of alternative methods.

In most virulence survey studies in NW-Europe, the principal aim is to detect new pathotypes of agronomic importance as soon as possible after they appear in the survey area (e.g. Bayles *et al.* 2000). Such new pathotypes may combine already known virulences into new combinations or they may possess a virulence character that has not been detected previously in the area. The use of molecular markers in such survey studies requires that the markers associate to the virulence characters considered or to the specific clones in which they appear.

The dataset of Hovmöller *et al.* (2001) comprise 100 isolates of NW-European origin collected between 1988 and 1998. These isolates were grouped into 15 pathotypes and 21 AFLP phenotypes; all isolates carried virulence for *Yr3* (present in the differentials Vilmorin 23 and Nord Desprez) and Strubes Dickkopf, and they were avirulent on the standard differential varieties possessing *Yr5, Yr7, Yr8* or *Yr10.* A preliminary analysis showed that:

- in one case only, a unique pathotype was exclusively a member of a unique clonal lineage, i.e. a unique pathotype was most often represented in more than a single AFLP-phenotype (clonal lineage) OR a unique AFLP-phenotype harboured more than a single pathotype.
- in three cases, a single AFLP fragment was associated with a particular clonal lineage; two of these groups had only a single isolate each, and the third group consisted of several isolates from distant areas and years.
- two independent AFLP-markers displayed a complete association with the group of 8 isolates carrying virulence for *Yr1.*
- no single AFLP fragment was associated with virulence for *Yr2, Yr4, Yr6, Yr9, Yr17 or Yr(CV).*

Conclusions

The results illustrate that it may be possible to find molecular markers that associate to a specific group of isolates, either sharing a single virulence character or being a member of a specific clone. However, it may require a very intensive search, and the markers detected may only apply to the population (or area) in

which they were developed. Further, a true genetic linkage between a specific DNA-fragment (marker) and a specific virulence character cannot be confirmed since no mapping tools are available in the wheat yellow rust fungus (entirely asexually reproduced). Finally, since most molecular markers are expensive to develop and require well-equipped laboratories, they may not be appropriate, for economic reasons, in routine surveys. Probably just as important in relation to surveys, molecular markers may tend to be conservative due to an inherent focus on already known virulence characters, whereas virulence matching the most recently-implemented sources of resistance will be the most relevant feature in most survey programmes. Therefore, there are several explanations why molecular markers are unlikely to replace successfully the traditional virulence surveys in which the virulence characters can be assayed directly on the most relevant plant genotypes. In contrast, molecular markers have proven very useful in migration studies, as demonstrated by the recent study of Hovmöller *et al.* (2001) where the spread of wheat yellow rust clones across the whole of North-west Europe was investigated.

References

Bayles RA, Flath K, Hovmöller MS, de Vallavieille-Pope C, 2000. Breakdown of the Yr17 resistance to yellow rust of wheat in northern Europe - a case study by the yellow rust sub-group of COST 817 . *Agronomie* **20**: 805-811.

Chen X, Line RF, Leung H, 1993. Relationship between virulence variation and DNA polymorphism in *Puccinia striiformis*. *Phytopathology* **83,** 1489-1497.

Hovmöller MS, Justesen AF, Brown JKM, 2001. Clonality and long-distance migration of *Puccinia striiformis* f.sp. *tritici* in NW-Europe. *Plant Pathology* (in press).

Jennings JM, Newton AC, Buck KW, 1997. Detection of polymorphism in *Puccinia hordei* using RFLP and RAPD markers, differential cultivars, and analysis of the Intergenic Spacer Region of rDNA. *Phytopathology* **145:** 511-519.

Justesen AFJ, Ridout CJ, Hovmöller MS, 2001. The recent history of *Puccinia striiformis* f.sp. *tritici* in Denmark as revealed by disease incidence and AFLP fingerprints. *Plant Pathology* (in press).

Newton AC, Caten CE, 1985. Variation for isozymes and double-stranded RNA among isolates of Puccinia striiformis and two other cereal rusts. *Plant Pathology* **34:** 235-247.

Shan W-X, Kang Z-S, Li Z-Q, 1998. Genetic diversity in *Puccinia striiformis* Westend. f.sp. tritici revealed by pathogen genome-specific repetitive sequence. *Canadian Journal of Botany* **76:** 587-95.

Steele KA, Humphreys E, Wellings CR and Dickinson MJ, 2001. Support for a stepwise mutation model for pathogen evolution in Australasian *Puccinia striiformis* f.sp. *tritici* by the use of molecular markers. *Plant Pathology* **50:** 174-180.

Molecular Markers in Wheat Yellow Rust: II. Long-Distance Spread of Clones in Northwest Europe

M. S. Hovmöller and A. F. Justesen

Research Centre Flakkebjerg, Danish Institute of Agricultural Sciences, Denmark

Introduction

It is well established that spores of plant pathogens may be spread over long distances by wind, plant products or man, thereby causing the spread of disease epidemics across large areas. Long-distance spore dispersal may take place in a single step or it may proceed through several successive steps. The former situation was the case when live spores of the fungi that cause powdery mildew and brown rust of barley and wheat were wind-transported for at least 600 km across the North Sea, between the UK and Denmark (Hermansen *et al.* 1978). The spread of wheat yellow rust epidemics in Europe in the late 1950s was most probably a result of several successive and individual dispersal events (Zadoks 1961). The emergence of new molecular techniques for fingerprinting individual isolates has greatly improved the possibility to detect migration in plant pathogen populations (e.g. Milgroom 1997).

A recent study comprised data, based on several years of yellow rust disease observations, and pathotype and AFLP phenotype dynamics in Denmark (Hovmöller 2001, Justesen *et al.* 2001). The latter study was extended to include AFLP phenotype diversity in three other NW-European countries, the UK, France and Germany (Hovmöller *et al.* 2001). The possible reintroduction of yellow rust into Denmark in 1997 was examined, after it was not found under natural conditions in 1996. This was associated with the spread in Denmark and NW-Europe of two pathotypes having the newly discovered virulence for *Yr17*. For some of the *Yr17*-virulent pathotypes it was found that isolates with identical AFLP-fingerprints were observed in all four countries, indicating a common origin of such isolates. Another example where pathogen clones had evidently been spread between the UK and Denmark was a *Yr9*-virulent clone, which caused *Yr9* resistance to become ineffective for yellow rust control in Denmark from 1988 and onwards. Virulence for *Yr9* was observed in the UK in the early 1980s and possibly also in the mid-1970s. Another example was a clone combining virulence for *Yr1* and for the cultivar Carstens V, first found in the UK. This clone affected, among others, the variety Kraka (Yr1, CV) which was grown at up to 80% of the Danish wheat area in the 1980s.

These examples demonstrate that long-distance migration of pathogen clones may cause the spread of virulence for the same resistance genes in different countries. The results emphasise the importance of maintaining diverse sources of resistance to important diseases across wide geographical areas, in order to limit the damage caused by long-distance dispersal of yellow rust clones.

References

Hermansen JE, Torp U, Prahm LP, 1978. Studies of transport of live spores of cereal mildew and rust fungi across the North Sea. *Grana* **17**: 41-46.

Hovmöller MS, 2001. Disease severity and pathotype dynamics of *Puccinia striiformis* f.sp. *tritici* in Denmark. *Plant Pathology* **50:** 181-189.

Hovmöller MS, Justesen AF, Brown JKM, 2001. Clonality and long-distance migration of *Puccinia striiformis* f.sp. *tritici* in NW-Europe. *Plant Pathology* (in press).

Justesen AFJ, Ridout CJ, Hovmöller MS, 2001. The recent history of *Puccinia striiformis* f.sp. *tritici* in Denmark as revealed by disease incidence and AFLP fingerprints. *Plant Pathology* (in press).

Milgroom MG, 1997. Genetic variation and the application of genetic markers for studying plant pathogen populations. *Journal of Plant Pathology* **78:** 1-13.

Zadoks JC, 1961. Yellow rust on wheat: studies in epidemiology and physiologic specialisation. *Tijdschrift over Planteziekten* **67:** 69-256.

Induced Transfer of Rust Resistance from *Aegilops* Species into Wheat and Characterisation of the Derivates Using Microsatellite Markers

M. Aghaee-Sarbarzeh[1], H.S. Dhaliwal[2]

[1] Dryland Agricultural Research Institute, Sararood, Kermanshah, Iran;
[2]Department of Biotechnology, Punjab Agricultural University, Ludhiana, India

Introduction

Among the wheat foliar diseases stripe rust (caused by *P. striiformis* Westend f.sp. *tritici)* and leaf rust (caused by *Puccinia recondita* Rob. ex Desm. f.sp. *tritici)* are the most damaging diseases of wheat (Knott 1989, McIntosh *et al.* 1995). During the last 3-4 decades a number of resistance genes have been transferred into cultivated wheat, but most of them have become ineffective due to emergence of new rust pathotypes thereby necessitating continuous search for new sources of resistance.

Related species of wheat, including both distantly related and progenitor species, represent a large reservoir of useful variability including rust resistance (Jiang *et al.* 1994; Friebe *et al.* 1996). Although a large number of transfers carrying useful alien genes have been produced, very few have been exploited commercially. Most alien segments either do not compensate well for the loss of wheat chromatin or contain undesirable genes causing depression in yield (Knott 1993, Jiang *et al.* 1994). This undesirable effect can be avoided by transferring the desired gene(s) without most of the unwanted alien chromatin.

Alien introgression has been facilitated by induced homoeologous chromosome pairing between wheat and alien chromosomes, and by identification of the alien chromatin in the recipient progenies by molecular cytogenetic techniques (Heslop-Harrison *et al.* 1990, Mukai and Gill 1991).

Recent studies have shown that the *Ph[1]* gene(s) from Ae. speltoides in the background of *T. aestivum* (Chen *et al.* 1994, Jiang *et al.* 1994) is useful in inducing pairing between homoeologous chromosomes in intergeneric crosses of Triticeae (Aghaee-Sarbarzeh *et al.* 2000) facilitating precise transfer of alien genes with least linkage drag.

Work at the Punjab Agricultural University has revealed that non-progenitor *Aegilops* species with C, U, and M genomes are good sources of resistance to stripe rust and leaf rust (Dhaliwal *et al.* 1993, Harjit-Singh *et al.* 1998). The aim of this study, therefore, was to transfer rust resistance from these species into wheat with the use of the *Ph*^{I} gene system.

Materials and Methods

Plant materials used in this investigation are listed in Table 1. Amphiploids were synthesized following the procedure used by Gill *et al.* (1988) after crossing susceptible *T. durum* cultivars as female parents with stripe rust and leaf rust resistant accessions of *Aegilops umbellulata* (Acc. 3732 and 13749) and *Ae. caudata* (Acc. 3556). These amphiploids were used as the bridge for the transfer of resistance genes into cultivated bread wheat.

Table 1. *Triticum durum* and *T. aestivum* cultivars, resistant accessions of wild species, and amphiploids used in this study.

Material	2n	Genomic Formula
Triticum durum		
cv. WH890	28	AABB
cv. A206	28	AABB
cv. WH868	28	AABB
cv. Bijaga Yellow	28	AABB
cv. Malvi Local	28	AABB
T. aestivum		
cv. Chinese Spring carrying *Ph*^{I} gene	42	AABBDD
cv. Chinese Spring	42	AABBDD
cv. WL711	42	AABBDD
Aegilops umbellulata		
Acc. 3732	14	UU
Acc. 13749	14	UU
Aegilops caudata Acc. 3556	14	CC
Amphiploids		
T. durum cv. WH890-Ae. *umbellulata* Acc. 3732	42	AABBUU
T. durum cv. Bijaga Yellow-Ae. *umbellulata* Acc. 13749	42	AABBUU
T. durum cv. Malvi Local-Ae. *umbellulata* Acc. 13749	42	AABBUU
T. durum cv. A206-Ae. *caudata* Acc. 3556	42	AABBCC
T. durum cv. WH868- Ae. *caudata* Acc. 3556	42	AABBCC

To induce homoeologous pairing, the amphiploids were first crossed with CS(*Ph*^{I}) and further backcrossed to either Chinese Spring (CS) or *T. aestivum* cv. WL711. Seeds of CS(*Ph*^{I}) were obtained from Dr. Bikram S. Gill of Kansas State University, USA.

The parents, F_1s, amphiploids, and the derivatives of crosses with CS*(Ph^I)* were scored for field and seedling reaction to stripe rust and leaf rust following standard procedures.

To identify possible wheat chromosome arms involved in translocation with alien chromosomes in the resistant derivatives, a set of 40 microsatellite markers (one marker per chromosome arm) was selected based on the linkage map developed by Roder *et al.* (1998) (Table 2). PCR was performed and the products were separated on a 3% high-resolution agarose gel. Ethidum bromide stained gels were visualized under UV light and photographed using UVP Gel Documentation System Model GDS 7600.

Results and Discussion

Aegilops caudata Acc. 3556 as well as *Ae. umbelulata* Acc. 3732 and Acc. 13749 exhibited consistent resistance to stripe rust and leaf rust under field conditions for many years. They were also resistant to individual pathotypes of stripe rust and leaf rust at seedling stage (Dhaliwal *et al.* 1993).

Table 2. Wheat microsatellite markers used for characterization of some of the resistant derivatives of crosses of amphiploids x CS*(Ph^I)*

Genome Location	STMS markers (gwm #)	Total
A	666, 136, 312, 359, 155, 369, 397, 601, 617, 304, 570, 334, 63, 60	14
B	140, 582, 388, 257, 340, 285, 251, 368, 335, 234, 219, 508, 577, 537	14
D	642, 33, 382, 261, 645, 114, 624, 583, 190, 469, 428, 350	12

Resistant derivatives were selected and studied cytologically (Table 3, and 4). These plants were partially to nearly fertile having 40 to 44 chromosomes. Although, some plants with 21 bivalents were also obtained (e.g. plant no.16, W1087-6, Table 4) additional backcrossing was, however, needed to restore chromosome balance, increase the fertility level and remove unwanted alien chromatin.

Table 3. Reaction of parental lines, *Aegilops species*, F_1s and amphiploids of *T. durum* with *Aegilops* species to leaf rust and stripe rust under field conditions and to individual pathotypes at seedling stage.

Materials	Rust reaction							
	Field		Individual pathotype+ Leaf rust				Stripe Rust	
	Leaf rust	Stripe rust	77-2	77-4	77-5	104B	104-2	P
T. aestivum cv. Chinese Spring	20S	60S	3	3	3+	3+	3+	3
T. aestivum cv. Chinese Spring (Ph^I)	20S	60S	3	3	3+	3+	3+	3
T. durum cv. WH890	20S	40S	3-3+	3+	2	3+	3+	3
Ae. umbellulata Acc. 3732	F	F	0;	0;	0	0;	0;	;
F_1 [T. durum-Ae. umbellulata]	F	F	-	-	-	-	-	-
Amphiploid [T. durum-Ae. umbellulata]	F	F	3	3+	3+	3+	3	3
T. durum cv. Bijaga Yellow	20S	60S	0;	0;	2-	3	3	;
Ae. umbellulata Acc. 13749	F	F	0;	0;	0;	0;	0;	;
Amphiploid [T. durum-Ae. umbellulata]	20S	60S	3	2-	3+	0;	0;	;
T. durum cv. Malvi Local	90S	40S	3	3	3+	0;	3	3
Ae. umbellulata Acc. 13749	F	F	0	0;	0;	0;	0;	;
Amphiploid [T. durum-Ae. umbellulata]	5S	60S	3	2-	3+	0;	3	3
T. durum cv. A206	90S	40S	4-4	3+-4-	0;	0;	3	3
Ae. caudata Acc. 3556	F	F	0;	0	0	-	-	-
F_1 [T. durum- Ae. caudata]	-	-	-	-	-	-	-	-
Amphiploid [T. durum- Ae. caudata]	80S	5S	3+	3	3+	3	3	3
T. durum cv. WH868	40S	40S	3++	X+	0;	0;	3	3
Ae. caudata Acc. 3556	F	F	0;	0	0	-	-	-
F1 [T. durum- Ae. caudata]	tS	20S	-	-	-	-	-	-
Amphiploid [T. durum- Ae. caudata]	60S	60S	0-4-	3	2-3	3	3	3

+ 0-2:Resistant, 3-4:Susceptible; X: Mesothetic, F: Free, tS: Trace of susceptibility, S: Susceptible

The results of molecular characterization, of some of the derivatives of CS*(Ph^I)* x amphiploid [*T. durum* cv. WH890 -*Ae. umbellulata* Acc. 3732] crosses indicated that out of 40 STMS markers examined, 23 (57.5%) amplified in *Ae. umbellulata* Acc. 3732. The amplification patterns of STMS markers revealed several cases in which wheat chromosome arms were missing or replaced by their homoeologous segments from *Ae. umbellulata* (Fig 1a-1d). The STMS markers used for chromosome 1A (gwm 666 for the long arm and gwm 136 for the short arm) showed that

the gwm 666 band was missing in plant no. 12, whereas, the wheat specific band of gwm136 was present in this plant but absent in plant no. 16 (Fig 1a and 1b). This suggests that in plant no. 12 the long arm and in plant no. 16 the short arm of chromosome 1A were involved in chromosomal exchanges.

Presence of both the donor and recipient parent specific bands synthesized by gwm136 in plants nos. 7, 18, and 19 (Fig 1b) may indicate addition of an alien chromosome carrying this marker, probably 1U, to the chromosome complement of these plants. In plant no. 19 chromosome number of 2n=44 (Table 4) further shows the addition of a pair chromosomal structural changes were also observed in chromosome 1BS of plant no 15 (Fig 1c), and 2DS of plants no. 19 (Fig 1d).

The chromosomal translocations must have been facilitated by the *Ph*^{I} gene. The molecular results of the present study provide further evidence of chromosomal exchanges between wheat chromosomes and those of its distantly related species, *Ae. umbellulata*, induced by the *Ph*^{I} gene of *Ae. speltoides*.

Synthetic amphiploids of *T. durum* and *Aegilops* species have been found to be very effective for transferring resistance to wheat diseases from non-progenitor C and U genome species. Compared with direct crosses of these species with wheat, induced homoeologous chromosome pairing in crosses of synthetic amphiploids with CS(*Ph*^{I}) and molecular characterization of the derivatives with wheat microsatellites would be a good strategy for transfer of desired variability from related species, such as *Ae. umbellulata* and *Ae. caudata*, into the wheat genome.

Resistance of the derivatives under field conditions and as seedlings indicate the presence of alien resistance gene(s). However, because of several chromosomal structural changes, monitored by cytological (Table 3) and molecular studies (Table 5, and Fig 1), it was difficult to associate any of the alien chromosomes or fragments with resistance genes. Further backcrossing and selection for resistance will be needed to reduce the unwanted alien chromatin. In this process, STMS markers identified for each chromosome are useful to follow the alien chromatin and select the derivatives with least linkage drag. Selecting the resistant derivatives, which still show the amplification pattern similar to that of recipient parent, indicates the presence of least alien chromatin in the derivatives.

Chromosome banding techniques, such as C-banding and *in situ* hybridization (Le *et al.* 1989) have been widely used for identification of wheat chromosomes involved in translocation, translocation breakpoints, and the sizes of transferred alien segments (Friebe *et al.* 1996).

Table 4. Resistant derivatives of crosses of amphiploid [*T. durum* cv. WH890 -*Ae. umbellulata* Acc. 3732] x CS (*Ph*^{I}), used for molecular characterization.

PAU Plant NO.in 1998-99	Reference	Pedigree
-	1	Triticum durum cv. WH890
-	2	Amph[T. durum cv. WH890-Ae. Umbellulata 3732]
-	3	Ae. Umbellulata 3732
-	4	Triticum aestivum cv. Chinese Spring [CS]
-	5	Triticum aestivum cv. Chinese Spring with Ph^{I} gene [CS(Ph^{I})]
-	6	Triticum aestivum cv. WL711
M618-1	7	F_3Amph[T. durum cv. WH890-Ae. umbellulata 3732]/ CS(Ph^{I})
M1673-1	8	F_3 CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]
M1673-2	9	F_3 CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]
MX1674-1	10	BC_1F_2 CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS
M1676-1	11	BC_1F_2CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS
M1676-2	12	BC_1F_2CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS
W1082-2	13	BC_2F_1CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS[2]
M1688-1	14	BC_1F_2CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS
W1087-1	15	BC_2F^{I} CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS/3/WL711
W1087-6	16	BC_2F^{I}CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS3/WL711
M1689-2	17	BC_1F_2 CS(Ph^{I})/Amph[T. durum cv.WH890-Ae. umbellulata 3732]//CS
M1689-3	18	BC_1F_2 CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS
M1691-1	19	F3 CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]
M1693-1	20	BC_1F_2 CS(Ph^{I})/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS
MX1693-1	21	BC_2F_1CS(S)/Amph[T. durum cv. WH890-Ae. umbellulata 3732]//CS/3/WL711

* Against leaf rust pathotype 104B

Contd....

(Contd Table 4)

PAU Plant No. in 1998-99	Reference	Chromosome Pairing							Rust Reaction		
									Field Data		Seedling test*
		2n	I	II			III	IV	Leaf Rust	Yellow Rust	
				Rod	Ring	Total					
-	1	28	-	-	-	-	-	-	10S	40	3+
-	2	42	-	-	-	-	-	-	0	0	3+
-	3	14	-	-	-	-	-	-	0	0	0;
-	4	42	-	-	-	-	-	-	20S	60S	3+
-	5	42	-	-	-	-	-	-	20S	60S	3+
-	6	42	-	-	-	-	-	-	90S	40S	3+
M618-1	7	-	-	-	-	-	-	-	tS	0	0;
M1673-1	8	-	-	-	-	-	-	-	tR	0	0;
M1673-2	9	40	6.58 (3-9)	3.97 (0-7)	12.63 (9-15)	26.6 0	0.07 (0-1)	- -	0	0	0;
MX1674-1	10	42	3.44 (1-6)	3.11 (1-9)	15.79 (10-18)	18.9 0	0.15 (0-1)	0.26 (0-1)	tR	0	0;
M1676-1	11	41	4.30 (3-5)	2.70 (0-6)	14.85 (12-17)	17.5 5	- -	0.20 (0-1)	0	tR	0;
M1676-2	12	42	6.06 (3-9)	4.71 (2-7)	13.16 (10-16)	17.8 7	0.13 (0-1)	0.13 (0-1)	0	tR	0;
W1082-2	13	42	0.53 (0-2)	2.50 (0-9)	17.93 (11-21)	20.1 3	0.13 (0-1)	0.07 (0-1)	0	0	0;
M1688-1	14	42	1.05 (0-2)	4.32 (1-6)	16.01 (13-21)	20.3 3	0.08 (0-1)	0.03 (0-1)	0	0	0;
W1087-1	15	-	-	-	-	-	-	-	0	0	0;
W1087-6	16	42	- -	1.90 (0-5)	19.10 (16-21)	21.0 0	- -	- -	0	0	0;
M1689-2	17	-	-	-	-	-	-	-	0	0	0;
M1689-3	18	-	-	-	-	-	-	-	0	0	0;
M1691-1	19	44	3.03 (1-4)	4.47 (1-7)	15.87 (13-19)	20.3 4	0.07 (0-1)	- -	0	0	0;
M1693-1	20	42	11.9 (9-15)	5.50 (0-10)	9.39 (3-18)	14.8 9	0.07 (0-1)	- -	5S	0	0;
MX1693-1	21	41	6.12 (3-9)	3.04 (1-7)	12.36 (9-15)	15.4 0	0.76 (0-2)	0.20 (0-1)	5S	0	0;

Against leaf rust pathotype 104B

Table 5. Presence (+) or absence (-) of microsatellite markers on short arm (S) and long arm (L) of chromosomes of resistant derivative of CS(*Ph*^{I}) x amphiploid [*T. durum* WH890-*Ae. umbellulata* Acc. 3732] crosses.

STMS		Plant / Lane No.										
		1	2	3	4	5	6	7	8	9	10	11
Gwm. no	Chr. arm	WH890	Amphiploid	Ae.umbellulata	CS(Ph^{I})	CS	WL711	M618-1	M1673-1	M1673-2	MX1674-1	M 1676-1
666	1AL	+	+	-	+	+	+	+	+	+	+	+
136	1AS	±	±	±	±	±	±	±	±	±	±	±
63	7AL	+	+	-	+	+	+	+	+	+	+	+
60	7AS	±	±	±	±	±	±	±	±	±	±	±
140	1BL	±	±	±	±	±	±	±	±	±	±	±
582	1BS	+	+	-	+	+	+	+	+	+	+	+
257	2BS	±	±	±	±	±	±	±	±	±	±	±
251	4BL	±	±	±	±	±	±	±	±	±	±	±
508	6BS	±	±	±	±	±	±	±	±	±	±	±
261	2DS	±	±	±	±	±	±	±	±	±	±	±
190	5DS	+	+	+	+	+	+	+	+	+	+	+
469	6DS	-	-	-	+	+	+	+	+	+	+	+

Fig 1. Amplification pattern of selected STMS markers a) gwm 666 on 1AL, b) gwm 136 on 1AS, c) gwm 582 on 1BS, d) gwm 261 on 2DS, STMS markers used for characterization of resistant derivative of amphiploid CS (*Ph*^{I}) x [*T. durum* cv. WH890 -*Ae. umbellulata* Acc. 3732] crosses. Lane no. 1) *T. durum* cv. WH890, 2) *T. durum* cv. WH890- *Ae. umbellulata* amphiploid, 3) *Ae. umbellulata,* 4) S(*Ph*^{I}), 5) CS, 6) WL711, 7-21). Resistant derivatives.

Contd....

Contd Table 5

STMS		Plant / Lane No.									
		12	13	14	15	16	17	18	19	20	21
Gwm.	Chr.										
no	*arm*	M1676-2	W1082-2	M1688-1	W1087-1	W1087-6	M1689-2	M1689-3	M1691-1	M1693-1	MX1693-1
666	1AL	-	+	+	+	+	+	+	+	+	+
136	1AS	±	±	±	±	±	±	±	±	±	±
63	7AL	+	+	+	+	-	-	+	+	+	+
60	7AS	±	±	±	-	±	±	±	-	-	-
		+	+	-	-	-	-	-	-	+	+
140	1BL	±	±	±	±	±	±	±	±	±	±
582	1BS	+	+	+	-	+	+	+	+	+	+
257	2BS	±	±	±	-	±	±	±	-	-	±
251	4BL	±	±	-	±	±	±	±	±	±	±
508	6BS	±	±	±	-	±	±	±	±	±	-
		±	±	±	-	±	±	±	±	±	+
		+	+	+	+	+	+	+	+	+	+
261	2DS	+	+	+	+	+	+	+	-	+	+
		±	±	±	±	±	±	±	±	±	±
		+	+	+	+	+	+	+	+	+	+
190	5DS	+	+	-	-	-	+	+	-	+	+
		+	+	+	+	+	+	+	+	+	+
469	6DS	+	+	+	-	+	+	+	-	+	+

Fig 1. Amplification pattern of selected STMS markers a) gwm 666 on 1AL, b) gwm 136 on 1AS, c) gwm 582 on 1BS, d) gwm 261 on 2DS, STMS markers used for characterization of resistant derivative of amphiploid CS *(Ph^I)* x [*T. durum* cv. WH890 -*Ae. umbellulata* Acc. 3732] crosses. Lane no. 1) *T. durum* cv. WH890, 2) *T. durum* cv. WH890- *Ae. umbellulata* amphiploid, 3) *Ae. umbellulata,* 4) S(*Ph^I*), 5) CS, 6) WL711, 7-21). Resistant derivatives.

The results of the present study, however, indicate indicate that wheat microsatellites can be efficiently used as a supplementary tool for easy and early identification of the wheat chromosomes involved in translocations.

References

Aghaee-Sarbarzeh M, Harjit-Singh, Dhaliwal HS, 2000. *Ph^{I}* gene derived from *Aegilops speltoides* induces homoeologous chromosome pairing in wide crosses of *Triticum aestivum*. *Journal of Heredity* **91**: 417-421.

Chen PD, Tsujimoto H, Gill BS, 1994. Transfer of *Ph^{I}* gene promoting homoeologous pairing from *Triticum speltoides* into common wheat and their utilization in alien genetic introgression. *Theoretical and Applied Genetics* **88**: 97-101.

Dhaliwal HS, Harjit-Singh, Gill KS, Randhawa HS, 1993. Evaluation and cataloguing of wheat germplasm for disease resistance and quality. In: Damania A B (ed) *Biodiversity and Wheat Improvement*. pp 123-140. John Wiley & Sons.

Friebe B, Jiang J, Raupp WJ, McIntosh RA, Gill BS, 1996. Characterization of wheat-alien translocation conferring resistance to disease and pest: current status. *Euphytica* **91**: 59-87.

Gill KS, Dhaliwal HS, Multani DS, 1988. Synthesis and evaluation of *T. durum-T. monococcum* amphiploids. *Theoretical and Applied Genetics* **76**: 912-916.

Harjit-Singh, Grewal TS, Dhaliwal HS, Pannu PPS, Bagga PS, 1998. Sources of leaf rust and stripe rust resistance in wild relatives of wheat. *Crop Improvement* **25**:26-33.

Heslop-Harrison J, Leitch AR, Schwarzacher T, Anamthawat-Jonsson K, 1990. Detection and characterization of 1B/1R translocations in hexaploid wheat. *Heredity* **65**:385-92.

Jiang J, Friebe B, Gill BS, 1994. Recent advances in alien gene transfer in wheat. *Euphytica* **73**:199-212.

Knott DR, 1989. *The Wheat Rusts, Breeding for Resistance*. Springer Verlag, Berlin, Germany.

Knott DR, 1993. Agronomic and quality characters of new isogenic lines of wheat carrying genes for stem rust resistance. *Euphytica* **68**: 33-41.

Le HT, Armstrong KC, Miki B, 1989. Detection of rye DNA in wheat-rye hybrids and wheat translocation stocks using total genomic DNA as a probe. *Plant Molecular Biology Report* **7**: 150-158.

McIntosh RA, Wellings CR, Park RF, 1995. *Wheat Rusts: An Atlas of Resistance Genes*. CSIRO Melbourne, Australia.

Mukai Y, Gill BS, 1991. Detection of barley chromatin added to wheat by genomic *In Situ* hybridization. *Genome* **34**: 448-52.

Roder MS, Korzun V, Wendehake K, Plaschke J, Tixier M, Leroy P, Ganal MW, 1998. A microsatellite map of wheat. *Genetics* **149**: 2007-23.

Cytogenetics and Genetic Analysis

Suppression of Rust Resistance Genes from Distantly Related Species in *Triticum durum-Aegilops* Amphiploids

M. Aghaee-Sarbarzeh[1], H.S. Dhaliwal[2] and Harjit-Singh[2]

[1] *Dryland Agricultural Research Institute, Sararood, Kermanshah, Iran*
[2] *Department of Biotechnology, Punjab Agricultural University, Ludhiana, India*

Introduction

Related wild and progenitor species of wheat represent a large reservoir of useful variability that can be exploited for wheat improvement. Wide hybridization has contributed significantly to germplasm enhancement of bread wheat. Many agronomically important traits, including resistance to diseases and pests, and abiotic stresses have been transferred from related species and genera into wheat (Knott and Dvorak 1976, Sharma and Gill 1983, Gale and Miller 1987, Jiang *et al.* 1994, Friebe *et al.* 1996). Alien resistance genes are useful only when they express in the cultivated background. Genetic suppression of disease resistance of related species by the D genome has frequently been reported (Kerber 1983, Bai and Knott 1992, Dhaliwal *et al.* 1993, Innes and Kerber 1994, Ma *et al.* 1997).

Studies at the Punjab Agricultural University (PAU), Ludhiana showed that among less closely related wild species, diploid *Aegilops species* with C, U, and M genomes are excellent sources of resistance to leaf rust and stripe rust (Dhaliwal *et al.* 1993, Harjit-Singh and Dhaliwal 2001). Therefore a study was initiated to transfer the rust resistance gene(s) from these species into hexaploid wheat. The present paper reports the suppression of resistance gene(s) of the C and U genomes of *Ae. caudata* and *Ae. umbellulata*, respectively, by gene(s) on the A/B genomes of wheat.

Materials and Methods

The plant materials are listed in Table 1. To transfer stripe rust and leaf rust resistance from diploid *Aegilops* species with C or U genomes to hexaploid wheat, amphiploids were developed between these species and susceptible *T. durum cultivars* as pollen parents. To synthesize amphiploids, the coleoptiles of 4-5 days old F_1 seedlings, obtained from these crosses, were treated with 0.25% colchicine in 2% DMSO solution for four hours (Gill *et al.* 1988). These amphiploids were used as the bridge for the transfer of leaf and stripe rust resistance genes into

cultivated hexaploid wheat. To induce homoeologous chromosome pairing the amphiploids were first crossed with a line *T. aestivem* cv. Chinese Spring carrying the *Ph*^{I} gene from *Ae. speltoides* (Aghaee Sarbarzeh *et al.* 2001) and further back-crossed to Chinese Spring.

The parents, F_1s, amphiploids, and the derivatives of crosses with Chinese Spring (CS) were scored for field reaction to leaf rust and stripe rust following modified Cobb's scale (Peterson *et al.* 1948). In addition, these materials were evaluated at the seedling stage against individual pathotypes of leaf rust and stripe rust using the standard procedure (Nayar *et al.* 1997).

Table 1. Plant materials along with their genomic formula used in the present study.

Materials	Genomic formula
Triticum aestivum cv. Chinese Spring	AABBDD
Triticum aestivum cv. Chinese Spring with *Ph*^{I} [CS(*Ph*^{I})]	AABBDD
Triticum durum cv. Bijaga Yellow	AABB
Triticum durum cv. Malvi Local	AABB
Triticum durum cv. A206	AABB
Triticum durum cv. WH868	AABB
Triticum durum cv. WH890	AABB
Aegilops umbellulata Acc. 13749	UU
Aegilops umbellulata Acc. 3732	UU
Aegilops caudata Acc. 3556	CC
Amph.[*T. durum* Bijaga Yellow-*Ae umbellulata* Acc. 13749]	AABBUU
Amph.[*T. durum* Malvi Local -*Ae umbellulata* Acc. 13749]	AABBUU
Amph.[*T. durum* WH890-*Ae. umbellulata* Acc. 3732]	AABBUU
Amph.[*T. durum* A206- Ae. *caudata* Acc. 3556]	AABBCC
Amph.[*T. durum* WH868- Ae. *caudata* Acc. 3556]	AABBCC

Results and Discussion

The accession 13749 of *Ae. umbellulata* was resistant under field conditions as well as at the seedling stage to pathotype N of stripe rust and pathotypes 77-4, 77-5, 104B, and 104-2 of leaf rust (Table 2). However, the durum wheat cvs Bijaga Yellow (BY) and Malvi Local (ML) were susceptible to both the rusts under field conditions. BY was resistant to races 77-4 and 77-5 of leaf rust and race N of stripe rust, but susceptible to races 104B and 104-2 of leaf rust, whereas ML was susceptible to all the pathotypes of rusts, except 104B of leaf rust, at the seedling stage (Table 2).

The amphiploid of BY with Acc. 13749 was susceptible under the field conditions and at the seedling stage to two races of leaf rust 77-5 and 104-2, and race N

of stripe rust (Table 2). The resistance of the amphiploid to race 77-4 may be from either of the parents, or a combination of resistance genes from both of them. However, resistance to race 104B, which was due to gene(s) from *Ae. umbellulata,* was expressed in the amphiploid. As CS was also susceptible to race 104B, this race can be used as the discriminating race to screen the segregating population for rust resistance of *Ae. umbellulata.* The amphiploid of ML with *Ae. umbellulata,* was also susceptible to both the rusts under the field conditions and at seedling stage to race 77-5 of leaf rust and race N of stripe rust, but resistant to races 77-4 and 104-2, to which ML was susceptible. The two races, 77-4 and 104-2 can therefore be used for screening purpose at seedling stage among the derivatives.

Susceptibility of the amphiploids and their F_1 hybrid with CS(*Ph^l)* indicated the presence of suppressor gene(s) on A/B genomes of the durum wheat cultivars for the resistance genes of *Ae. umbellulata* Acc. 13749. Consistent resistance of the donor species, *Ae. umbellulata* Acc. 13749, to all the races, and differential reaction of the amphiploid to different pathotype of leaf rust at seedling stage (Table 2) may indicate that at least two different genes for rust resistance were present in the U genome of *Ae. umbellulata* Acc. 13749. For instance, in amphiploid Bijaga Yellow-*Ae. umbellulata* Acc. 13749 one of the genes was effective against race 104B, which was expressed in the amphiploid, and the other was effective against races 104-2 and 77-5, but was suppressed in the amphiploid due to gene(s) on A and/or B genome of durum wheat. This may be attributed to selective specificity of the suppression system. Differential specificity of suppressor genes has already been reported. Nelson *et al* (1997) reported a locus designated as *SuLr23*, from *T. tauschii*, which suppressed *Lr23* of durum wheat in an amphiploid of these two species. However, the gene *SuLr23* could not suppress other *Lr* gene in the F_1 hybrid of the amphiploid and hexaploid wheat. Susceptibility of the amphiploid Bijaga Yellow-*Ae. umbellulata* to the race N of stripe rust and 77-5 of leaf rust, to which both the parents were resistant, may be attributed to gene interaction in the amphiploids.

The *Ae. caudata* accession 3556 showed consistent resistance to prevalent races of leaf rust and stripe rust under the field conditions (Dhaliwal *et al.* 1993) and to pathotypes 77A-1, 77-1, 77-2, 77-4, and 77-5 of leaf rust at seedling stage (Table 2). Whereas, *T. durum* cvs A206 and WH868 were susceptible to these rusts under the field conditions but resistant to races 77-5 and 104B at seedling stage. The amphiploids of these two cultivars with *Ae. caudata,* and their F_1 hybrid with CS(*Ph^l)* were also susceptible in the field as well as to individual pathotypes of leaf rust to which the durum wheat were susceptible.

Susceptibility of the amphiploids and their F_1 hybrid with CS(*Ph^1*) under the field conditions as well as to individual patotypes of leaf rusts at seedling stage, clearly indicated suppression of resistance gene(s) from the resistant *Aegilops* species by gene(s) on A/B genome of durum wheat. Suppression of rust resistance genes by the A or B genome of wheat has already been reported (Kerber 1983, Ma *et al* 1997). Innes and Kerber (1994) reported suppression of resistance to leaf rust in amphiploid of susceptible durum wheat and resistant *Ae. squarrosa.*

Recovery of resistant plants from the F_2 and subsequent backcross generations (BC_1 and BC_2) with CS in crosses involving *Ae. caudata,* suggests the absence of suppression gene(s) on A/B genomes of CS, so that in these generations resistance gene(s) could segregate from the suppression gene(s) of durum wheat. It also indicates the absence of suppressor genes in the D genome of CS for resistance gene(s) of *Ae. caudata.*

T. durum cv. WH890 was also susceptible under the field conditions and to most of the pathotypes of leaf rust and pathotype P of stripe rust (Table 2). However, in contrast to the previous amphiploids, the resistance gene(s) from U genome of *Ae. umbellulata* Acc. 3732 was expressed in the amphiploid *T. durum* WH890 - *Ae. umbellulata* Acc. 3732 under the field condition, whereas, at the seedling stage the amphiploid was susceptible to all the individual pathotypes of leaf rust, except race 77-3. As the donor species, *Ae. umbellulata,* Acc. 3732 was resistant to all the races of leaf rust (Table 2), but the amphiploid was resistant only to race 77-3, it may be concluded that the donor species carried at least two different genes. One of the genes expressed throughout the life of the plant, which was also effective against race 77-3, and the other gene(s) was an adult plant resistance (APR) gene(s), which expressed only at the adult plant stage. The possibility of suppression of this gene(s) at seedling stage also can not be ruled out. Recovery of plants in later generation (e.g. F_2 and backcross generations) that were resistant to races to which durum wheat, amphiploid, and hexaploid wheat were susceptible (Table 2) further confirms the suppression of some of the resistance gene(s) of *Ae. umbellulata* by the A/B genome of *T. durum* parents and that the Chinese Spring did not carry any suppressor for resistance gene(s) of *Ae. umbellulata.*

Although suppression of alien resistance gene(s) by gene(s) present on the genomes of cultivated species is a barrier in alien gene transfer, but this obstacle can be avoided by selecting wheat stocks or cultivars lacking the suppressing genes.

Table 2. Rust reaction of *T. durum, Aegilops* species, their amphiploids, and derivatives of crosses of amphiploids with *T. aestivum* Chinese Spring.

Materials	Field Reaction*		Seedling Reaction against Individual Race **								
			Leaf Rust								Stripe Rust
	Leaf Rust	Stripe Rust	77A-1	77-1	77-2	77-3	77-4	77-5	104B	104-2	N/P
T. aestivum cv. Chinese Spring	20S	60S	3+	-	3+	-	3+	3+	3+	3+	3+
T. aestivum cv. Chinese Spring CS(Ph^1)	220S	60S	3+	-	3+	-	3+	3+	3+	3+	3+
Aegilops umbellulata Acc. 13749	0	0					0;	2-	3	0;	0
T. durum cv. Bijaga Yellow	20S	60S					0;	0;	0;	3	;
BY-*Ae. umbellulata* Amphiploid	80S	60S	-		-	-	2-	3+3+	0;	3	3
F_1 CS(Ph^1)5Amphiploid	40S	5S					-	3-3+	-	-	-
T. durum cv. Malvi Local	90s	40S					3	3+	0;	3	3
ML *Ae. umbellulata* Amphiploid	40S	60S	-	-	-	-	2-	3+	0;	2	3
F_1 CS(Ph^1) 5 Amphiploid	40S	5S					-	3-3+	-	-	-
	90S										
T. durum cv. A206	0	40s	3+-4	-	44	-	3+-4+	0;	0;	3	3
Ae. caudata Acc. 3556	5S-	0	0;	0-0;	0;-		0	0	-	-	-
Amph.[*T. durum- Ae. caudata*]	80S	5S		-	3+	0-0;	3	3+	3	3	3
F_1 CS(Ph^1) / Amphiploid	20S	40S		-	-	-		-	-	-	-
F_2 CS(Ph^1) / Amphiploid	0-20S	0-20S	-	-	-	-		-	-	-	-
F_3 CS(Ph^1) / Amphiploid	0 90S	0-60S	-	-	0;-3	-		-	-	-	-
BC_1 CS(Ph^1) / Amphiploid // CS	0-40S	0-20S	-	-	-	-	-	-	-	-	-
BC_1F_2 CS(Ph^1) / Amphiploid // CS	0-80S	0-10S	-	-	-	-	0;-3	-	0;-3	0; 3	0;-3
BC_2 CS(Ph^1) / Amphiploid // CS[2]	0 20S	0-5S	-	-	-	-	-	-	-	-	-
	40s										
T. durum cv. WH868	0	40s	3-	3+ 4-	3+	-	x+	0;	0;	3	3
Ae. caudata Acc. 3556	20-	0	0;	0;	0;	-	0	0	-	-	-
Amph.[*T. durum Ae. caudata*]	60s	5-20s	-	-	4-	-	3	2--3-	3	3	3
F_1 CS(Ph^1) / Amphiploid	40s	60s	-	-	-	0-0;	-	-	-	-	-
F_2 CS(Ph^1) / Amphiploid	0-10s	0-20s	-		-	-	-		-	-	-
F_3 CS(Ph^1) / Amphiploid	0-20s	0-90s	-	-	0;-3	-	-	-	-	-	0;-3
BC_1 CS(Ph^1) / Amphiploid // CS	0 80s	0-20s	-	-	-	-		-	-	-	-
BC_1F_2 CS(Ph^1)) / Amphiploid // CS	0 80s	0-5s	-	-	-	-	0; 3	-	0;-3	0;-3	-
BC_2 CS(Ph^1) / Amphiploid // CS[2]	0-90s	0-10s	-	-	-	-	-	-	-	-	-
T. durum cv. WH890	20s	20s	-	x+	3 3+	3-3	3+	2	3+	3+	3
Ae. umbellulata Acc. 3732	0	0	0;	0-0;	0;	0	0;	0	0;	0;	0;
Amph. [T. durum-Ae. umbellulata]	0	0		-	3	0-0;	3+	3+	3+	3+	3
F_1 CS(Ph^1) / Amphiploid	0	-		-	-	-	-	-	-	-	-
F_2 CS(Ph^1) / Amphiploid	0-40s	0-20s	-	-	-	-		-	-	-	-
F_3 CS(Ph^1) / Amphiploid	0 60s	0-20s	-	-	-	-	-	-	-	-	-
BC_1 CS(Ph^1) / Amphiploid // CS	0-10s	0-5s	-	-	0;-3	-	0; 3	-	0;-3	0;-3	0;-3
BC_1F_2 CS(Ph^1) / Amphiploid // CS	0-90s	0-20s		-	0;-3	-	0;-3	-	0;-3	0;-3+	0;-3
BC_2 CS(Ph^1) / Amphiploid // CS2	0-90s	0-10s	-	-	0;-3	-	0;-3+	-	0;-3+	0;-3	0;-3

0:Free, tS:Trace of Susceptibiliy, S: Susceptible,** + 0-2:Resistant, 3-4:Susceptible ; X: Mesothetic.

References

Aghaee-Sarbarzeh M, Harjit-Singh, Dhaliwal HS, 2001. *Ph*^{I} gene derived from *Aegilops speltoides* induces homoeologous chromosome pairing in wide crosses of *Triticum aestivum. Journal of Heredity* (in press).

Bai DP, Knott DR, 1992. Suppression of rust resistance in bread wheat (*Triticum aestivum)* by D-genome chromosomes. *Genome* **35**: 276-82.

Dhaliwal HS, Singh H, Singh KS, Randhawa, HS 1993. Evaluation and cataloguing of wheat germplasm for disease resistance and quality. In: Damania AB (ed.) *Biodiversity and Wheat Improvement.* pp 123-140. John Wiley & Sons.

Friebe B, Jiang J, Raupp WJ, McIntosh RA, Gill BS, 1996. Characterization of wheat-alien translocations conferring resistance to disease and pest: current status. *Euphytica* **91**: 59-87.

Gale MD, Miller TE, 1987. The introduction of alien genetic variation into wheat. In: *Wheat Breeding*: *Its Scientific Basis* (Lupton FGH , ed), UK, Chapman and Hall, 173-210.

Gill KS, Dhaliwal HS, Multani DS, 1988, Synthesis and evaluation of *T. durum-T. monococcum* amphiploids. *Theoretical and Applied Genetics* **76**: 912-16.

Harji-Singh, Dhaliwal HS, 2001. Interspecific genetic diversity for resistance to wheat rusts in wild *Triticum and Aegilops species. Wheat Information Service* **91** (in press).

Innes RL, Kerber ER, 1994. Resistance to wheat leaf rust and stem rust in *Triticum tauschii* and inheritance in hexaploid wheat of resistance transferred from *T. tauschii. Genome* **37**: 813-22.

Jiang J, Friebe B, Gill BS, 1994. Recent advances in alien gene transfer in wheat. *Euphytica* **73**:199-212.

Kerber ER, 1983. Suppression of rust resistance in amphiploids of *Triticum.* In: Sakamoto S (ed) *Proceedings of the 6*th *International Wheat Genetics Symposium.* pp 813-814. Kyoto, Japan.

Knott DR, Dvorak J, 1976. Alien germplasm as a source of resistance to disease. *Annual Review of Phytopathology* **14**: 211-235.

Ma H, Singh RP, Mujeeb-Kazi A, 1997. Resistance to stripe rust in durum wheat, A-genome diploids, and their amphiploids. *Euphytica* **94**: 279-86.

Nayar SK, Prashar M, Bhardwaj SC, 1997. Manual of current techniques in wheat rusts. *Research Bulletin* No.2. Regional Station, DWR, Flowerdale, Shimla , India.

Nelson JC, Singh RP, Antrique JE, Sorrells ME, 1997. Mapping genes conferring and suppressing leaf rust resistance in wheat. *Crop Science* **37:** 1928-23.

Peterson RF, Campbell AB Hannah AE, 1948. A diagrammatic Scale for estimating of rust intensity of leaves and stem of cereals. *Canadian Journal of Research C* **26:** 469-500.

Sharma HC, Gill BS, 1983. Current status of wide hybridization in wheat. *Euphytica* **32**: 17-31.

Analysis of Resistance to Yellow Rust in Wheat Lines by a Test for Resistance and a Diallel Cross

A. Barati[1] and S. Aeineh[2]

[1] *University of Mazandaran, College of Agriculture, Sari, Iran.*
[2] *University of Razi, College of Agriculture, Kermanshah, Iran.*

Introduction

Nearly 90% of human food is provided from plants and 85% is provided from ten plant species, among which wheat and rice are the most important. Therefore, it is important to protect crop plants from diseases and pests. Wheat is major crop in Iran and yellow rust is one of its most important diseases.

Investigation of factors that cause yellow rust epidemics in Mazandaran showed that, when rainfall was high, yellow rust developed rapidly (Foroutan and Torabi 1995). Because chemical fungicides may have deleterious effects, the best method for controlling this disease is the production and use of resistant varieties. This study was conducted to determine relative resistance of 40 lines of wheat and to investigate general combining ability and specific combining ability of five cultivars (Rashid, Sardari. Sabalan and Falat) and the type of gene action responsible for resistance to yellow rust by a diallel cross.

Materials and Methods

To test for resistance, 40 cultivars and lines of wheat, including hexaploids and tetraploids, were planted in a random complete block design with 3 replications in Kermanshah Agricultural College. The amount infection under natural conditions was measured as percentage leaf area infected and scored from 1 to 10 (Moghaddam 1994). Analysis of variance and comparison of means with Duncan's multiple range test and T-test were used to analyze the data. In the diallel design Sardari, Rashid, Navid, Sabalan and Falat with their crosses (without reciprocals) were also planted in a completely randomized design with 3 replications and scored in the same way in late May.

Statistical methods for the diallel cross were analysis of variance and comparison of means by LSD. To investigate general combining ability and specific combining ability the Griffing method (Griffing 1956, method 2 and model B) and gene action was assessed with the method of Hayman (1954a,b).

Results and Discussion

Cultivar test for resistance

Variance analysis (Table 1) indicated significant differences between cultivars for yellow rust resistance. The Lowest resistance was observed in Ghods and the highest in Golsiah (Table 2). The T-test showed that differences between tetraploid and hexaploid cultivars were not significant.

Table 1: Variance analysis of the test for resistance

Source of variation	Df	SS	MS	F
Replication	2	7.42	3.71	0.21ns
Cultivars/Lines	39	2401.6	52.35	3.09**
Error	78	1320.95	16.93	
Total	119	3370.24		

Table 2. Resistance ranking of cultivars/lines in the resistance test (1= lowest, 40= highest)

1- Ghods	9- 3b	17- 20-1-55	25- 6B	33- 10-B
2- Omid	10- 3-1-3	18- 9-B	26- Shirodi	34- 12-B
3- 110-x	11- Navid	19- 2-B	27- M73-4	35- Chanab
4- Rashid	12- 19-32	20- 5-B	28- 11-B	36- Mahdavi
5- Sardari	13- Zardak	21- Yavarous	29- Sabalan	37- 1-B
6- Ghafghaz	14- 25-1-49	22- 113-X	30- 4-1-36	38- Zarrin
7- Falat	15- 4B	23- 7-B	31- 20-1-54	39- Nik nezhad
8- Alemot	16- Gaskogen	24- 8-B	32- Chamran	40- Golsiah

Diallel analysis

Variance analysis (Table 3) indicated significant differences between crosses for yellow rust resistance. The highest resistance was observed in Falat and the lowest was observed in Sardari X Rashid (Table 6). The T-test again showed no significant difference between crosses with tetraploid cultivars and crosses with hexaploid cultivars. The Griffing analysis showed that GCA and SCA variances were significant (Table 4), and indicated that genes with both additive and non-additive effects were important, but that the non-additive effects were more important. Sardari, with low resistance, had the highest general combining ability (Table 5) and Falat with high resistance had the lowest general combining ability. The Navid X Sabalan hybrid, with the lowest specific combining ability, showed high resistance but Navid X Falat, Sabalan X Falat and Sardari X Falat, with high specific combining ability, showed low resistance. The T^2-test and b coefficient showed that Hayman's hypothesis was true.

In a graphic analysis, the position of the regression line showed that the type of gene action was over-dominance. Graphic analysis also indicated that the majority of the genes responsible for the resistance were recessive.

The amount of D, H_1, H_2, h^2 and E indicated that genes with additive and non-additive effects were important. The mean degree of dominance showed that the gene action was over-dominance. The proportion of genes with positive and negative effects varied in the parents. The number of recessive genes in the parents was greater than the number of dominant genes. Heritability in the narrow sense was 48% and in the broad sense was 73%, which indicated that selection would be useful in early generations.

Tetraploid cultivars are a suitable source of resistance for transferring resistance to other cultivars and selection is more useful than hybrid production for increasing resistance to yellow rust.

Table 3. Variance analysis of the diallel design

Source of Variation	Df	SS	MS	F
Replication	2	4.15	2.07	0.59^{ns}
Crosses	14	200.14	14.29	4/10**
Error	28	97.52	3.48	
Total	44	301.82		

Table 4. Variance analysis of combining ability in the diallel cross

Source of Variation	Df	SS	MS	F
GCA	4	17.79	4.45	3.83**
SCA	10	48.91	4.89	4.21**
Error	28	32/50	1.16	

Table 5. GCA effects (diagonal) and SCA effects (super diagonal) for the diallel cross

Parents	1	2	3	4	4
Navid (1)	-0.112^{ns}	-2.294**	-0.346^{ns}	1.259^{ns}	2.949**
Sabalan (2)	0.00	0.030ns	0.011^{ns}	-0.884^{ns}	2.640**
Sardari (3)	0.00	0.00	0.883**	0.497^{ns}	1.854**
Rashid (4)	0.00	0.00	0.00	0.445^{ns}	1.059^{ns}
Falat (5)	0.00	0.00	0.00	0.00	-1.246**

Table 6. Resistance arrangement of hybrids in diallel design (1= lowest, 15= highest)

1- Sardary X Rashid	6- Sabalan X Sardary	11- Rashid
2- Falat X Navid	7- Sardary	12- Rashid X Sabalan
3- Naivd X Rashid	8- Navid X Sardary	13- Navid
4- Falat X Sardary	9- Sabalan	14- Navid X Sabalan
5- Falat X Sabalan	10- Rashid X Falat	15- Falat

References

Foroutan A, Torabi M, 1995. Investigation of factors with effects on yellow rust in mazandaran. *Proceedings of the 12th Iranian Plant Protection Congress.* Karaj Agricultural College.

Griffing B, 1956. Concepts of general and specific combining ability in relation to diallel crossing systems. *Australian Journal of Biological Science* **9**: 463-493.

Hayman BI, 1954a. The analysis of variance of diallel tables. *Biometrics* **39**: 235-244.

Hayman BI, 1954b. The theory and analysis of diallel crosses. *Genetics* **39**: 789-809.

Moghaddam M, 1994. Plant breeding notebook. Tabriz University.

Diallel Analysis of Pustule Size and Pustule Density of Stripe Rust in Wheat

M. R. Naghavi[1], M. R. Ghannadha[1], M. Torabi[2] and K. Nazari[2]

[1]Agriculture College, Tehran University, Karaj, Iran;
[2]Seed and Plant Improvement Institute, Karaj, Iran

Introduction

Yellow rust, caused by *Puccinia striiformis* Westend., is a common disease of wheat in cool, humid climates. In Iran it is one of the main biotic factors limiting wheat production, with yield losses of over 30% reported in 1993 and 1995 (Torabi *et al*, 1995).

The breeding of resistant varieties offers an effective approach to eliminate the use of fungicides and to minimize crop losses from this disease. However, most of the known major genes for resistance to yellow rust have become ineffective and vulnerable to one or more pathogenic races (Fahlma *et al.*1998).

There is no published information on the inheritance of pustule size and pustule density as two possible components of resistance, to yellow rust. In the investigation presented here, information was sought on the inheritance of pustule size and pustule density to this disease.

Materials and Methods

Four wheat lines (M-73-7, M-73-6, M-73-4 and M-73-3), which had resistance to yellow rust, and one susceptible cultivar, Bolani, were chosen for this investigation. The parents were intercrossed and selfed in a half-diallel crossing scheme in 1995. Two cultures of *P. striiformis* (134E150A+ and 6E18A+) from the rust culture collection of the Seed and Plant Improvement Institute in Karaj, were used. Pathotype identification procedures were carried out according to Johnson *et al.* (1972). Seedlings of the parents and F1 progenies were planted in a randomized complete block design and inoculated with two pathotypes, separately, when the first leaf was fully expanded. Inoculated seedlings were left in a dew chamber for 24 h at 10 °C in dark and saturated moisture conditions. The seedlings were then transferred to a greenhouse with 16h light/8h dark photoperiod. Resistance components including pustule size and pustule density were recorded on detached leaves in the laboratory.

The Data were analyzed using the method developed by Hayman (1954) and described in detail by Mather and Jinks (1982). The General and Specific combining ability effects were calculated according to Griffing (1956).

Results and Discussion

Significant differences existed among genotypes within parents and hybrids for both resistance components and both pathotypes to warrant the diallel analysis (Table 1).

Table 1. Duncan's multiple ranges of diallel components for pustule size and pustule density to two *P.striiformis* pathotypes. Column values followed by the same letter are not significantly different.

Diallel Components	Pathotype 134E150A+		Pathotype 6E18A+	
	Pustule Size ($mm^2 \times 100$)	Pustule Density (mm^2)	Pustule Size ($mm^2 \times 100$)	Pustule Density (mm^2)
Bolni	3.95 a	1.2 0a	1.90 abc	1.31 a
M-73-7xBolani	3.07 b	1.17 a	2.20 ab	1.21 a
M-73-3xBolani	0.00 d	0.71 c	2.26 a	0.96 b
M-73-4xBolani	2.74 b	1.15 a	0.00 e	0.71 c
M-73-6xBolani	1.73 c	1.05 b	1.66 cd	1.21 a
M-73-7	1.93 c	1.0 0b	0.00 e	0.71 c
M-73-7xM-73-3	0.00 d	0.71 c	0.00 e	0.71 c
M-73-7xM-73-4	3.02 b	1.01 b	0.00 e	0.71 c
M-73-7xM-73-6	0.00 d	0.71 c	1.55 cd	0.92 b
M-73-3	0.00 d	0.71 c	0.00 e	0.71 c
M-73-3xM-73-4	0.00 d	0.71 c	0.00 e	0.71 c
M-73-3xM-73-6	0.00 d	0.71 c	1.31 d	0.87 bc
M-73-4	0.00 d	0.71 c	0.00 e	0.71 c
M-73-4xM-73-6	1.54 bc	0.99 b	1.54 cd1.	24 a
M-73-6	1.67 d	0.81 c	1.74 bcd	0.98 b

According to Table 2, the mean squares of general combining ability (GCA) were larger than those for specific combining ability (SCA) for pathotype 134E50A+, indicating the relative importance of additive versus non-additive effects. It should be possible, therefore, to manipulate the resistance genes in a breeding program because of the high level of additive gene effects (Ghannadha *et al.* 1995). In contrast the GCA and SCA of pustule size and pustule density for pathotype 6E18A+ showed that the non-additive variance was more important.

Table 2. Mean squares of General (GCA) and Specific (SCA) Combining Ability for pustule size and pustule density of two *P. striformis* pathotypes

Genetic Parameters	DF	Pathotype 134E150A+		Pathotype 6E18A+	
		Pustule Size ($mm^2 \times 100$)	Pustule Density (mm^2)	Pustule Size ($mm^2 \times 100$)	Pustule Density (mm^2)
GCA	4	11.99**	0.286**	1.13**	0.064**
SCA	10	2.54**	0.052**	5.80**	0.15**
Error	28	0.19	0.002	0.073	0.026

** Significant at 1% level

Table 3. Genetic statistic analysis for pustule size and pustule density for two *P. striiformis* pathotypes

Genetic Parameters	Pathotype 134E150A+		Pathotype 6E18A+	
	Pustule Size ($mm^2 \times 100$)	Pustule Density (mm^2)	Pustule Size ($mm^2 \times 100$)	Pustule Density (mm^2)
D ± S.E (D)	1.79 ± 0.20	0.005 ± 0.004	0.42 ± 0.26	0.002 ± 0.003
H1 ± S.E(H)	3.80 ± 0.54	0.082 ± 0.011	7.10 ± 0.61	0.222 ± 0.008
H2 ± S.E(H)	2.75 ± 0.49	0.058 ± 0.010	6.63 ± 0.55	0.211 ± 0.007
h^2 b.s (%) [1]	92	95	97	96
h^2 n.s (%) [2]	62	62	16	1
Correlation	0.92**		0.86**	

** Significant at 1% level ; [1] Broad sense heritability; [2] Narrow sense heritability

The diallel statistics for pustule size and pustule density in response to two pathotypes are presented in Table 3. Dominant genetic variances (H1 and H2) were greater than additive genetic variance (D) for both components. Narrow-sense heritability values were high or moderately high for pathotype 134E150, indicating that selection for resistant genotypes would be effective. Because the narrow-sense heritability was less for pathotype 6E18A+, it would be more difficult to make successful selection for resistance. However the heritability of a given trait depends on the material under test, the environment in which material is being grown and the experimental conditions used to produce the phenotypic expression of the trait (Hill *et al.* 1998). Even published estimates of heritability for the same trait in the same crop may differ widely (Wagoire *et al.*1999).

The significant and high positive correlation coefficients between pustule size and pustule density suggest that diallel components with smaller pustule size generally have lower pustule density for both pathotypes (Table 3). The high correlation between pustule size and pustule density may be related to linkage or pleiotropic

effects (Ohm and Shaner 1976). The result of this study is in agreement with other reports (Gannadha *et al.* 1995, Ohm and Shaner 1976).

References

Fahlma T, Roder MS, Grama A, 1998. Microsatellite DNA polymorphism divergence in Triticum dicoccoides accessions highly resistant to yellow rust. *Theoretical and Applied Genetics* **96**:187-195.

Johnson R, Stubbs RW, Fuchs E, Chamberlain NH, 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. *Transactions of the British Mycological Society* **58**: 475-480.

Hayman BI, 1954. The theory and analysis of diallel crosses. *Genetics* **39:** 789-805.

Ghannadha MR, Gordon IL, Cromey MG, McKewan JM, 1995. Diallel analysis of the latent period of stripe rust in wheat. *Theoretical and Applied Genetics* **90:**1471-476.

Griffing B, 1956. Concept of general and specific combining ability in relation to diallel crossing systems. *Australian Journal of Biological Science* **9:** 463-493.

Hill J, Becker HC, Tigersted PMA, 1998. *Quantitative and Ecological Aspects of Plant Breeding*. Chapman and Hall. London.

Mather K, Jinks JL, 1982. *Biometrical Genetics-the Study of Continuous Variation.* Chapman and Hall, London. pp 255-291.

Ohm HW, Shaner GE, 1976. Three components of slow leaf-rusting at different growth stages in wheat. *Phytopathology* **66**: 1356-1360.

Torabi M, Mardoukhi V, Nazari K, 1995. Effectiveness of wheat yellow rust resistance genes in different parts of Iran. *Cereal Rusts and Powdery Mildews Bulletin* **23**: 9-12.

Wagoire WW, Ortiz R, Hill J, Stolen O, 1999. Comparison of methods for calculating the heritability of adult field resistance to yellow rust and grain yield in spring wheat. Theoretical and Applied Genetics **99**: 1075-1079.

Seedling and Adult Plant Reactions of Some Rainfed Bread Wheat Advanced Lines/Cultivars to Two Pathotypes of Yellow Rust (*Puccinia Striiformis* f. sp. *Tritici*) by Measuring Components of Resistance

M. Hassanpour Hosni[1] and M. Torabi[2]

[1]Dryland Agricultural Research Institute (DARI), Maragheh, Iran;
[2]Seed and Plant Improvement Institute (SPII), Karaj, Iran.

Introduction

Yellow or stripe rust of wheat (caused by *Puccinia striiformis* f.sp. *tritici* Westend.), is an important disease in many wheat growing regions of the world, especially in areas with cool and wet environmental conditions (Roelfs *et al.* 1992). Epiphytotics have been reported in diverse areas such as China (Saari and Prescott 1985), Australia, Continental Europe and Ethiopia (Johnson 1992) and Iran. In Iran epiphytotics of the disease have occurred frequently since 1991. The most severe years were 1993 and 1995 (Nazari *et al.* 1998). Losses caused by the disease could be up to 75% (Roelfs 1978). In Iran the loss observed in 1993 was approximately 1.5 million tonnes of the total production of the country.

Genetic resistance is the most economical and environmental safe approach for disease control. Genes conferring seedling and adult plant resistance are known to occur in wheat (*Triticum aestivum* L.). Seedling resistance is usually race-specific and can be recognized by its characteristic low infection type at all plant stages. Adult plant resistance can be either race-specific or race non-specific and is usually better recognized after the seedling stage (Johnson 1980). Although more than 20 genes for seedling resistance to stripe rust are currently known (McIntosh *et al.* 1993), most are ineffective due to the corresponding virulence in the pathogen. Some known adult plant resistance that have remained durable appear to involve slow rusting mechanisms conferred by minor additive gene and may be temperature sensitive (Singh and Rajaram 1994). Utilization of durable resistance for the global control of stripe rust is a major objective CIMMYT's bread wheat breeding program (Rajaram *et al.* 1988).

The objectives of this study were to evaluate the resistance of some rainfed bread wheat advanced lines/cultivars with two pathotypes of yellow rust, by measuring components of resistance.

Materials and Methods

A total of 7 advanced, promising and current commercial bread wheat cultivars including a susceptible check "Bolani" were tested at the seedling stage in the greenhouse and at the adult plant stage in the field. The names of lines/cultivars used in this study were:

1- Sabalan//1-27-5614, **2**- Anza/3/Pi/Hys/4/Sefid, **3**- Sbn//Trm/K253
4- Kvz/Tm 71/3/Maya "S" /Bb/Inia/4/Sefid, **5**- Shahi (Lr64....Ste)...
6- Agri/093.44/Momtchil, **7**- Sabalan, **8**- Bolani.

Two isolates of yellow rust from Maragheh (6E6(134)A^+) and Moghan (134E134A^+) regions were used in the greenhouse tests and the pathotype 6E6(134)A^+ was used in the field experiment. Both pathotypes were virulent on genes *Yr* (2, 6, 7, 9, 22, 23, 24, 25), *YrCle*, but they differed from each other by presence or absence of virulence factors on *YrCle*, *Yr HVII* and *Yr24*.

For evaluation of some factors of resistance, sets of the cultivars and lines were inoculated at the second leaf stage with the two yellow rust pathotypes in six replications. Samples taken at different times after inoculation, were examined microscopically for measurement of germination (%), penetration (%), colony size, number of pustules per leaf unit area, pustule size and spore production. The latent periods were recorded in hours and infection type on a 0-9 scale based on McNeal *et al.* (1971). The latent period of cultivars with no apparent pustules was given as an LP of 20 days.

For resistance evaluation at the adult plant stage in the field, all cultivars/lines were planted in three replications, based on a randomized block design, and inoculated three times with spores of pathotype 6E6(134)A^+ for estimation of Area Under the Disease Progress Curve (AUDPC). Four readings were used to calculate the AUDPC using a program developed by CIMMYT. The relative AUDPC(%) of each entry was obtained by dividing its AUDPC by that of Bolani and multiplying by 100. The AUDPC(%) and Final Disease Severity (FDS) of flag leaves were used for statistical analysis.

Results and Discussion

Greenhouse studies

The results of measuring components of resistance with two yellow rust pathotypes and some bread wheats are given in table 1. As the results show, germination (%) and penetration (%) could not be criteria for resistance evaluation, because the resistance phenomenon starts after penetration.

Also the results of a T-Test showed significant differences between the two pathotypes of yellow rust. Pathotype 134e134A+ was more aggressive than pathotype 6E6(134)A+ (Table 2).

Field studies

The results of field studies showed significant differences between lines/cultivars. Rust symptoms did not appear on lines/cultivars No. 1, 5 and 7; other lines differed significantly in different components of resistance at (a=1%). However, results of cluster analysis on 11 components of resistance classified cultivars to 4 cluster groups (Figure 1). Group 1 included line No. 1, 5 and 7 with no infection at the seedling and adult plant stages, which were considered as resistant; group 2 included lines No. 2, 3 and 6 with susceptible or moderately susceptible reactions at seedling and resistant reactions at the adult plant stage. These lines had partial resistance or slow rusting resistance characteristics; group 3 included line No. 4 with a susceptible reaction at the seedling and moderately susceptible (rAUDPC=62.33%) response at the adult plant stage with slow rusting characteristics; group 4 included cultivar No. 8 (Bolani), with susceptible reactions at both seedling and adult plant stages.

Table 1. The reactions of some rainfed bread wheat advanced lines/cultivars. Interaction with two pathotypes of yellow rust at the seedling stage in greenhouse conditions.

Line No.	Germination %	Penetration %	Colony Size µ	LP (h)	Spores x 1000 per leaf	Pustule Number per cm^2	Pustule Size µ	Infection Type (0-9)
1	46.53	11.37	0	480	0	0	0	3
2	47.99	26.14	155.84	202.01	17.168	82.43	0.035	6
3	43.15	22.14	205.15	258	25.848	48.04	0.04	6.5
4	46.09	28.72	289.35	198	35.047	40.4	0.044	6.5
5	40.07	13.42	0	480	0	0	0	3
6	44.29	25.93	135.91	222	38.292	39.02	0.048	6.5
7	39.37	10.54	0	480	0	0	0	2
8	55.38	33.90	355.31	186	76.751	37.61	0.052	8

Table 2. The results of a T-Test

Resistance Factors	Pathotype 6E6(134)A+	Pathotype 134E134A+	T-Test
Germination(%)	43.16	47.55	-3.31**
Colony size	132.66	152.73	-4.509**
No. of Pustule	28.80	33.07	-2.030**
Infection Type	4.94	5.44	-4.657**

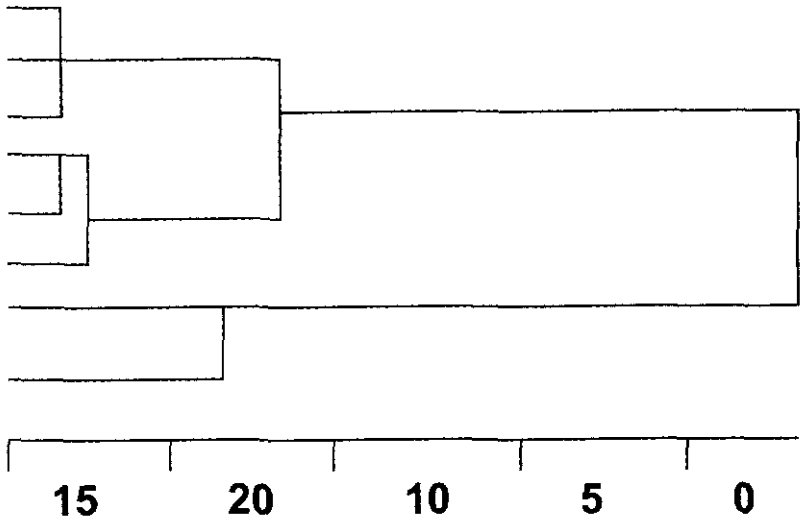


Figure 1. Cluster analysis of 8 lines/cultivars based on 11 components of resistance

Among the resistance components, measurement of latent period and pustule size in the greenhouse, at the seedling stage, and calculation of the area under the disease progress curve (AUDPC) in the field, at the adult stage, are suggested as suitable criteria for resistance measurement because of ease of measuring and high correlation with other components.

References

Johnson R, 1980. Genetics of adult-plant resistance to yellow rust in winter wheat cultivars. Pages 59-63 In: *Proceedings of the 5th Cereal Rusts Conference, 5th* Italy

Johnson R, 1992. Past, present and future opportunities in breeding for disease resistance, with examples from wheat. *Euphytica* **63:** 3-22

McIntosh R A, Hart GE, Gale MD, 1993. Catalogue of gene symbols for wheat. Z. S. Li and Z. Y. Xin, eds. *Proceedings of the 8th International Wheat Genetics Symposium,* Beijing, China.

McNeal FH, Konzak CF, Smith EP, Tate WS, Russell TS, 1971. A uniform system for recording and processing cereal research data. *United States Department of Agricultural Research Service,* ARS 34-121. 42pp.

Nazari K, Saidi A, Torabi M, 1998. Seedling and adult plant reactions of some

bread wheat lines/ cultivars to different pathotypes of *Puccinia striiformis* Westend. Pp. 300-302 in: A. E. Slinkard. ed., Poster Presentation. *Proceedings of the 9th International Wheat Genetics Symposium.* Vol. 3., Saskatoon, Saskatchewan, Canada, 2-7 August.

Rajaram S, Singh RP, Torres E, 1988. Current CIMMYT approaches in breeding wheat for rust resistance. Pages 101-118 in: *Breeding strategies for Resistance to the Rust of Wheat.* N. W. Simmonds and S. Rajaram, Eds. CIMMYT, Mexico. D. F., Mexico.

Roelfs AP, 1978. Estimated losses caused by rust in small grain cereals in the United States-1918-1976. *Miscellanous Publications of the United States Department of Agriculture.* **1363:** 1-85.

Roelfs AP, Singh RP, Saari EE, 1992. Rust Diseases of Wheat: Concepts and methods of disease management. Mexico, D.F.: CIMMYT. 81 pp.

Saari EE, Prescott JM, 1985. World distribution in relation to economic losses. Pp. 259-298 in A. P. Roelfs and W. R. Bushnell, eds. *The Cereal Rusts vol. II; Diseases, Distribution, Epidemiology, and Control.* Academic Press, Orlando.

Singh RP, Rajaram S, 1994. Genetics of adult-plant resistance to stripe rust in ten spring bread wheats. *Euphytica* **72:**1-7.

Torabi, M, Mardoukhi V, Nazari K, Afshari FA, Foroutan AR, Ramai MA, Golzar H, Kashani AS, 1995. Effectiveness of yellow rust resistance genes in different parts of Iran. *Cereal Rusts and Powdery Mildews Bulletin.* **23:** 9-12.

Epidemiology

The Importance of Yellow Rust in Rainfed-Wheat Areas of Central and West Asia and North Africa (CWANA)

H. Ketata

International Center for Agricultural Research in the Dry Areas (ICARDA) Aleppo, Syria.

Introduction

Yellow rust, caused in wheat by the fungus *Puccinia striiformis* f. sp. *tritici* has recently attracted more attention from researchers and production managers, due to important wheat losses it caused in several countries, particularly in the last two decades (Mamluk *et al* 1997, Ketata *et al.* 2001) . The development and spread of this disease is conditioned by the simultaneous occurrence of cool weather and sufficient moisture on the plants during the growing season, and the large-scale cultivation of genetically susceptible cultivars. As a result of the recent yellow rust epidemics, breeders in the CWANA region increased efforts to develop resistant wheat germplasm. This led to the release of a number of resistant cultivars, constantly threatened by the ever-changing genetic makeup of the pathogen (Danial *et al.* 1994; Hakim and Mamluk 1998). Terminal drought-and-heat stress is a more frequent phenomenon in the CWANA region than yellow rust. Temperatures above 27-30 °C, more and more common in CWANA at and past anthesis, inhibit the multiplication of the yellow rust pathogen and arrest the dissemination of the disease. The relative importance of the drought-and-heat stress (DH) and of yellow rust (YR) and the priority of yellow rust research in CWANA are discussed in this paper.

Materials and Methods

Data and information presented in the paper are derived from observations made during the past two decades in several countries of the CWANA region, and from experiments conducted at ICARDA research stations, mainly Tel Hadya, Syria (36°01'N,36° 56'E) during the period 1995-2000. A particular reference is made to field tests conducted in a relatively wet season (1997-1998) in contrast to a typical, dry season (1998-1999).

In the experiments conducted at Tel Hadya, several hundred of wheat breeding lines were grown under two different water-supply regimes, *i.e.* either "rain only" (rainfed, RF) or "rain plus one or two supplemental irrigations of 25 mm, applied

during the jointing-anthesis period" (SIR). Plants were subjected to heavy or moderately-heavy disease pressure. Disease severity (%), grain yield (kg/ha), and days to heading were scored on a plot basis in replicated trials. Additional traits (such as 1000-kernel weight) were occasionally measured in specific experiments. Tested wheat entries were divided into resistant, intermediate and susceptible depending on yellow rust reaction and into early, intermediate and late, based on days to heading. Resistant and early types present obvious advantages in the experimental conditions. The relative importance of resistance versus earliness was assessed by comparison of effects on grain yield of either of these two characters.

Results and Discussion

The widespread cultivation over millions of hectares of susceptible but otherwise good cultivars, known for their adaptation to abiotic stresses in rainfed wheat-areas, is perhaps the major reason for yellow rust outbreaks that occurred in North Africa during the 1970's (Morocco and Tunisia) and in Central and West Asia during the 1980's and the 1990's (Afghanistan, Azerbaijan, Iran, Lebanon, Pakistan, Syria, Tajikistan, Turkey, and Uzbekistan). Such cultivars, including those with the 1BL.1RS translocation, relied on specific source(s) of resistance that failed because of the genetic change of the pathogen to virulence for them. Although good progress has recently been achieved in developing resistant germplasm by breeders in the region and in international centers (e.g. CIMMYT and ICARDA), such yellow rust epidemics will reoccur if a single host cultivar predominates in the region.

Table 1. Monthly maximum (Max) and minimum (Min) temperature (°C) and rainfall (Rf, mm) distribution at 3 rainfed-wheat sites in West Asia, 1997-1998.

Month	Tel Hadya, Syria			Maragheh, Iran			Konya, Turkey		
	Max	Min	Rf	Max	Min	Rf	Max	Min	Rf
Sept	37.8	9.4	18				30.0	1.2	16
Oct	36.4	7.8	36	25.0	0.5	7	27.0	0.4	93
Nov	24.8	0.8	38	13.5	-7.0	48	19.4	-4.2	16
Dec	18.2	-3.3	62	13.0	-15.0	23	14.2	-11.4	23
Jan	17.4	-5.8	84	2.5	-18.5	64	13.8	-11.7	9
Feb	20.8	-6.2	38	5.0	-17.6	34	15.6	-8.8	18
March	25.9	-1.1	59	14.5	-11.0	96	19.8	-10.4	37
Apr	34.1	3.7	64	24.5	-2.0	52	30.0	-2.7	28
May	35.5	7.0	12	28.0	1.5	17	27.8	5.2	59
June	41.5	11.9	0	35.0	5.0	6	33.0	9.4	17
July	45.5	18.6	0	35.5	9.5	1	38.2	13.0	7
Total			340			411			316

Table 2. Monthly maximum (Max) and minimum (Min) temperature (°C) and rainfall (Rf, mm) distribution at 3 rainfed-wheat sites in West Asia, 1998-1999.

Month	Tel Hadya, Syria			Maragheh, Iran			Konya, Turkey		
	Max	Min	Rf	Max	Min	Rf	Max	Min	Rf
Sept	43.1	11.6	0				32	5.8	6
Oct	34.6	6.0	2	26.0	0.0	1	31.4	-0.3	48
Nov	28.2	4.4	39	19.0	-2.0	26	20.4	-1.4	20
Dec	19.0	-2.8	88	18.0	-9.0	28	13.3	-8.1	114
Jan	16.6	-3.0	40	5.0	-11.5	37	14.8	-9.1	21
Feb	20.0	-4.4	51	11.5	-13.5	27	14.2	-7.0	20
March	25.5	-0.5	62	15.5	-7.0	27	18.6	-8.4	22
Apr	33.9	2.5	25	23.0	-3.5	44	25.5	-1.0	9
May	40.0	6.7	0	32.0	-3.5	8	31.0	2.2	15
June	38.8	13.2	0	32.0	7.0	0	32.9	9.6	20
July	41.4	18.4	0	35.0	9.5	6	37.5	12.0	7
Total			307			202			301

Rainfed wheat in CWANA is almost all sown in the fall, generally coinciding with the onset of the rainy season. Winter is generally wet and cool or cold. Rain in early spring and temperature in late spring and early summer, to a large extent determine the likelihood of both high yield and yellow rust development. Tables 1 and 2 show the temperature and rainfall distribution at each of three typical sites in West Asia during two consecutives seasons: 1997-1998, a relatively wet season, and 1998-1999, a dry season. The first season, generally favorable in most of CWANA was marked by heavy yellow rust epidemics in several countries of the region, including Afghanistan, Azerbaijan, Syria, Tajikistan and Turkey. In contrast, during 1998-1999, drought, combined with terminal heat, was so severe in several countries that grain yield dropped to below 0.5 t/ha in large areas of Iran and Turkey. No yellow rust developed in this season on known susceptible cultivars or landraces. Elsewhere in the Caucasus and Central Asia, where most wheat is irrigated and the winter was mild in 1999, yellow rust developed to an epidemic level in Azarbaijan, Uzbekistan, and Tajikistan.

Table 3 shows the main characteristics of field experiments conducted in each of two seasons at Tel Hadya, Syria. Except for a limited number of checks, entries differ across experiments.

Table 3. Characteristics of field experiments conducted during 1997-1998 and 1998-1999 at ICARDA research station, Tel Hadya, Syria.

Season	Experiment	No. of entries	Water source	
			Rainfall (RF) mm	SIR (RF + irrigation) mm
1997-1998	1	125	340	340 + 25
	2	123	340	340 + 25
	3	230	340	340 + 25
1998-1999	4	128	307	307 + 25 + 25
	5	92	307	307 + 25 + 25

Overall grain yields in the 1997-1998 season were 4520kg/ha for the irrigated (SIR) experiments and 3899 kg/ha for the rainfed (RF) experiments, in comparison with 2339 kg/ha and 2168 kg/ha for the same environments in 1998-1999, respectively. Higher yields in the first season are attributed to better rainfall distribution, and cooler weather during grain filling. Comparison of the resistant entry groups with the susceptible groups in the first season showed an overall grain yield reduction due to yellow rust (YR) of 12% for the SIR environment and 7% for the RF environment. The corresponding figures in the second, dry season are 16% for SIR and 5% for RF (Table 4).

Table 4. Wheat-yield reduction (%) due to drought-and-heat (DH) stress and to yellow rust (YR) under rainfed (RF) conditions and supplementary irrigation (SIR) in field experiments conducted in two seasons, at Tel Hadya, Syria.

Season	No. of entries	DH		YR	
		SIR	RF	SIR	RF
1997-1998	470	3	7	12	7
1998-1999	320	22	25	16	5

This clearly shows that the effect of YR is negligible in areas receiving 300-350 mm or less of annual rainfall. Comparison of the early versus late groups showed an overall yield reduction due to lateness, equivalent to the drought-and-heat (DH) stress, of 3 and 7% for SIR and RF, respectively, in the first, better watered season, as compared to 22 and 25% in the second, dry season (Table 4). It therefore appears that, in areas receiving 300-350 mm rainfall, 1-2 supplemental irrigations will increase YR damage but may or may not decrease the DH stress, depending on rainfall distribution and high temperature pattern. In the present experimental conditions, the DH stress is more important than the YR effect in the drier season. The small magnitude of these computed reductions is due to the fact that they are averages of a large number of entries, and most of the test entries have been previously selected for good levels of resistance to YR and, to a

lesser extent DH. Similar experiments conducted in 1994-1995 with less improved cultivars (ICARDA 1995) showed that both YR and DH affected grain yield under supplemental irrigation (by 18 and 30%, respectively), but only DH affected yield, by 39%, under rainfed conditions. In 1997-1998, when 124 entries were grown in a fully-irrigated, disease-free environment, the kernel weight of the same entries under SIR, YR-infected environment, suffered a 40% reduction, attributed equally to each of YR and DH. In the favorable season 1995-1996, a controlled crop-loss assessment study, using improved and unimproved cultivars, showed grain yield reductions due to YR varying between 0 and 50% (Table 5).

Table 5. Grain yield loss (%) of wheat cultivars due to yellow rust infection, ICARDA, Tel Hadya, Syria, 1995-1996[a].

Cultivar	Yield (kg/ha) in disease-free field	Yield (kg/ha) in yellow rust-infected field	Percent yield loss
Bezostaya 1	4732	4063	14 ns
Bolal	3882	2809	28 *
Dagdas	3664	3242	12 ns
Gerek	3988	3039	24 *
95-129	5938	5000	16 *
Zargoon	4626	4464	4 ns
Seri82	4848	3211	34 **
95-13	3837	1850	52 **

a: annual rainfall: 404 mm, one irrigation of 40 mm was applied at booting.
ns: nonsignificant at P= 0.05; *: significant at 0.05 ; and **: significant at 0.01.

Environmental conditions in rainfed-wheat areas with a Mediterranean climate generally are not conducive to an effective yellow rust development. Indeed, a major portion of those areas is characterized by a drought-and-heat stress that frequently occurs towards flowering or early grain filling. Although this stress depresses grain yield significantly, its frequent nature limits the occurrence of yellow rust epidemics to one in five years or less. This may lead to the conclusion that yellow rust research may not be a priority for those areas. Yet, yellow rust can and does occur and impart non-negligible losses on seedlings or young plants in semi-arid areas, where the pathogen survives and thrives on spring dew when cool temperatures prevail. In rainfed-wheat areas with cool spring, and a Mediterranean annual rainfall above 400 mm, the risk of yellow rust epidemics is high. In drier zones, supplementary irrigation, more and more practiced in many of the traditionally-rainfed areas, generally creates a favorable environment for yellow rust development and dissemination. Results show yield losses of 30 percent or more may be caused by supplementary sprinkler irrigation of susceptible wheat cultivars in such areas of the CWANA region. The use of early, widely-based resistant or slow-rusting cultivars (Ketata *et al.* 2001), combined with the

avoidance of relying on a specific cultivar, and the adoption of raised-bed technique instead of sprinklers, where irrigation is practiced, should minimize the risk of yellow rust epidemics in the predominantly-rainfed wheat areas.

References

Danial DL, Stubbs RW, Parlevliet JE, 1994. Evolution of virulence patterns in yellow rust races and its implications for breeding for resistance in wheat in Kenya. *Euphytica* **80:** 165-170.

Hakim MS, Mamluk OF, 1998. Monitoring the pathotypes and virulences of *Puccinia striiformis* f. sp. *tritici* in Syria. *Phytopathologia Mediterranea* **37:** 106-110.

ICARDA. 1995. Germplasm Program Annual Report, p-167-170. International Center for Agricultural Research in the Dry Areas, Aleppo, Syria.

Ketata H, Yahyaoui A, Jarrah M, Braun H-J, Mergoum M, Morgounov A, Cetin L, Dusunceli F, 2001. Slow rusting in winter and facultative wheat infected with yellow rust. In: Z. Bedo and L. Lang (Eds.), *Wheat in a Global Environment,* p. 391-395. Kluwer Academic Publishers. Dordrecht, The Netherlands.

Mamluk OF, Cetin L, Braun H-J, Bolat N, Bertschinger L, Makkouk KM, Yildirim AF, Saari EE, Zencirci N, Albustan S, Cali S, Beniwal SPS, Dusunceli F, 1997. Current status of wheat and barley diseases in the Central Anatolian Plateau of Turkey. *Phytopathologia Mediterranea* **36:** 167-181.

Study of Yellow Rust Epidemiology in Khuzestan Province of Iran

S. T. Dadrezaei[1] and M. Torabi [2]

[1]Agricultural Research Center of Khuzestan, Ahwaz, Iran;
[2]Seed and Plant Improvement Institute, Karaj, Iran

Introduction

Stripe or yellow rust is the most important disease of wheat in Iran. It appears periodically and causes severe epiphytotics in different parts of the country and reduces total yield. For example, it occurred in 1962, 1966, 1972, 1979 and 1981 in northern, western and the northwest of the country (Bamdadian 1983). Damage caused by this disease in 1994 was more than 1.5 million tonnes of wheat grain, indicating its economic importance (Torabi *et al.* 1995). An epidemiological investigation was carried out, to investigate environmental factors favoring the development of the disease, to discover primary sources of inoculum, and also to compare its development in Iran with development of the disease in other parts of the world. Stripe rust has a low optimum temperature for development that limits it as a major disease in many areas of the world. It is principally an important disease of wheat during the winter or early spring or at high elevations (Roelfs *et al.* 1992). *Puccinia striiformis* has the lowest temperature requirements of the three wheat rust pathogens, with minimum, optimum and maximum temperatures of 0, 11 and 23 °C, respectively (Hogg *et al.* 1969). The present study was conducted in order to assess relations between environmental factors and development of disease and the role of primary inoculum sources in Khuzestan province of Iran.

Materials and Methods

In an attempt to assess field infection to yellow rust and its relation to environmental factors, one or two fields were chosen and their disease development was measured in four cities of Khuzestan province. Date of appearance and type of disease reaction (Infection type) and severity were assessed by random quadrate at regular weekly intervals in these fields. This work was conducted by the W or M method. Results were converted to a single variable by application of coefficients of infection based on infection type (0 = Immune, R = Resistant, MR = Moderately Resistant, MS = Moderately Susceptible and S = Susceptible) and percentage infection according to the Modified Cobb scale. To obtain the

coefficient of infection, the percentage was multiplied by fractions assigned to the infection types, which were 0 = 0.0, R = 0.2, MR = 0.4, MS = 0.8 and S = 1.0. A disease progress curve was obtained and its relation to environmental factors can be shown as a two-variable curve.

Results and Discussion

Yellow rust epidemiology was studied during a seven-year period in Khuzestan wheat fields. Appearance and development of disease were compared with environmental conditions. The severity of epidemics varied on different cultivars, depending on time of appearance and environmental factors in the area. When yellow rust was observed in almost all the fields by late February, its severity was intense on susceptible cultivars. In contrast, when appearance of the rust was delayed until mid-March, severity remained low in this study. Yellow rust did not appear before the second half of February in the region. If, in the two month period before the disease appears (first of January till late February) temperature and humidity are ideal and, if disease appears in this period, its development can occur and an epidemic is possible. However, it usually appears after the first of March or later. At this time, the environmental conditions become unsuitable for development of yellow rust (Figure 1) because the maximum temperature goes above that tolerated by yellow rust.

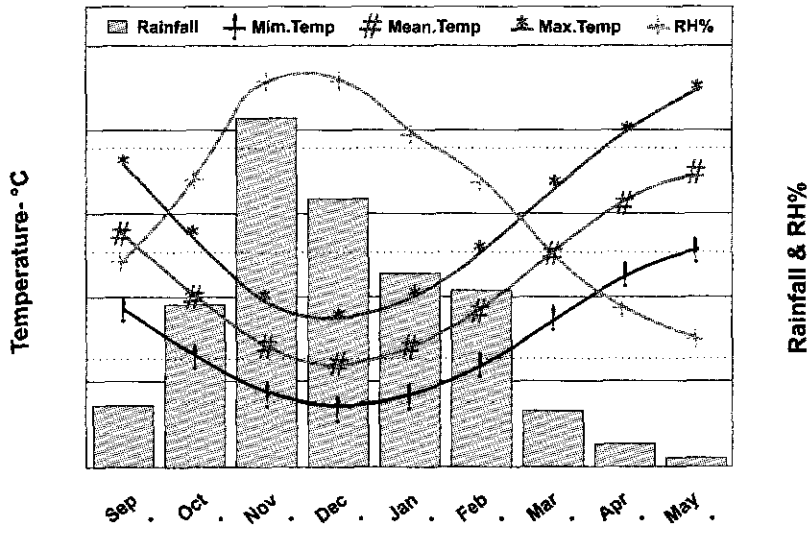


Fig.1. Temperature (°C), rainfall (mm) and relative humidity (%) in Khuzestan, averages 1975-1995

Thus activity of yellow rust, including germination, penetration and development, are limited to night and low temperature hours. Gradually, this limitation increases

so that the minimum environmental temperature is above the maximum temperature for yellow rust in late April, preventing further development of the disease.

When these environmental conditions exist and there is no inoculum, then no infection is observed. Progress of yellow rust showed large differences between susceptible cultivars (such as Azadi) and more resistant cultivars, such as Falat, in the same region (Figure 2). Appearance of yellow rust was delayed on Falat compared with development on susceptible cultivars and its progress on Falat was relatively slow. For example in Behbahan, on the second of March, average disease severity on Azadi was 80S when in nearby fields, Falat showed maximum 5MS of disease severity. Also the maximum infection on Falat was 25MS in Howayzeh when disease severity on Chenab reached 45S in mid-April (Fig 3).

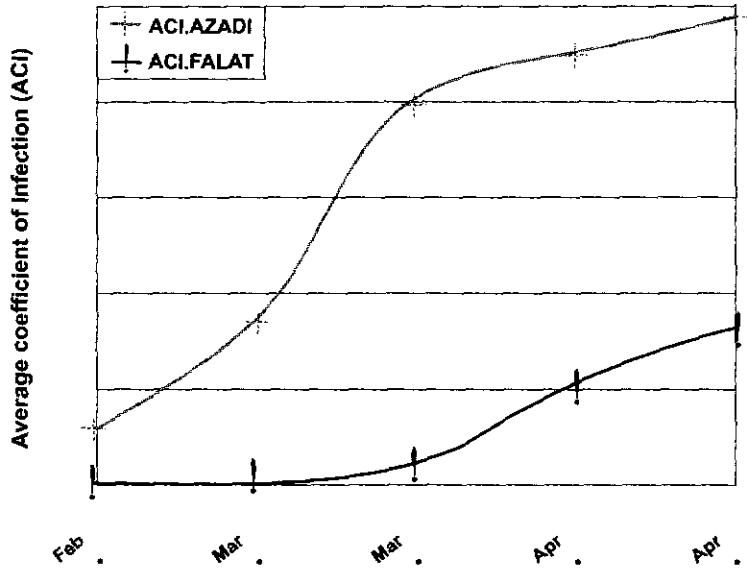


Fig.2 Yellow rust progress curve of Falat and Azadi cultivars (Behbahan-1994)

Delay in the appearance of yellow rust and its slow development on Falat resulted in secondary infections being delayed until unfavorable climatic conditions occurred and the disease was controlled by heat and dryness. Falat is a semi-susceptible cultivar and is the main variety in Khuzestan. Slow development of disease on this variety and the restricted optimum period for development of yellow rust provided a disease escape situation for Falat in this province. The complex interactions of these factors prevented a severe epidemic of yellow rust in the

wheat fields of Khuzestan, and the restricted cultivation of susceptible cultivars probably also helped.

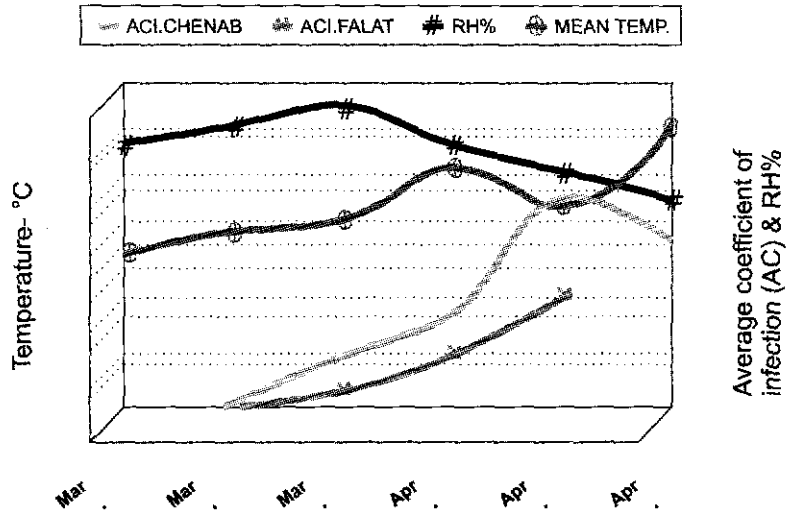


Fig.3. Yellow rust progress curve for Chenab and Falat cultivars in comparison with temperature and relative humidity curves (Howayzeh-1996)

References

Bamdadian,A.1982. *Study on yellow rust epidemioloy in Iran.* PhD. Thesis, Tehran University, 225pp.

Hogg WH, Houmm CE, Mallik AK, Zadoks JC.1969. Meteorological factors affecting the epidemiology of wheat rusts. *WMO Technical Note* **99**.143pp.

Roelfs AP, Singh RP, Saari EE.1992. *Rust Diseases of Wheat: Concepts and Methods of Disease Management.* Mexico, D.F.:CIMMYT. 81pp.

Torabi M, Mardoukhi V, Nazari K, Afshari F, Forotan AR, Ramai, MA, Golzar H, Kashani HS. 1995. Effectiveness of wheat yellow rust resistance genes in different parts of Iran. *Cereal Rusts and Powdery Mildews Bulletin* **23**:9-12.

BARLEY YELLOW RUST

Occurrence and Spread of Barley Yellow Rust in the Americas and the Genetic Basis of Resistance

F. Capettini[1], W. M. Brown[2] and V. R. Velasco[2]

[1] ICARDA/CIMMYT Latin American Regional Barley Breeding Program, Mexico D.F., Mexico

[2] Department of Bioagricultural Sciences and Pest Management, Colorado State University, Fort Collins, USA

Introduction

Barley yellow (stripe) rust, is caused by *Puccinia striiformis* Westend. f. sp. *hordei* Eriksson & Henning. A highly virulent race of this pathogen was first introduced into the Americas in 1975 near Bogotá, Colombia. From this region, the pathogen spread south to Ecuador one year later and was found in Peru in 1977, in Bolivia in 1978, in Chile in 1980 and in Argentina in 1982 (Velasco and Brown 1982, Dubin and Stubbs 1986). The pathogen moved north in the following years: in 1987 it was identified in Mexico (Navarro and Zamora 1990) and was first found in the USA in Texas in 1991. In 1993 yellow rust was found in another seven states in the USA and Canada (Line and Chen 1999).

Historically, barley yellow rust occurred in the Middle East, South Asia, and East Africa for many years. Two European races designated as R-23 and R-24 were known to infect mainly barley. Isolates found in South America appeared to be similar to those in Europe (Dubin and Stubbs 1986). Initially, only R-24 was thought to be present in the Americas, but more recently extensive analysis has revealed considerable variation in pathogen isolates (Chen *et al,* 1995).

Yellow rust development is more severe in the cool barley production areas and in mountain regions worldwide. Survival and primary sources of barley yellow rust inoculum are evidently wild grasses and winter barley. *Hordeum muticum* was widely infected with R-24 of yellow rust in the department of Puno, Peru, demonstrating that the spread of the rust through the Andes Mountains did not depend only on cultivated barleys (Dubin and Stubbs 1986).

Barley yellow rust in South America

Until the early 80's most of the barley cultivars planted in Ecuador, Peru, and Bolivia were land varieties. The new disease affected nearly all the commercial

varieties planted in the region, causing severe yield losses. Only two improved cultivars presented moderate levels of resistance at that time, i.e. Zapata in Peru and Dorada in Ecuador. The French variety Grignon was also found to have some resistance in Peru and released in Bolivia with the name of IBTA 80. Estimates of losses based on field surveys in Colombia showed that about 70% of barley yield was lost in 1975. The national barley average yield in Ecuador dropped from 0.88 to 0.62 t/ha from 1976 to 1980. Yield losses in Bolivia in fungicide trials using land varieties varied between 47 to 61% of that in fungicide-treated plots. Although data obtained at that time were imprecise, all indicated that yellow rust had a severe impact on all the barley production areas of the Andean countries (Dubin and Stubbs 1986).

The high susceptibility found in the extremely diverse land varieties indicates that yellow rust was an exotic pathogen in South America, with lack of co-evolution of host and obligate parasite. It was hypothesized that it was introduced to Colombia carried in the undercarriage of airplanes or on contaminated clothes of travelers (Dubin and Stubbs 1986). Within 7 years yellow rust traveled at least 6000 km and infected all major growing areas in South America and continued spreading to North America. This would support Zadoks' suggestion that primary inoculum can travel long distances and cause infection far from where the urediospores were produced. Mountains and deserts were not sufficient barriers to prevent continental dispersal of a windborne pathogen.

Screening barley for resistance to yellow rust

Extensive field screening trials of all available barley germplasm in the USDA/ARS National Small Grains Collection at Aberdeen, Idaho and other barley breeding programs was initiated in 1990. Over 44000 accessions were screened since then and all the information is available in the GRIN data base (Germplasm Resource Information Network: http://www.ars-grin.gov/npgs/) (Brown *et al.* 2001). Barley germplasm resistant to most prevalent pathotypes of the Americas have been also developed in the ICARDA/CIMMYT Barley Breeding Program and has been successfully adopted by many collaborators.

During 2000, collaborators in Peru reported important changes in the resistance patterns of well-known genotypes in that country, which were personally observed in a recent visit in April of this year. Differences in reaction among genotypes in the two main barley experiment stations of the La Molina University were observed. Yellow rust differentials and trap nurseries will be distributed to the Andean countries next year to determine if changes in disease levels on culltivars are due to a new race. The importance of international cooperation became evident in the study and fight against this important barley disease.

Genetics of resistance to yellow rust in barley

Unlike many other fungal diseases of small grains, the genetics of the barley yellow rust resistance and the pathogen are not well known (Brown *et al.* 2001). The first genetic studies cited in the literature were from India and described two dominant resistance genes in the American cultivar Alpha (Chen and Line, 1999). Later work in the same country described 8 resistance loci, Ps1 through Ps8. Ps1 and Ps3 had multiple alleles for a total of 11 genes for resistance, 8 dominant and 3 recessive. The work done in Europe in the 60s described the recessive genes yr1, yr2, and yr3. A fourth gene, yr4 was described later (Chen and Line, 1999). In 1995 two quantitative trait loci in the arms of chromosome 4 and 7 were reported in a study carried out in the US with a six-row spring feed barley line (Chen *et al.* 1995). A more recent study of 18 genotypes found 20 different resistance genes (Chen and Line, 1999). Among those 18 genotypes, 3 had a single resistance gene, and 15 had at least 2 or more genes for resistance, most of them recessive. There were also reports of different types of epistasis in the expression of resistance. Very low correlation between seedling resistance and adult plant resistance has been reported (Sandoval-Islas *et al.* 1998).

Breeding for resistance to yellow rust of barley

Breeding for genetic resistance is the most cost-effective and environmentally appropriate technique for crop disease management. The first step in any resistance breeding program is to obtain sources of resistance to the major pests or diseases. Germplasm collections are important sources of resistance of yellow rust (*Puccinia striiformis* f.sp. *hordei*), and most of the resistant lines identified for this disease are from Ethiopia (Brown *et al.* 2001). Several USDA National Small Grain Collection lines located in Aberdeen, ID, USA, various private and public sector cooperators, and other elite barley lines and cultivars were also identified with differing levels of resistance.

According to the data from the USA (Chen *et al.* 1995), *P. striiformis* f. sp. *tritici* can form numerous physiologic races or pathotypes. There is also evidence of durable adult plant resistance to the disease, and breeding for it would seem to be the best approach. Pyramiding quantitative resistance genes into one genotype should result in better disease control than when such genes are present alone, especially with highly variable pathogen populations, such as yellow rust of barley. The manipulation of quantitative resistance in breeding programs is more difficult than for those genes effective in seedlings but usually providing race-specific resistance. Pyramiding quantitative resistance genes in to one genotype should

result in better disease control than where such genes are present singly (Brown *et al.* 2001). Marker-based strategies can be of great help in a breeding program to produce commercial lines with good quantitative resistance to yellow rust. Castro *et al.* (2000) reported on the effectiveness of pyramiding resistance QTL in a cross of Orca barley, (Cali-sib/Bowman*)/Harrington, finding a relationship between QTL number and level of resistance in the germplasm. The Orca allele in chromosome 4 (4H) was estimated to contribute a 57% decrease in disease severity. The allele in chromosome 5 (1H) reduced severity by 38% and the allele in chromosome 7 (5H) reduced severity by 23%. There was a cumulative effect of pyramiding different resistance QTL. This approach can be used to create genotypes with good levels of resistance, and because the genes are of the quantitative and additive type, it is hoped that they will provide durable resistance.

References

Brown WM, Hill JP, Velasco VR, 2001. Importance of identification of stripe rust resistance in barley. *Proceedingss of the Barley Improvement Workshop*, Cd. Obregon, Mexico. CIMMYT, (in press).

Castro A, Corey A, Filichkin T, Hayes P, Sandoval-Islas JS, Toojinda T, Vivar H, 2000. Development and characterization of stripe rust resistance QTL pyramids in barley. *Plant and Animal Genome* VIII.

Chen XM, Line RF, 1999. Recessive genes for resistance to *Puccinia striiformis* f.sp. *hordei* in barley. *Phytopathology* **89**: 226-32.

Chen XM, Line RF, Leung H, 1995. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f.sp. *hordei* to other rusts. *Phytopathology* **85**:1335-42.

Dubin HJ, Stubbs RW, 1986. Epidemic spread of barley stripe rust in South America. *Plant Disease* **70**:141-144.

Line RF, Chen XM, 1999. Epidemiology and control of barley stripe rust in North America. *Proceedings of the 16th American Barley Researchers' Workshop.* Idaho Falls, ID. July 11-15, 1999. p. 24.

Navarro FM, Zamora MD, 1990. Control quimico de la roya lineal amarilla de la cebada en la Mesa Central. SARH. INIFAP-CIFAEM. Mexico, 10 pp.

Sandoval-Islas JS, Osada KS, Vivar HV, Riquelme IB, 1999. Correlacion entre resistencia en plantula y resistencia en planta adulta a la roya amarilla y a la escaldadura de la cebada. *Agrociencia* **33**:415-422.

Velasco VR, Brown WM, 1982. Stripe rust occurrence and control in Bolivia. *Phytopathology* **72**:975 (Abstr).

Pathogen Variability and the Role of Resistance in Barley Yellow Rust Management

W.M. Brown Jr, J.P. Hill and V.R. Velasco

Department of Bioagricultural Sciences and Pest Management, Colorado State University, Fort Collins, USA.

Introduction

The authors first noted considerable variation between host reactions to yellow rust in their field plots in Bolivia, Colorado and Ecuador (Brown *et al.* 1993, Hill *et al.* 1995). The variation appeared to vary from country to country and in each country from year to year. Variation was also noted by Marshall and Sutton (1995) in their work in Texas. In order to confirm this apparent variation a special differential nursery was developed in cooperation with Dr. Mareike Johnston at Montana State University (Hill *et al.* 1995). The differential nursery was composed of cultivars selected originally by R.W. Stubbs (Hill *et al.* 1995) and additional lines selected by Dr. Johnston. A local variety was also included at each site. The nursery planting of each line consisted of meter long rows and not replicated. The entire nursery was no longer than approximately 10 meters in length.

Race variation studies

The differential nurseries were planted in Bolivia, Colorado, Ecuador, and Germany. Considerable variation was observed both between field nurseries (Table 1) and also under controlled conditions in greenhouse trials. Dr. Johnston tested isolates from Colorado, Montana and Utah against the differentials and when these were compared to the known race 24 and 23 in tests carried out by Dr. Walthers in Germany, considerable variation was again seen (Table 2).

Germplasm evaluation and screening in Mexico, Peru and elsewhere continue to identify a wide range of barley stripe rust resistance sources. Of note though, is that our work, and the work of others show that there is considerable variation in what has been called "race 24". Variation in "race 24" is also supported by observations and work reported in 1994 by Marshall and Sutton (1995) and by Chen and Line (Chen *et al.* 1995, Line and Chen 1996, 1999). It is clear that the barley stripe rust pathogen in North America is a very heterogeneous population.

Discussion and Conclusions

Considerable progress has been made in the identification of sources of resistance to barley stripe rust "race 24" (Brown *et al.* 1993, Hill *et al.* 1995, Velasco *et al.* 1991). The observed variability and the conclusion by the authors and others that the "race 24" designation is not just one race, but potentially many, brings in to question the value of searching for genes that are race-specific. The importance of stripe rust resistance depends on all or any of several characteristics:

Location and environment
Other pests and diseases present
Total context of the cropping system
Economics

Table 1. Yellow rust susceptibility in field differential nurseries in Bolivia, Ecuador and Colorado, 1994.

Differentials	Bolivia	Ecuador	Colorado
Emir	S	MS	MS
Mazurka	MS	MS	S
Zephyr	S	S	S
I 5	MS	S	S
BBA 2890	MS	MS	MS
Trumpf	MS	0	S
Hor 4020	R	MS	MS
Bigo	MS	R	MS
Abed Binder 12	S	S	MS
Cambrinus	S	S	S
Luttichauer Landgerste	MS	S	MS
Heils Franken	MS	MR	S
S 3170 (Hor 3209)	S	MR	S
S 3192 (Hor 2926)	MS	R	S
Grannelose Zweizeilige	R	0	S
Weisse Von Fong Tien	S	S	S
Varunda	MS	R	S
Stauffers Obersulzer	S	MS	S
Hor 1428	MS	S	MS
Morex	S	S	S
Steptoe	S	S	S
Larker	S	S	S
Abyssinian 14	MS	0	MS
Topper	S	MS	S
BBA 809	MR	R	MS
Hokkaido Chevalier	MS	R	S
Hiproly	MS	S	S

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

Each of the above affects the potential usefulness of developing stripe rust resistance in barley. Barley stripe rust is favored by humid and cooler climates, but these do not necessarily prevail in many of the locations in which malting and feed barleys are produced.

Other pests and diseases affecting the crop, such as Russian wheat aphid *(Diuraphis noxia)* and scab *(Fusarium graminearum)* are more important in many areas. Putting a high priority on stripe rust resistance may not be appropriate or the best use of limited resources. Looking at the total cropping system is also important. In some areas such as Colorado, barley stripe rust is only a problem if barley is planted late and even then only in some years. Use of early planting provides an early period when the fungus is not present and allows the major development of the crop in the absence of inoculum.

Table 2. Yellow Rust Selected Differential Reactions in Greenhouse Inoculations

Differentials	GER24	GER23	WPD93	PF94	Utah	Colo
Mazurka	R	S	R	R		R
HOR 4020	R		R, S	I	R	S
Cambrinus	S	R	S	S	R	R
Heils Franken	S	R	R	I	R	I
HOR 1428	R	S	R	R	R	R

The economics of fungicide use are also important. Effective fungicides are available for stripe rust. But in less affluent societies economic resources of the growers prohibit the use of fungicides. Even in more affluent agriculture, the cost benefit ratio of the new generation strobilurin based fungicides may not justify fungicide use. Use of a fungicide to manage stripe rust will not be necessary on a yearly basis due to the sporadic development of the disease in many areas. Thus in those years when the fungus does develop early enough to become a threat, a timely fungicide application may be economic and outweigh the expense and priority of resistance development for stripe rust.

Additionally, other pest or disease problems may not be as amenable to pesticide treatment. Therefore, development and deployment of resistance to those problems may be more important in the total management context.

Knowledge gained from barley screening trials over 10 years, field observations of the epidemiology and work with both seed and foliar fungicide trials suggest that a long range integrated approach is best. Such an approach puts emphasis on the use of trace to moderately susceptible barley lines in combination with other suppression tactics.

Even in using such an integrated approach it is still critical to manage host plant resistance (Brown 1976, Brown 1993, Brown *et al.* 1996) by the use of:

gene rotation, where different sources of resistance are moved in and out of the host plant population,
gene diversity, where a range of resistance genes are present in different cultivars within an area at any one time,
gene protection, where continued and careful monitoring of fields are routinely carried out and initial outbreaks are detected early and spot treated with appropriate fungicides early before the disease can spread.

Therefore a stripe rust program is recommended that uses the following integrated management tactics:

USE A SLOW RUSTING (TMS-5MS) LINE.
TREAT SEED WITH APPROPRIATE FUNGICIDE.
PLANT EARLY.
INSPECT AND APPLY APPROPRIATE FUNGICIDE IF 5% BSR PRIOR TO BOOT.

Aknowledgements

The authors would like to acknowledge and thank our cooperators:
Dr. Ursula Walther (Germany), Ing. Juan Cardova (Bolivia), Ing. Miguel Rivadeneira (Ecuador). Dr. Hugo Vivar and Dr. Flavio Capettini (Mexico), Dr. Luz Gomez Pando (Peru), Dr. Mareike Johnston, Montana State University, USA, Dr. Darrell Wesenberg and Dr. Harold Bockelman at the USDA/ARS Small Grains Laboratory, Aberdeen, ID for providing and preparing barley lines used in this program, Dr. Rollie Line, USDA/ARS at Washington State University for identifying the multiple "races" of barley stripe rust from Colorado and other U.S. collections.

This work is supported by the USDA/ARS Small Grains Laboratory, Aberdeen, ID and the American Malting Barley Association (AMBA).

References

Brown WM Jr, 1976. Emerging Disease Problems in Korean Rice and Their Origins. *Korean Journal of Plant Protection.* 15:39-46.

Brown WM, 1993. The Role of IPM in Contemporary Agriculture and Environmental Issues. In: *Successful Implementation of Integrated Pest Management for Agricultural Crops*. ed, AR Leslie, GW Cuperus. Lewis Publishers, CRC Press, Boca Raton, FL. pp 171-79.

Brown WM, Velasco VR, Hill JP, 1996. Integrated management of barley stripe rust disease: A Case Study in Successful International Cooperation. *Technical Quarterly* **33**:241-45.

Brown WM, Velasco VR, Hill JP, Marshall D, Wesnberg DM, Bockelman HC, 1993. Responses of national small grain collection and other elite barley germplasm to stripe rust race 24. 15th North America Barley Research Workshop, Guelph, Canada. 1992. *Barley Newsletter* **36**:183 (Abstr.)

Chen XM, Line RF, Leung H, 1995. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f.sp. *hordei* to other rusts. *Phytopathology* **85**:1335-42.

Hill JP, Johnston MR, Velasco VR, Brown WM, 1995. Response of selected barley lines to barley stripe rust in Bolivia, Ecuador, Colorado, and Germany. *Phytophathology* **85**:1040.

Line RF, Chen XM, 1996. Wheat and barley stripe rust in North America. *Proceedings of the Ninth Europe and Mediterranean Cereal Rusts and Powdery Mildews Conference*. pp 2-6.

Line RF, Chen XM, 1999. Epidemiology and control of barley stripe rust in North America. *Proceedings of the 16th American Barley Researchers' Workshop*. Idaho Falls, ID. July 11-15, 1999. p. 24.

Marshall D, Sutton RL, 1995. Epidemiology of stripe rust virulence of *Puccinia striiformis* f.sp. *hordei*, and yield loss in barley. *Plant Disease* **79**:732-37.

Velasco VR, Brown WM, Hill JP, Bockelman HE, Wesenberg DM, 1991. Barley stripe rust race 24 resistance identified in field screening trials in Bolivia, SA. *Phytopathology* **81**:1347 (Abstr.)

Identification of Barley Yellow Rust Resistance in the Americas: a Case Study in Successful International Cooperation

W. M. Brown, Jr., J. P. Hill and V. R. Velasco

Department of Bioagricultural Sciences and Pest Management, Colorado State University, Fort Collins, USA.

Introduction

Barley yellow rust, caused by *Puccinia striiformis* f.sp. *hordei,* was introduced into South America from Europe in 1975 (Dubin and Stubbs 1986). It spread to the U.S. in 1991 (Marshall and Sutton 1995) and since that time has been found in all principal barley growing areas of the west. It is now endemic to the northwest United States (Oregon and Washington) and has caused significant damage in the Central Valley of California over the last several seasons. In other malt barley producing areas of the U.S. stripe rust has not yet developed into a significant problem.

In 1990, Dr. Darrell Wesenberg, Director of the USDA/ARS Small Grain Germplasm Collection (NSGC) Research Laboratory at Aberdeen, ID contacted the Colorado State University (CSU) barley disease research team to initiate field trials. The authors and their Bolivian associates were familiar with the fungus and had initiated some of the first studies of the disease in the late 1970s in Bolivia (Velasco and Brown 1982, Velasco and Brown 1980).

Materials and Methods

Field work was established in 1991 in Cochabamba, a high Andean valley approximately 7,500 feet above sea level in Bolivia, South America, which shows a high endemic occurrence of barley yellow rust and heavy infections are a yearly occurrence. Germplasm evaluations and associated studies were continued from in the 1991 growing season (January-May) until 1996. USDA/ARS NSGC staff prepared selections for planting in the trials, while CSU and host country staff managed the field trials.

All trials were planted in non-replicated 1meter long rows (5 gms per entry). Disease severity was recorded as percentage of rust infection on the plants according to a modified Cobb scale. Response, referring to infection types, was recorded

according to the pictorial scale developed by Stubbs and Vecht and used as a standard by the International Center for Maize and Wheat Improvement (CIMMYT). Trials were composed of Group I selections from the National Small Grain Collection that had not been previously evaluated, Group II selections were lines showing resistance or zero reaction from the prior year Group I test. Group III selections were developed and submitted by cooperators from: USDA/ARS (Aberdeen, ID), University of California-Davis, North Dakota State University, Oregon State University, Utah State University, Washington State University, Adolf Coors, Busch Agricultural Resources, Plant Breeders 1, and Western Plant Breeders and some USDA/ARS re-selections.

Group II lines were also tested in Colorado, Ecuador and Germany. In 1995, Ecuador was deleted in favor of adding the Tuloca Valley of Central Mexico. A winter nursery was planted in December 2001 for the first time in Peru. The latter site is also at approximately 7,500 feet and provides an excellent alternative season (winter) location for further screening and accelerated development of germplasm.

Results

Over 44,000 barley accessions were screened from the USDA and other public and private barley breeding programs in the Western United States, resulting in identification of sources of resistance to barley yellow rust through the cooperative efforts of CSU and the USDA/ARS. All this information is available in the USDA/ARS **GRIN** (Germplasm Resource Information Network: http://www.ars-grin.gov/npgs/) data base.

The percentage of lines with resistance to barley stripe rust from all sources became higher with the passing years. This was the result of the successful identification and selection of lines in prior years by the participating breeders in the various programs. This has consistently proved to be an effective method for rapid identification of sources of resistance for cooperating breeders. Large numbers of lines were quickly evaluated, especially in the first 5 years (Table 1). This was especially true of Cooperators' lines submitted (Table 2).

Table 1. Initial Screening for Barley Yellow Rust Resistance at Cochabamba, Bolivia, 1991-1994.

Source	1991	1992	1993	1994	Total
NSGC	7,198	4,112	3,000	2,968	17,278
ELITE	523	2,252	520	680	3,975
COOP		1,019	1,084	2,117	4,220
ADV			598	200	798
Total	7,721	7,383	5,202	5,965	26,271

Table 2. Comparison of Barley Yellow Rust Cooperator results, Cochabamba, Bolivia, 1991-1994

Year	Entries	0	R	MR	%
1991	523	42	27	18	16.6
1992	2,252	208	123	162	21.9
1993	520	86	54	5	27.9
1994	680	152	51	31	34.4

Lines tested in the Toluca Valley of Mexico have consistently been exposed to high levels of natural inoculum and good infection rates have resulted. Selection pressure has been clear and results distinct (Table 3).

Discussion

In the Toluca Valley of Mexico, the environmental conditions and the high level of natural inoculum continue to be conducive to disease, providing good pathogen pressure and subsequent disease development. Susceptible controls consistently show a very high degree of infection (100% in many instances) while the newly released Brancroft (being used as a resistant check) is generally resistant or has, at most, a trace of rust.

Table 3. Number of barley lines tested (#) for yellow rust and percentage in resistance classes 0, R, MR and MS in the Toluca Valley, Mexico, 1995-1999

Year	0		R		MR		MS		Total	
	#	%	#	%	#	%	#	%	#	%
1995	205	78.5	11	4.2	1	0.4	12	4.6	229	87.7
1996	133	32.2	44	12.6	35	10.1	79	16.4	291	71.3
1997	124	47.3	14	5.4	9	3.4	71	27.1	146	83.2
1998	217	79.2	4	1.5	2	0.7	38	13.9	261	95.3
1999	35	41.7	7	8.3	3	3.6	23	27.4	68	81.0

While significant progress has been made identifying barley stripe rust resistance (Brown *et al.* 1996, Brown *et al.* 1993, Velasco *et al.* 1991), there is still a major problem in developing reliable resistant lines for deployment in the near future. There still are just a few commercially available lines with sufficient levels of resistance and the essential qualities necessary for a good malting barley. Bancroft was a joint release from the USDA-ARS at Aberdeen, ID, Colorado State University, Idaho State University and Oregon State University. Additionally,

numerous sources of resistance have been identified and several are being incorporated into commercially available malting varieties to provide sufficient levels of resistance. Levels of resistance being observed in field trials in cooperator lines are very encouraging. This cooperative program is an example of a successful international screening cooperative effort that can be developed in other areas targeting similar or different problems of international significance (Brown *et al.* 1996).

Acknowledgements

Dr. Ursula Walther (Germany), Ing. Juan Cardova (Bolivia), Ing. Miguel Rivadeneira (Ecuador), Dr. Luz Gomez Pando (Peru), Dr. Hugo Vivar and Dr. Flavio Capettini, ICARDA/CIMMYT, (Mexico), Dr. Darrell Wesenberg, USDA/ARS Small Grains Laboratory, Aberdeen, ID, USA, Dr. Harold Bockleman, USDA/ARS Small Grains Laboratory, Aberdeen, ID, USA.
This project is supported by the USDA/ARS Small Grains Laboratory, Aberdeen, ID and the American Malting Barley Association (AMBA).

References

Brown WM, Velasco VR, Hill JP, 1996. Integrated management of barley stripe rust disease: A Case Study in Successful International Cooperation. *Technical Quarterly* **33**:241-45.

Brown WM, Velasco VR, Hill JP, Marshall D, Wesnberg DM, Bockelman HC, 1993. Responses of national small grain collection and other elite barley germplasm to stripe rust race 24. 15th North America Barley Research Workshop, Guelph, Canada. 1992. *Barley Newsletter* **36**:183 (Abstr.).

Dubin HJ, Stubbs RW, 1986. Epidemic spread of barley stripe rust in South America. *Plant Disease* **70**:141-44.

Marshall D, Sutton RL, 1995. Epidemiology of stripe rust virulence of *Puccinia striiformis* f.sp. *hordei*, and yield loss in barley. *Plant Disease* **79:**732-37

Velasco VR, Brown WM, 1982. Stripe rust occurrence and control in Bolivia. *Phytopathology* 72:975 (Abstr.).

Velasco VR, Brown WM, Hill JP, Bockelman HE, Wesenberg DM. 1991. Barley stripe rust race 24 resistance identified in field screenIng trials in Bolivia, SA. *Phytopathology* **81**:1347 (Abstr.).

Velasco VR, Brown WM Jr., 1980. Incidencia y Control de la Raza Nueva de *Puccinia striiformis* en Cebada. III *Seminario Nacional de Investigaciones Agropecuarias y Forestales*. Santa Cruz de la Sierra, Bolivia. March 14-16, 1980.

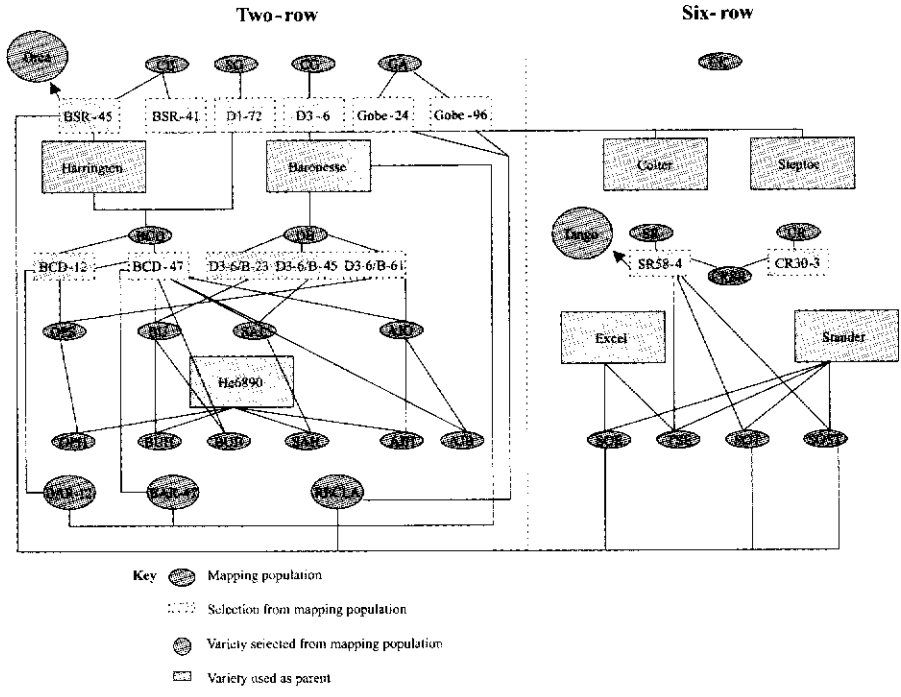
Collaborative Barley Stripe Rust Resistance Gene Mapping and Deployment Efforts

P. Hayes[1], F. Capettini[2], A. Castro[1], A. Corey[1], T. Filichkin[1], M. Johnston[3], C. Rossi[1], S. Sandoval-Islas[4], I. Vales[1], H. Vivar[2] and J. Von Zitzewitz[1]

[1] *Department of Crop and Soil Science, Oregon State University, Corvallis, USA,*
[2] *ICARDA/CIMMYT Latin American Regional Barley Breeding Program. Mexico D.F., Mexico,*
[3] *Dept. of Plant Pathology, Montana State University, Bozeman, Montana, USA,*
[4] *Instituto de Fitosanidad, Colegio de Postrgaduados, Montecillo, Texcoco, Mexico*

Introduction

The collaborative stripe rust resistance research projects in barley described here have multiple objectives. The first and foremost is to provide agronomically-competitive, disease-resistant varieties to a range of clients from the Saraguro Indians of Ecuador to North American malting barley producers. A second objective is to broaden the genetic base of our barley germplasm via the systematic characterization and introgression of unique alleles. A third objective is to contribute to a better understanding of the genetic basis of durable disease resistance in crop plants. Three key components to this research are: (i) phenotyping at ICARDA/CIMMYT facilities at Toluca, Mexico, an environment in which the heritability of disease symptom expression is maximized; (ii) genotyping with molecular markers; and (iii) accelerated germplasm advance. Initially, resistance genes were mapped in ICARDA/CIMMYT germplasm and then introgressed into North American germplasm via marker-assisted selection. More recently, quantitative and qualitative resistance genes were pyramided, with attempts to integrate gene discovery and deployment. The objective of this report is to summarize multiple areas of endeavor that converge on stripe rust resistance gene mapping and deployment: (i) stripe rust epidemiology, (ii) quantitative vs. qualitative resistance, (iii) and linkage mapping and QTL analysis efforts in barley. The results of this collaborative stripe rust resistance mapping and germplasm improvement efforts are summarized in Figure 1. Because this stripe rust resistant germplasm development is an ongoing process, updated summaries will be maintained on the Internet at http://www.css.orst.edu/barley/orbarley/collab.htm.



. Follow links under Summaries: OSU/ICARDA/CIMMYT collaborative disease resistance mapping efforts

Barley stripe rust

Barley stripe rust (*Puccinia striiformis* f.sp. *hordei)* has caused serious yield losses in Europe, in the Asian subcontinent, and in the Americas (Dubin and Stubbs 1986, Hayes *et al.* 1993). The disease was first reported in South America in 1975 and in the U.S. in 1991 (Marshall and Sutton 1995) and by 1995, it was reported in every state of the western U.S. Commercial-scale epidemics have occurred annually in California and Oregon since 1995. Environments in the Pacific Northwest favor the disease: wheat stripe rust (*Puccinia striiformis* f.sp. *tritici)* is the most important disease of wheat in the Pacific Northwest (Line 1993). The two f.sp. of *Puccinia striiformis* are, however, highly specialized: cross-infection is not of economic importance. Initially, only race 24 of *P. striiformis* f.sp. *hordei* was thought to be present in the Americas (Dubin and Stubbs 1986). Recently, more extensive analysis has revealed considerable variation in pathogen isolates collected in the U.S. (Chen *et al.* 1995, Roelfs and Huerta-Espino 1994).

Genetic resistance is the most cost-effective and environmentally appropriate technique for crop disease management. Durability of resistance is a key consideration in disease resistance breeding. Unfortunately, durability can only be demonstrated in hindsight. Barley germplasm developed by the ICARDA/CIMMYT pro-

gram in Mexico allows limited symptom development when exposed to the spectrum of virulence encountered in field tests in South America, Mexico, and the U.S. The fact that this germplasm has remained resistant over a 15-year period may be grounds for describing it as "durable". Sandoval-Islas *et al.* (1998) provided additional evidence for the quantitative and durable nature of the resistance of genotypes in the ICARDA/CIMMYT program.

Quantitative and qualitative resistance

There is an extensive literature on the merits of different types of resistance, and much of the debate is phrased in the context of probable durability (Johnson, 1981). The terminology of disease resistance genetics does justice to the complexity of the subject. The terms "quantitative", "qualitative", "vertical", "horizontal", "partial", and "tolerance" have precise definitions (Browning *et al.* 1977). Resistance phenotypes are a continuum ranging from the hypersensitive response (HR) to a modest reduction in the rate of epidemic development. Throughout the continuum, there are cases representing permutations of locus number, allele effect, race-specificity, stage of expression, and durability (Browning *et al.* 1977). Therefore, locus number, allele effects, stage of expression, and race-specificity need to be defined on a case-by-case basis.

In the case of cereal rusts, a large body of theory has developed regarding the risks associated with race-specific resistance genes (Johnson 1981, Parlevliet 1983, Vanderplank 1978). There is evidence that pathogen virulence can evolve more quickly than plant breeders can deploy single resistance genes in new varieties (Parlevliet 1983). Accordingly, a number of alternative disease resistance breeding strategies have been proposed, and in some cases implemented. One approach is to pyramid multiple race-specific genes into a single genotype (Huang *et al.* 1997, McIntosh and Brown 1997, Mundt 1994).

Another approach is to use resistance genes that do not exhibit gene-for-gene relationships. Distinctions between quantitative and qualitative resistance, adult plant and seedling resistance, and partial and complete resistance are important considerations within this general approach to disease management. At the risk of oversimplification, there is empirical evidence that non-race-specific resistance genes may be more durable than race-specific single genes (Parlevliet 1983). Despite the existence of considerable on quantitative resistance, there are relatively few actual data on the inheritance and mechanism of quantitative resistance. Molecular tools have revealed some unexpected results: unsuspected complexities in some gene-for-gene resistance systems and unsuspected large-effect determinants in some quantitative resistance systems (see reviews by Michelmore 1995, Young 1996). If

quantitative resistance genes are to be useful, it is necessary to understand their effects, interactions, and relationships with genes determining other economically important, quantitatively inherited phenotypes.

The characterization of plant resistance genes at the molecular level has provided information upon which to develop models involving signal detection, signal transduction, and response (Beynon 1997). These studies (Buschges *et al.* 1997, Martin *et al.* 1993, Salmeron *et al.* 1994, Schulze-Lefert *et al.* 1997, Zhou *et al.* 1995) have provided molecular evidence confirming hypotheses based on whole plant data (summarized by Ellingboe 1976, Gabriel and Rolfe 1999) indicating that "monogenic" gene-for-gene relationships are recognition processes that turn on multiple genes in a resistance pathway. At the same time, QTL analysis procedures have facilitated dissection of quantitative disease resistance (see reviews by Michelmore 1995, Young 1996). In some cases, a significant proportion of the total variance in the expression of quantitative traits may be attributable to one locus or a few loci (Chen *et al.* 1994; Hayes *et al.* 1996b, Michelmore 1995, Young 1996), confirming classical quantitative genetic studies (summarized by Vanderplank 1978). This may be an oversimplification due to overestimation of locus effects and underestimation of locus numbers (Beavis 1998, Jansen and Stam 1994, Kaeppler 1997, Melchinger et al. 1998, Utz et al. 2001, Visscher et al. 1996 Zeng 1994). However, the overall picture is one of converging lines of evidence supporting complexity in some qualitative models and simplicity in some quantitative models.

Differentiation of qualitative versus quantitative resistance has long been a source of controversy. At one extreme is the view that these classifications represent genes with distinctly different mechanisms and race specificity (Vanderplank 1968, 1978). At the other extreme is the view that all resistance genes are similar, but are merely expressed differently in different combinations and in different genetic backgrounds (Nelson 1978). Unfortunately, three decades of debate have failed to resolve the issue. Wang *et al.* (1994) used recombinant inbred lines of rice *(Oryza sativa)* to show that a cultivar with durable resistance (*sensu* Johnson, 1981) to rice blast (caused by *Magnaporthe grisea*) contains two race-specific, qualitative genes for resistance and ten QTL contributing to partial resistance *(sensu* Parlevliet 1989). Qi *et al.* (1999) presented evidence for race-specific QTL and argued that quantitative resistance to leaf rust is an example of minor gene-for-minor gene interaction. However, the contribution of the qualitative or quantitative genes to resistance remains unclear.

Barley linkage maps and QTLs

Barley is an excellent system for genome mapping and map-based analyses. This diploid (2n = 14) species has seven cytologically distinct chromosomes containing

approximately 5.3 x 10^9 bp DNA (Bennett and Smith 1976). Although barley is an autogamous species, there is sufficient DNA-level diversity for efficient linkage map construction in populations derived from crosses between related genotypes (Graner *et al.* 1991, Kleinhofs *et al.* 1993, Kasha *et al.*1995, Becker and Heun 1995, Hayes *et al.* 1997). The North American Barley Genome Mapping Project (NABGMP) has focused on building maps in elite germplasm in order to facilitate the direct application of these maps to plant breeding (reviewed by Hayes *et al.* 1996a). Several thousand loci have been placed on these maps, providing a comprehensive catalog of markers. Higher through put markers, such as AFLPs, have been used for barley map construction (Becker and Heun 1995, Hayes *et al.* 1997). Microsatellite polymorphism has been demonstrated (Saghai-Maroof *et al.* 1994) and used for barley germplasm characterization and map construction (Powell *et al.* 1996, Russell *et al.* 1997, Becker and Heun 1995, Toojinda *et al.* 2000). The Scottish Crop Research Institute (SCRI) has a very productive SSR development program (http://www.scri.sari.ac.uk/SSR/). There is currently cooperation with the SCRI in an international barley SSR characterization effort with systematic mapping the SCRI SSRs on NABGMP populations (Toojinda *et al.* 2000).

Linkage maps are useful for understanding genome organization, establishing synteny, as a platform for map-based cloning, and for QTL detection. For a review of QTL detected in barley with references and links, see Hayes *et al.* (1996a), GrainGenes (http://wheat.pw.usda.gov/graingenes.html); and the NABGMP home pages (http://www.css.orst.edu/barley/nabgmp/nabgmp.htm; http://gnome.agrenv.mcgill.ca). In barley, as in other crop species, much of the activity in QTL mapping has been descriptive. The experiments required for validation of estimates of QTL number, effect, and interaction are just coming to fruition. Larson *et al.* (1996), Romagosa *et al.* (1996), Spaner *et al.* (1999) and Zhu *et al.* (1999a,b) have conducted marker-assisted selection experiments to verify QTL for agronomic traits in barley. Han *et al.* (1997) and Marquez-Cedillo *et al.* (2001) have conducted similar experiments for malting quality traits. In all cases, marker-assisted selection was effective for some, but not all QTL. For stripe rust, resistance QTL alleles have been successfully introgressed into a susceptible genotype (Toojinda *et al.* 1998). The limited population sizes used in many of the reported QTL detection experiments may have led to underestimation of QTL number, overestimation of QTL effects, and a failure to quantify QTL interactions (Beavis 1998, Jansen and Stam, 1994, Kaeppler 1997, Melchinger *et al.* 1998, Utz *et al.* 2001, Visscher *et al.* 1996. Zeng 1994).

Mapping and deployment of stripe rust resistance genes in barley

Four qualitative resistance genes, *Yr1* - *Yr4*, were described by Lehmann *et al.* (1975). Of these genes, only the *Yr4* locus has been mapped, and it is on the short arm of chromosome 5 (1H) (von Wettstein-Knowles 1992). A qualitative resistance gene was recently mapped to the long arm of chromosome 1 (7H) in CI10587 (Hayes *et al.* 1999), but the identity of this gene relative to the *Yr1* - *Yr3* genes remains to be established. Resistance QTL were mapped on chromosomes 4 (4H) and 7 (5H) in Calicuchima-sib (Cali-sib), an ICARDA/CIMMYT germplasm line (Chen *et al.* 1994, Hayes *et al.* 1996b). A major adult plant stripe rust resistance QTL was mapped on the short arm of chromosome 5 (1H) in the ICARDA/CIMMYT-derived variety Shyri and smaller-effect QTL were mapped on chromosomes 2 (2H), 3 (3H), and 6 (6H) (Toojinda *et al.* 2000). At the level of resolution afforded by the available maps, the chromosome 5 (1H) QTL coincides with the position of the *Yr4* locus. *Yr4* is reported to confer resistance to race 23 (von Wettstein-Knowles 1992) while the virulence spectrum in the Americas is described in terms of race 24 and its variants (Chen *et al.* 1995). Thomas *et al.* (1995) also mapped an adult plant resistance QTL in the same region in the variety Blenheim and hypothesized that it was an effect of an allele at the *Yr4* locus. A QTL was mapped to the same region on chromosome 5 (1H) in the winter six-row variety Kold and a resistance QTL on chromosome 7 (5H) (at the same position as the chromosome 7 (5H) QTL in Cali-sib), in the CIMMYT/ICARDA spring two-row germplasm CMB643 (Hayes *et al.* 1999). These stripe rust resistance mapping results are summarized in Figure 2.

Deployment of resistance genes has involved marker-assisted selection in various germplasm advance strategies - backcrossing in the case of the six-row variety "Tango" (Toojinda *et al.* 1998) - and more recently pyramiding. The pyramiding efforts, focused on two-row barley, are summarized in Figure 2. The first step in construction of resistance gene pyramids involved positioning the resistance QTL alleles from Shyri and Cali-sib in a malting quality background contributed by Harrington and Galena. these resistance QTL allele pyramid lines constitute the "BCD" population. The acronym has local athletic allusions and stands for "Beavers conquer Ducks". This germplasm has been extensively phenotyped and its allelic structure at the target stripe rust resistance QTL is currently being defined (Castro *et al.* 2000). The second step in the construction of the resistance gene pyramids involved adding a qualitative resistance gene, contributed by CI10587. Because CI10587 is agronomically, the qualitative resistance gene was transferred to Baronesse, the leading feed variety in the Pacific Northwest of the U.S. The quantitative/qualitative resistance gene pyramids are referred to as the "Ajo, Sal, Bu, and Ops" germplasm. These germplasm descriptors have gastro-

nomic allusions; the germplasm itself has been extensively phenotyped. Genotyping is in progress. The third step in pyramid construction has involved He6890, an agronomically attractive Czech selection with uncharacterized stripe rust resistance. The stripe rust resistance breeding effort in six-row barley is not as advanced as the effort in two-row barley. As shown in Figure 2, the six-row effort is currently directed to deploying resistance QTL alleles, tracing to Cali-sib, in a Midwestern malting quality background.

In summary, the ICARDA/CIMMYT program has been very successful in accumulating resistance to multiple diseases in single genotypes. Although this review has focused on stripe rust, this germplasm is also rich in genes conferring resistance to a range of diseases including Barley Yellow Dwarf Virus, leaf rust, net blotch, scald, and spot blotch. This accumulation of disease resistance alleles has been accomplished based on phenotypic data alone, and this success is testimony to Dr. Hugo Vivar's sharp eye and powers of recollection, and to the dedication of his staff. The genotypic characterization of this germplasm should be of assistance to all participants. Faced with a field nursery of agronomically promising germplasm, all of which is phenotypically resistant to diseases, knowledge regarding the genetic architecture of each germplasm accession can be invaluable in deciding which genotypes to advance as varieties, and which genotypes to use as parents in order to continue the allele accumulation process. Knowledge regarding the genetic architecture of exotic germplasm should increase its utility, ensuring a broader germplasm base and providing an impetus for germplasm conservation.

This collaboration has succeeded thanks to the efforts of numerous individuals and to support from a number of organizations. Special thanks to all who have contributed to this research over the years, including Fuqiang Chen, Xianming Chen, Jeanine DeNoma, Aihong Pan, Mareike Johnston, John Korte, Rollie Line, Vicente Morales, Chris Mundt, Doris Prehn, Bernardo Ramirez, and Theerayut Toojinda. For the OSU participants, this research was made possible by the barley growers of Oregon, Washington, and Idaho; the American Malting Barley Association; the Anheuser Busch Company; Busch Agricultural Resource, Inc.; Great Western Malting/ConAgra Malt; the North American Barley Genome Mapping Project; and the Oregon Agricultural Experiment Station. The Colegio de Postgraduados and ICARDA/CIMMYT provided support for their participating staff.

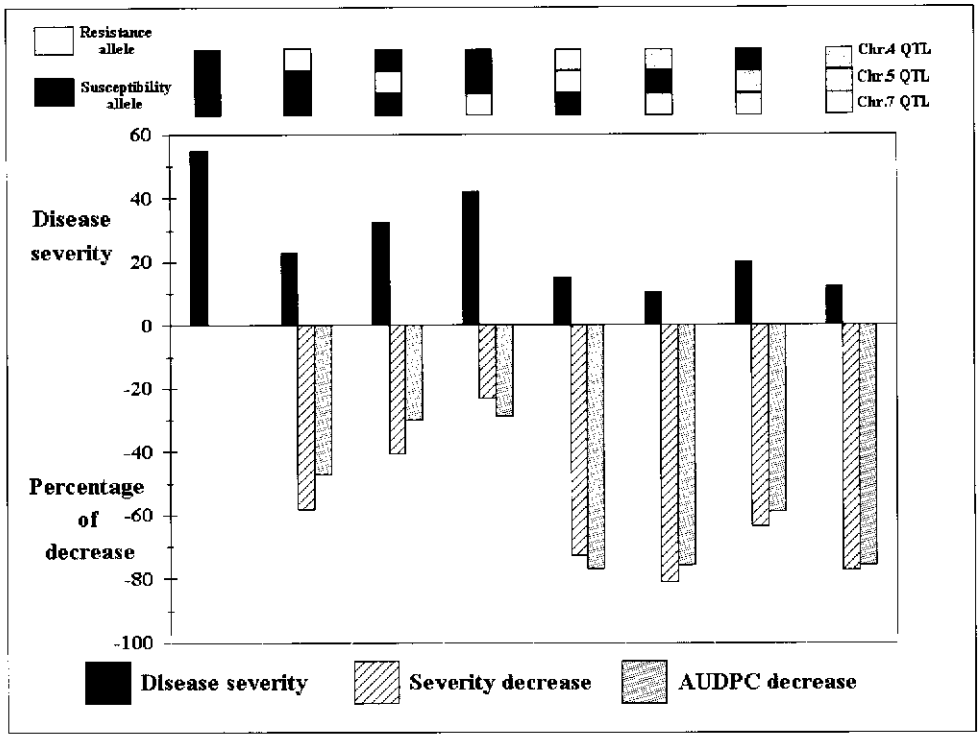


Figure 2. Effects of different combinations of resistance alleles at three BSR resistance QTL on disease severity and decrease of disease severity and area under the disease progress curve (AUDPC). Disease severity values are plotted in the upper panel. The percentage decrease in severity and AUDPC relative to the average of the lines with susceptible alleles at all three QTL (54.8% severity and 912 AUDPC) are shown in the lower panel.

References

Beavis WD, 1998. QTL analyses: power, precision, and accuracy. In. A. H. Patterson (ed). *Molecular Dissection of Complex Traits*. CRC Press, Boca Raton.

Becker J, Heun M, 1995. Barley microsatellites: allele variation and mapping. *Plant Molecular Biology* **27**: 835-845.

Bennett MD, Smith JB, 1976. Nuclear DNA amounts in angiosperms. *Philosophical Transactions of the Royal Society London Biology* **274**: 227-274.

Beynon JL, 1997. Molecular genetics of disease resistance: and end to the gene-for-gene concept? In: I.R. Crute, E.B. Holub, and I.J. Burdon (eds.). *The Gene-for-Gene Relationship in Plant-Parasite Interactions.* CAB International, Wallingford, UK.

Browning JA, Simons MD, Torres E, 1977. Managing host genes: epidemiologic and genetic concepts. In J.G. Horsfall and E.B. Cowling (eds). *Plant Disease: an Advanced Treatise.* Vol. 1. Academic Press, New York.

Buschges R, Hollricher K, Panstruga R, Simons G, Wolter M, Frijters A, Daelen R van, Lee T va de, Diergaarde P, Groenendijk J, Topsch S, Vos P, Salamini F, Schulze-Lefert P, 1997. The barley *Mlo* gene: a novel control element of plant pathogen resistance. *Cell* **88**: 695-705.

Castro A, Corey A, Filichkin T, Hayes P, Sandoval-Islas JS, Toojinda T, Vivar H, 2000. Development and characterization of stripe rust resistance QTL pyramids in barley. *Plant and Animal Genome* VIII.

Chen F, Prehn D, Hayes PM, Mulrooney D, Corey A, Vivar H, 1994. Mapping genes for resistance to barley stripe rust (*Puccinia striiformis* f. sp. *hordei). Theoretical and Applied Genetics.* **88**: 215-219.

Chen X., Line RF, Leung.H, 1995. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f. sp. *hordei* to other rusts. *Phytopathology* **85**: 1335-1342.

Dubin HJ, Stubbs RW, 1986. Epidemic spread of barley stripe rust in South America. *Plant Disease* **70**:141-144.

Ellingboe AH, 1976. Genetics of host-parasite interactions. In: R. Heitefuss and P.H. Williams, (eds). *Physiological Plant Pathology.* Springer-Verlag, Berlin.

Gabriel DW, Rolfe BG, 1990. Working models of specific recognition in plant-parasite interactions. *Annual Review of Phytopathology* **28**: 365-391.

Graner A, Jahoor A, Schondelmaier J, Siedler H, Pillen K, Fischbeck G, Wensel G, Herrmann RG, 1991. Construction of an RFLP map of barley. *Theoretical and Applied Genetics* **83**: 250-256.

Han F, Romagosa I, Ullrich S, Jones B, Hayes P, Wesenberg D, 1997. Molecular marker-assisted selection for malting quality traits in barley. *Molecular Breeding* **3**: 427-437.

Hayes PM, Cerono J, Witsenboer H, Kuiper M, Zabeau M, Sato K, Kleinhofs A, Kudrna D, Kilian A, Saghai-Maroof M, Hoffman D and the North American Barley Genome Mapping Project 1997. Characterizing and exploiting genetic diversity and quantitative traits in barley (*Hordeum vulgare). Journal of Quantitative Trait Loci* http://probe.nalusda.gov:8000/otherdocs/jqtl/jqtl1997-02/

Hayes PM, Chen FQ, Kleinhofs A, Kilian A, Mather D, 1996a. Barley genome mapping its applications. In: P.P. Jauhar (ed.). *Methods of Genome Analysis in Plants*: CRC Press, Boca Raton, USA.

Hayes PM, Chen X, Corey A, Johnston M, Kleinhofs A, Korte J, Kudrna D, Toojinda T, Vivar H, 1999. A summary of barley stripe rust mapping efforts. *Plant and Animal Genome* VII. http://www.intl-pag.org/

Hayes PM, Liu BH, Knapp SJ, Chen F, Jones B, Blake T, Franckowiak J, Rasmusson D, Sorrells M, Ullrich SE, Wesenberg D, Kleinhofs A, 1993. Quantitative trait locus effects and environmental interaction in a sample of North American barley germplasm. *Theoretical and Applied Genetics* **87**: 392-401.

Hayes PM, Prehn D, Vivar H, Blake T, Comeau A, Henry I, Johnston M, Jones B, Steffenson B, 1996b. Multiple disease resistance loci and their relationship to agronomic and quality loci in a spring barley population. *Journal of Quantitative Trait Loci.* http://probe.nalusda.gov:8000/otherdocs/jqtl/index.htm

Huang N, Angeles ER, Domingo J, Magpantay G, Singh S, Zhang G, Kumaradivel N, Bennett J, and Khush GS 1997. Pyramiding of bacterial blight resistance genes in rice:marker-assisted selection using RFLP and PCR. *Theoretical and Applied Genetics*. **95**: 313-320.

Jansen RC, Stam P, 1994. High resolution mapping of quantitative traits into multiple loci via interval mapping. *Genetics* **136**: 1447-1455.

Johnson R, 1981. Durable resistance: definition of, genetic control, and attainment *Phytopathology* **71**: 567-568.

Kaeppler S, 1997. Power analysis for quantitative trait locus mapping in populations derived by multiple backcrosses. *Theoretical and Applied Genetics* **95:** 618-621.

Kasha KJ, Kleinhofs A, Kilian A, Saghai Maroof M, Scoles.GJ, Hayes PM, Chen FQ, Xia X, Li XZ, Biyashev RM, Hoffmann D, Dahleen L, Blake TK, Rossnagel BJ, Steffenson BJ, Thomas PL, Falk DE, Laroche A, Kim W, Molnar SJ, 1995. The North American barley genome map on the cross HT and its comparison to the map on cross SM. In: K. Tsunekawi (ed.) *Plant Genome and Plastome: Their Structure and Evolution.* Kodansha Scientific, LTD, Tokyo, JP.

Kleinhofs A., Kilian A, Saghai Maroof M, Biyashev R, Hayes P, Chen F, Lapitan N, Fenwick A, Blake T, Kanazin V, Ananiev E, Dahleen L, Kudrna D, Bollinger J, Knapp S, Liu B, Sorrells M, Heun M, Franckowiak J, Hoffman D, Skadsen R, Steffenson B, 1993. A molecular, isozyme, and morphological map of the barley *(Hordeum vulgare)* genome. *Theoretical and Applied Genetic* **86**: 705-712.

Larson SR, Kadyrzhanova D, McDonald M, Sorrells M, Blake TK, 1996. Evaluation of barley chromosome-3 yield QTLs in a backcross F2 population using STS-PCR. *Theoretical and Applied Genetics* **93**: 618-625.

Lehman E, Nover I, Scholz F, 1975. The Gaterslaben barley collection and evaluation. In: Gaul H. (ed.) *Proceedings Symposium Barley Genetics III*. Karl Theimig, Munich, pp 64-69.

Line RF 1993. Durability of resistance to *Puccinia striiformis* in North American wheat cultivars. In: *Durability of Disease Resistance*. T. Jacobs and J.E. Parlevliet (eds.). Kluwer Academic Publishers, The Netherlands.

Marquez-Cedillo L.A, Hayes PM, Jones PL, Kleinhofs A, Legge WD, Rossnagel BG, Sato K, Ullrich SE, Wesenberg DM, and the NABGMP, 2001. QTL analysis of malting quality in barley based on the doubled haploid progeny of two elite North American varieties representing different germplasm groups. *Theoretical and Applied Genetics* (in press).

Marshall D, Sutton RL, 1995. Epidemiology of stripe rust, virulence of *Puccinia striiformis* f. sp. *hordei,* and yield loss in barley. *Plant Disease* **70**: 732-737.

Martin GB, Brommonschenkel SH, Chunwongse J, Frary A, Ganal MW, Spivey R, Wu T, Earle ED, Tanksley SD, 1993. Map-based cloning of a protein kinase gene conferring disease resistance in tomato. *Science* **262**:1432-1436.

McIntosh RA, Brown GN, 1997. Anticipatory breeding for resistance to rust diseases in wheat. *Annual Review of Phytopathology*. **35**: 311-326.

Melchinger AE, Utz HF, Schon CC, 1998. Quantitative trait locus (QTL) mapping using different testers and independent population samples in maize reveals low power of QTL detection and large bias in estimates of QTL effects. *Genetics* **149**: 383-403.

Michelmore RW, 1995. Molecular approaches to manipulation of disease resistance genes. *Annual Review of Phytopathology* **33**: 393-428.

Mundt CC, 1994. Use of host genetic diversity to control cereal diseases: implications for rice blast. In: R.S. Zeigler, S.A. Leong, and P.S. Teng (eds). *Rice blast disease*. CAB International, Wallingford, UK.

Nelson RR, 1978. Genetics of horizontal resistance to plant diseases. *Annual Review of Phytopathology* **16**: 359-378.

Parlevliet JE, 1983. Race-specific resistance and cultivar-specific virulence in the barley-leaf rust pathosystem and their consequences for the breeding of leaf rust resistant barley. *Euphytica* **32**: 367-375.

Parlevliet JE, 1989. Identification and evaluation of quantitative resistance. In: *Plant disease, epidemiology: genetics, resistance, and management*, vol. II. K.J. Leonard and W.E. Fry (eds). McGraw-Hill, New York.

Powell W, Baird E, Booth A, Lawrence P, MacAulay M, Bonar N, Young G, Thomas WTB, McNicol JW, Waugh R, 1996. Single and multi-locus molecular assays for barley breeding and research. In: Barley Genetics VII *Proceedings of the International Symposium*. Saskatoon, Canada. July, 1996.

Qi, X., G. Jiang, W. Chen, R.E. Niks, P. Stam, Lindhout P, 1999. Isolate-specific QTLs for partial resistance to Puccinia hordei in barley. *Theoretical and Applied Genetic* **99**: 877-884.

Roelfs AP, Huerta-Espino J, 1994. Seedling resistance in *Hordeum* to barley stripe rust from Texas. *Plant Disease* **78**: 1046-1049.

Romagosa I, Ullrich SE, Hayes PM, Wesenberg DM, and the NABGMP, 1996. Verification of quantitative trait loci for grain yield in the 'Steptoe'/'Morex' barley cross. In (eds.) A.Slinkard, G.Scoles, and B. Rossnagel. V International Oat Conference & VII International Barley Genetics Symposium: Proceedings. University of Saskatchewan, Saskatoon. Poster Sessions Vol. 1: 298-300.

Russell J, Fuller J, Young G, Thomas B, Taramino G, Macaulay M, Waugh R, Powell W, 1997. Discriminating between barley genotypes using microsatellite markers. *Genome* **40**: 442-450.

Saghai-Maroof M.A, Biyashev RM, Yang GP, Zhang Q, Allard RW, 1994. Extraordinarily polymorphic microsatellite DNA in barley: species diversity, chromosomal locations, and population dynamics. *Proceedings of the National Academy of Science,* USA **91**: 5466-5470.

Salmeron JM, Barker SJ, Carland FM, Mehta AY, Staskawicz BJ, 1994. Tomato mutants altered in bacterial disease resistance provide evidence for a new locus controlling pathogen recognition. *The Plant Cell*. **6:** 511-520.

Sandoval-Islas JS, Broers L, Vivar H, Osada K, 1998. Evaluation of quantitative resistance to yellow rust in ICARDA/CIMMYT's barley breeding programme. *Plant Breeding* **117**: 127-130.

Schulze-Lefert C, Haensel P, Freialdenhoven A, 1997. Mutation analysis for the dissection of resistance. 1997. In: I.R. Crute, E.B. Holub, and I.J. Burdon (eds.). *The Gene-for-Gene Relationship in Plant-Parasite Interactions.* CAB International, Wallingford, UK.

Spaner D, Rossnagel BG, Legge WG, Scoles GJ, Eckstein PE, Penner GA, Tinker NA, Briggs KG, Falk DE, Afele JC, Hayes PM, Mather DE, 1999. Verification of a quantitative trait locus affecting agronomic traits in two-row barley. *Crop Science* **39**: 248-252.

Thomas WTB, Powell W, Waugh R, Chalmers KJ, Barua UM, Jack P, Lea V, Forster BP, Swanston JS, Ellis RP, Hanson PR, Lance RCM, 1995. Detection of quantitative trait loci for agronomic, yield, grain and disease

characters in spring barley (*Hordeum vulgare* L.). *Theoretical and Applied Genetics* **91**: 1037-1047.

Toojinda T, Baird E, Booth A, Broers LHM, Hayes PM, Powell W, Thomas W, Vivar H, Young G, 1998. Introgression of quantitative trait loci (QTLs) determining stripe rust resistance in barley: an example of marker-assisted line development. *Theoretical and Applied Genetics* **96**: 123-131.

Toojinda T, Broers LHM, Chen XM, Hayes PM, Kleinhofs A, Korte J, Kudrna D, Leung H, Line RF, Powell W, Vivar H, 2000. Mapping qualitative and quantitative disease resistance genes in a doubled haploid population of barley. *Theoretical and Applied Genetics* **101**: 580-589.

Utz HF, Melchinger AE, Schon CC, 2001. Bias and sampling error of the estimated proportion of genotypic variance explained by QTL determined from experimental data in maize using cross validation and validation with independent samples. *Genetics,* (in press).

Vanderplank JE, 1968. *Disease Resistance in Plants.* Academic Press, New York.

Vanderplank JE, 1978. *Genetic and molecular basis of plant pathogenesis.* Springer-Verlag. Berlin, Heidelberg, New York.

Visscher PM, Haley SC, Thompson R, 1996. Marker-assisted introgression in backcross breeding programs. *Genetics* 144:1923-1932.

Von Wettstein-Knowles P, 1992. Cloned and mapped genes: current status. In: Shewry, P. R., (Ed) *Barley Genetics, Biochemistry, Molecular Biology and Biotechnology*, CAB International, Wallingford, UK, pp. 73-98.

Wang GL, Mackill DJ, Bonman JM, McCouch SR, Champoux MC, Nelson RJ, 1994. RFLP mapping of genes conferring complete and partial resistance to blast in a durably resistant rice cultivar. *Genetics* **136**: 1421-1434.

Young ND, 1996. QTL mapping and quantitative disease resistance in plants. *Annual Review of Phytopathology*. **34**: 479-501.

Zeng ZB, 1994. Precision mapping of quantitative trait loci. *Genetics* **136**: 1457-1468.

Zhou J, Loh YY, Bressan RA, Martin GB, 1995. The tomato gene *Pti1* encodes a serine/threonine kinase that is phosphorylated by *Pto* and is involved in the hypersensitive response. *Cell* **83:** 925-935.

Zhu H, Briceño G, Dovel R, Hayes PM, Liu PH, Liu CT, Toojinda T, Ullrich SE, 1999a. Molecular breeding for grain yield in barley: an evaluation of QTL effects in a spring barley cross. *Theoretical and Applied Genetics* **98**: 772-779.

Zhu H, Cilchrist L, Hayes P, Kleinhofs A, Kudrna D, Liu Z, Prom L, Steffenson B, Toojinda T, Vivar H, 1999b. Does function follow form? QTLs for Fusarium Head Blight (FHB) resistance are coincident with QTLs for inflorescence traits and plant height in a doubled haploid population of barley. *Theoretical and Applied Genetics* **99**: 1221-1232.

Stripe Rust Resistance QTL Pyramids in Barley

A. Castro[1], F. Capettini[3], A. Corey[1], T. Filichkin[1], P.M. Hayes[1], J.S. Sandoval-Islas[2] and H. Vivar[3]

[1]Dept. of Crop and Soil Science, Oregon State University, Corvallis, USA;
[2] Instituto de Fitosanidad, Colegio de Postgraduados, Chapingo, Mexico;
[3]ICARDA/CIMMYT, Mexico, DF, Mexico.

Introduction

Barley stripe rust (*Puccinia striiformis* f.sp. *hordei)* (BSR) is an important barley disease that has caused serious yield losses in Europe, in the Asian subcontinent, and in the Americas (Dubin and Stubbs 1986). It was first reported in South America in 1975, and in the U.S.A. in 1991 (Marshall and Sutton 1995). In 1995, the disease was reported in every state of the western U.S. Commercial-scale epidemics have occurred annually in California and Oregon since then. At least one million of the approximately 3.2 million acres of barley in the western U.S. could be considered at risk to BSR. BSR in the Americas was first described in terms of race 24, which was reported in Europe in the early 1960's and first detected in Colombia in 1975 (Dubin and Stubbs 1986). Considerable variation has been reported in pathogen isolates collected in the U.S. (Chen *et al.* 1995, Marshall and Sutton 1995, Roelfs and Huerta-Espino 1994).

Genetic resistance is the most cost-effective and environmentally appropriate technique for crop disease management. Molecular markers provide the option to select on the genotypic rather than on the phenotypic level, theoretically increasing selection response. However, success in the application of marker-assisted selection depends heavily on the availability of appropriate markers tightly linked to genes conferring resistance (Graner 1996). The use of a gene-for-gene system has the implication that resistance will not remain effective if the pathogen acquires the corresponding virulence by losing the avirulence allele that elicits resistance in the host (Johnson 1992). In order to increase the durability of resistance, two alternatives have been suggested: a) the use of multiple race-specific genes in a single genotype to develop gene pyramids (Mundt 1991); and b) the use of resistance genes that do not show gene-for-gene relationships. There is empirical evidence that such types of resistance genes may be more durable than race-specific single genes (Parlevliet 1983).

In this report "quantitative resistance" will be used to describe resistance that defies easy rating (i.e. resistant vs. susceptible) in the homozygous progeny of a resistant x susceptible cross. This type of resistance can be described in terms of a scale, such as percent severity on a plot basis. A quantitatively resistant genotype will allow some symptom development under intense epidemic conditions. The barley germplasm developed by the ICARDA/CIMMYT program in Mexico allows limited symptom development when exposed to the spectrum of virulence encountered in field tests in South America, Mexico, and the U.S. Sandoval-Islas *et al.* (1998) studied an extensive sample of this germplasm and found that most accessions showed a susceptible reaction when inoculated at the seedling stage under controlled environment conditions with a variant of race 24. However, when the same accessions were inoculated with the same isolate and tested under field conditions, 76% of the lines had adult plant disease severities less then or equal to 10%. ICARDA/CIMMYT germplasm has remained resistant to the spectrum of virulence encountered in the Americas over a 15-year period.

Using germplasm of ICARDA/CIMMYT origin, the Oregon State University (OSU) Barley Project has mapped resistance QTL in several regions of the barley genome (Chen *et al.* 1994, Hayes *et al.* 1996, Toojinda *et al.* 1998, Toojinda *et al.* 2000). Other resistance QTL were mapped by Thomas *et al.* (1995). It is not clear how many genes and what kinds of genes determine a QTL. The relationship between the determinants of quantitative resistance and the single genes that confer complete resistance ("major" or race-specific genes) is still unclear. Von Wettstein-Knowles (1992) reported the only mapped qualitative resistance gene, *Yr4*, located on the short arm of chromosome 5 (1H). The genome-wide distribution of BSR resistance loci is grounds for optimism: multiple resistance genes, both quantitative and qualitative can be combined in single genotypes.

Materials and Methods

BSR resistance QTL in chromosomes 4 (4H) and 7 (5H) were introgressed into 2-row barley germplasm via one cycle of marker assisted backcrossing. Orca, a BSR resistant variety (Hayes *et al.* 2000) selected from the Cali-sib/Bowman mapping population (Chen *et al,* 1994) was used as the resistance donor and Harrington (the U.S. 2-row malting barley standard) was the recurrent parent. One cycle of marker-assisted backcrossing for the chromosome 4 (4H) and 7 (5H) resistance QTL was conducted. Four BC1 plants with the Orca alleles at marker loci flanking the chromosome 4 (4H) and 7 (5H) QTL were crossed with D1-72, a doubled haploid (DH) line from the Shyri x Galena mapping population with a BSR resistance QTL on the short arm of chromosome 5 (1H) (Toojinda *et al.*

2000). One hundred and fifteen DH lines were derived from the cross. The DH lines and the three parents were phenotyped at Toluca, Mexico in 1996, 1998 and 1999 for BSR severity, in a total of seven experiments. Epidemics were generated with local bulk isolates. This procedure was used to detect and measure resistance in the original mapping populations. BSR severity ratings made at multiple growth stages allowed calculation of the area under the disease progress curve (AUDPC) in six of the seven experiments.

The objective is to determine the relationship between QTL number and level of resistance in this germplasm. Phenotype and QTL data on this material could help to answer several questions related with the nature of stripe rust resistance QTL: Are the most resistant lines those with resistance alleles at three QTL (4(4H), 5(1H) and 7(5H)? What is the individual contribution of each resistance QTL allele? Are the effects of the resistance alleles additive?
The DH population was genotyped with markers defining QTL on chromosomes 4 (4H), 5 (1H), and 7 (5H). For bracketing the QTL regions, the strategy was to first screen markers of known map position on Orca, Harrington, and D1-72. As the interest is to define the region of chromosomes 4 (4H) and 7 (5H) that were introgressed from Orca, map positions of markers in these regions was confirmed in the Cali-sib x Bowman mapping population. In chromosome 5 (1H), the interest is in saturating the region tracing to Shyri (via D1-72). The reference population for confirming map position is Shyri x Galena. For genotyping the 115 DH lines, the ideal marker is one that distinguishes among Harrington, Orca, and D1-72. SSRs are the best candidates, in that they may reveal multiple alleles per locus. With co-dominant markers, the minimum ability will be to distinguish between D1-72 vs. Orca or Harrington for chromosome 5 (1H) and Orca vs. D1-72 or Harrington for chromosomes 4 (4H) and 7 (7H). The long-term goal is to define the parental source of the genome segment for each resistance QTL region in each DH line.

Results and Discussion

The population is being genotyped for the target regions in chromosomes 4(4H), 5(1H) and 7(5H). To date in chromosome 4 HvMLO3 was used. In chromosome 5 (1H) Bmac213 was used, a SSR proximal to the resistance QTL mapped in Shyri/Galena. In chromosome 7(5H) three SSRs were used (Bmag337, Bmac303 and HVM30).

Based on the joint ANOVA of the seven experiments (six for AUDPC), the presence of the resistance alleles at the resistance QTL (Orca alleles in chromosomes 4(4H) and 7(5H), and the Shyri (via DI-72) allele in chromosome 5(1H)) is

associated with a lower disease severity and AUDPC (Figure 1). The individual effects of each allele were estimated using multiple regression models. The presence of the Orca allele in chromosome 4(4H) was associated with a significant decrease of disease severity (57%) and AUDPC (49%) compared with lines having susceptibility alleles at all three loci. In the case of the Shyri allele on chromosome 5(1H), its presence was also associated with a significant, but smaller, decrease in disease severity (38%) and AUDPC (28%). The Orca allele in chromosome 7(5H) had the smaller effect: a significant decrease in disease severity of 23% and in AUDPC of 31%.

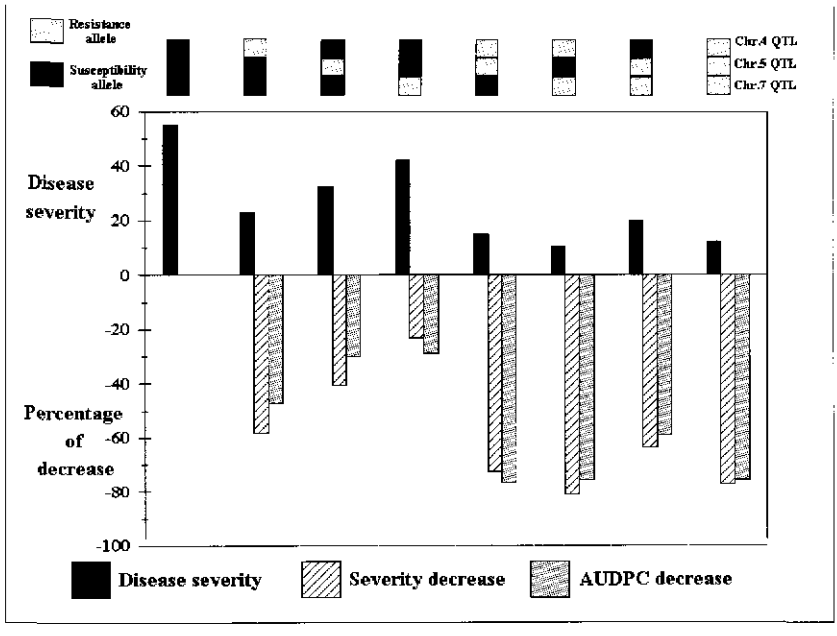


Figure 1. Effects of different combinations of resistance alleles at three BSR resistance QTL on disease severity and decrease of disease severity and area under the disease progress curve (AUDPC). Disease severity values are plotted in the upper panel. The percentage decrease in severity and AUDPC relative to the average of the lines with susceptible alleles at all three QTL (54.8% severity and 912 AUDPC) are shown in the lower panel.

The ANOVA revealed a significant effect of the resistance alleles in terms of decreasing both disease severity and AUDPC, and comparatively little interaction among alleles at the three loci. In the case of the chromosome 4 and 5 QTL for severity, and for all three loci for AUDPC, genotypes with two or three alleles had

similar resistance phenotypes. Otherwise, there was a cumulative effect of pyramiding different resistance QTL alleles in the same line. Genotypes with resistance alleles at two or more loci were always the most resistant and this trend was most apparent from the test in which disease pressure was highest (1996, 1st planting date 1998, 3rd planting date 1999). This could also be attributable to the fact that it is more difficult to rate symptoms consistently at low and intermediate disease levels than at high levels (van Ginkel *et al.* 1996) These data indicate that introgression and pyramiding of resistance alleles is possible and lowers disease severity and AUDPC. The long-term goal is to determine the function and specificity of alleles at each QTL region. The data presented are consistent with a model in which quantitative resistance can be explained by minor genes with additive effects as suggested by Singh *et al.* (This volume) (Parlevliet 1989).

References

Chen FQ, Prehn D, Hayes PM, Mulroney D, Corey A, Vivar H, 1994. Mapping genes for resistance to barley stripe rust *(Puccinia striiformis* f. sp. *hordei). Theoretical and Applied Genetics* **88**: 215-219.

Chen X, Line RF, Leung H, 1995. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f. sp. *hordei* to other rusts. *Phytopathology* **85**: 1335-1342.

Dubin HJ, Stubbs RW, 1986. Epidemic spread of barley stripe rust in South America. *Plant Disease* **70**: 141-144.

Graner AA, 1996. Molecular mapping of genes conferring disease resistance: The present state and future aspects. In: Scoles G. and Rossnagel B. (eds), *V International Oat Conference and VII International Barley Genetics Symposium. VI.- Invited papers.* P.157-166.

Hayes PM, Prehn D, Vivar H, Blake T, Comeau, Henry I, Johnston M, Jones B, Steffenson B, 1996. Multiple disease resistance loci and their relationship to agronomic and quality loci in a spring barley population. *Journal of Agricultural Genomics* **2**. http://www.ncgr.org/research/jag/papers96/paper296/indexp296.html

Johnson R, 1992. Past, present and future opportunities in breeding for disease resistance, with examples from wheat. *Euphytica* **63**. 3-22.

Hayes PM, Corey AE, Dovel R, Karow R, Mundt CC, Rhinart K, Vivar H, 2000. Registration of Orca barley. *Crop Science* **40:** 849-851.

Marshall D, Sutton RL, 1995. Epidemiology of stripe rust, virulence of *Puccinia striiformis* f. sp. *hordei,* and yield loss in barley. *Plant Disease* **79**: 732-737.

Mundt CC, 1991. Probability of mutation to multiple virulence and durability of resistance gene pyramids: further comments. *Phytopathology* **81**: 240-242.

Parlevliet JE, 1983. Race-specific resistance and cultivar-specific virulence in the

barley-leaf rust pathosystem and their consequences for the breeding of leaf rust resistant barley. *Euphytica* **32**: 367-375.

Parlevliet JE, 1989. Identification and evaluation of quantitative resistance. In: K.J. Leonard and W.E. Fry (eds) *Plant Disease Epidemiology: Genetics, Resistance, and Management,* vol. II. McGraw-Hill, New York.

Roelfs AP, Huerta-Espino J, 1994. Seedling resistance in *Hordeum* to barley stripe rust from Texas. *Plant Disease* **78**: 1046-1049.

Sandoval-Islas JS, Broers LHM, Vivar H, Osada KS, 1998. Evaluation of quantitative resistance to yellow rust in ICARDA/CIMMYT's barley breeding programme. *Plant Breeding* **117**: 127-130.

Thomas WTB, Powell W, Waugh R, Chalmers, U.M. Barua UM, P. Jack P, Lea V, Forster BP, Swanston JS, Ellis RP, Hanson PR, Lance RCM, 1995. Detection of quantitative trait loci for agronomic, yield, grain and disease characters in spring barley *(Hordeum vulgare* L.). *Theoretical and Applied Genetics* **91**: 1037-1047.

Toojinda T, Baird E, Booth A, Broers LHM, Hayes PM, Powell W, Thomas WTB, Vivar H, Young G, 1997. Introgression of quantitative trait loci (QTLs) determining stripe rust resistance in barley: an example of marker-assisted line development. Theoretical and Applied Genetics **96**: 123-131.

Toojinda T, Broers LHM, Chen XM, Hayes PM, Kleinhofs A, Korte J, Kudrna D, Leung H, Line RF, Powell W, Vivar H, 2000. Mapping quantitative and qualitative disease resistance genes in a doubled haploid population of barley. *Theoretical and Applied Genetics* **101**: 580-589.

Van Ginkel M, Van der Schaar W, Yang Z, Rajaram S, 1996. Inheritance to scab in two wheat cultivars from Brazil and China. *Plant Disease* **80**: 863-867.

Von Wettstein-Knowles P, 1992. Introgression of quantitative trait loci (QTLs) determining stripe rust resistance in barley: an example of marker-assisted line development. In: Shewry P.R. (ed.) *Barley: Genetics, Biochemistry, Molecular Biology and Biotechnology.* CAB International, Wallingford, UK.

Chemical Suppression of Barley Yellow Rust

W.M. Brown Jr. and V.R. Velasco

Department of Bioagricultural Sciences and Pest Management, Colorado State University, Fort Collins, USA.

Introduction

Early indications in 1979 (Velasco and Brown 1982, Velasco *et al.* 1980) in Bolivia showed that seed treatments with fungicides might be effective in controlling yellow rust on barley led to re-evaluation of this approach.

Methods and Materials

Seed Treatment

Seed treatment field trials were hand planted in Bolivia in January and April in 1998. Seed was commercially treated by the Gustafson Corporation, as indicated in Table 1. The planting in January was at the normal time for the area, prior to the development of high stripe rust. At the second planting, in April, the fungus was very active and producing extremely high stripe rust inoculum pressure.

Foliar fungicide trials

Plots were hand planted and sprays were applied with a manual backpack sprayer at 33 psi at label rates. No adjuvants were used. Disease assessments and yields were recorded. Fungicide trials initiated in 1978 showed at least 2 fungicide applications of tridimefon (Bayleton) were needed to keep the disease below a potentially damaging level (Velasco and Brown 1982, Velasco *et al.* 1980). This and subsequent work by others confirms this, but it is unlikely that two field applications of any fungicides would be economically feasible for barley stripe rust control.

Combination seed treatment and foliar fungicide trials

Integrated trials using seed treatments and foliar spray combinations were carried out in Bolivia from January through April, 1994 and 1995. The trials were planted in January (the normal planting period for the area) and foliar fungicide propiconazole (Tilt) was applied at first sign of stripe rust.

One trial was planted with a local Criolla (land-race) barley and had no seed treatment other than noted (Table 2). The other trial was planted with the variety

Russell. The seed for this trial was supplied by the ARS/USDA Small Grains Laboratory in Aberdeen, ID and had been treated with carboxin (Vitavax). All plots were hand planted and sprays were applied as described above.

Results

Seed treatment

In all cases triadimenol (Baytan) gave significantly enhanced stripe rust protection after emergence (Table 1).

Table 1. Effect of fungicide seed treatment on stripe rust race-24, 1994 at different planting times.

Treatment	January*	April
CBG** 3.00	45 a	23 a
CBG 2.00	45 a	22 a
CBG 1.50	43 a	22 a
CBG 1.00	43 a	23 a
CBG 0.75	43 a	23 a
Captan/Baytan	40 a	19 a
Thiram/Raxil	39 b	14 c
Vitavax extra	39 b	13 c
Vitavax 200	34 c	13 c
Untreated	34 c	13 c

*days after emergence when first pustules observed
**Captan/Baytan/Gaucho

Foliar fungicides

These trials demonstrated that foliar applications of propiconazole (Tilt), tebuconazole (Folicur) or tridimefon (Bayleton) applied at first sign of stripe rust were effective against stripe rust (Brown *et al.* 1996, Velasco and Brown 1982, Velasco *et al.* 1993). Other trials have demonstrated effective control with additional fungicides, including strobilurins (Calhoun *et al.* 1988, Line 1998, 1999, Navarro and Zamora 1990).

Combination of seed treatment and foliar fungicide

All combinations of triadimenol (Baytan) and foliar fungicides enhanced disease suppression and resultant yield (Table 2). Of considerable interest is a trial (Table 3) where a combination of carboxin (Vitavax) and triadimenol Baytan gave better protection than two foliar sprays without seed treatment. When compared to the efficacy of triadimenol (Baytan) alone in the trial with a Criolla cultivar (Table 2), it indicated a possible synergistic effect between carboxin (Vitavax) and triadimenol (Baytan). Subsequent trials in 1995 supported this initial observation with specially chosen treatment combinations.

Table 2. Barley Stripe Rust R-24 Fungicide Trial on a Criolla cultivar, 1994

Treatment	Applications	Rate (kg/ha)	Disease severity (%) at Milk Stage	Yield (kg/ha)
B/T	2	**	0.55 a	3,537.66 a
B/T	1	**	7.35 a	3,177.50 a b
Baytan		*	12.72 a	3,140.00 a b
Tilt	2	0.6	13.22 a	2,532.00 a b
Tilt	1	0.6	66.77 b	2,499.16 b
Control	-	-	91.05 c	1,094.16 c

* as seed treatment only, 200g/100kg seed.
** Baytan/Tilt at 0.6kg/ha and 200g/100kg seed.

Discussion and Conclusions

Chemical suppression of yellow rust is possible. Fungicide trials initiated in 1978 showed that at least two applications of tridimefon (Bayleton) were necessary to keep the disease below a potentially damaging level (Velasco and Brown 1982, Velasco *et al.* 1980). Subsequent work by others supports this, but it is unlikely that two field applications of any fungicides would be economically feasible for barley stripe rust control.

All combinations of triadimenol and foliar fungicides enhanced disease suppression and resultant yield. A combination of carboxin and triadimenol gave better protection than two foliar sprays without seed treatment (Table 3) indicating a possible synergistic effect between carboxin and triadimenol.

Table 3. Barley Stripe Rust R-24 Fungicide Trial on cultivar Russell, 1994

Treatment	Applications	Rate (kg/ha)	Disease severity (%) at Milk Stage	Yield (kg/ha)
Baytan		*	0.82 a	3,352.66 a
Tilt	2	0.600	7.90 a b	3,075.83 a b
Bayleton	2	1.000	8.20 a b	2,715.83 b c
Folicur	2	0.750	25.45 b c	2,553.33 b c d
Manzate	2	2.000	27.47 c	2,240.83 c d
Control	-		73.77 d	2,002.50 d

*seed treatment only, 200g/100kg seed.
All seed treated with Vitavax.

Acknowledgement

This work was supported by the USDA/ARS Small Grains Laboratory, Aberdeen, ID and the American Malting Barley Association (AMBA).

References

Brown WM, Velasco VR, Hill JP, 1996. Integrated management of barley stripe rust disease: A Case Study in Successful International Cooperation. *Technical Quarterly* **33**:241-45.

Calhoun DS, Arhana, Vivar HE, 1988. Chemical control of barley stripe rust, a new disease for North America. *Barley Newsletter* **32**:109-12.

Line RE, 1998. Control of Stripe Rust and Stem Rust of Spring Barley with Seed Treatments and Foliar Fungicides, 1997. F & N *Tests* **53**:383-386.

Line RE, 1999. Control of Stripe Rust of spring Barley with Foliar Fungicides, 1998. *F & N Tests* **54**:299-303.

Navarro FM, Zamora MD, 1990. Control quimico de la roya lineal amarilla de la cebada en la Mesa Central. SARH. INIFAP-CIFAEM. Mexico. 10 pp.

Velasco VR, Brown WM, 1982. Stripe rust occurrence and control in Bolivia. *Phytopathology* **72**:975 (Abstr.).

Velasco VR, Hill JP, Brown WM, 1993. Barley stripe rust-race 24 control with triazole fungicides. *Phytopatholgy* **83**:1335 (Abstr.).

Velasco VR, Brown WM Jr, 1980. Incidencia y Control de la Raza Nueva de *Puccinia striiformis* en Cebada. III *Seminario Nacional de Investigaciones Agropecuariasy* Forestales. Santa Cruz de la Sierra, Bolivia. March 14-16, 1980.

Building up Multiple Disease Resistance in Barley

F. Capettini[1], H. Vivar[2], L. Gilchrist[2] and M. Henry[2]

[1] ICARDA/CIMMYT Latin American Regional Barley Breeding Program, Mexico D.F., México;
[2] CIMMYT, Mexico D.F., México.

Introduction

Breeding to incorporate multiple disease resistance has been one of the main goals in the ICARDA/CIMMYT Barley Breeding Program. An enhanced genetic pool and varieties released with resistance to the main barley diseases were generated in an agronomically improved genetic background. Assessing in adequate environments that assured higher heritability for selecting the different diseases was one of the main reasons for the success obtained.

The Diseases

Yellow Rust *(Puccinia Striiformis)*
Selection for yellow rust has been carried out since its appearance in the region in 1984. Sources of resistance were obtained after the screening of approximately 20000 samples in Colombia and Mexico. Important material is inoculated at the Toluca Experiment Station through the creation of infection borders and hills planted with susceptible genotypes. Partial resistance to this disease was found and contrasting cultivars with differences in the latent period of infection were characterized, obtaining what was hoped to be a more durable type of resistance.

Leaf Rust *(Puccinia hordei)*
Results obtained on Leaf rust research from Montana and Holland helped to identify parents to use in the breeding program. Every year 10-12 ha of segregating populations and yield experiments in the Yaqui valley are artificially inoculated through infection borders using fresh pathogen spores. Epidemics are almost always present and selection is efficiently carried out for this disease.

Scald *(Rhyncosporium secalis)*
In the 1980s, the sample of entries from the world collection showing resistance after being screened in California were introduced to Mexico as sources of resistance to the program. The environmental conditions at Toluca are usually optimal for the development of this important barley disease. Every year 7-10 ha of

experiments and segregating populations are artificially inoculated, creating relatively high epidemics that easily differentiate genotypes with different levels of resistance. Previous research found that disease development of resistant genotypes had small AUDPC values as compared to susceptible ones. This slow-scalding genetic pool is frequently used in the program.

Fusarium Head Blight *(Fusarium graminearum)*
Selection for this devastating disease (FHB) started in 1995 in response to its rapid increase in importance in North and South America. Twenty-three lines presenting different degrees of resistance after screenings carried out in Japan and Mexico were used as initial sources for the resistance program. Genotypes were screened under artificial epidemics created at Toluca. The ICARDA/CIMMYT program was among the pioneers in screening and describing the independently inherited Type I (initial infection) and Type II (fungus spreading) types of resistance in barley, which had been previously described in wheat. Genotypes having both types of resistance were identified and confirmed through several years of testing, and are widely used as resistance sources.

Barley Yellow Dwarf Virus
Research on BYDV aims to characterize genotypes for their individual reaction to three biotypes: MAV, PAV and RPV. BYDV symptoms are frequent under natural conditions at Toluca and selection against susceptible genotypes are usually carried out, but artificial inoculation with greenhouse-reared aphids is done in screening nurseries under field conditions to assure uniform infection, to differentiate biotype reaction and reduce the risk of escapes. Four plots are planted with each genotype and three of them are inoculated with one biotype each. The fourth plot is a check kept free of aphids by insecticide applications.

Assembling Resistant Genotypes
To incorporate resistance to all these diseases into a high yielding genetic background, the approach of creating templates was followed. At the first stage, resistance to scald and leaf rust were incorporated, followed by templates where resistance to yellow rust and to other diseases were added. This process was continued for 20 years, with two generations per year, to pyramid resistance to diseases described above and also net blotch, spot blotch and stem rust.

One of the examples of success was the variety Shyri, released in Ecuador in 1989. The disease resistance present in the variety Shyri was studied in detail at Oregon State University (OSU) using molecular markers. QTLs for resistance to scald, net blotch, BYDV, yellow rust and leaf rust were found. Shyri was found to be also resistant to FHB and partially resistant to leaf rust.

Table 1. Sample six- and two-row genotypes resistant to at least five diseases and with high yield.

Variety or Pedigree	BYDV PAV	BYDV MAV	BYDV RPV	Yellow Rust	Leaf Rust	Scald	Grain Type	Stem Rust	Yield (t/ha)
Six-row									
EGYPT4/TERAN78//P.STO/3/QUINA	R	R	R	R	40S	TR	C		9.0
BELLA UNION	R	R	R	30S	TR	TR	C		8.2
ALPHA/DURRA//CORACLE/3/ALELI									
4/MPYT169.1Y/LAUREL//OLMO/5/									
GLORIA-BAR..	R	R	R	R	TR	R	C		8.0
DC-B/SEN/3/AGAVE/YANALA//									
TUMBO/4/CEN-B/2*CALI92	R	R	R	5S	TR	MS	C		7.3
PETUNIA 1	R	R	R	5MS	TR	R	D	R	7.1
BBSC/CONGONA	R	R	R	R	TR	TR	D		6.8
CARDO/VIRDEN//ALOE	R	R	R	-	TR	-	C		6.7
PALTON	R	R	R	TR	TR	TR	C		6.6
DC-B/SEN/3/AGAVE/YANALA//									
TUMBO/4/CEN-B/2*CALI92	R	R	R	5MS	TR	TR	C		6.5
QUINN/ALOE//CARDO	R	R	R	TR	TR	TR	C		6.4
SEN/SLLO/3/RHODES/CI14100/									
/LIGNEE527	R	R	R	30S	TR	R	C		6.4
MONROE/4/ASE/3CM//RO-B/3/SMA1									
/5/MATICO	R	R	R	R	TR	R	C		6.3
Two-row									
MADRE SELVA	R	R	R	R	TR	R	C	TS	7.1
ABN-B/KC-B//RAISA/3/ALELI	R	R	R	TR	TR	R	C		6.9
CONDOR-BAR/3/PATTY.B/RUDA/									
/ALELI/4/ALELI	R	R	R	TR	TR	R	C		6.7
ARUPO*2/KC-B//ALELI	R	R	R	R	TR	S	C		6.7
LIMON	R	R	R	TR	TR	R	C	TS	6.6
INCIENSO	R	R	R	5MS	TR	TR	C	TS	6.5
COMINO/3/MATICO/JET//SHYR1/4/									
ALELI	R	R	R	R	TR	R	C		6.5
POROTILLO	R	R	R	R	TR	TR	D		6.3
HLLA/GOB//HLLA/3/CANELA	R	R	R	-	10MS	-	C		5.8
CALENDULA	R	R	R	R	TR	R	D		5.7
GOBERNADORA/HUMAI10/									
/CANELA/3/ALELI	R	R	R	-	TR	-	C		5.4
DUMARI	R	R	R	10S	TR	TR	D	VS	5.3

Other examples of success of the program occurred in China. The area planted in that country in with germplasm developed by this program is estimated to account for 40% of the one million ha planted with barley. The main reason for this is the resistance of the germplasm to FHB, tolerance to barley yellow mosaic virus and high yield potential. In several provinces the variety Zhenmai-1 (Gobernadora) had 20-25% higher yield than the local varieties. In a genetic study carried out by OSU, a large effect QTL was found for FHB Type II resistance near the centromeric region of chromosome 2.

Besides these examples of success of the breeding program through the impact obtained by the release of cultivars in different countries, success could be also

measured by the large germplasm pool with resistance to different diseases in an improved agronomic background that is available to breeding programs world-wide.

Table 2. Genotypes with higher levels of resistance to FHB in more than three years of testing. Many genotypes combine 2-3 different sources of resistance.

	Pedigree	Head Type	Damage % Type I	Damage % Type II
1	TOCTE//GOB/HUMAI10/3/ATAH92/ALELI	2	5.6	7.07
2	PENCO/CHEVRON-BAR	6	1.51	17.32
3	ZHEDAR#1/SHYRI//OLMO	2	5.68	8.04
5	ATAH92/GOB	2	4.88	4.27
6	CANELA/ZHEDAR#2	2	5.28	5.33
7	MNS1	6	3.43	17.12
8	ZHEDAR#1/4/SHYRI//GLORIA-BAR/COPAL /3/SHYRI /GRIT/5/ARUPO/K8755//MORA	2	3.21	4.03
9	SVANHALS-BAR/MSEL//AZAF/GOB24DH	2	3.29	8.76
	CHECKS			
	AZAFRAN (MR-R)	2	8.5	8.3
	GOBDH83(R-R)	2	5.1	7.6
	GOBDH89(S-S)	2	13.4	27.7
	PENCO/CHEVRON-BAR (R-MR)	6	4.69	12.05

Concluding Remarks on the First Regional Yellow Rust Conference for the CWANA Region

R. Johnson

The major contribution of this conference was the opportunity it provided to share experiences and views about how to control the yellow (stripe) rust diseases of wheat and barley. The broad consensus of opinion was that the best option for control of these diseases is the incorporation of genetically controlled resistance by plant breeding. This apparently simple option is not so easy to apply because of the variation exhibited by the pathogens *Puccinia striiformis* f. sp. *tritici* and *hordei*. Resistance incorporated by breeders is too often rendered ineffective when new virulence in the pathogen appears and spreads in epidemic proportions. Numerous recent epidemics of yellow rust on wheat in the CWANA region are noted in the papers. It is evident that these epidemics were favoured by the widespread cultivation of single cultivars, not only nationally but also internationally, as illustrated by the selection of Seri 82 under different names by many national programmes, such as Falat in Iran. Seri 82 and its related Veery lines possessed two race-specific genes *Yr7* and *Yr9* and as virulence for this combination spread, the lines were too susceptible for commercial use in many areas and suffered serious losses of yield. Combined with increasing use of irrigation, which favours the development of the disease, this generated a large population of the pathogen. This, in turn, led to great diversity in the pathogen population so that virulence for other cultivars appeared, and other areas, normally less affected by yellow rust, were also affected. In the context of effects of environment on the amount of yellow rust affecting cultivars, it is interesting to note that Falat, on which there were epidemics near the Caspian Sea, is still grown successfully, and is recognised as slow-rusting, in Khuzestan Province of Iran. There, the environment is less favourable to yellow rust, but cultivars more susceptible than Falat can suffer severe infections.

Despite the diversity of the pathogen population, it is not unlimited, and as so often, cultivars from other areas in trials remain resistant. Some of these may be highly susceptible in their own original areas, but the evidence for this may remain unappreciated, despite the distribution of the seeds. Examples in this volume show how important communication is between the countries of the CWANA region, and with the rest of the world and the literature to prevent recurrent problems, already known in one area, spreading to other areas. Problems of the uncontrolled spread of Seri 82 derivatives through the region years after the first epidemics of yellow rust had occurred in other areas amply demonstrate the need for communication.

While the diversity of the pathogen population creates problems, there is emerging, slowly, more information about the examples of durable resistance to yellow rust of wheat, and more information about the genetic control of such resistance, as reported in several contributions. The evidence is that adequate levels of resistance could be obtained with a few additive genes each of small to moderate effect. Despite this increasing information, the application of programmes to achieve durable resistance to yellow rust of wheat needs to be improved, as noted in the first short paper from CIMMYT. It is to be hoped that some co-operative programmes, with the objective of finding out more about durable resistance to yellow rust of wheat and how to use it, will be stimulated by this regional meeting.

There are several papers on barley yellow rust, all from America. These indicate diversity in the pathogen population, and the occurrence of important epidemics after arrival of the barley-attacking strain (f. sp. *hordei*) in South America in 1975. There were no corresponding reports from outside America, and it should perhaps be noted that epidemics of yellow rust on barley are rare in the old world. This is because most barley cultivars of the old world possess durable adult-plant resistance to the disease. It is to be hoped, therefore, that research in America will not follow the path of introducing major resistance genes of unknown provenance, that may well prove to be race-specific, to control this disease, but will concentrate on the exploitation of durable resistance.

List of Participants

A

Dr. Colin R. Wellings
Senior Research Scientist
Plant Breeding Institute, Cobbitty
Private Bag 11, Camden 2570
Australia

Dr. Harbans Bariana
National Cereal Rust Control Program
Plant Breeding Institute-Cobbitty
Private Bag 11, Camden, NSW 2570
Australia

Dr. Ehtibar Ibragimov
Azerbaijan Research Institute
Sovkhoz 2, Baku, 370098
Azerbaijan

C

Dr. L. Lamari
Dept. of Plant Science
University of Manitoba
Winnipeg MB.R 322N2
Canada

D

Dr. Mogens S. Hovmöller
Research Center Flakkebjerg
Danish Institute of Agricultural Sciences
DK-4200
Denmark

E

Dr. S. Sherif
Plant Pathology Research Institute
Cereal Disease Research Department
Agricultural Research Cooperation (ARC)
Giza,
Egypt

I

A. Akbari
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. Alizadeh
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. Arzani
Department of Agronomy, College of Agriculture,
Isfahan University of Technology
Isfahan,
Iran
a_arzani@cc.iut.ac.ir

A. Barati
College of Agriculture
University of Mazandaran,
Sari, Mazandaran,
Iran

A. Dariaee
Research Institute of Dryland Agriculture
Sararoud, Kermanshah,
Iran
Dariaee342@yahoo.com

A. Foroutan
Seed and Plant Improvement Institute
Karaj,
Iran
Foroutn_2000@yahoo.com

A. Ghandi
Agricultural Research Center of Isfahan
Isfahan,
Iran

A. Heidari
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. Keshavarz
Seed and Plant Improvement Institute
Jaraj,
Iran

A. S. Kashani
Agricultural Research Center of Kermanshah
Kermanshah,
Iran

A. *Malihipour*
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. Mohamadi
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. Naderi
Agricultural Research Center of Khuzestan
Ahwaz,
Iran

A. A. Noorinia
Gorgan Agricultural Research Center
Gorgan,
Iran
noorinia@areeo.or.ir

A. Pakniat
Agricultural Research Center of Fars
Zarghan,
Iran
Pakniat @yahoo.com

A. Saidi
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. S. Pourshahbazi
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. *Rezaei*
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. Yazdansepas
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

A. Yousefi
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

B. Ghareyazie
Agricultural Research Extension and Education Organization (AREEO)
Tehran,
Iran

B. Sharifnabi
College of Agriculture
Isfahan University of Technology
Isfahan,
Iran
Sharifna@cc.iut.ac.ir

B. Sohayli
Agricultural Research Station of Ardabil
Ardabil,
Iran

E. Alizadeh
Agricultural Research Station of West Azarbiajan
P.O. Box 383
Khoy,
Iran

F. Afshari
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

F. Khelghati
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

F. Talaei
Agricultural Research, Education and Extension
Tehran,
Iran

H. Asadi
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

G. Aminzadeh
Agricultural Research Station of Ardabil
Ardabil,
Iran

H. Ghazvini
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

H. Jafari
Zanjan Agricultural Research Center
Zanjan,
Iran

Dr. H. Ketata
ICARDA
AREEO, Tehran
Iran
H.Ketata@cgiar.org

H. Mohammadkhani
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

H.R. Niknejad
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

J.A. Jafarbai
Agricultural Research Center of Golestan
Gorgan,
Iran

K. Keshavarz
Gachsaran Agricultural Research Center
Gachsaran,
Iran

K. Nazari
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

K. Nourollahi
Agricultural Research Center of Ilam
Ilam,
Iran

K. Rashmekarim
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

M. Aghaee Sarbarzeh
Dryland Agricultural Research Institute
Kermanshah,
Iran
Maghaee@yahoo.com

M. Ata-Hossaini
Khorasan Agricultural Research Center
Mashhad,
Iran

M. Damadzadeh
Agricultural Research Center of Isfahan
Isfahan,
Iran

M. Esmaeilzadeh
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

M. Hassanpour Hosni
Dryland Agricultural Research Institute (DARI)
Maragheh,
Iran
m-hassanpour@areeo.or.ir

M. Kalateh-Arabi
Agricultural Research Center of Golestan
Golestan,
Iran

M. Khodarahmi
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

M. Nasrollahi
Agricultural Research Station of Boroujerd
Boroujerd,
Iran

M. Patpour
Seed and Plant Improvement Institute
Karaj,
Iran

M. Radmehr
Agricultural Research Center of Khuzastan
Ahwaz,
Iran

M. Seraj-Azari
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

M. Torabi
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

M. Vahabzadeh
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

M. Zamani
Maize and Forage Crop Research Department
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

M.A. Dehghan
Agricultural Research Center of Golestan
Gorgan,
Iran

M.R. Jalal Kamali
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

M.R. Moini
Agricultural Research Center of Zanjan
Zanjan,
Iran

M. R. Naghavi
College of Agriculture
Tehran University
Karaj,
Iran

M.S. Ahmadian Moghadam
Agricultural Research Center of Mazandaran
Sari,
Iran
spii.cereal@abdnet.com

R. Aghnoom
Agricultural Research Center of Khorasan
Mashhad,
Iran

R. Houshyar
Agricultural Research Station of Miandoab
Miandoab,
Iran

R. Joukar
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

R. Roohparvar
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

S.A. Safavi
Agricultural Research Station of Ardabil
Ardabil,
Iran

S.A. Kouhkan
Agricultural Research Center of Zabol
Zabol,
Iran

S.T. Dadrezaei
Agricultural Research Center of Khuzestan
Ahwaz,
Iran

V. Mardokhi
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

V. Rahjou
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

Y. Arshad
Genetics and Plant Genetic Resources Department
Seed and Plant Improvement Institute
Karaj,
Iran

Z. Nategh
Seed and Plant Improvement Institute
Karaj,
Iran
spii.cereal@abdnet.com

K

Dr. Alma Kokhmetova
Institute of Plant Physiology,
Genetics and Bioengineering 45,
Timiryasev Street, Almaty 480090,
Kazakhstan

Dr. A. Kokhmetov
Kazak Research Institute of Farming,
Almaty,
Kazakhstan

Dr. M. Koishibayev
National Academic Center of Agrarian Research (NACAR)
Republic of Kazakstan
79, Abylai Khan Ave., Almaty,
480091,
Kazakhstan

Dr. Myra Junusova
Kyrgyz Agrarian Academy
68, Mederova Street, Bishkek, 720005,
Kyrgyz Republic

M

Dr. F. Capettini
ICARDA/CIMMYT Latin American
Regional Barley Breeding Program
Apartado Postal 6-641
Mexico D.F.,
Mexico

Dr. R.P. Singh
International Maize and Wheat
Improvement Center (CIMMYT)
Apartado Postal 6-641
Mexico D.F.,
Mexico

P

Dr. Iftikhar Ahmad
Crop Diseases Research Institute,
Islamabad,
Pakistan

S

Dr. William Erskine
Assistant Director General for
Research
ICARDA
P.O. Box 5466
Aleppo,
Syria
W.Erskine@cgiar.org

Dr. Amor Yahyaoui
ICARDA
P.O. Box 5466
Aleppo,
Syria
A.Yahyaoui@cgiar.org

Dr. Michael Baum
ICARDA
P.O. Box 5466
Aleppo,
Syria
M. Baum@cgiar.org

Mr. Munzer El-Naimi
ICARDA
P.O. Box 5466
Aleppo,
Syria
M.Naimi@cgiar.org

T

Dr. Farkhod Kosimov
Research Institute of Crop
Husbandary, V. Sharora, Gissarskiy,
distr.,
Tajikistan
Dr. Fazel Dusunceli
Central Research Institute for
Field Crops, 4 Ulus, Ankara,
Turkey

U

Dr. R. Johnson
16, Coppice Avenue, Great Shelford,
Cambridge CB2 5AQ,
UK

Dr. William M. Brown, Jr.
Professor
Dept. of Bioagricultural Sciences and
Pest Management
Colorado State University,
Fort Collins, Colorado 80623-1177,
U.S.A.

Dr. Aziz Nurbekov
Galla-Aral Branch of Andijan Research
Institute of Grain,
Olimlar Makhallasi, 704620 1
M. Amonov Street, Djizzak Region,
Uzbekistan

Dr. Valentina Khokhlacheva
Institute of Genetics and
Experimentation
Under the Academy of Sciences of the
Republic of Uzbekistan
702151, P.O. Yukori-Yuz, Kibray
District, Tashkent Region,
Uzbekistan

ICARDA-17/350/Nov.2002