Identification and characterization of resistance to yellow rust and powdery mildew in wild emmer wheat and their transfer to bread wheat

Promotor: dr. ir. J. E. Parlevliet,
hoogleraar in de plantenveredeling

C. H. van Silfhout

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Proefschrift
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op gezag van de rector magnificus,
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This thesis has been accomplished at the Research Institute for Plant Protection (IPO). The research was part of a cooperative project between this institute and the Agricultural Research Organization (ARO) in Israel. The project was co-funded by the Netherlands Minister for Development Cooperation.

STELLINGEN

1. De hypothese verwoord door Dinoor en Eshed dat resistentiegenen in natuurlijke ecosystemen zorgen voor een evenwicht tussen waard en parasiet is in zijn algemeenheid niet juist.
   Dit proefschrift
   A. Dinoor and N. Eshed. 1984. Ann. Rev. Phytopathol. 22:443-66

2. De stelling dat, ten behoeve van resistentieveredelingsprogramma's, hoog-resistente planten van wilde emmer tarwe het beste kunnen worden verzameld op plaatsen waar deze soort een optimale habitat bezet, gaat voorbij aan het feit dat veredelaars veel meer geïnteresseerd zijn in genetische diversiteit.
   Moseman, J.G., E. Nevo, M.A. El Morshidy and D. Zohary, 1984. Euphytica 33:41-47

3. Het in zijn algemeenheid afwijzen van het gebruik van overgevoeligheids-resistentiegenen ('major genes') in de plantenveredeling is niet verstandig.
   Dit proefschrift

4. De aanwezigheid van overgevoeligheids-resistentiegenen tegen *Phytophthora infestans* in geavanceerd uitgangsmateriaal van aardappel moet vermeden worden.

5. Ook in de toekomst blijven chemische bestrijdingsmiddelen noodzakelijk.

6. Het postuleren van resistentiegenen op basis van corresponderende bekende virulentiegenen, na toetsing met verschillende fysio's, leidt vaak tot verkeerde conclusies.
   M.S. Perwaiz and R. Johnson. 1986. Plant Breeding 97:289-296
   C.R. Wellings, R.A. McIntosh and M. Hussain. 1988. Plant Breeding 100:88-96

7. De uitspraak dat partiële resistentie van gerst tegen dwergroest "bijna fysio-niet-specifiek" blijkt te zijn, is een contradictio in terminis.
   J.E. Parlevliet. 1983. In: Durable resistance in crops. Eds. F. Lamberti, J.M. Waller and N.A. van der Graaff, Plenum Press, New York and London. Pp. 57-80

8. Het gegeven dat larven van vele tweevleugelige insekten in de bodem leven, maakt dat het inbouwen van het Bt-toxinegen in een bodembacterie ter bestrijding van emelten ongekende risico's met zich meebrengt.

9. Er is een discrepantie tussen de definitie van de termen virulentie en agressiviteit, zoals gegeven in de lijst van gewasbeschermingskundige termen van de Nederlandse Planteziektenkundige Vereniging, en het internationaal gebruik in de literatuur over obligate parasieten van granen.

10. Het is een slecht gebruik om in plaats van de naam van de vegetatieve fase, de naam van de generatieve fase van schimmels te vermelden in publikaties betreffende onderzoek over de aantasting door deze schimmels in hun vegetatieve fase.

Z. Eyal, A.L. Sharen, M.D. Huffman and J.M. Prescott. 1985. Phytopathology 75:1456-62
G. Shaner and R.E. Finney. 1982. Phytopathology 72:154-158

11. Het begrip zelfvoorzieningsgraad in publikaties over voedselproduktie in ontwikkelingslanden, geeft vaak een schromelijke onderschatting van de werkelijke behoefte.

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Stellingen behorende bij het proefschrift van C.H. van Silfhout, getiteld "Identification and characterization of resistance to yellow rust and powdery mildew in wild emmer wheat and their transfer to bread wheat", te verdedigen op 23 november 1989 in de Aula van de Landbouwuniversiteit te Wageningen.

AUTHOR'S ABSTRACT

In wild emmer wheat three different kinds of genes for resistance to yellow rust were found, namely genes causing overall resistance, genes causing adult-plant resistance and genes which induce resistance detectable at higher temperatures. At least eleven different and probably novel major genes for overall resistance to yellow rust were detected. The inheritance of overall resistance genes and temperature-sensitive genes was found to be mainly dominant, while the latter showed an additive gene action as well. Several new sources of resistance to powdery mildew were found. These include overall resistance, true seedling resistance, adult-plant resistance and partial resistance. There proved to be no serious crossing barrier between tetraploid wild emmer and hexaploid bread wheat. It was shown that genes for resistance as well as other positive traits are readily transferable to bread wheat, while the negative traits of wild emmer can be easily removed. Two or three consecutive crosses with bread wheat appeared to be optimal for developing wild emmer-derived germplasm or varieties.

**ACKNOWLEDGEMENT**

This thesis presents part of the results of a cooperative research project between the Research Institute for Plant Protection (IPO), Wageningen, The Netherlands and the Agricultural Research Organisation (ARO), The Volcani Center, Bet Dagan, Israel. This project was financially supported by the Netherlands Minister for Development Cooperation to improve the nutritional value, baking quality and the yield stability of spring wheat, especially in (sub)tropical areas of developing countries where yellow rust and powdery mildew are important diseases.
In addition to the above-mentioned cooperation, a second cooperative project was carried out by the Research Institute for Plant Protection (IPO), the Foundation for Agricultural Plant Breeding (SVP), the Department of Plant Breeding of the Agricultural University of Wageningen, the Research Institute for Cereal Quality (TNO-IGMB) and the private breeding companies Cebeco, Van der Have, Semundo, and Zelder. The aim of this project was to transfer yellow rust resistance and other valuable traits of wild emmer to winter bread wheat.

The investigators at ARO were dr. Adriana Grama, senior wheat breeder, and dr. Zeev Amitai, senior wheat pathologist, with whom I worked together for many years and who have become personal friends. On the many field trips with dr. Amitai to collect seeds from wild emmer wheat and to evaluate the collected material in nurseries, I have learned much about the botany, evolution and characteristics of the wild cereals in Israel and of the coevolution with their rust diseases. During those trips he also introduced me sideways into the flora, fauna, geology and archeology of this interesting area. Dr. Adriana Grama introduced me into the more general aspects of breeding and growing wheats for semi-arid environments especially with respect to the characters grain protein content and quality, which became important aspects of the project in later phases. The results of these aspects of the project are published elsewhere. I also would like to thank the Israeli assistants; ms. Dvora Benyamin, ms. Vera Fein, Ir. Tineke Wieringa, ms. Ziva Nave and ms. Frida Kleitman. They took care of the planting, inoculating, crossing, harvesting, of the very laborious peeling of the seeds, of the maintenance of the experiments carried out in Israel (chapter 3-6), and of the supply of the seeds for most of the experiments including the experiments which were mainly carried out in the Netherlands (chapter 2 and 7-12). Especially, I would like to thank ms. Kleitman, who was attached to the project during the whole period as coordinator of the professional staff and who took care of the administration and storage of the seed stocks.
Besides for their professional contribution to the project, I am very much indebted to all of the Israeli project members for their generous hospitality.

The initiative for the second-mentioned cooperation was born at the trial fields of Zelder Plant Breeding Station, where Ir. L.J.M. Groenewegen has given room and maintenance for the wild emmer screening nurseries and for some of the experiments described in chapter 10-12. His interest, from the beginning, has stimulated me very much and after we showed that the possibilities of using wild emmer wheat in a winter wheat breeding programme were exciting, he became an important stimulator in the resulting cooperative project. Although the winter wheats which are developed will not be of direct use in most developing countries, the methods,

strategies and ideas which were developed in cooperation with the dutch breeders are transferable. Moreover the demonstration of the possibilities to improve winter wheat with the use of wild emmer has encouraged me to stimulate breeders in developing countries to use wild emmer and its spring wheat derivatives in their breeding programmes. For their contribution in the development of methods, strategies and ideas to reach the main aim of the first-mentioned project the members of the second project-team are also gratefully acknowledged.

Much of the experimental work in the Netherlands was carried out by ms. Margriet Huisman-de Lange, mr. Michiel Werner and Ing. Gert Kema who worked in succession on the projects. Ms. Huisman-de Lange did much of the initial screening of the wild emmer material, which was continued by mr. Werner who became also involved in the selection for resistance. Ing. Kema was especially involved in the cooperation with the dutch breeders and SVP. He worked in particular on the selection of appropriate isolates to test the success of the transfer of resistance to yellow rust from wild emmer to the winter bread wheats. I would like to thank all three of them for their outstanding dedication to the work and for the very pleasant personal contacts.

Dissemination of the results has not only been accomplished through publications and reports but also by distributing seeds. Cooperators who received seed of wild emmer wheat or derivatives of crosses between wild emmer wheat and cultivated wheat are :
Nepal : drs. C.B. Karki, A. Mudwari (ARS), H.J. Dubin (CIMMYT)
Pakistan : drs. M. Aslam, M.A.S. Kirmani (CDRI), N.I. Hashmi (NARC)
Paraguai : dr. M. M. Kohli (CIMMYT)
Kenya : mr. P. Arama Msc. (NPBS)
Mexico : dr. S. Rajaram (CIMMYT)
USA : dr. J.G. Moseman, Plant Genetics and Germplasm Institute, Beltsville

I especially would like to thank those cooperators who have already sent back the results of their tests, which will help to progress faster towards our mutual aim, the improvement of quality and yield of wheat varieties and in particular of the yield stability under disease pressure.

Last but not least I would like to thank my promotor Professor J.E. Parlevliet and my coleages Dr. R.P. Baayen, Ir. R.A. Daamen, Ing. G.H.J. Kema and R.W. Stubbs at the IPO for their interest in the present study, critically reviewing of the manuscript and helpful comments.

**CONTENTS**

## GENERAL INTRODUCTION

Cereals are worldwide the most important staple food. World production in 1986 amounted to 1867 million metric tons (MMT), of which about 30 per cent was contributed by wheat, while rice and maize each contributed about 25 per cent (Anonymus, 1988). Wheat production in the developing countries contributed about 40 per cent to the total wheat production which stood at 535 MMT in 1986. Wheat production has enormously increased since 1970 when 319 MMT was produced. About half of this increase has been grown in the Third World (Curtis and Klatt, 1988). Although several of the developing countries have become self-sufficient, they remain the largest importing group (including China), having received about two-thirds of the total shipments of 84 MMT in 1985. Real shortages of staple food are still higher than this figure indicates, because in many developing countries the mean daily caloric intake is below the necessary level.

One way to overcome the present shortages and to answer to the demands of the still growing world population, is to increase the yields per hectare and to decrease the yearly fluctuations due to adverse conditions. In general, the gap between the genetic yield potential and the actual yield is quite large in many developing countries. Even under the present socio-economic conditions at the small farms, considerable yield improvements can be obtained. Production constraints are drought, heat, floods, rains during harvest, suboptimal cultural practises, weeds, diseases and pests. Some improvements with respect to the first four mentioned constraints can be expected from more adapted varieties, but the main improvements can be expected from better farm management with respect to cultural practises and control of weeds, diseases and pests. The use of resistant varieties will be the main method to control diseases and pests in developing countries, because chemicals for crop protection are in most cases not available to farmers, and their application is expensive. In areas where these chemicals are available, resistant varieties can reduce the dependence of the farmer on these chemicals and reduce the environmental pollution.

The most important wheat diseases in developing countries are caused by the following pathogens; *Puccinia recondita* (brown rust), *P. striiformis* (yellow rust), *P. graminis* (black rust), *Erysiphe graminis* (powdery mildew), *Septoria tritici* (septoria tritici leaf blotch), *Fusarium graminearum* (scab), *Helminthosporium sativum* (spot blotch) and *Ustilago tritici* (loose smut) (Rajaram et al., 1988).

Among these pathogens yellow (stripe) rust (*Puccinia striiformis* Westend. f.sp. *tritici* Eriks.) is considered the most or second most important pathogen in six out of nine agroecological regions as defined by Rajaram et al. (1988), while powdery mildew (*Erysiphe graminis* DC. ex Merat f.sp. *tritici* [E. Marchal]) is an important pathogen in four regions.

In the developed countries the above mentioned pathogens are also causing economic losses, although black (stem) rust, spot blotch and loose smut are relatively less important.

Losses due to yellow rust in Asia and Africa since 1970 are given by Saari and Prescott (1985). In this period 16 moderate to major epidemics have been recorded with estimated yield losses of 5 to 30 per cent.

Losses due to powdery mildew are reviewed by Jenkyn and Bainbridge (1978). Annual yield losses range from 1-5 % in areas where mildew is a

problem, but in localities where the disease is severe, losses greater than 20 % have been recorded. A recent disease-loss relationship (- 0.013 kg per are per pustule-day per leaf) has been given by Daamen (1989).

The scientific name of yellow rust, and also its other common name (stripe rust), has been derived from the linear arrangement (stripes) of the yellow pustules on the leaves of adult plants . This phenomenon is caused by the growth process of yellow rust, which also differentiates this fungus from the other wheat rusts. The mycelium of the fungus grows longitudinally between the mesophyl cells, while transversal growth is inhibited by the vascular bundles which fill the space from upper epidermis to lower epidermis in adult-plant leaves. Germination of uredospores on the wheat leaf is initiated by contact with water, after which the germ tubes penetrate the leaf through the stomata. For a successful infection, about eight hours of leaf wetness are necessary. The life cycle of *Puccinia striiformis* is hemiform; only the uredial and telial stages are known. In the epidemiology of this fungus only the uredial stage is important. Therefore the development of variation for virulence must be due to non-sexual mechanisms, such as mutation, somatic recombination and parasexuality. Evidence has been given that the first two mechanisms may play a role in the formation of new races; mutation being considered the most important (Stubbs, 1985). Hundreds of different physiologic races have been detected worldwide, of which many have been stored in the gene bank of the Institute for Plant Protection in Wageningen, the Netherlands.

Hyphal growth of *Erysiphe graminis* is superficial and haustoria are normally restricted to the epidermal cells. Optimal conditions for infection are 15-20 °C and 100% relative humidity, while free water hampers the infection proces. After the formation of the first haustoria, hyphae begin to grow from the appressorium over the leaf surface and further epidermal cells are penetrated as the colony spreads over the leaf surface. Young colonies are usually white but become grey or reddish brown when sporulating. Conidial chains develop from a characteristic basal cell; mature spores at the apex of the chain are easily detached by wind. Cleistothecia are usually formed in the mycelial mat after conidial production has ceased, each cleistothecium containing up to 25 asci with normally eight ascospores. The cleistothecia are considered to play an important role in the oversummering of the fungus; the role in overwintering seems to be negligible. New virulence genes arise most probably from mutations (mutation rates of up to 2000 per locus per hectare per day have been recorded) and can be recombined during the generative stage, making powdery mildew even more flexible than the rusts. Numerous physiologic races have been detected in all the major cereal growing areas (Jenkyn and Bainbridge, 1978).

Because resistance to several of these pathogens is scarcely available in bread wheat (*Triticum aestivum* (L.) Thell.) or has become ineffective (e.g. Stubbs, 1988), the search for sources of resistance has been extended to the wild relatives of wheat. Lange and Balkema-Boomstra (1988) have given a concise review of the taxonomy and phylogeny of bread wheat and its diploid and tetraploid ancestors. Among the wild relatives of bread wheat, wild emmer wheat (*Triticum dicoccoides* Körn., or *Triticum turgidum* L. ssp. *dicoccoides* [Körn] Bowden) is the most important ancestor; two (genome A and B) of the three genomes (A, B and D) of bread wheat are derived from this wild species. The donor of genome D is the wild species

*Aegilops squarrosa* L. (*Triticum tauschii*).

Wild emmer wheat has been divided into two races by Harlan and Zohary (1966), i.e. the Palestine race and the Turkish-Iraqi race, the former race characterized by a remarkably large and robust plant type with large seeds, heavy awns, wide leaves and thick stems; the latter characterized by small and slender plants with small spikes and grains. Within Israel, besides the typical type of the Palestine race, several different forms have been found which indicate that the botanical identification of wild emmer wheat in Israel might be more complex than described previously (Z. K. Gerechter-Amitai, pers. comm.).

Wild emmer wheat was discovered by Aaronsohn (1910, 1913) in northern Palestine, and he already at that time indicated the importance of this species for breeding, especially for resistance to rust. After this discovery it took about half a century before substantial research on this species was continued. Among the pioneer researchers were several Israeli scientists like D. Zohary, who studied its distribution, ecology and evolution (Zohary and Brick, 1961; Harlan and Zohary, 1966), and I. Wahl and Z.K. Gerechter-Amitai who studied the relation between this wild species and wheat pathogens (Gerechter-Amitai and Wahl, 1966; Gerechter-Amitai and Stubbs, 1970). The latter report has been the first publication resulting from a cooperation between Israeli and Dutch scientists on research in wild emmer wheat which started in the early sixties and has been continued up to the present day and is reflected in the present study.

The primary objectives of the present study were to investigate the variation in wild emmer wheat with respect to resistance to yellow rust and powdery mildew, and to transfer the resistance to these pathogens to bread wheat. This included the selection of resistant plants from the wild emmer populations, and the study of the effectiveness, the possible race-specificity, the mechanism, and the inheritance of the resistance. For the transfer of the resistance to bread wheat, research was focussed on possible crossing barriers, hybrid necrosis, breeding strategies, and selection procedures.

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# Major genes for resistance to yellow rust in wild emmer wheat

C.H. VAN SILFHOUT[1], G.H.J. KEMA[1] AND Z.K. GERECHTER-AMITAI[2]

[1] Research Institute for Plant Protection (IPO), P.O. Box 9060, 6700 GW Wageningen, the Netherlands
[2] Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan 50250, Israel

## Abstract

In a search for new sources of resistance to yellow rust from wild emmer wheat, about 850 samples were collected from wild emmer populations in Israel. Of these circa 10% proved to be resistant to the Israeli test isolates used in this study. Further tests with 28 isolates from 19 countries in South America, Africa, Asia, Australia and Europe revealed that 30 selections were resistant to all 28 isolates, while another 38 selections were in various ways susceptible to one or more isolates. The reaction patterns of 19 of the latter selections were different from each other, as well as from the reaction patterns which are shown by the European differential varieties for yellow rust when tested with the same isolates. A Person-analysis of the results revealed that at least eleven different factors for resistance are present in the tested wild emmer selections. Strategies for the use of the involved genes are discussed.

*Additional keywords: Triticum dicoccoides*, *Puccinia striiformis*, stripe rust.

## Introduction

With the recent breakdown of *Yr5* in India and Australia (Nagarajan, 1983; Wellings, 1986), all the described major genes for resistance to yellow rust (*Puccinia striiformis* Westend. f.sp. *tritici* Eriks.) in wheat have become ineffective to one or more of the known pathogenic races when acting singly (Stubbs, 1985). In all wheat-growing areas of the world, the virulence of the races has been shown to increase step by step (Stubbs, 1985), often breaking down even combinations of resistance genes. In order to be able to protect the world's wheat crop also in the future by virtue of major genes, it is necessary to obtain novel resistance genes.

In this respect, several wild relatives of wheat have shown promising resistance to the wheat rusts (Gerechter-Amitai, 1967). A particularly promising source for yellow rust resistance, which until recently has barely been utilized in breeding, is wild emmer wheat (*Triticum dicoccoides* Körn.). This wild species was discovered in northern Palestine by Aaronsohn (1910). He observed that wild emmer wheat excelled in rust resistance, obviously referring to yellow rust. Already at that time he realized how great the potential importance was of combining the rust resistance of wild emmer wheat with agronomic traits of commercially grown wheats (Aaronsohn, 1913). Also Crepin (1924) observed that wild emmer wheat, experimentally grown near Paris, repeatedly proved resistant to yellow rust.

During the sixties, the study of resistance to yellow rust in wild emmer was revived by the Israeli scientists Z.K. Gerechter-Amitai and I. Wahl in cooperation with R.W. Stubbs from the Netherlands. Preliminary results in Israel revealed that among 55 seed samples of wild emmer wheat, 17 contained seedlings which were resistant, while in the Netherlands 21 of the 57 accessions exhibited resistance to yellow rust. One accession which was more extensively tested (*T. dicoccoides* sel. G-25) proved to be highly resistant to all 21 races and field races used in the tests (Gerechter-Amitai and Stubbs, 1970). In the mid-seventies the above-mentioned cooperation evolved into a formalized cooperative research project on wild emmer wheat, part of which is reflected in the present study.

The objectives of the present study were
1, to collect a large and representative sample of the population of wild emmer wheat growing in Israel;
2, to select from this collection plant-progenies which are homogeneously resistant to yellow rust;
3, to investigate the effectiveness of the resistance in these wild emmer selections to isolates of yellow rust from important wheat growing areas in the world, and
4, to compare the resistance of these selections with the resistance due to already described Yr-genes for resistance to yellow rust in wheat.

**Materials and Methods**

In the course of this study, about 850 accessions of wild emmer wheat have been collected. The collection is of wide geographic origin, extending from the Judean desert in the south to Upper Galilee, the Golan Heights and Mt. Hermon in the north, where wild emmer is most abundant. Topographically, the seed has been collected from -125 m alt. near Lake Kinneret (Sea of Galilee) to approximately 1500 m alt. on Mt. Hermon. The specimens showed a wide variation for morphological traits and spike color characteristics (Gerechter-Amitai, unpublished).

After collection, the accessions were tested in Israel for resistance to local isolates of yellow rust. Tests were carried out in the seedling stage and the adult-plant stage; in the seedling stage with one or more of the isolates WYR-004 (race 2E18), WYR-295 (race 2E0) and GYR-22 (race 2E0) and in the adult-plant stage with a mixture of races, including 2E0, 2E16, 2E18 and 82E16. The seedlings were tested in growth rooms at 15 °C and a daily photoperiod of 14 h, including 12 h at a maximum light intensity (22000 lux, combined fluorescent and incandescent light) and a step-wise light increase and decrease, respectively, of one hour each. The seedlings were inoculated when the second leaf appeared. Incubation of the plants occurred in dew chambers at 9 °C during 24 h in the dark after which the plants were grown under the same conditions as before inoculation. Observations on infection types were made during the third week, usually 16-18 days after inoculation. From non-homogeneous entries, resistant plants were selected and the progenies of these plants were retested. This procedure was repeated when necessary until progenies were obtained which were homogeneous for resistance to yellow rust.

The resistant entries, which were obtained from these tests, were then tested in the Netherlands with 28 isolates of yellow rust from the Gene Bank of Yellow Rust (GBYR) maintained at the Institute for Plant Protection (IPO). These isolates have been collected in different countries and carry the matching virulence genes for all described major genes for over-

all resistance to yellow rust (Table 1). Of each entry, about 10 seedlings were grown in plastic pots and inoculated just before the second leaf appeared. The plants were incubated in a controlled environment chamber at L/D: 12/12 h, RH: 100% and a temperature of 9 °C for 24 h. Before and after incubation the seedlings were kept in climate rooms at L/D: 16/8 h, RH: 70%, a temperature of 17/15 °C, and a light intensity of appr. 25000 lux. Infection types were scored, using the 0-9 scale (McNeal et al., 1971) at 17 days after inoculation. If entries did not perform uniformly, further selections were made, either in the Netherlands or in Israel.

Table 1. Geographic origin, culture number, race designation and determined virulence factors of 28 isolates of yellow rust used in wild emmer seedling tests

| Isolate code | Country of origin | Isolate number | Race designation | Virulence factors[1] and virulence genes |
|---|---|---|---|---|
| A | Pakistan | 75059 | 67E(16) | S/O, 1, 7, (8) |
| B | Netherlands | 78627 | 234E139 | S/O,SD, 2,3,3N,4b, 7,7R, 9 |
| C | Kenya | 80022 | 38E22 | SD, 6,6P,7,7R,8 |
| D | Chili | 75002 | (108)E(205) | S/O,SD, 2,(3),3N,4b,6,6P, (SP) |
| E | Chili | 74210 | 108E173 | S/O,SD, 2,3,3N,4b,6,6P CV |
| F | Kenya | 75147 | 38E16 | SD, 6, 7, 8 |
| G | Egypt | 75080 | 82E16 | S/O, 7, 8, 10 |
| H | India | 76033 | (71)E0 | S/O, (1), (6), 7 |
| I | Netherlands | 78527 | 108E169 | S/O,SD, 2,3,3N,4b,6, CV |
| J | Tunesia | 76078 | 6E16 | 6, 7, 8 |
| K | Afghanistan | 81065 | 7E134 | 1,2, 6,6P,7,7R |
| L | Ecuador | 81047 | 66E0 | S/O, 7 |
| M | Algeria | 80093 | 41E136 | SD,1,2,3,3N |
| N | China | 82015 | 15E158 | 1,2,3,3N,6,6P,7,7R,8 |
| O | Tanzania | 80100 | 38E(18) | SD, 6, 7,7R,8 |
| P | Nepal | 80054 | 4E16 | 6, 8 |
| Q | Ethiopia | 81038 | 70E148 | S/O, 2, 6,6P,7, 8 |
| R | Peru | 81035 | 104E9 | S/O,SD, 3,3N,4b |
| S | Iraq | 77167 | 82E16 | S/O, 7, 8, 10 |
| T | Colombia | 74176 | 12E132 | 2,3, 6,6P |
| U | Iran | 78098 | 82E16 | S/O, 7, 8, 10 |
| V | Netherlands | 87506 | 169E168 | SD,1,2,3,3N, 9,CV |
| W | Netherlands | 85537 | 234E171 | S/O,SD, 2,3,3N,4b, 7,7R, 9,CV |
| X | Netherlands | 88547 | 105E169 | S/O,SD,1,2,3,3N,4b, CV |
| Y | Netherlands | 88507 | 108E141 | S/O,SD, 2,3,3N,4b,6,6P |
| Z | Australia | 85569 | 360E137 | S/O,SD, 2,3,3N,4b,5 |
| AA | Pakistan | 86026 | 7E150 | 1,2, 6,6P,7,7R,8 |
| AB | Netherlands | 58017 | 104E9 | S/O,SD, 3,3N,4b |

[1] Virulence factors indicated by the corresponding differential variety or known *Yr*-gene; S/O - Suwon/Omar, SD - Strubes Dickkopf, 1 to 10 - *Yr1* to *Yr10*, 3N - *Yr3* and Nord Desprez, 4b - *Yr3* and *Yr4b*, 6P - *Yr6* and Peko, 7R - *Yr7* and Reichersberg 42, CV - Carstens V, SP - Spaldings Prolific

∞ Table 2. Reaction patterns of 68 selections of wild emmer to 28 isolates of yellow rust [1,2]

| Wild emmer selection | Isolate | | | | | | | | | | | | | | | | | | | | | | | | | | | Tot R | Tot S |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | K | O | Z | A | F | J | C | H | N | AA | Q | G | S | R | P | U | I | T | B | E | Y | X | M | AB | D | W | V | L | | |
| G303-1M-4-3-2 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 28 | 0 |
| G340-3B-3B-6 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 28 | 0 |
| G344-1-1-3M-1 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 28 | 0 |
| G363-5M | R | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 27 | 0 |
| G148-1-2 | R | R | 0 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 27 | 0 |
| G298-8-1-1-W-WM | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 27 | 0 |
| G149M | R | R | 0 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 27 | 0 |
| G040-1-2B-1 | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 27 | 0 |
| G416-5-1 | R | R | 0 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | | R | R | | 26 | 0 |
| G313-9-1-1 | R | | R | R | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 26 | 0 |
| G090-1-1B | R | R | 0 | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 26 | 0 |
| G283-10-2 | R | * | 0 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 26 | 0 |
| G117-1-1-1-2M | S | R | 0 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 26 | 1 |
| G309-8-1B | R | R | 0 | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 26 | 0 |
| G312-10-5B-4 | R | R | 0 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 0 | R | R | R | R | R | R | R | 26 | 0 |
| G025-78-35 | S | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 26 | 1 |
| G288-3-5M | R | R | 0 | R | R | R | R | R | R | R | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 26 | 0 |
| G021-1-1BM | * | * | R | R | R | R | R | R | R | R | R | R | R | * | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 25 | 0 |
| G194-3M | R | R | 0 | R | R | R | R | R | | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 25 | 0 |
| G316-2M | R | R | 0 | R | R | R | | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 0 | R | R | R | 24 | 0 |
| G484-6M | S | R | 0 | | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 24 | 1 |
| G168-1-2-4B | R | R | 0 | R | R | R | R | R | S | R | R | R | R | R | 0 | 0 | R | R | R | R | R | R | R | R | R | R | R | R | 24 | 1 |
| G007-2-6B-3 | R | R | 0 | R | R | | R | | R | R | R | R | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | 24 | 0 |
| G007-2-4 | S | R | R | R | | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | | R | R | R | R | R | 24 | 1 |
| G485-5 | R | R | | R | | R | | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | | R | R | 23 | 0 |
| G121-1-3-1-1 | R | R | R | R | R | R | R | R | R | R | | R | R | R | | | R | R | R | R | | | R | R | R | R | R | R | 23 | 0 |
| G193-1M | S | S | 0 | | R | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 23 | 2 |
| G348-10BM | R | R | 0 | R | R | | R | R | | R | | R | | * | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 22 | 0 |
| G084-1M | R | R | 0 | R | R | R | R | R | | R | R | R | R | R | | R | R | R | R | | | R | | R | R | R | R | R | 22 | 0 |
| G342-2-2 | S | | R | R | R | R | R | | | R | | R | R | R | | R | R | R | R | R | R | | R | R | R | R | R | R | 21 | 1 |
| G029-1M-8-2 | S | R | R | S | S | R | R | | R | | R | S | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | 21 | 4 |
| G004M-1M | | | | R | R | | R | | | | R | R | R | R | | R | R | R | R | R | R | R | R | R | R | R | R | R | 20 | 0 |
| G503 | R | * | 0 | R | R | R | R | | R | R | 0 | R | R | * | R | 0 | R | R | R | R | 0 | R | 0 | R | R | R | R | R | 20 | 0 |
| G387-3-6 | R | R | | | | R | | R | R | R | R | R | R | R | R | | R | | R | R | | | R | R | R | R | | R | 19 | 0 |
| G179-2-1M-4M | S | | R | R | | | | | | | R | R | R | R | R | R | R | R | R | R | R | R | R | 0 | R | R | R | R | 19 | 1 |
| G297-3M | | | 0 | | R | | | | R | | R | R | R | S | R | R | R | R | R | R | R | R | R | R | R | R | R | R | 18 | 1 |
| G495-7M | | | 0 | R | R | | | R | | | R | R | | R | | R | | R | R | R | R | R | R | R | R | R | R | R | 18 | 0 |
| G023-1M | * | * | R | R | R | R | R | R | R | R | R | R | R | * | R | * | R | R | R | R | * | * | R | * | R | * | * | 0 | 18 | 0 |

Table 2. Cont.

| Wild emmer selection | Isolate | | | | | | | | | | | | | | | | | | | | | | | | | | | Tot R | Tot S |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | K | O | Z | A | F | J | C | H | N | AA | Q | G | S | R | P | U | I | T | B | E | Y | X | M | AB | D | W | V | L | | |
| G714M | | * | 0 | | S | R | | | | R | R | R | R | * | R | R | R | R | R | R | R | R | R | R | R | R | R | | 18 | 1 |
| G747-3 | S | R | | R | R | R | R | | R | | R | R | R | R | R | | R | R | | R | | | R | | R | | R | R | 18 | 1 |
| G326-1-4-5-3M | S | S | R | | R | | R | | | R | | S | | R | R | R | | R | R | R | R | R | | R | R | R | R | R | 17 | 3 |
| G693M | | R | 0 | R | | R | | R | | | R | | | R | | R | R | R | | R | | R | R | R | R | R | R | R | 17 | 0 |
| G713M | S | S | R | | | | S | | | R | R | R | R | R | R | | R | R | 0 | | R | R | R | R | R | | R | R | 16 | 3 |
| G695-1-1 | S | R | 0 | | | | R | | S | | | S | S | R | R | R | | R | R | R | R | R | R | R | R | R | R | R | 16 | 4 |
| G507-6-3 | S | | R | | S | | R | | | R | | | | | | R | | R | R | R | R | R | R | R | R | R | R | R | 15 | 2 |
| G802 | | | R | | S | | | | | | | S | S | R | R | R | | R | R | | R | R | R | R | R | R | R | R | 14 | 3 |
| G395 | | R | | R | | R | S | | | | | S | S | | | R | R | | R | R | R | R | R | R | R | R | R | R | 15 | 3 |
| G345-2-W-WM | | R | R | | | R | | | | | | R | | R | | | R | | R | R | R | | R | | R | R | R | R | 14 | 0 |
| G348-6-2-1M | * | * | R | | R | | | | R | | | * | S | * | R | R | | R | * | R | R | R | R | R | * | R | R | R | 14 | 1 |
| G487 | * | * | 0 | R | 0 | 0 | 0 | R | R | S | | * | R | * | R | R | R | | * | R | R | S | R | R | * | 0 | 0 | R | 12 | 2 |
| G493-1M | S | | R | | | | | | R | S | R | S | | | R | R | R | R | S | R | R | | | R | S | R | R | | 12 | 5 |
| G315-3M-W | S | S | R | | | R | | | | S | | | | | | | R | | R | R | R | | R | R | R | R | R | R | 12 | 3 |
| G281-3M | * | * | 0 | | | | | | R | | | S | R | * | | | | | R | | R | R | R | R | R | R | R | R | 11 | 1 |
| G506-1M | | | 0 | | | | | R | | S | | R | | R | | | R | | R | | R | R | R | | R | | R | R | 11 | 1 |
| G351 | S | | | S | S | S | | | | R | | S | | S | R | R | R | | R | R | R | R | | | S | R | R | R | 11 | 7 |
| G306-12-3-1M | | S | R | | | S | S | | S | R | | S | | R | | | | | | R | R | R | R | | R | R | R | R | 11 | 5 |
| G305-3M | S | S | R | | | | | | S | S | | S | | R | | S | | R | | | R | R | R | R | S | R | R | R | 10 | 7 |
| G323-1-2-2 | R | | R | | | | R | | S | | S | S | | | R | | | | | R | | R | R | | R | 0 | R | | 9 | 3 |
| G789M | S | | | S | | | | | R | | S | | | R | | | | R | * | R | | * | R | R | R | | * | R | 8 | 3 |
| G356-5-3B | S | | R | | | | | | | S | | S | S | S | | R | | R | S | | R | R | | | S | R | R | R | 8 | 7 |
| G240-5-3-2M | | R | | | S | | | | | | | S | | R | | | R | R | R | | 0 | 0 | | R | R | | | | 7 | 2 |
| G716M | | S | | | | | | R | S | S | | | R | | R | | | | | | | | R | | | R | R | R | 7 | 3 |
| G481-3-W-WM | S | S | 0 | | | | | | S | S | S | S | | | | | | | | | R | R | | R | S | R | R | R | 6 | 7 |
| G354-9-1M | S | S | R | | S | | | | | | | S | | S | | | R | | | | | R | | R | S | R | R | | 6 | 6 |
| G642M | | | S | | S | S | | R | R | | S | S | | | | S | R | | | | R | R | | 0 | | R | 0 | | 6 | 6 |
| G410-1 | S | S | R | S | S | | | S | | R | R | R | | S | R | R | | S | S | | S | S | S | S | S | S | S | | 6 | 15 |
| G744M | S | S | R | S | S | S | | | | | | S | R | S | R | R | | | * | | | S | | R | S | S | | | 5 | 10 |
| G411-1 | S | S | S | S | | S | S | | | | S | | S | S | | R | | | S | | | | | R | S | | | | 2 | 11 |
| Total R | 25 | 32 | 30 | 37 | 35 | 35 | 36 | 35 | 36 | 38 | 40 | 43 | 42 | 45 | 44 | 48 | 51 | 51 | 50 | 53 | 52 | 53 | 54 | 54 | 55 | 55 | 59 | 58 | 1246 | |
| Total S | 24 | 12 | 2 | 6 | 10 | 5 | 4 | 1 | 7 | 8 | 5 | 17 | 6 | 7 | 0 | 2 | 0 | 1 | 4 | 0 | 1 | 3 | 1 | 1 | 9 | 2 | 1 | 0 | | 139 |

[1] For isolate codes see Table 1

[2] R - resistant; S - susceptible; * - not tested; 0 - no symptoms; space - results inconclusive

## Results

The results of the seedling tests with 28 isolates of yellow rust carried out in the Netherlands are summarized in Table 2. Only those 68 wild emmer selections are presented which showed promising resistance or which indicated the presence of different resistance genes. Conclusive results were obtained in 1385 out of the 1904 possible host-parasite interactions. In the other 519 instances it could not be established conclusively whether the entry was resistant or susceptible for the particular isolate used. In these cases the corresponding places in the table were left blank or were marked with an asterix or zero. Inconsistent responses such as nonuniformity in reaction (even though in many cases repeated re-selections had been made) and contradictory results between repetitions were marked by a blank space. An asterix was used to indicate that the wild emmer selection was not tested to a particular isolate due to lack of seed or bad germination. In case of a zero all tested plants were free of visible symptoms, due to a very strong hypersentivity reaction or, perhaps, to escape from infection.

As shown in Table 2, 30 wild emmer selections were resistant to all isolates for which conclusive results were obtained. Thirteen selections were susceptible to only one isolate, four to two isolates, and eight to three isolates, while the remaining 13 selections were susceptible to more than three isolates. Selection G410-1 was susceptible to the largest number of isolates, namely for 15 out of the 21 isolates with which conclusive results were obtained.

Among the 38 wild emmer selections which were susceptible to one or more isolates, at least 18 definitely different reaction patterns can be discerned, implying at least 18 different compositions of resistance genes in the entries studied. Resistance to all 28 isolates as expressed by selections G303-1M-4-3-2, G340-3B-3B-6 and G344-1-1-3M-1 represents a nineteenth reaction pattern. All patterns discerned are shown in Table 3.

The various reaction patterns of the wild emmer entries were compared with the reaction patterns of the European set of differential varieties in order to determine whether the resistance in the wild emmer selections could be similar to the resistance in the differential varieties with already described genes for resistance to yellow rust (Table 4). Not a single reaction pattern appeared similar to that of a European differential variety.

The reaction pattern of each of the 19 type-selections which showed an unique pattern was compared with that of all other selections. Selections which showed similar reactions to the isolates for which conclusive information was available were grouped together with the corresponding type-selection (Table 5). Several entries could be placed in more than one group; for instance, selection G4-1M could be grouped with selection G168-1-2-4B, G25-78-35, G193-1M or G506-1M. In addition, selection G4-1M could even have a different resistance genotype than any of these four type-selections. Only when additional information becomes available on those interactions with selection G4-1M where conclusive information is now lacking, final conclusions can be drawn whether selection G4-1M belongs to any of these groups, and if so, to which of them. Clearly the same holds for the other selections with incomplete data as well.

Table 3. Reaction patterns of 19 selections of wild emmer to 15 isolates of yellow rust, defining all unambiguously different reaction patterns present in Table 2

| Wild emmer entry | Isolate[1] | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | N | K | O | F | R | AA | G | C | S | X | D | J | A | B | Z |
| G303-1M-4-3-2 | R[2] | R | R | R | R | R | R | R | R | R | R | R | R | R | R |
| G168-1-2-4B | S | R | R | R | R | R | R | R | R | R | R | R | R | R | 0 |
| G25-78-35 | R | S | R | R | R | R | R | R | R | R | R | R | R | R | R |
| G193-1M | R | S | S | R | R | R | R | R | R | R | R | R | | R | 0 |
| G714M | | | * | S | * | R | R | | R | R | R | R | | R | 0 |
| G297-3M | R | | | R | S | | R | | R | R | R | | | R | 0 |
| G506-1M | | | | | R | S | R | | | R | R | | | R | 0 |
| G281-3M | R | * | * | | * | | S | | R | R | R | | | R | 0 |
| G323-1-2-2 | S | R | | | | | S | R | | R | R | | | R | R |
| G395 | | | R | | | | S | S | S | R | R | R | R | R | |
| G695-1 | S | S | R | | R | | S | R | S | R | R | | | 0 | R |
| G487 | R | * | * | 0 | * | S | * | 0 | R | S | * | 0 | R | * | 0 |
| G305-3M | S | S | S | | R | S | S | | | R | S | | | | R |
| G306-12-3-1M | S | | S | | R | R | S | S | | R | R | S | | | R |
| G351 | | S | | S | S | R | S | | | R | S | S | S | R | |
| G410-1 | | S | S | S | S | R | R | | | S | S | | S | S | R |
| G744M | | S | S | S | S | | S | | R | S | S | S | S | * | R |
| G356-5-3B | | S | | | S | S | S | | S | R | S | | | S | R |
| G411-1 | | S | S | | S | | | S | S | | S | S | S | S | S |

[1] For isolate codes see Table 1. [2] Responses as in Table 2

Table 4. Reaction patterns of the European yellow rust differential varieties with identified *Yr*-genes to 15 isolates of yellow rust

| Differential variety | Yr-gene | Isolate[1] | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | N | K | O | F | R | AA | G | C | S | X | D | J | A | B | Z |
| Chinese 166 | 1 | S | S | R | R | R | S | R | R | R | S | R | R | S | R | R |
| Lee | 7 | S | S | S | S | R | S | S | S | S | R | R | S | S | S | R |
| Heines Kolben | 6 | S | S | S | S | R | S | R | S | R | R | S | S | R | R | R |
| Vilmorin 23 | 3 | S | R | R | R | S | R | R | R | R | S | S | R | R | S | S |
| Moro | 10 | R | R | R | R | R | R | S | R | S | R | R | R | R | R | R |
| Strubes Dickkopf | | R | R | S | S | S | R | R | S | R | S | S | R | R | S | S |
| Suwon/Omar | | R | R | R | R | S | R | S | R | S | S | S | R | S | S | S |
| Clement | 9 | R | R | R | R | R | R | R | R | R | R | R | R | R | S | R |
| T. spelta album | 5 | R | R | R | R | R | R | R | R | R | R | R | R | R | R | S |
| Hybrid 46 | 3b+4b | R | R | R | R | S | R | R | R | R | S | S | R | R | S | S |
| Reichersberg 42 | 7+? | S | S | S | R | R | S | R | S | R | R | R | R | R | S | R |
| Peko | 6+? | S | S | R | R | R | S | R | S | R | R | S | R | R | R | R |
| Nord-Desprez | 3+? | S | R | R | R | S | R | R | R | R | S | S | R | R | S | S |
| Compair | 8 | S | R | S | S | R | S | S | S | S | R | R | S | S | R | R |
| Carstens V | | R | R | R | R | R | R | R | R | R | S | R | R | R | R | R |
| Spaldings Prolific | | R | R | R | R | R | R | R | R | R | R | S | R | R | R | R |
| Heines VII | 2 | S | S | R | R | R | S | R | R | R | S | S | R | R | S | S |

[1] R - resistant; S - susceptible; for isolate codes see Table 1

Table 5. Wild emmer entries which could have similar resistance genotypes according to their seedling reaction to 28 isolates of yellow rust

| Type selection | Similar selections[1] |
|---|---|
| G303-1M-4-3-2 | 340,344,....[2] |
| G168-1-2-4B | 4,84,194,316,345,348,495,693 |
| G25-78-35 | 4,7-2-4,21,23,40,117,179,345,484,495,693,747 |
| G193-1M | 4,21,23,179,342,495 |
| G714M | 7-2-4,179,345,387,484,485,507,693,713 |
| G297-3M | 21,23,315,348,503 |
| G506-1M | 4,179,194,315,345,348,485,495,693,716,747,789 |
| G281-3M | 29,240,315,326,507,642,693,789 |
| G323-1-2-2 | |
| G395 | 240,348,693,802 |
| G695-1 | |
| G487 | 240,315,345,493,747 |
| G305-3M | 481,716 |
| G306-12-3-1M | 802 |
| G351 | 354 |
| G410-1 | |
| G744M | |
| G356-5-3B | 348,354,481,493 |
| G411-1 | 481 |

[1] Selection numbers in full are given in Table 2
[2] All other 27 entries with merely R-reactions

A Person-analysis (Person, 1959), based on the gene-for-gene theory, was carried out on the data given in Table 2, using a computer programme designed by Kampmeijer (1980). The analysis revealed that at least eleven different resistance factors must be involved in the 68 wild emmer selections to explain the various responses of the wild emmer selections to the test isolates. Of these eleven postulated resistance factors, nine would be present singly in nine out of the nineteen different type-selections (data not shown).

**Discussion**

There are different approaches to determine whether new collections of wild emmer wheat, which were found to be resistant to Israeli isolates of yellow rust, differ from each other with respect to their resistance genes. The most secure approach is the application of a diallel crossing scheme, which however is also the most laborious technique. This technique can in fact only be applied with a limited number of entries. Therefore, three other approaches have been applied at first. One of these is to determine the number of resistance genes and their gene action by analyzing the offspring of crosses between the test entries and a susceptible parent (Van Silfhout et al., 1989). A second approach is to cross new sources of resistance with a wild emmer selection for which it has been shown already that its resistance gene is different from the described genes for yellow rust resistance (Gerechter-Amitai et al., 1989). A third approach, which is the subject of the present study, is to compare the reaction of the new sources of resistance to different isolates of yellow rust. This technique

has the advantage, in comparison with the other techniques, that already an indication can be given concerning the effectiveness of the involved resistance. Disadvantages of this method are that no conclusions can be drawn when entries are resistant to all test isolates, that only the number of different resistance factors can be estimated (each of which may contain one or more resistance genes), and that no conclusions can be drawn with respect to gene action and possible allelism. A comparison of the most promising sources of resistance using a diallel crossing scheme is in preparation.

From the results obtained in this study, one of the drawbacks mentioned above is very evident. From the tested selections about 50% was resistant to all isolates with which conclusive results were obtained, but nevertheless nothing could be concluded about the number and identity of the resistance factors involved in these selections. The resistance in the latter selections may be based on various single genes which completely differ from each other, but the resistance may also be based on one or more resistance factors which are similar to each other or on the presence of some of the few unmatched combinations of the resistance factors found in the selections which were susceptible to one or more isolates. Anyhow, the resistance in these selections appears to be widely effective and may therefore be very useful in breeding for resistance.

Although 19 different reaction patterns were found in the tested wild emmer selections, only eleven different resistance factors were assigned by the Person-analysis to explain the results. The reason is that several entries may have genes in common, although in different combinations. Whether this is really the case can not be discerned with certainty in such an incomplete matrix. Moreover, in the application of this method only the minimum number of resistance factors is ascertained, as is common practice in other genetical methods as well. In crossing experiments with wild emmer selections (van Silfhout et al., 1989; Gerechter-Amitai et al., 1989) including several of the same selections, only one or two resistance genes were found in most wild emmer selections. Only in a few cases three or more genes were postulated. With the Person-analysis on the data from the present study, one or two resistance factors were indicated in most of the studied selections as well. Nine of the postulated resistance factors were assumed to occur singly in certain selections. This may indicate that in these cases each resistance factor represents a single gene. If so, at least nine of the eleven factors would be novel genes differing from the known *Yr*-genes. In a few selections up to five resistance factors were postulated, which seems to be an unlikely high number. This might in part be due to the Person-analysis which is designed to search for the minimum number of factors; if more resistance factors would have been postulated, less complex genotypes would have resulted from the analysis.

In the present study major attention was given to the expression of resistance in the seedling stage. However, as stated in the description of the methods used in this study, all entries were also tested in the adult-plant stage to a mixture of local yellow rust isolates in Israel and in many cases also in the Netherlands. The results of these tests indicated that the resistance detected in the seedling stage was also effective in the adult-plant stage. Therefore it is assumed that the resistance found in the studied selections can be designated as overall resistance.

The resistance in the studied wild emmer selections induced a very low infection type, mostly without sporulation. Together with the apparent single occurrence of most of the postulated resistance factors in specific wild emmer selections, and the conclusion reached above that these resist-

ance factors probably represent single genes, it is reasonable to designate the resistance genes present in the studied selections as major genes.

The fact that several entries were found to be susceptible to one or more isolates does not mean that the resistance in these selections is of less importance. For example, 32 of the 38 selections of wild emmer which were susceptible to one or more of the 28 isolates, proved to be resistant to eight of the main yellow rust races from the Netherlands. The resistance of these entries can still be used in this area. Although for other areas smaller numbers of isolates have been used, the same is probably true for these areas as well. In addition, the fact that different resistance factors can be assigned to these selections opens the possibility to combine these factors, either in single varieties or in variety mixtures. The different resistance factors can be of use in diversification schemes as well.

In conclusion, it has been shown that the resistance in the studied wild emmer selections is based on at least eleven resistance factors which represent probably single genes differing from the known *Yr*-genes. These novel genes can be designated as race-specific major genes for overall resistance. Most of these genes appeared to be widely effective to virulent isolates from different continents and can be of great value in wheat breeding for resistance to yellow rust.

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# Resistance to yellow rust in Triticum dicoccoides. I. Crosses with susceptible Triticum durum

C.H. VAN SILFHOUT[1], ADRIANA GRAMA[2], Z.K. GERECHTER-AMITAI[2] and FRIDA KLEITMAN[2]

[1] Research Institute for Plant Protection (IPO), P.O. Box 9060, 6700 GW Wageningen, the Netherlands
[2] Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan 50250, Israel

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**Abstract**

Out of 71 selections of wild emmer wheat which were involved in this study on the inheritance of resistance to yellow rust, 45 selections gave conclusive results. The most common finding was that the observed segregation ratios in the $F_2$-progenies indicated that the resistance is based on one or more dominant genes (67%). Less frequently, resistance may be conferred by one or more recessive genes (18%), or a combination of both dominant and recessive genes (15%). At least eight different genes have to be involved in the 45 wild emmer selections in order to account for our findings.

*Additional keywords: Puccinia striiformis*, stripe rust, wild emmer wheat, genetics, inheritance, major genes.

## Introduction

For breeding bread wheat cultivars resistant to yellow rust (*Puccinia striiformis* Westend.), the need for yet unused genes has been emphasized (Van Silfhout and Groenewegen, 1984). Since cultivated wheat as a donor of effective resistance genes appears to be greatly exhausted, the necessity for a new source was indicated.

Gerechter-Amitai and Stubbs (1970) found a valuable source of resistance to yellow rust in Israeli populations of wild emmer wheat (*Triticum dicoccoides* Körn.). In these initial screening tests including 55 entries, 17 were found to contain resistant seedlings; two in particular, G-7 and G-25, proved to be resistant to many races and field-races, both in the seedling and in the adult plant stages. The inheritance in one of these accessions, sel. G-25, was subsequently studied in crosses with a susceptible *T. durum* cultivar, revealing that the resistance in this wild emmer selection is probably conferred by one dominant gene (Gerechter-Amitai and Grama, 1974). Using *T. durum* as a bridge, and also by direct crosses with *T. aestivum*, it was shown that the resistance can be transferred to bread wheat (Grama and Gerechter-Amitai, 1974). In further work, Grama et al. (1984) demonstrated that the resistance could be incorporated into an agronomically desirable type of bread wheat, with good baking quality.

The initial study of a limited number of wild emmer accessions was expanded into a comprehensive research project, including country-wide collections of *T. dicoccoides* in Israel, and screening of this material with local isolates of yellow rust. Eighty-two selections which had proved resistant in this research were further analyzed with 20 isolates of yellow rust from different countries and continents. Concurrently, 71 of these resistant selections were subjected to genetic analysis. In 26 of these selections, the $F_2$-progenies presented a wide range of infection-types and the segregation ratios indicated the presence of modifier, suppressor or minor-effect genes, a complexity requiring further study. The present analysis deals with the 45 selections for which conclusive results were obtained.

The objective of the present investigation was to study the mode of inheritance of yellow rust resistance in wild emmer, and to make the resistance genes accessible for wheat breeding.

## Materials and methods

The seed of wild emmer used in the present study consisted of 45 selections from 38 collection sites in Israel and two in Lebanon. The collection was of wide geographic origin, extending from Mt. Hermon in the north to the Judean Desert in the south. Topographically, the seed had been collected from sea-level at the Mt. of Beatitudes to approximately 1400 m alt. on Mt. Hermon. The entries showed a wide range of variability for morphological traits and for colour characteristics.

In the crossing programme, *T. durum* Desf. cv. D447 (= LD393/2 Langdon ND58-322) was used as the susceptible parent. When crossed with the resistant wild emmer selections, the durum cultivar served usually as the female parent. The crosses were carried out in a wire-screen-protected nethouse.

Parents and progenies were tested in the seedling stage for yellow rust reaction in growth chambers under controlled light and temperature conditions. The seedlings were grown at 15 °C and a daily photoperiod of 14 h, including 12 h at a maximum light intensity (22 000 lux, combined fluorescent and incandescent light) and a step-wise light increase and decrease, respectively, of one hour each. Each of the $F_2$-progenies was inoculated with one of the following isolates; GYR-22 and WYR-295, belonging to race 2E0, and WYR-004, belonging to race 2E18, which were avirulent to all parents. The seedlings were inoculated when the second leaf appeared. For incubation the plants were kept in dew chambers at 9 °C for a 24 h dark period and afterwards grown under the same conditions as before inoculation. Notes on the infection-types (Gassner and Straib, 1928, 1932) were taken during the third week, usually 16-18 days after the inoculation.

In the segregating $F_2$-populations, non-sporulating (I.T. 00-0²) plants were considered to be resistant and sporulating (I.T. 1-4) plants to be susceptible. This borderline between resistance and susceptibility was chosen because it coincided with the infection-type of the resistant parent or of the $F_1$-population, if the $F_1$ was resistant. Expected ratios as well as the respective chi-squares were calculated for each of the crosses.

## Results

The observed and expected $F_2$ segregation ratios in the 45 crosses between selections of resistant wild emmer and susceptible durum wheat are given in Table 1. In several of the crosses studied, two or more genotypes could be postulated on the basis of the observed segregation ratios. In these instances, the theoretical segregation for the simplest genotype was shown in Table 1, unless additional data would suggest otherwise. More complex genotypes were postulated when three or more distinct infection-types, in particular of the intermediate range, were observed in the $F_2$-populations. In a few cases also the data obtained in crosses with resistant sel. G-25 were taken into consideration in postulating the genotypes.

On the basis of the theoretical segregation ratios, nine different groups could be discerned. The largest of these (group 1) consists of 22 entries, each of which does not deviate significantly from what is expected under the hypothesis that the resistance is based on one dominant gene (3 : 1). Next (group 7), the $F_2$-populations of six entries showed segration ratios indicating the presence of one dominant and one recessive gene (13 : 3) and in five entries (group 6) the resistance seems to be based on two recessive complementary genes (1 : 15). These are followed by two groups (nos. 3 and 4), each including three entries, which probably have two dominant genes; in the former independently inherited (15 : 1), and in the latter complementary (9 : 7). Two entries (group 5) may be carrying two recessive genes each (7 : 9). The three remaining groups were represented by single entries only; in one of these (group 2), one recessive gene seems to be present (1 : 3), whereas in the other (group 8), one dominant and two recessive genes were indicated (55 : 9). In one cross (group 9), all 256 plants proved to be resistant, showing that probably at least three dominant genes are involved.

In all 45 crosses, the chi-square values (Table 1) were not significant at the 5% level.

## Discussion

Although in our genetic study of resistance in wild emmer $F_3$-populations could not be tested due to the large number of selections included, additional studies of some of the same selections furnished supporting evidence on our hypothetical genotypes. By crossing 17 of the same selections with the resistant wild emmer sel. G-25, in 13 of these crosses confirmatory results were obtained (Gerechter-Amitai et al., 1989). Only in four of the crosses different genotypes were indicated in the two tests.

Each plant in an $F_2$-population with an infection-type lower than I.T. 3 has inherited part of the genes which are involved in the resistance of the parent. Therefore the borderline between the susceptible class and the resistant class can be set between any two consecutive infection-types. If the border is set between I.T. 00 and I.T. $0^2$ one can expect segregation ratios which indicate the highest number of genes and indications for interaction between the genes. With a border at higher infection-types one will find a lower number of genes and fewer interactions between the loci. By using this method of shifting border-lines, supporting evidence can be found for the conclusions which were drawn at the lower infection-types (Van Silfhout and Drenth, 1987). Because in this paper too many crosses were analyzed to go into detail, one fixed border-line between I.T. $0^-$ and I.T. 1 was chosen for all entries. In most cases this will give a good estimate of the number of genes and the mode of inheritance.

Table 1. Genetic analysis of resistance to yellow rust in 45 crosses between resistant wild emmer selections and susceptible *Triticum durum* cv. D447.

| Group | Wild emmer selection | $F_2$ segregation ratio (R : S) | | | Chi-square | P value |
|---|---|---|---|---|---|---|
| | | observed | theoretical | expected | | |
| 1 | G 025-5B | 36: 9 | 3: 1 | 33.75: 11.25 | 0.600 | 0.30-0.50 |
| | G 029-1 | 59: 17 | 3: 1 | 57.00: 19.00 | 0.281 | 0.50-0.70 |
| | G 197-2-1B | 111: 28 | 3: 1 | 104.25: 34.75 | 1.748 | 0.10-0.20 |
| | G 240-5 | 64: 25 | 3: 1 | 66.75: 22.25 | 0.453 | 0.50 |
| | G 288-3-5 | 80: 19 | 3: 1 | 74.25: 24.75 | 1.781 | 0.10-0.20 |
| | G 298-8-1 | 90: 30 | 3: 1 | 90.00: 30.00 | 0 | 1.00 |
| | G 303-1-4-3-2 | 32: 7 | 3: 1 | 29.25: 9.75 | 1.034 | 0.30-0.40 |
| | G 305-1B | 117: 41 | 3: 1 | 118.50: 39.50 | 0.076 | 0.70-0.80 |
| | G 313-9-1-1 | 25: 11 | 3: 1 | 27.00: 9.00 | 0.593 | 0.30-0.40 |
| | G 314-4-7 | 106: 36 | 3: 1 | 106.50: 35.50 | 0.009 | 0.90-0.95 |
| | G 315a-3 | 141: 48 | 3: 1 | 141.75: 47.25 | 0.016 | 0.90 |
| | G 332-1-2 | 21: 11 | 3: 1 | 24.00: 8.00 | 1.500 | 0.20-0.30 |
| | G 351-3-1 | 68: 26 | 3: 1 | 70.50: 23.50 | 0.355 | 0.50-0.70 |
| | G 360-1-3-2 | 111: 29 | 3: 1 | 105.00: 35.00 | 1.371 | 0.20-0.30 |
| | G 363-4-3-1 | 38: 12 | 3: 1 | 37.50: 12.50 | 0.027 | 0.80-0.90 |
| | G 368-6-1 | 18: 7 | 3: 1 | 18.75: 6.25 | 0.120 | 0.70-0.80 |
| | G 436-4 | 48: 21 | 3: 1 | 51.75: 17.25 | 1.087 | 0.30 |
| | G 476-10 | 34: 12 | 3: 1 | 34.50: 11.50 | 0.029 | 0.80-0.90 |
| | G 484-6 | 96: 30 | 3: 1 | 94.50: 31.50 | 0.095 | 0.70-0.80 |
| | G 485-5 | 100: 30 | 3: 1 | 79.50: 32.50 | 0.256 | 0.50-0.70 |
| | G 487-11-3-3 | 32: 12 | 3: 1 | 33.00: 11.00 | 0.121 | 0.70-0.80 |
| | G 498-2 | 19: 12 | 3: 1 | 23.25: 7.75 | 3.108 | 0.05-0.10 |
| | G 503-2-1-1 | 59: 24 | 3: 1 | 62.25: 20.75 | 0.679 | 0.40-0.50 |
| 2 | G 411-1-4-1-2 | 9: 46 | 1: 3 | 13.75: 41.25 | 2.188 | 0.10-0.20 |
| 3 | G 117-1-1-1-3 | 32: 4 | 15: 1 | 33.75: 2.25 | 1.452 | 0.20-0.30 |
| | G 168-1-2 | 63: 6 | 15: 1 | 64.69: 4.31 | 0.704 | 0.30-0.50 |
| | G 309-8-1B-1 | 95: 11 | 15: 1 | 99.38: 6.62 | 3.082 | 0.05-0.10 |
| 4 | G193-1 | 49: 34 | 9: 7 | 46.69: 36.31 | 0.262 | 0.60-0.70 |
| | G 327-6 | 116:103 | 9: 7 | 123.19: 95.81 | 0.959 | 0.30-0.40 |
| | G 340-3 | 52: 34 | 9: 7 | 48.38: 37.62 | 0.621 | 0.40-0.50 |
| 5 | G 121-1-3-1-1-5-3 | 12: 25 | 7: 9 | 16.19: 20.81 | 1.926 | 0.10-0.20 |
| | G 493-1-2 | 26: 31 | 7: 9 | 24.94: 32.06 | 0.080 | 0.70-0.80 |
| 6 | G 213-2-8 | 7:132 | 1:15 | 8.69:130.31 | 0.350 | 0.50-0.60 |
| | G 281-3-4 | 3:100 | 1:15 | 6.44: 96.56 | 1.958 | 0.10-0.20 |
| | G 474-4-1-4 | 5: 92 | 1:15 | 6.06: 90.94 | 0.199 | 0.60-0.70 |
| | G 507-6-3 | 11:100 | 1:15 | 7.56:113.44 | 1.667 | 0.20 |
| | G 695-1 | 2: 19 | 1:15 | 1.31: 19.69 | 0.384 | 0.50-0.60 |
| 7 | G 028-3-1-3 | 119: 25 | 13: 3 | 117.00: 27.00 | 0.182 | 0.60-0.70 |
| | G 040-1-2 | 106: 21 | 13: 3 | 103.19: 23.81 | 0.409 | 0.50-0.60 |
| | G 156-2 | 159: 31 | 13: 3 | 154.38: 35.62 | 0.739 | 0.30-0.50 |
| | G 194-3 | 79: 17 | 13: 3 | 78.00: 18.00 | 0.068 | 0.80 |

Table 1. Continued.

| Group | Wild emmer selection | $F_2$ segregation ratio (R : S) | | | Chi-square | P value |
|---|---|---|---|---|---|---|
| | | observed | theoretical | expected | | |
| 7 (cont.) | G 316-2-5 | 120: 29 | 13: 3 | 121.06: 27.94 | 0.050 | 0.80-0.90 |
| | G 345-1 | 152: 36 | 13: 3 | 152.75: 35.25 | 0.020 | 0.80-0.90 |
| 8 | G 156-3 | 126: 19 | 55: 9 | 124.61: 20.39 | 0.110 | 0.70-0.80 |
| 9 | G 303-3 | 256: 0 | | | | |

From our study of the 45 selections of wild emmer it became evident that we are dealing with a number of different resistance genes. On the basis of only four of the postulated genotypes, namely, two dominant genes, two complementary dominant genes, two recessive genes and two complementary recessive genes, it can be concluded that at least eight different genes must be involved.

Based on these results, a backcross breeding programme was initated by us to transfer the resistance genes from wild emmer to high-yielding spring wheat cultivars.

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**Samenvatting**

*Resistentie van wilde-emmer tarwe tegen gele roest. I. Kruisingen met een vatbare durum tarwe*

In dit onderzoek werden 45 resistente wilde-emmer selecties (*Triticum dicoccoides*) gekruist met de vatbare *Triticum durum* cv. D447 om na te gaan hoe de resistentie van de wilde-emmer selecties overerft. De ouders, de $F_1$- en $F_2$-populaties van een bepaal-

de selectie werden in het kiemplantstadium getoetst met één Israëlisch gele-roest isolaat van fysio 2E0 of van fysio 2E18. In de uitsplitsende $F_2$-populaties werden de niet-sporulerende planten als resistent beschouwd en de sporulerende als vatbaar.

De waargenomen uitsplitsingsverhoudingen komen overeen met 9 verschillende theoretische uitsplitsingsverhoudingen in de $F_2$. Het meest voorkomend (23 herkomsten) was een R : S = 3 : 1 uitsplitsing, wijzend op één dominant gen. In zes herkomsten lijken twee dominante genen aanwezig te zijn, die in drie gevallen onafhankelijk overerven (15 : 1) en in de andere drie complementair (9 : 7). In één herkomst lijkt de resistentie op tenminste drie dominante genen te berusten, daar alle 256 getoetste planten resistent waren. Een uitsplitsing wijzend op één dominant gen samen met één of twee recessieve genen (13 : 3 of 55 : 9) werd in zeven herkomsten gevonden. Resistentie berustend op uitsluitend recessieve genen werd aangenomen in acht herkomsten. In drie gevallen erven deze genen onafhankelijk over (1 : 3 of 7 : 9), in de overige vijf complementair (1 : 15).

Concluderend kan worden gesteld dat de resistentie in dit wilde-emmer materiaal in de meeste herkomsten (67%) op uitsluitend dominante genen lijkt te berusten, of op een combinatie van dominante en recessieve genen (15%). In de overige herkomsten (18%) lijkt de resistentie te berusten op uitsluitend recessieve genen. Om de gevonden resultaten te kunnen verklaren moet worden aangenomen dat er tenminste acht genen zijn betrokken bij de resistentie in deze wilde-emmer herkomsten.

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# Resistance to yellow rust in Triticum dicoccoides. II. Crosses with resistant Triticum dicoccoides sel. G-25

Z.K. GERECHTER-AMITAI[1], ADRIANA GRAMA[1], C.H. VAN SILFHOUT[2] and FRIDA KLEITMAN[1]

[1] Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan 50250, Israel
[2] Research Institute for Plant Protection (IPO), P.O. Box 9060, 6700 GW Wageningen, the Netherlands

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**Abstract**

A comparison was made between the genes in 29 new selections of wild emmer wheat resistant to yellow rust over wide geographic areas and the previously extensively studied selection *Triticum dicoccoides* G-25. In 23 selections the resistance may be conferred by 1 dominant gene; these include 11 selections in which the gene is different from the dominant gene in sel. G-25 and two others in which the genes were closely linked or allelic to the gene in G-25, differing from sel. G-25 by race-specificity. Two dominant genes different from the gene in sel. G-25 seem to be present in one selection. In five selections the resistance may be conferred by one or two recessive genes, including three instances in which the recessive gene was associated with a dominant gene. Our findings show that at least 19 out of the 29 selections studied possess genes which are different from the gene in *T. dicoccoides* sel. G-25.

*Additional keywords: Puccinia striiformis*, stripe rust, wild emmer wheat, genetics, inheritance, major genes.

## Introduction

Gradual depletion of gene resources for resistance to yellow rust (*Puccinia striiformis* Westend. f.sp. *tritici*) in cultivated wheat prompted an extensive search among the wild relatives of wheat for still unused genes (Gerechter-Amitai, 1967).

In a study of 55 accessions of wild emmer wheat (*Triticum dicoccoides* Körn.), Gerechter-Amitai and Stubbs (1970) found that one accession in particular, sel. G-25, exhibited resistance to a wide range of isolates of yellow rust from Israel, the Netherlands, Kenya, Japan, Chile and the United States. Gerechter-Amitai and Grama (1974) demonstrated that the resistance of sel. G-25 is based on one dominant gene.

In extensive inoculation experiments, including 82 selections of wild emmer, it was proven that there are many more accessions of wild emmer which carry effective resistance genes to yellow rust. Studying the inheritance of resistance in these accessions, Van Silfhout et al. (1989) found that the resistance in these selections was conferred by both dominant and recessive genes, singly or in combination. They concluded that at least eight different genes were involved in the wild emmer accessions studied.

The objective of the present study was to investigate whether the diverse populations of wild emmer in Israel possess genes for resistance which differ from the dominant gene in *T. dicoccoides* sel. G-25.

## Materials and methods

The present study included 29 new selections of wild emmer and the previously extensively studied sel. G-25. The new accessions originated from 27 collection sites in Israel and two sites in Lebanon. Topographically, the seed had been collected from sea-level near the Mt. of the Beatitudes to about 1400 m alt. on Mt. Hermon. The entries displayed a considerable range of variability for morphological traits of the plants and for colour characteristics of the spike. Selection G-25 belongs to var. *aaronsohni* Perc., originally collected near Rosh Pinna in the Lower Galilee, at approximately 500 m altitude.

The wild emmer accessions which were included in the present study were selected by the first author from his collection on the basis of their resistance to Israeli test isolates.

For genetic analysis, each of the new selections was crossed with *T. dicoccoides* sel. G-25. The crosses were carried out in a wire-screen-protected nethouse.

Parents and progenies were tested for seedling reaction to yellow rust in temperature- and light-controlled growth chambers; the growth conditions of the seedlings were described elsewhere (Van Silfhout et al., 1989). The parents and progenies of 26 selections were inoculated with isolate WYR-004 (race 2E18). For the three remaining selections two other isolates were used; namely isolate WYR-295 (race 2E0) for parents and progenies of selections G-288-3 and G-305-1B while isolate GYR-22 (race 2E0) was used for selection G-168-1. Notes on the infection-types (Gassner and Straib, 1928, 1932) were taken during the third week, usually 16-18 days after the inoculation.

For genetic analysis, the segregating $F_2$-populations were divided into non-sporulating (I.T. 00-0$^+$) and sporulating (I.T. 1-4) seedlings. This border-line between resistance and susceptibility was chosen because it coincides with the infection-type of the resistant parent or of the $F_1$-populations, if the $F_1$ was resistant. Expected ratios as well as the respective chi-square and probability values were determined for each of the crosses.

## Results

Genetic analysis of the 29 wild emmer selections studied shows that in 23 accessions the resistance to yellow rust was probably conferred by one dominant gene (Table 1). These include 12 entries in which only resistant plants were obtained, indicating that the gene conferring the resistance is located at the same locus as the gene in *T. dicoccoides* sel. G-25 or is closely linked to this gene. Two of these entries (group 2) had shown a reaction pattern different of sel. G-25 when inoculated in the seedling stage with isolates 75059 and 80022 of yellow rust. While sel. G-25 was resistant to both isolates, sel. G-029-1 was susceptible to the former isolate and sel. G-395-7 to the latter. Therefore it may be assumed that the resistance in these entries is conferred by alleles of the gene in G-25 or by a gene which is closely linked to the gene in G-25. In the remaining ten entries (group 1), no evidence for different reaction patterns was obtained and thus the resistance gene in these entries may be identical to the gene in sel. G-25.

Table 1. Genetic analysis of resistance to yellow rust in 29 crosses between resistant selections of wild emmer and resistant *Triticum dicoccoides* sel. G-25.

| Group | Wild emmer selection | $F_2$ segregation ratio (R : S) | | | Chi-square | P value |
|---|---|---|---|---|---|---|
| | | observed | theoretical | expected | | |
| 1 | G 028-3[2] | 200: 0 | | | | |
| | G 288-3[1] | 190: 0 | | | | |
| | G 305-1B[1] | 240: 0 | | | | |
| | G 313-9-1-1[1] | 136: 0 | | | | |
| | G 416-4 | 182: 0 | | | | |
| | G 476-10[1] | 133: 0 | | | | |
| | G 484-6[1] | 185: 0 | | | | |
| | G 485-5[1] | 98: 0 | | | | |
| | G 486-5 | 127: 0 | | | | |
| | G 503-2[1] | 185: 0 | | | | |
| 2 | G 029-1-8[1] | 111: 0 | | | | |
| | G 395-7-1 | 205: 0 | | | | |
| 3 | G 068-1-5-1 | 185:12 | 15: 1 | 184.7:12.3 | 0.008 | 0.90-0.95 |
| | G 168-1[2] | 128:11 | 15: 1 | 130.3: 8.7 | 0.657 | 0.30-0.50 |
| | G 197-2-1B-4[1] | 156:12 | 15: 1 | 157.5:10.5 | 0.229 | 0.50-0.60 |
| | G 298-13-1 | 130: 6 | 15: 1 | 127.5: 8.5 | 0.784 | 0.30-0.50 |
| | G 332-1[1] | 111: 6 | 15: 1 | 109.7: 7.3 | 0.251 | 0.60-0.70 |
| | G 348-4 | 160:12 | 15: 1 | 161.2:10.8 | 0.155 | 0.60-0.70 |
| | G 487-11-2[1] | 196:10 | 15: 1 | 193.1:12.9 | 0.685 | 0.30-0.50 |
| | G 493-4 | 68: 4 | 15: 1 | 67.5: 4.5 | 0.059 | 0.80-0.90 |
| | G 695-1[2] | 167: 9 | 15: 1 | 165.0:11.0 | 0.388 | 0.50-0.60 |
| | G 714-1 | 140:10 | 15: 1 | 140.6: 9.4 | 0.044 | 0.80-0.90 |
| | G 716-2 | 118: 8 | 15: 1 | 118.1: 7.9 | 0.002 | 0.95-0.98 |
| 4 | G 040-1[1] | 88: 2 | 61: 3 | 85.8: 4.2 | 1.224 | 0.20-0.30 |
| | G 194-3-6-17 | 69: 3 | 61: 3 | 68.6: 3.4 | 0.044 | 0.80-0.90 |
| | G 240-5[2] | 119: 7 | 61: 3 | 120.1: 5.9 | 0.213 | 0.50-0.60 |
| 5 | G 156-6 | 163: 4 | 63: 1 | 164.4: 2.6 | 0.753 | 0.30-0.50 |
| 6 | G 721-3-2 | 60:13 | 13: 3 | 59.3:13.7 | 0.043 | 0.80-0.90 |
| 7 | G 213-2[1] | 149:44 | 49:15 | 147.8:45.2 | 0.044 | 0.80-0.90 |

[1] Results confirmed in cross with susceptible *T. durum* (Van Silfhout et al., 1989).
[2] Results not confirmed in cross with susceptible *T. durum*.

In the 11 other crosses (group 3) a dihybrid ratio of 15R : 1S was found, indicating the presence of one dominant gene in each of these entries in addition to the dominant gene from G-25.

Three accessions, i.e. G-040, G-194 and G-240 (group 4), presented a trihybrid ratio of 61 : 3, agreeing with one dominant gene different from the gene in sel. G-25 and one recessive gene. In one entry, G-156-6 (group 5), the segregating population fitted a 63 : 1 ratio for three dominant genes, meaning that it probably has two dominant

genes different from the gene in sel. G-25. In one other accession, G-721 (group 6), the segregation ratio did not deviate significantly from 13 : 3, indicating one recessive gene. Finally, in entry G-213-2 (group 7), the $F_2$-population fitted a 49 : 15 ratio, attesting to the presence of two complementary recessive genes.

## Discussion

The main purpose of the present study was to determine whether some of our wild emmer selections resistant to yellow rust isolates from different geographic regions possess resistance genes which are different from the gene in *T. dicoccoides* sel. G-25; the first wild emmer selection in which a very effective resistance was found (Gerechter-Amitai and Stubbs, 1970) and which is now being used for resistance-breeding in various countries. We have shown that in 19 out of 29 selections, the resistance was definitely not due to the gene in selection G-25. Moreover, the findings that in two entries (G-029-1-8 and G-395-7-1) the resistance seems to be based on a gene which is allelic to the gene in selection G-25, in one entry (G-156-6) on two dominant genes different from the gene in G-25, in a second entry (G-721-3-2) on a recessive gene and in a third entry (G-213-2) on two complementary recessive genes, indicate that we are dealing with at least six genes different from the gene in G-25. The results of the present study thus provides good evidence that the effective resistance to yellow rust in wild emmer has a wide genetic base.

The accessions which were included in the present study were also crossed for genetic analysis with a susceptible *durum*-cultivar; however, for technical reasons, sometimes different selections from the same accession had to be used for the two studies (Van Silfhout et al., 1989). A comparison between the results obtained for those accessions in which the same selection was used (Table 1), showed good agreement in 13 out of 17 cases. In two of the four remaining cases (G-240-5 and G-168-1-2) the conclusions were partly in agreement indicating a difference of one gene. Although the same selection was used as parent, it was not the same plant. Because the percentage of outcrossing is rather high in many accessions of *T. dicoccoides*, it can be expected that in some plants one or more genes from the original accession have been lost. The difference between the conclusions for selection G-028-3 and G-695-1 is difficult to explain.

These results further coroborate that *T. dicoccoides* constitutes a valuable source of resistance to yellow rust for wheat breeding.

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**Samenvatting**

*Resistentie van wilde-emmer tarwe tegen gele roest. II. Kruisingen met de resistente wilde-emmer sel. G-25*

In dit onderzoek werden 29 nieuwe resistente wilde-emmer selecties (*Triticum dicoccoides*) gekruist met de reeds uitvoerig bestudeerde resistente selectie G-25, om na te gaan of de resistentie van de nieuwe selecties wordt veroorzaakt door genen op dezelfde locus als het dominante gen in sel. G-25 of dat er andere loci bij zijn betrokken. De ouders, de $F_1$- en $F_2$-populaties van een bepaalde selectie werden in het kiemplantstadium getoetst met één Israëlisch gele-roest isolaat van fysio 2E0 of van fysio 2E18. In de uitsplitsende $F_2$-populaties werden de niet-sporulerende planten als resistent beschouwd en de sporulerende als vatbaar.

In de $F_2$-populaties van 12 herkomsten werden geen vatbare planten gevonden, hetgeen er op duidt dat de resistentie wordt veroorzaakt door een gen op dezelfde locus als het gen in G-25 of door een gen dat nauw gekoppeld is aan het gen in G-25. Voor twee van deze herkomsten kan op basis van een fysio-specifieke interactie worden vastgesteld dat de resistentie berust op allelen die verschillen van het allel in sel. G-25. In 11 herkomsten werd een uitsplitsing voor twee dominante genen gevonden (R : S = 15 : 1), waarbij het tweede dominante gen uit de getoetste nieuwe selectie afkomstig is. De aanwezigheid van twee dominante genen verschillend van het gen in sel. G-25 werd gevonden in één herkomst (63 : 1). In de overige vijf selecties bleek de resistentie te worden veroorzaakt door één of twee recessieve genen waarnaast in drie gevallen ook nog een dominant gen werd gevonden.

De resultaten tonen aan dat tenminste 19 van de 29 bestudeerde selecties resistentiegenen bezitten die verschillen van het gen in *T. dicoccoides* sel. G-25. Slechts in twee van deze selecties kan het gen allel zijn met het gen in sel. G-25.


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# Yr*15* – a new gene for resistance to *Puccinia striiformis* in *Triticum dicoccoides* sel. G-25

Z.K. Gerechter-Amitai[1], C.H. van Silfhout[2], Adriana Grama[1] and Frida Kleitman[1]
[1] *Agricultural Research Organization (ARO), The Volcani Center, P.O.Box 6, Bet Dagan 50 250, Israel;*
[2] *Research Institute for Plant Protection (IPO), P.O.Box 9060, 6700 GW Wageningen, the Netherlands*

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*Key words: Triticum dicoccoides*, wild emmer wheat, *Puccinia striiformis*, stripe rust of wheat, yellow rust of wheat, Yr genes

**Summary**

In a comparative study of reaction patterns and by analysis of segregation ratios in cross progenies, *Triticum dicoccoides* Koern. sel. G-25 was shown to possess a yet unknown gene for resistance to yellow rust. It is suggested to assign provisionally the symbol Yr*15* to this gene.

## Introduction

Depletion of effective genes for resistance to yellow rust, caused by *Puccinia striiformis* West., in cultivated wheat led to a search for new genes among close relatives still growing in the wild. Screening an original collection of wild emmer wheat, *Triticum dicoccoides* Koern., Gerechter-Amitai & Stubbs (1970) found that the populations indigenous to Israel were valuable sources of yellow rust resistance. One accession, *T. dicoccoides* G-25, proved to be highly resistant to all 21 races from six countries, used in their tests. In crosses with a susceptible *T. durum* Desf. cultivar, Gerechter-Amitai & Grama (1974) showed that this resistance was conferred by one dominant gene. The most widely effective resistance gene to yellow rust of wheat described so far is Yr5 which occurs in *T. spelta album* and is located on chromosome 2BL (Macer, 1966; Anon., 1976).

The objective of the present investigation was to determine whether *T. dicoccoides* G-25 carries a previously unknown gene for yellow rust resistance. As a first step the reactions of *T. dicoccoides* G-25 were compared with those of wheats with known Yr genes when inoculated with cultures having the virulence genes matching the designated resistance gene(s). A unique reaction pattern for *T. dicoccoides* G-25 would indicate an undescribed gene. On the other hand, if the pattern for G-25 was similar to that of one or more of the varieties with known Yr genes, a cross(es) between the two would show whether different genes were involved.

## Materials and methods

Three single-plant selections of *T. dicoccoides* G-25 (originally collected by the first author at Rosh Pinna in Israel) – G 25-4-46, G 25-4-48 and G 25-22, were crossed with *T. spelta* L. *album* Perc. Selection G 25-4 served in the comparative study of reaction patterns.

For the latter study, 24 isolates of yellow rust from 18 countries in Africa, Asia, Europe and South America were used. These isolates, belonging to 21 races or race-groups, were chosen on the

basis of their wide virulence spectrum. Isolate 77141 (race 2E18), was used for the study of segregation ratios. This isolate had been used to establish that G-25 had one dominant gene (Gerechter-Amitai & Grama, 1974).

The reaction pattern of *T. dicoccoides* G 25-4 to the yellow rust isolates was compared with the reaction patterns of 17 wheat varieties possessing known Yr genes. These included the 15 varieties recommended for yellow rust race identification (Johnson et al., 1972), 'Clement' (Yr9) and *T. spelta album* (Yr5).

Inoculations were performed on seedlings, just before the second leaf appeared. Pre- and post-inoculation conditions in the growth chambers included a temperature of 15° C and a light intensity of approximately 22,000 lux. Infection-types were scored in the third week after inoculation on a scale of 0 to 9 (McNeal et al., 1971).

In our study of reaction patterns, infection types 0–6 were considered resistant and 7–9 susceptible. In genetic experiments, non-sporulating plants were classified as resistant and sporulating plants as susceptible.

## Results

The reactions of *T. dicoccoides* G 25-4 and 17 wheats with known Yr genes to 24 yellow rust isolates are given in Table 1. *T. dicoccoides* G 25-4 proved resistant to all 24 isolates. In contrast, 16 of the wheats with known Yr genes were susceptible to at least one of the isolates. Evidently, the resistance in *T. dicoccoides* G-25 was not conferred by any of the resistance genes in those 16 differential varieties: nor could it be due to a combination of resistance genes, since accession G-25 was known to possess a single resistance gene (Gerechter-Amitai & Grama, 1974). Only *T. spelta album* showed a similar reaction pattern to *T. dicoccoides* G-25. Thus, if *T. dicoccoides* G-25 possessed a gene already known to occur in cultivated wheat, it could be only that gene carried by *T. spelta album*.

In order to find out whether the gene in *T. dicoccoides* G-25 is different from that in *T. spelta album*, three crosses were made and the resulting progenies were analysed (Table 2). In cross no. 1, only one $F_1$ plant was obtained; unexpectedly, this pentaploid – derived from tetraploid *T. dicoccoides* and hexaploid *T. spelta* – was relatively fertile, giving 68 seeds. The cross was subsequently twice repeated and both crosses in which the $F_1$ plants were bagged, again gave a high seed-set. In cross no. 2, three $F_1$ plants were obtained, giving rise to 60, 63 and 122 seeds per plant, respectively. In cross no. 3, four $F_1$ plants were obtained, resulting in 38, 55, 66 and 83 seeds per plant. In a few instances where the numbers of spikelets and grains per head were counted, the average fertility was slightly above 50%. Morphologically, the $F_1$ plants showed characteristics of both parents.

$F_2$ and $F_3$ progenies segregated for reaction to yellow rust. The distribution pattern of the $F_2$ segregates showed a good fit for a 15 (resistant) : 1 (susceptible) ratio (Table 2). Overall, 590 seedlings were resistant and 42 were susceptible ($X^2$ 15 : 1 = 0.17; $P > 0.6$).

$F_3$ lines (Table 2) exhibited segregation ratios of 1 : 0, 15 : 1 and 3 : 1, as expected on the basis of a segregation at two loci.

## Discussion

The combined $F_2$ and $F_3$ results demonstrated that Yr5 and the gene in G-25 were not only different but were also genetically independent. Since the gene in G-25 was also distinguishable from all other named seedling resistance factors, it has been proposed (R.A. McIntosh, personal communication) to designate it Yr*15*.

The present study (Table 1) further corroborates our previous findings (Gerechter-Amitai & Stubbs, 1970) that Yr*15* is effective over a wide range of yellow rust races. In a series of crosses, Grama and Gerechter-Amitai (1974) incorporated this gene into several *T. aestivum* L. lines. By backcrossing to superior bread wheat cultivars, Grama et al. (1984) obtained lines combining good baking quality, high yield, yellow rust resistance, high protein content, good agronomic characters, resistance to stem rust and in some instances, resistance to leaf rust. During this process the negative traits

of the wild parent, such as the disarticulating rachis and poor threshability, were eliminated (Gerechter-Amitai & Grama, 1974). Thus Yr*15* could be easily used in wheat breeding.


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*Table 1.* Reaction patterns of *Triticum dicoccoides* sel. G-25 and wheat varieties possessing the known Yr genes, to 24 cultures of yellow rust

| Country of origin | Race no. | Culture no. | Test varieties and Yr genes represented* | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Chinese 166 1 | Lee 7 | Heines Kolben 6 | Vilmorin 23 3 | Moro 10 | Strubes Dickkopf | Suwon/Omar | Clement 9 | Hybrid 46 3b + 4b | Reichersberg 42 7 + ? | Peko 6 + ? | Nord-Desprez 3 + ? | Compair 8 | Carstens V | Spaldings Prolific | Heines VII 2 | *T. spelta album* 5 | *T. dicoccoides* G-25 |
| Afghanistan | 7E134 | 81065 | ● | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ | ● | ● | ○ | ○ | ○ | ○ | ● | ○ | ○ |
| Algeria | 41E136 | 80093 | ● | ○ | ○ | ● | ○ | ● | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ● | ○ | ○ |
| Chile | 108E173 | 74210 | ○ | ○ | ● | ● | ○ | ● | ● | ○ | ● | ○ | ● | ● | ○ | ● | ○ | ● | ○ | ○ |
| Chile | 108E205 | 75002 | ○ | ○ | ● | ● | ○ | ● | ● | ○ | ● | ● | ○ | ● | ○ | ○ | ● | ● | ○ | ○ |
| China | 15E158 | 82015 | ● | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ● | ● | ● | ● | ○ | ○ | ● | ○ | ○ |
| Colombia | 12E132 | 74176 | ○ | ○ | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ● | ○ | ○ |
| Ecuador | 66E0 | 81047 | ○ | ● | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Egypt | 82E16 | 75080 | ○ | ● | ○ | ○ | ● | ○ | ● | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Ethiopia | 70E148 | 81038 | ○ | ● | ● | ○ | ○ | ○ | ● | ○ | ○ | ○ | ● | ○ | ● | ○ | ○ | ● | ○ | ○ |
| India | 66E0 | 76033 | ○ | ● | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Iraq | 82E16 | 77167 | ○ | ● | ○ | ○ | ● | ○ | ● | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Israel | 2E0 | 77142 | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Israel | 2E16 | 77136 | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Israel | 2E18 | 77141 | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Israel | 82E16 | 75098 | ○ | ● | ○ | ○ | ● | ○ | ● | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Kenya | 38E16 | 75147 | ○ | ● | ● | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Kenya | 38E22 | 80022 | ○ | ● | ● | ○ | ○ | ● | ○ | ○ | ○ | ● | ● | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Nepal | 4E16 | 80054 | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Netherlands | 108E169 | 78527 | ○ | ○ | ● | ● | ○ | ● | ● | ○ | ● | ○ | ○ | ● | ○ | ● | ○ | ● | ○ | ○ |
| Netherlands | 234E139 | 78627 | ○ | ● | ○ | ● | ○ | ● | ● | ● | ● | ● | ○ | ● | ○ | ○ | ○ | ● | ○ | ○ |
| Pakistan | 67E0 | 75059 | ● | ● | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ |
| Peru | 104E9 | 81035 | ○ | ○ | ○ | ● | ○ | ● | ● | ○ | ● | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ |
| Tanzania | 2E16 | 80100 | ○ | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |
| Tunisia | 6E16 | 76078 | ○ | ● | ● | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ○ | ● | ○ | ○ | ○ | ○ | ○ |

* ● – susceptible; ○ – resistant.

*Table 2.* Segregation of seedlings in three crosses between *Triticum spelta album* and selections of *T. dicoccoides* G-25, inoculated with *Puccinia striiformis* race 2E18 (isolate 77141)

| Cross No. | Selection | Cross generation | Segregation ratios | | | |
|---|---|---|---|---|---|---|
| | | | Observed | | Theoretical | |
| | | | Resistant | Susceptible | R : S | $\chi^{2*}$ |
| 1 | *T. dicoccoides* G 25-4-46 | | | | | |
| | | $F_1$ | 1 | | | |
| | | $F_2$ | 41 | 2 | 15 : 1 | 0.2 |
| | | $F_3$ | 46 | 0 | 1 : 0 | – |
| | | | 7 | 2 | 3 : 1 | 0.0 |
| | | | 21 | 2 | 15 : 1 | 0.2 |
| | | | 14 | 1 | 15 : 1 | 0.0 |
| 2 | *T. dicoccoides* G 25-4-48 | | | | | |
| | | $F_1$ | 3 | | | |
| | | $F_2$ | 196 | 13 | 15 : 1 | 0.0 |
| 3 | *T. dicoccoides* G 25-22 | | | | | |
| | | $F_1$ | 6 | | | |
| | | $F_2$ | 353 | 27 | 15 : 1 | 0.5 |
| | | $F_3$ | 25 | 0 | 1 : 0 | – |
| | | | 30 | 13 | 3 : 1 | 0.6 |
| | | | 31 | 0 | 1 : 0 | – |
| | | | 12 | 0 | 1 : 0 | – |
| | | | 10 | 2 | 3 : 1 | 0.4 |
| | | | 24 | 4 | 3 : 1 | 1.7 |
| | | | 15 | 1 | 15 : 1 | 0.0 |
| | | | 18 | 0 | 1 : 0 | – |
| | | | 9 | 0 | 1 : 0 | – |

* Value for significance with 1 d.f. is 3.8 at 5% level.

ions expressed rests solely with the authors: publication does not constitute an endorsement by the Netherlands Minister for Development Cooperation.'

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# Adult-plant resistance to yellow rust in wild emmer wheat

C.H. VAN SILFHOUT[1] and Z.K. GERECHTER-AMITAI[2]

[1] Research Institute for Plant Protection (IPO), P.O. Box 9060, 6700 GW Wageningen, the Netherlands
[2] Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan 50 250, Israel

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**Abstract**

Seventy-six entries of wild emmer, susceptible to a culture of yellow rust, race 39E134, in the seedling stage at a low temperature-profile, were tested with the same culture for field resistance at two locations in the Netherlands. While most entries were susceptible also in the adult-plant stage, 15 showed an intermediate or resistant infection-type in Flevoland and 18 at Wageningen. In subsequent seedling tests at low and high temperature-profiles, including 20 entries which at either of the two locations had displayed field resistance, 16 entries were shown to possess temperature-sensitive genes, whereas four appeared to have true host stage-bound adult-plant resistance.

*Additional keywords: Triticum dicoccoides, Puccinia striiformis,* stripe rust, temperature-sensitive genes.

## Introduction

Among 58 accessions of wild emmer wheat (*Triticum dicoccoides* Körn), Gerechter-Amitai and Stubbs (1970) found two accessions to be very resistant when tested with many isolates of yellow rust in both seedling and field tests. In later work it was shown that numerous selections of wild emmer were highly resistant to isolates of yellow rust from many countries (Van Silfhout and Gerechter-Amitai, in preparation). In addition to this 'seedling' or overall resistance conferred by major genes, Gerechter-Amitai et al. (1984) described in wild emmer also another kind of resistance conferred by temperature-sensitive 'minor-effect' genes, which are race-specific (Gerechter-Amitai and Van Silfhout, in press) and, when combined, display additive gene action (Grama et al., 1984). Gerechter-Amitai et al. (1984) concluded that these genes are effective not only in the seedling stage but also in the adult-plant stage, in the field.

In cultivated wheat, breeding to reduce yield losses caused by yellow rust (*Puccinia striiformis* Westend.) is based mainly on three kinds of resistance. One of these, referred to above as overall resistance, operates in all growth stages of the plant and is effective under a wide range of environmental conditions. The other two kinds of resistance are often detected in the field in the adult-plant stage and are therefore called 'field resistance'. One being conferred by genes sensitive to environmental conditions which prevail in summer and coincide with the adult-plant stage, the other by genes which come to expression only in the adult-plant stage. Transitions and intermediate situa-

tions between the latter two kinds of resistance may exist. Pope (1968), Sharp and Fuchs (1982), Qayoum and Line (1985), Milus and Line (1986) and others described a type of field resistance induced by an increase in temperature during the growing season. The induction of resistance by increasing the temperature could also been shown in the seedling stage in growth rooms with the same lines as used in some of these experiments. Slovenčikova (1972) found that the amount of light can also influence the expression of field resistance. Studying the resistance in the wheat cultivar Bellevue, Pochard et al. (1962) showed that the field resistance of this cultivar was not influenced by temperature. With respect to most cultivars which possess field resistance it is not known, however, whether this resistance is related to certain environmental conditions or is due to adult-plant stage proper.

So, overall resistance and temperature-sensitive resistance have been reported both in cultivated wheat as well as in wild emmer wheat. However the type of field resistance caused by genes which come to expression only in the adult-plant stage has not yet been described for wild emmer wheat. The objective of the present study was to investigate whether in wild emmer also true adult-plant resistance occurs.

**Materials and methods**

The seed of wild emmer used in this study consisted of 79 entries from 62 collection sites in Israel. The sites range from −125 m alt. near Lake Kinneret (Sea of Galilee) to approximately 1500 m alt. on Mt. Hermon. The specimens showed a wide variation for morphological traits and spike color characteristics.

The entries were selected by the second author from his collection of wild emmer on the basis of their susceptibility to Israeli isolates of yellow rust when previously tested in the seedling stage in growth chambers at a constant temperature of 15 °C and a light intensity of 22 000 lux at plant height.

The studies of field resistance were carried out in the Netherlands with the Dutch isolate 68009 of race 39E134. All entries were first tested in the seedling stage, in growth rooms at temperatures of 17/15 °C day/night and a light intensity of 24 000 lux, in order to ascertain the absence of overall resistance genes to this rust isolate.

After vernalization, the seedlings were planted out at two locations. In Flevoland, the nursery was established in an isolated plot in an oil-seed rape field; in Wageningen, at the IPO, the nursery was established in a plastic tunnel. Ten plants of each entry were grown in an one-meter row, spacing between rows 30 cm. To ensure a constant inoculum pressure on all entries during the experiment, a spreader row was planted along the nursery. Field inoculations were made, when the plants were in the tillering stage, with the same isolate previously used in the seedling test. The inoculum was suspended in mineral oil and applied onto the nursery using a low volume sprayer. When the yellow rust had developed optimally and the plants were in the water-ripe to early-milk growth stage, readings were taken on infection-types using the 0-9 scale of McNeal et al. (1971).

Entries which in the nurseries were more resistant than in the seedling stage were tested once more in the seedling stage, 10-15 plants per entry, for the possible presence of temperature-sensitive genes. One set of the entries was kept at a high temperature-profile with a maximum of 24 °C and a minimum of 15 °C, while a duplicate set was tested at a low temperature profile with a maximum of 18 °C and a minimum of 4 °C.

In both profiles, the maximum temperature was maintained for 10 h and the minimum for 9 h, with a gradual increase and decrease, respectively, for 2.5 h. Light conditions in both growth chambers were 23 000 lux for 6 h, a dark period for 12 h, and a gradual increase and decrease for 3 h in between.

## Results

In the seedling trials, out of the 79 entries of wild emmer which had been uniformly susceptible to yellow rust in Israel, 76 proved to be likewise susceptible also to the Dutch test isolate. In the nurseries, 20 of these entries showed in at least one of the two locations a more resistant infection-type in the adult-plant stage than they had displayed in the seedling stage (Table 1). In Flevoland, under field conditions, 15 entries had a resistant or intermediate infection-type; at Wageningen, in a plastic tunnel, 18 entries fell into this category. These include 13 entries which at both locations had either a resistant or intermediate infection-type in the adult-plant stage. Usually, the resistance was higher at Wageningen than in Flevoland, but in three instances (entries 12, 13, 17) the reverse was observed.

In a further study to elucidate whether the field resistance observed in the 20 entries was due to the higher temperatures in summer or to true adult-plant resistance, the entries were tested in the seedling stage, at two temperature-profiles (Table 1). While all entries were susceptible at the low temperature-profile, 16 showed a shift towards resistance at the high temperature-profile (11 displaying an intermediate and five a resistant infection-type), indicating in these entries the presence of temperature-sensitive genes. Four entries (4, 5, 7, 19) were susceptible also at the higher temperature-profile.

## Discussion

In our experiments, field resistance in the adult-plant stage showed up in 20 out of 76 entries tested, suggesting that this phenomenon is of rather frequent occurence in wild emmer.

The results of the seedling tests at two temperature-profiles demonstrate that in the majority of these cases, the resistance could be ascribed to temperature-sensitive genes. This conclusion is confirmed by the field test. It was found that at Wageningen, where the nursery was grown in a plastic tunnel, the resistance was often higher than in Flevoland, where the nursery was grown in the open field. Since in the plastic tunnel the temperature was higher than in the open, it may be assumed that the greater resistance was conferred by temperature-sensitive genes in these entries. However, in three instances (entries 12, 13, 17), temperature-sensitive resistance was shown in the seedling tests, but in the field tests the resistance at Wageningen was lower than in Flevoland. This could be the result of other environmental conditions, such as a lower light intensity at Wageningen, due to the plastic tunnel.

In four instances (entries 4, 5, 7, 19) there was no indication of the presence of temperature-sensitive resistance in the seedling stage. The resistance which was observed in the field is thus most probably true adult-plant resistance. The resistance in entries 5 and 7 seems to be most valuable since it is highly effective (infection-types 2-3) and showed up in both nurseries. In two other instances (entries 3, 6), the field resistance is probably a combination of temperature-sensitive resistance and true adult-plant

Table 1. Infection-types on 20 entries of wild emmer in the seedling stage in growth cabinets at two temperature-profiles, and in the adult-plant stage in field nurseries at two locations, scored after inoculation with the same isolate of yellow rust.

| Entry number | Wild emmer selection | Seedling reaction | | Adult-plant reaction | |
|---|---|---|---|---|---|
| | | 18/4 °C | 24/15 °C | Flevoland | Wageningen |
| 1 | G 108-5B | 9[1] | 1 | 8 | 5 |
| 2 | G 110-2M | 9 | 6 | 8 | 6 |
| 3 | G 111-3M | 8 | 6 | 2 | 2 |
| 4 | G 112M | 9 | 8 | 7 | 2-3 |
| 5 | G 123-1M | 9 | 9 | 3 | 3 |
| 6 | G 136-1M | 8 | 5 | 2 | 2-3 |
| 7 | G 150-1M-4M | 9 | 8 | 2-3 | 2-3 |
| 8 | G 153-1M | 9 | 4 | 7 | 2-3 |
| 9 | G 186-1M | 9 | 6 | 9 | 5 |
| 10 | G 224-1B | 9 | 6 | 6 | 7 |
| 11 | G 258-1BM | 9 | 4 | 6 | 5 |
| 12 | G 260-1M | 9 | 2-3 | 2-3 | 6 |
| 13 | G 301-1B | 8 | 6 | 2-3 | 6 |
| 14 | G 322-5-1B | 9 | 4 | 3 | 3 |
| 15 | G 382M | 8 | 4 | 4 | 4 |
| 16 | G 386M | 8 | 2-3 | 3-6 | 4-6 |
| 17 | G 404M | 7 | 1-2 | 2-3 | 8 |
| 18 | G 496M | 8 | 2-3 | 4-6 | 2 |
| 19 | G 570M | 8 | 8 | 6 | 6 |
| 20 | G 697M | 9 | 6 | 6 | 5 |

[1] Infection-types according to the scale of McNeal et al. (1971).

resistance. In these entries, in the seedling tests, the effect of the temperature-sensitive genes appears to be of only minor magnitude (IT 5-6). Therefore it is assumed that the additional resistance (IT 2-3) found in the field tests can also be ascribed to adult-plant resistance.

Since temperature-sensitive genes will mask adult-plant resistance under summer conditions in the field and also under simulated summer conditions in growth chambers, it may well be that the frequency of adult-plant resistance is greater than could be shown in our experiments. Only when temperature-sensitive genes give intermediate resistance and adult-plant genes give complete resistance, can the presence of adult-plant resistance be detected. In our experiments in at least two of the entries, the presence of both temperature-sensitive genes and true adult-plant resistance genes is indicated (entries 3, 6).

In conclusion, it can be stated that wild emmer may serve as a useful source not only for major genes and temperature-sensitive genes of minor or major effect, but also for true adult-plant genes for yellow rust resistance.

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**Samenvatting**

*Volwassenplantresistentie tegen gele roest in wilde emmer tarwe*

Voor dit onderzoek werden 76 herkomsten van wilde emmer tarwe (*Triticum dicoccoides* Körn.) geselecteerd op basis van vatbaarheid voor gele roest (*Puccinia striiformis* Westend. f.sp. *tritici*) in het kiemplantstadium. In een veldtoets met hetzelfde gele roest isolaat (fysio 39E134) werd de reaktie in het volwassenplantstadium bepaald. Deze toets werd in Wageningen in een plastic kas uitgevoerd en in Flevoland in een geïsoleerd veld.

In Flevoland vertoonden 15 herkomsten een intermediaire of resistente reaktie, in Wageningen werd dit bij 18 herkomsten waargenomen. Om na te gaan of de waargenomen veldresistentie mogelijk berust op temperatuurgevoelige genen, werd de resistentie vergeleken bij hoge en lage temperatuur. Van de 20 herkomsten, die resistentie vertoonden in de veldtoets in Wageningen of in Flevoland, bleken er 16 temperatuurgevoelige resistentiegenen te hebben. Vier herkomsten lijken echte, stadium afhankelijke, volwassenplantresistentie te vertonen.

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# ADDITIVE GENE ACTION FOR RESISTANCE TO *PUCCINIA STRIIFORMIS* F.SP. *TRITICI* IN *TRITICUM DICOCCOIDES*

ADRIANA GRAMA[1], Z. K. GERECHTER-AMITAI[1] and C. H. VAN SILFHOUT[2]

[1] Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan, Israel
[2] Research Institute for Plant Protection, P.O. Box 9060, 6700 GW Wageningen, the Netherlands

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INDEX WORDS

*Triticum dicoccoides*, wild emmer wheat, *Puccinia striiformis*, yellow rust, stripe rust of wheat, minor genes.

SUMMARY

Seven single-plant selections of wild emmer, with temperature-sensitive minor-effect genes for stripe rust resistance, were intercrossed in eight combinations. The resulting progenies were studied for a possible additive gene action.

The transgressive segregation towards resistance in $F_2$ observed in all the combinations indicates that additive gene action for resistance indeed occurs in wild emmer. The common occurrence of this phenomenon in random combinations suggests further that several minor-effect genes are involved.

Following selection of the most resistant plants in $F_2$, a marked shift towards resistance was noted in $F_3$, which demonstrates a positive response to selection. In some instances, additive resistance selected for (in $F_2$) at the high temperature-profile was expressed (in $F_3$) also at the low temperature-profile. This kind of resistance, when utilized in breeding programmes, promises therefore to be effective over a range of temperatures.

## INTRODUCTION

Resistance to stripe rust in cultivated wheat is based mainly on major genes. In addition to major gene resistance, LEWELLEN et al. (1967) found another type of resistance based on what they called 'minor genes'. These genes conferred greatest resistance at relatively high temperatures. Later, SHARP & VOLIN (1970) showed that if such temperature-sensitive minor genes are accumulated, an additive effect of resistance was expressed in some of the offspring. They also reasoned that the accumulation of minor-effect genes should result in longer-lasting resistance.

GERECHTER-AMITAI & STUBBS (1970) showed that wild emmer (*T. dicoccoides*) is a valuable source of major genes for stripe rust resistance. One of the major genes found in wild emmer was successfully transferred to both *T. durum* (GERECHTER-AMITAI & GRAMA, 1974) and *T. aestivum* (GRAMA & GERECHTER-AMITAI, 1974). GERECHTER-AMITAI et al. (1981) proved that temperature-sensitive minor-effect genes exist also in wid emmer wheat.

The purpose of the present study was to investigate whether these minor-effect genes are additive in resistance and whether the high levels of resistance demonstrated in cultivated wheat (KRUPINSKY & SHARP, 1978) can be attained also by combining different minor-effect genes present in *T. dicoccoides*.

## MATERIALS AND METHODS

The seed of wild emmer used in the present study was obtained from seven single-plant selections which were chosen from a larger collection previously tested for the presence of temperature-sensitive minor-effect genes for stripe rust reaction (GERECHTER-AMITAI et al., 1981). Each of these selections originates from a different collection site in Israel and has been shown to be temperature-sensitive and homogeneous with regard to stripe rust reaction.

The seven selections were crossed in eight different combinations (Table 1). Progeny testing was commenced in $F_2$, plants with the lowest infection type being selected to produce the next generation. The resulting $F_3$ was tested simultaneously with reserve $F_2$, together with their respective parents.

Inoculations were made with an isolate of *Puccinia striiformis* GYR-22 (2E0(16)), originally collected on *T. dicoccoides* in northern Israel. All the tests were carried out in growth chambers under controlled light and temperature conditions. The seedlings were grown at a daily photo-period of 14 hours, including 12 hours at a maximum light intensity (22000 lux, combined fluorescent and incandescent light), and a stepwise light increase and decrease, respectively, for one hour each. Pre-inoculation temperature was 15 °C. The seedlings were inoculated when the second leaf appeared. After inoculation the plants were kept in dew chambers at 9 °C for a 24-hour dark period, followed by a stepwise increase in light and temperature. The plants were then maintained at a low temperature profile of 15 °C day and night or at a high temperature-profile of 15/24 °C dark/light for a 10/14 hours diurnal cycle. Notes on the infection-types (GASSNER & STRAIB, 1928, 1932) were taken during the third week after inoculation, usually after 16–18 days.

## RESULTS

The $F_2$ populations showed a wide range of infection-types (Table 1). Whereas the parents of the eight crosses tested together with the $F_2$ at the high temperature-profile displayed intermediate types of reactions (moderate resistance to moderate susceptibility), the $F_2$ progenies segregated in most instances from high resistance (non-sporulating types) to various degrees of susceptibility. The occurrence of hybrid progenies with infection-types lower than those of their respective parents clearly indicates additive gene-action for stripe rust resistance in these wild emmer crosses.

To illustrate further the relative proportion of $F_2$ plants which were more resistant, similar to, or more susceptible than their parents, the segregating progenies in three of these crosses were arranged according to reaction group (Fig. 1). In each of the crosses, relatively many plants were more resistant than their respective parents. Whereas the occurrence of segregants more susceptible than either parent (reaction-group C) indicates that different genes are involved, transgressive segregation towards higher resistance (group A) demonstrates an additive effect of these genes.

In five of the crosses in which the $F_3$ was tested at the high temperature-profile together with reserve $F_2$ and the respective parents, the proportion of plants more resistant than the parents was larger in the $F_3$ population than in the $F_2$ (Table 2). In one of these crosses (no. 3), all the $F_3$ plants were more resistant than their parents;

Table 1. Distribution of infection-types in eight crosses between wild emmer selections possessing temperature-sensitive minor-effect genes, at the high temperature-profile (15/24 °C night/day).

| Cross no. | | | Reaction class | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | resistant | | | | | intermediate | | | susceptible | | |
| | | Infection | v 00 | 0− | 0± | 0+ | 1 | 2− | 2 | 2+ | 3− | 3 | 34[a] |
| | Cross | type | v 1 | 1 | 1–2 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 8–9[b] |
| 1 | G 14-1-2 | $P_1$ | | | | | | | | | X | | |
| | G 50-1-1 | $P_2$ | | | | | | | X | | | | |
| | | $F_2$ | | | | 1 | 1 | 5 | | 3 | 2 | | 2 |
| 2 | G 50-1-2 | $P_1$ | | | | | | | X | | | | |
| | G 131-1-1 | $P_2$ | | | | | | | | | X | | |
| | | $F_2$ | | | | | 6 | | 11 | | | 7 | |
| 3 | G 76-1-2 | $P_1$ | | | | | | X | | | | | |
| | G 128-1-1 | $P_2$ | | | | | | | X | | | | |
| | | $F_2$ | 5 | 9 | 10 | 2 | 17 | 2 | 8 | | 4 | 2 | 1 |
| 4 | G 121-1-2 | $P_1$ | | | | | | | X | | | | |
| | G 76-1-1 | $P_2$ | | | | | X | | | | | | |
| | | $F_2$ | 5 | 26 | | 14 | | | 8 | | | | |
| 5 | G 121-1-2 | $P_1$ | | | | | | | X | | | | |
| | G 128-1-1 | $P_2$ | | | | | | | X | | | | |
| | | $F_2$ | 1 | 4 | | 2 | | | 8 | | | | |
| 6 | G 121-1-2 | $P_1$ | | | | | | | X | | | | |
| | G 131-1-2 | $P_2$ | | | | | | | X | | | | |
| | | $F_2$ | | | 1 | | 8 | | 12 | | 15 | | |
| 7 | G 128-1-1 | $P_1$ | | | | | | | X | | | | |
| | G 14-1-1 | $P_2$ | | | | | | | | X | | | |
| | | $F_2$ | | 5 | 5 | 6 | 4 | 5 | 24 | 1 | | 1 | |
| 8 | G 128-1-2 | $P_1$ | | | | | | | | | X | | |
| | G 158-1-2 | $P_2$ | | | | | | | | | X | | |
| | | $F_2$ | | | 1 | 1 | | 2 | 1 | 5 | 6 | 6 | |

[a] GASSNER & STRAIB (1928) code.
[b] MCNEAL et al. (1971) index value.

in two other crosses (nos. 1 and 4) the plants were either more resistant than their parents or similar to them in their reaction. In the remaining two crosses (nos. 2 and 8), while still some $F_3$ seedlings were more susceptible than their respective parents, the proportion of plants in this reaction group (C) had decreased considerably in the $F_3$. Since the $F_3$ was obtained from the most resistant plants of the $F_2$ population, the observed shift towards resistance in $F_3$ demonstrates a positive response to selection. However, while the proportion of plants which were more resistant than their parents increased in the $F_3$, no plants were found with an infection-type lower than that observed in $F_2$.

When several of the crosses were tested in $F_3$ at the low temperature-profile, some of the seedlings displayed a resistant or intermediate reaction, whereas the parents

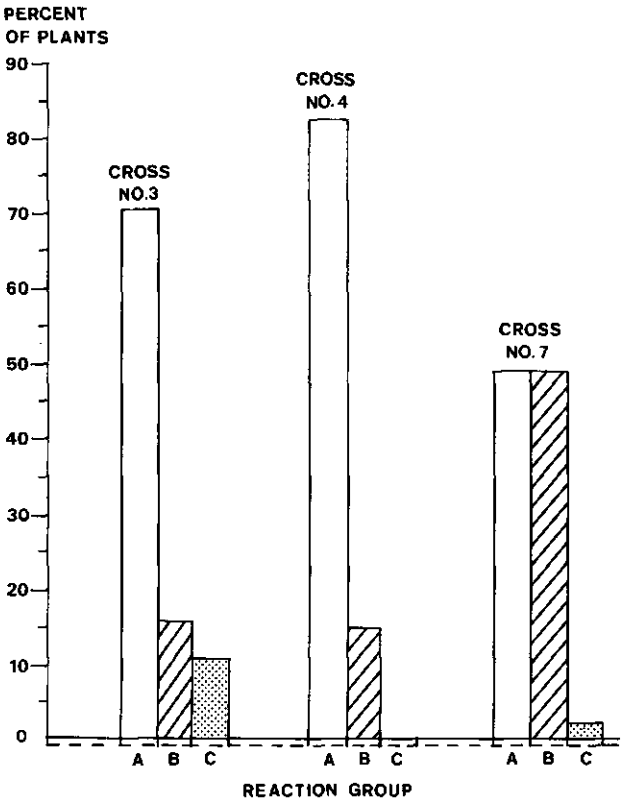


Fig. 1. Additive gene-action in $F_2$ populations of three crosses between temperature-sensitive selections of *Triticum dicoccoides*.
A = More resistant than the parents; B = Within the range of the parents; C = Less resistant than the parents.

of the crosses, as expected, were all susceptible (Table 3). In one of the crosses (no. 5), even more than half the number of the plants were resistant. In comparison, no such shift towards resistance, at the low temperature-profile, was observed in the $F_2$. Evidently, although the selections in the $F_2$ for the lowest infection type were made at the high temperature-profile, the additive gene-action for resistance in the $F_3$ was expressed also at the low temperature-profile.

## DISCUSSION

In our investigation, eight crosses between single-plant selections of wild emmer were studied for additive gene action with regard to stripe rust resistance. Each of the seven wild emmer selections used in these crosses were obtained from a different collection site and were found to possess minor-effect genes conditioning temperature-sensitive reactions. The existance of additive gene action for stripe rust resistance in wild emmer was established in our studies by the occurrence of transgressive segregation towards

Table 2. Effect of selection of the most resistant seedlings in $F_2$ on the response of the resulting $F_3$ population, in tests performed at the high temperature-profile.

| Cross no. | Pedigree | Population | Total number of plants | % of plants* | | |
|---|---|---|---|---|---|---|
| | | | | A | B | C |
| 1 | G 14-1-2X | $F_2$ | 27 | 42 | 22 | 26 |
| | G 50-1-1 | $F_3$ | 13 | 46 | 54 | |
| 2 | G 50-1-2X | $F_2$ | 22 | 18 | 50 | 32 |
| | G 131-1-1 | $F_3$ | 13 | 62 | 31 | 7 |
| 3 | G 76-1-2X | $F_2$ | 40 | 67 | 15 | 18 |
| | G 128-1-1 | $F_3$ | 19 | 100 | | |
| 4 | G 121-1-2X | $F_2$ | 25 | 44 | 48 | 8 |
| | G 76-1-1 | $F_3$ | 23 | 61 | 39 | |
| 8 | G 128-1-2X | $F_2$ | 13 | 15 | 39 | 46 |
| | G 158-1-2 | $F_3$ | 9 | 56 | 33 | 11 |

*A = more resistant than parents; B = within the range of the parents; C = less resistant than parents.

Table 3. Expression of additive resistance in $F_3$ populations from crosses between wild emmer selections possessing temperature-sensitive minor-effect genes, tested at the low temperature-profile (15°/15°C night/day).

| Cross no. | Pedigree | | Number of plants | | |
|---|---|---|---|---|---|
| | | | resistant | inter-mediate | susceptible |
| 5 | G 121-1-2 | $P_1$ | | | 10 |
| | G 128-1-1 | $P_2$ | | | 10 |
| | 9,1 | $F_2$ | | | 10 |
| | 9,1-1 | $F_3$ | 7 | 3 | 2 |
| 6 | G 121-1-2 | $P_1$ | | | 10 |
| | G 131-1-2 | $P_2$ | | | 10 |
| | 10,2 | $F_2$ | | | 10 |
| | 10,2-1 | $F_3$ | 1 | 2 | 7 |
| 7 | G 128-1-1 | $P_1$ | | | 10 |
| | G 14-1-1 | $P_2$ | | | 10 |
| | 11,2 | $F_2$ | | | 10 |
| | 11,2-2B | $F_3$ | 1 | 1 | 10 |

resistance in the $F_2$ populations in each of the eight crosses.

The occurrence of additive resistance shows that the two parents, in each of the eight crosses, possess different genes. The fact that different genes are involved is also demonstrated for six of the crosses by the occurrence of susceptible segregants in the $F_2$ populations. The absence of susceptible plants in two of the crosses (nos. 4 and 5, Table 1) may be an indication that a larger number of minor.-effect genes are in-

volved in these combinations but the small number of $F_2$ seedlings tested does not warrant definite conclusions.

Although the $F_1$ was not tested for stripe rust reaction in our studies, the relatively large proportion of $F_2$ plants more resistant than their respective parents suggests that we deal with dominant genes. In this respect, the minor-effect genes in wild emmer appear to be different from those described in cultivated wheat (SHARP, 1973).

The high temperature-profile, although not simulating actual growing conditions, proved to be useful for the detection of additive resistance and for the selection of the most effective minor gene combinations. Since the additive resistance detected at the high temperature-profile (in $F_2$) was expressed also at the lower temperature-profile (in $F_3$), the utilization of the minor-effect genes in breeding wheat cultivars resistant under a relatively wide range of temperatures appears to be feasible.

The fact, that in each of our eight random crosses – as indicated by the additive gene action in each of the combinations – the two parents possessed different minor-effect genes, suggests that there exists in *T. dicoccoides* a multitude of minor-effect genes. A more detailed study of the minor-effect genes present in our temperature-sensitive wild emmer selections, including a scheme of diallel crosses, is in progress (VAN SILFHOUT, GRAMA & GERECHTER-AMITAI, unpublished). This study should result in more information on the wild emmer selections included in our investigation – both with regard to the number of detectable minor-effect genes involved and their distribution among the selections studied.

In our work, the additive effect of resistance was found in each of the crosses already in the $F_2$ populations. By selection in $F_2$, a rapid shift towards resistance was achieved, indicating that rapid progress can be made in the introduction of these genes into cultivated wheat.

Recently, these wild emmer lines were crossed with two durum wheats and several bread wheats (REINHOLD et al., 1983) known to possess additive minor-effect genes for stripe rust resistance. Although it has not been proved that these genes in wild emmer do not occur in cultivated wheat, it has been shown that they can act additively when combined either with minor-effect genes or with major-effect genes present in cultivated wheat.

The common occurrence of additive gene action for stripe rust resistance in crosses involving wild emmer possessing temperature-sensitive genes establishes *T. dicoccoides* as a rich resource of minor-effect genes endowed with good combining ability.

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# Race-specificity of temperature-sensitive genes for resistance to *Puccinia striiformis* in *Triticum dicoccoides*

Z.K. Gerechter-Amitai[1] and C.H. van Silfhout[2]
[1] *Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan 50250, Israel;* [2] *Research Institute for Plant Protection, P.O. Box 9060, 6700 GW Wageningen, the Netherlands*

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**Summary**

Twenty-four entries of wild emmer possessing temperature-sensitive genes for resistance to yellow rust were studied in the seedling stage, at two temperature-profiles, with 15 pathogenic races from 11 countries in South America, Africa, Asia and Europe. It was shown that the majority of the resistance genes in these wild emmer entries were race-specific. In most of these entries a more resistant reaction was displayed at the higher temperature-profile; however in three entries a shift in reaction towards resistance was observed with certain races but towards susceptibility with some of the other races, suggesting that two different kinds of temperature-sensitive genes were involved in each of these entries. The similarity of temperature-sensitive genes occurring in wild emmer and in cultivated wheat is discussed.

## Introduction

In cultivated wheat, breeding for resistance to yellow rust (*Puccinia striiformis* West.) is based mainly on major genes. In addition to major gene resistance, Lewellen et al. (1967) found another type of resistance based on what they called 'minor genes'. These genes conferred greatest resistance at relatively high temperatures. Later, Sharp & Volin (1970) demonstrated that such temperature-sensitive minor genes when accumulated, showed additive effects.

Testing wheat lines possessing one, two or three minor genes with 11 races of yellow rust from the United States, they came to the conclusion that the genes involved are race non-specific in action. Stubbs (1977), using the same three wheat lines both in race nurseries in the Netherlands and in the International Yellow Rust Trials for three years, also concluded that the minor gene resistance is race non-specific. In a study designed to evaluate the effectiveness of minor genes, Sharp & Fuchs (1982) tested 49 $F_6$ wheat lines – derived from crosses between 12 cultivars and the three wheat lines possessing one, two and three minor genes, respectively – with seven isolates of yellow rust. These test isolates, from Europe and the United States, together carried virulence to all cultivars of the World Differential Set for race determination, except for 'Lee' (Johnson et al., 1972). They found that in these crosses the progenies were resistant or moderately resistant to the different rust isolates, which again suggests absence of race-specific interactions.

Gerechter-Amitai et al. (1984) found in wild emmer wheats resistance conferred by temperature-

sensitive 'minor' genes – possibly of the same kind as described by Lewellen et al. (1967) in cultivated wheat. In a further study, Grama et al. (1984) combined these temperature-sensitive genes by means of intercrossing and demonstrated that additive gene action could provide enhanced resistance.

By crossing selections of wild emmer with lines of cultivated wheat – both possessing temperature-sensitive minor genes – Reinhold et al. (1983) found in many combinations an additive effect of resistance, thus proving that the minor genes from wild emmer act together with those from cultivated wheat.

Comparing the characteristics of minor genes present in wild emmer with those occurring in cultivated wheat, it was found that both confer a temperature-sensitive reaction (Gerechter-Amitai et al., 1984; Lewellen et al., 1967) and both, when accumulated, bring about an additive effect on resistance (Grama et al., 1984; Sharp et al., 1982; Sharp et al., 1970). With regard to these minor genes in cultivated wheat, it was concluded that they are race non-specific (Sharp et al., 1982; Sharp et al., 1970; Stubbs, 1977). Concerning those minor genes in wild emmer little information is available. In previous work on the existence of temperature-sensitive genes in wild emmer, Gerechter-Amitai et al. (1984) tested a number of entries both in Israel and the United States, with two different isolates of yellow rust. In general, there was good agreement between the results obtained with both isolates but in a few instances, a significant shift in reaction was recorded only at one location but not at the other. The authors concluded that the few exceptions could be due to the somewhat different environmental conditions or the existence of race-specificity (Gerechter-Amitai et al., 1984).

*Table 1.* Collection sites and geographic origin of 24 entries of wild emmer used in the inoculation tests

| Entry No. | Wild emmer selection* | Collection site | Geographic origin |
|---|---|---|---|
| 1 | G 014-1-2-1 | Sede Eliezer | Eastern Upper Galilee |
| 2 | G 015–1M | Sede Eliezer, west | Eastern Upper Galilee |
| 3 | G 020M-2-1 | Hatsor | Eastern Upper Galilee |
| 4 | G 032-1M-2-6-1 | Ammiad | Korazim Sill |
| 5 | G 076-1-2-2-1-3 | Mt. of Beatitudes, north | Korazim Sill |
| 6 | G 086-1 | Mitspe Hayammim, Amirim | Upper Galilee |
| 7 | G 088-1M-5-2-3 | Jaba | Judean Mountains |
| 8 | G 092-1 | Wered Hagalil | Korazim Sill |
| 9 | G 094-1M-1B | Almagor – Kefar Nahum | Korazim Sill |
| 10 | G 128-1-2-2M-2 | Mapal Nehoshet | Golan Plateau |
| 11 | G 131-1-1M | Mt. of Beatitudes | Korazim Sill |
| 12 | G 158-1-2-1M | Majdal Shamms | Mt. Hermon |
| 13 | G 228-1-4-2 | Har Adami – Yavneel | Eastern Upper Galilee |
| 14 | G 279-1M-3-1 | Hushniyya | Golan Plateau |
| 15 | G 448-1M | Duma, Allon Road | Judean Mountains |
| 16 | G 531M | Nurit | Mt. Gilboa |
| 17 | G 552M-3 | Semadar | Eastern Upper Galilee |
| 18 | G 582M | Huqoq | Lower Galilee |
| 19 | G 587M-1 | Tel Kinnorot | Lower Galilee |
| 20 | G 649M-1 | Nov | Golan Plateau |
| 21 | G 687M-1 | Qazrin | Golan Plateau |
| 22 | G 725M | Kefar Hittim | Eastern Lower Galilee |
| 23 | G 726M | Kinneret | Eastern Lower Galilee |
| 24 | G 766M | Giv'at Hamore | Eastern Lower Galilee |

* First number indicates collection site, next numbers indicate single plant selection cycles, mass plant selection cycle (M) or bulk increase without selection (B).

The objective of the present study was to establish by a more extensive experiment whether the temperature-sensitive minor genes in wild emmer are race-specific or whether they confer resistance to all virulence types with which they are tested.

**Materials and methods**

In the present study 24 accessions of wild emmer were studied which in previous experiments (Gerechter-Amitai, unpublished; Gerechter-Amitai et al., 1984; Grama et al., 1984) had been shown to possess temperature-sensitive resistance genes to yellow rust. The accessions were collected in Israel by the first author and originated from altitudes of −160 to 1000 m. They included a diversity of morphological forms and color characteristics. The collection sites, the geographic regions from which the wild emmer entries were obtained, and the number of single-plant selection cycles, are given in Table 1.

Inoculations were made with 15 isolates of yellow rust, covering – with respect to major resistance genes – a wide spectrum of virulence. The virulence formula can be deduced from the race identification number (Johnson et al., 1972). The isolates originated from South America, Africa, Asia and Europe; their race identification numbers and the countries of their origin are given in Table 2.

For testing the reactions to these 15 isolates, 30 similar sets of wild emmer seedlings, each set containing 24 entries, were grown simultaneously under the same environmental conditions. In most instances, 10–15 seeds were sown of each entry. After sowing, the trays were kept for 24 h at room temperature and then maintained for 4 days at 7° C in order to break the dormancy in some of the entries. On the fifth day the plants were placed in growth chambers at a constant temperature of 15° C until inoculation. For inoculation, the spores were suspended in mineral oil and sprayed evenly on the seedlings – each isolate on two similar sets of plants. After incubation at 9° C for 24 h, one set of each isolate was kept at a high temperature profile of a maximum of 24° C and a minimum of 15° C. The other set was kept at a low temperature profile of a maximum of 18° C and a minimum of 4° C. In both profiles, the maximum temperature was maintained for 10 h and the minimum for 9 h, with a gradual increase and decrease, respectively, for 2.5 h. Light conditions in both growth chambers were 23000 lux for 6 h, a dark period for 12 h, and a gradual increase and decrease for 3 h in between.

Notes on the infection-types, using the 0–9 scale

*Table 2.* Countries of origin and race identification numbers of 15 isolates of yellow rust used in the inoculation tests

| Code No. | Country of origin | Race No. | Isolate No. |
|---|---|---|---|
| A | Netherlands | 234E139 | 78627 |
| B | Chile | 108E205 | 75002 |
| C | Chile | 108E173 | 74210 |
| D | Egypt | 82E16 | 75080 |
| E | Netherlands | 108E169 | 78527 |
| F | Tunesia | 6E16 | 76078 |
| G | Afghanistan | 7E134 | 81065 |
| H | Ecuador | 66E0 | 81047 |
| I | Nepal | 4E16 | 80054 |
| J | Ethiopia | 70E148 | 81038 |
| K | Peru | 104E9 | 81035 |
| L | Iraq | 82E16 | 77167 |
| M | Netherlands | 106E139 | 76515 |
| N | Israel | 2E18 | 77141 |
| O | Israel | 2E0 | 77142 |

of McNeal et al. (1971), were taken the third week after inoculation, when the yellow rust had developed optimally. For each host-parasite interaction, the reading of the infection-types at the two temperature-profiles was made at the same time and the reactions were compared. In this comparison, a shift of two or more scale units on the scale of infection-types was considered a significant temperature-sensitive reaction.

## Results

The classification into significant and non-significant shifts in reaction at the two temperature-profiles, for the 24 entries of wild emmer and 15 isolates of yellow rust, are given in Table 3.

Conclusive results were obtained in 314 out of the 360 possible host parasite interactions. In the other 46 instances it could not be established conclusively whether a significant shift in reaction occurred at one of the temperature-profiles and therefore the corresponding places in the table were left blank. The inconclusive results were due to a number of reasons, including non-uniformity in reaction, expression of major genes in certain combinations of host parasite interactions, and escape from infection due to slow and non-uniform

*Table 3.* Pattern of temperature-sensitive reactions in 24 entries of wild emmer inoculated with 15 isolates of yellow rust at two temperature-profiles[1]

| Entry No. | Yellow rust isolates | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | A | B | C | D | E | F | G | H | I | J | K | L | M | N | O |
| 1 | − | − | − | + | − | − | + | + | − | + | − | + | − | − | − |
| 2 | + | + | + | − | + | + | + | + | + | + | | − | + | + | + |
| 3 | − | × | − | + | − | + | + | − | + | + | − | + | − | + | + |
| 4 | + | + | − | + | + | + | + | + | − | + | + | − | + | | |
| 5 | + | + | + | − | + | + | + | + | + | | + | + | + | − | − |
| 6 | | + | + | + | + | | + | − | | | + | + | | + | + |
| 7 | − | − | − | | − | − | + | + | | + | − | − | − | + | − |
| 8 | + | − | + | − | − | − | − | | − | − | − | − | | − | − |
| 9 | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − |
| 10 | + | − | + | − | − | − | − | + | − | + | − | + | + | − | + |
| 11 | × | | − | − | − | − | − | + | + | − | | + | − | + | + |
| 12 | + | | | + | + | | − | + | | + | | | + | − | + |
| 13 | + | + | + | + | + | − | − | + | − | + | + | − | + | − | + |
| 14 | + | | + | + | − | − | − | + | − | | + | + | + | | + |
| 15 | − | − | − | + | − | − | + | + | − | + | − | + | − | − | |
| 16 | + | + | + | + | + | + | + | + | − | + | + | + | | + | + |
| 17 | + | + | + | + | + | + | − | + | − | | + | − | + | − | |
| 18 | | − | + | + | + | + | + | − | + | | − | + | − | + | + |
| 19 | | − | − | + | − | + | − | − | − | | + | + | − | − | + |
| 20 | × | × | | − | × | × | + | + | − | + | × | × | | | + |
| 21 | + | + | + | − | + | + | + | + | | − | + | | | + | − |
| 22 | − | − | | − | − | − | − | + | − | | − | − | + | + | − |
| 23 | + | − | + | − | + | + | + | + | + | | | + | − | − | + |
| 24 | | − | − | | + | + | + | + | − | − | + | | | + | − |
| Mean shift[2] | 2.0 | 1.6 | 2.0 | 2.0 | 1.6 | 1.8 | 1.8 | 2.6 | 1.3 | 2.2 | 1.8 | 1.8 | 1.8 | 1.5 | 1.9 |

[1] + = Significant shift in reaction: more resistant at higher temperature;
× = Significant shift in reaction: more resistant at lower temperature;
− = No significant shift.

[2] Mean shift in scale units of scale of infection types for all 24 entries (reversed shifts and missing data are excluded from calculations).

germination in some of the entries.

As shown in Table 3, all the entries – except the control (no. 9) – displayed a temperature-sensitive reaction to some of the rust isolates but not to all of them. Variation was expressed not only by the number of isolates with which significant shifts did occur but also by the distribution pattern of these reactions. These patterns were different for all entries except for entry 15 which shows a pattern almost similar to entry 1. Also all isolates are different from each other. Among the different patterns a large number of race-specific interactions can be detected, e.g. entries 1 and 2 with isolates C and D, entries 2 and 3 with isolates D and E, entries 1 and 3 with isolates H and I.

Actual infection-types at the low and high temperature-profiles of part of the results are shown in Table 4. An example of race-specific interaction is shown with entries 1 and 8 tested with isolates A and D. The moderate resistance at the high temperature-profile of entry 1 against isolate D is ineffective against isolate A. Similarly, the moderate resistance of entry 8 against isolate A is ineffective against isolate D.

Besides the interactions shown by occurrence or non-occurrence of a shift, also interactions are shown which are due to differences in the magnitude of the shift (Table 4). The shift in infection-type of entry 6 tested with isolates B, G and O is 6, 2 and 4 scale units respectively, while the mean shifts of these isolates are quite similar (Table 3). Moreover the ranking of entry 6 and 20 are reversed when tested with isolates G and O.

In most instances in which temperature-sensitive reactions occurred the entry was more resistant at the higher temperature-profile, but in three entries a reversed shift was displayed (Table 3). In one of these (no. 20), a reversed shift took place with six isolates, a non-reversed shift with four isolates and no shift at all with two isolates. In two other entries (nos. 3, 11), a reversed shift was recorded with only one isolate (B and A, respectively). In Table 4 some examples of the magnitude of the reversed shifts are given; entry 3 with isolate B, entry 8 with isolate G (not significant) and entry 20 with isolates A and B.

Although in some combinations already at the low temperature-profile moderate to intermediate resistance is found (entry 8 with isolate B and entry 3 with isolate A) and no higher resistance at the high temperature-profile, in other combinations even then a shift towards higher resistance was found (entry 6 with isolate D).

## Discussion

In order to determine whether the temperature-sensitive genes in *T. dicoccoides* are indeed race non-specific or not, it was necessary to secure both selections of wild emmer and isolates of yellow rust which were of different genotypes. Therefore a number of wild emmer entries were included in the present study which in previous work had proved to possess different temperature-sensitive genes (Grama et al., 1984). Having had no previous

*Table 4.* Infection types of some selected entries with different isolates of yellow rust at two temperature-profiles

| Entry No. | Yellow rust isolates | | | | |
|---|---|---|---|---|---|
| | A | D | B | G | O |
| 1 | 9–8* | 8–4 | 9–8 | 7–5 | 8–8 |
| 8 | 9–5 | 9–8 | 6–6 | 7–8 | 9–8 |
| 3 | 4–4 | 8–4 | 3–5 | 8–5 | 9–4 |
| 6 | | 5–2 | 9–3 | 9–7 | 9–5 |
| 20 | 2–6 | 9–9 | 2–7 | 9–5 | 9–7 |
| 9 | 9–8 | 9–8 | 9–9 | 9–8 | 8–8 |

* Infection type at low and high temperature-profile respectively.

knowledge on the test races with regard to their virulence to the temperature-sensitive 'minor-effect' genes in wild emmer, the isolates were chosen on the basis of their diverse geographic origin and their wide variation in virulence with respect to major genes.

Generally observations were made on 10–15 seedlings per entry-isolate combination. Usually these seedlings were uniform in reaction, showing that there was very little variance within the host-parasite combinations. Therefore most differences in reaction observed between specific host-parasite combinations, tested at the two temperature-profiles, can be ascribed to the action of temperature-sensitive resistance genes. However, to allow for some subjectivity we only consider differences of two scale units on the scale of infection-types a significant shift in reaction.

Comparing the entries with regard to the frequency occurrence and distribution pattern of significant shifts (Table 3), the wide variation observed shows that different genotypes of resistance are involved. Similarly, the isolates which were known to differ with regard to major gene virulence, also differed in virulence with respect to temperature-sensitive 'minor-effect' gene action. Using '+' (shift) and '−' (no shift) as a simplified representation of our data (Table 3), or a shift of two scale units (Table 4), it becomes obvious from the distribution pattern of the shifts that the variation observed cannot be explained solely by the main effects of the entries and isolates. This differential behaviour within an entry rather proves that the temperature-sensitive gene involved is race-specific. This race-specificity finds expression not only in the occurrence or non-occurrence of significant shifts but also in variations in the magnitude of shifts (Table 4).

The fact that the temperature-sensitive resistance in wild emmer is race-specific may prove to be advantageous for wheat breeding. Although it is generally believed that race-specific resistance is not very durable, in this case, where we deal with genes which are both race-specific and additive in action (Grama et al., 1984), there seems to be a better chance for stability. Parlevliet & Zadoks (1977) compared the level and durability of resistance based on race-specific minor genes with additive effects with that of resistance based on true race-non-specific minor genes with additive effects. They concluded that the race-specific minor genes confer on average a higher level of resistance and a higher degree of durability.

In 23 of the entries a shift in reaction towards resistance occurred with at least one of the races, but in entry 9 (infection type 8 or 9 at the low temperature-profile) no such shift was observed with any of the races. However, in a previous study (Gerechter-Amitai et al., 1984) this entry had shown a temperature-sensitive reaction with an American race not included in the present study. Since a temperature-sensitive gene may not show up even when testing with 15 different races, it can not be concluded definitely that an entry in which no shift in reaction is observed has indeed no temperature-sensitive resistance gene.

In Table 3 it was shown that in many instances a significant shift in reaction occurred when the entries were tested with the various races at two temperature-profiles. As already mentioned, the magnitude of the shift varied often widely both within an entry and among different entries (Table 4). The fact that the shift in infection types in some of the instances was quite large – four to six scale units – makes it questionable whether it is justified here to speak of minor-effect genes. On the other hand, although a large shift occurred in certain entries with some races, a small shift was observed with other races – a fact making it unreasonable to ascribe these shifts to major genes. This considerable variation in the magnitude of the shifts in some entries may also indicate the presence of two or more 'minor-effect' genes, each reacting specifically to the different test races. As demonstrated in previous work (Grama et al., 1984), temperature-sensitive genes are of common occurrence in wild emmer and often differ from one another in diverse selections; it is therefore conceivable that different temperature-sensitive genes may be present in the same entry.

In cultivated wheat, Sharp (1965) described in one of the cultivars tested, Rego, a temperature-sensitive reaction in which a higher resistance was expressed at the lower temperature-profile. In the

present work, a similar reversed shift was observed by us in three entries of wild emmer. However, these reversed shifts occurred in two entries (nos. 3, 11) only with one, and in one entry (no. 20) with six, of the test races, while with the other races a 'regular' shift or no shift at all was observed. This phenomenon, i.e., that in one and the same entry shifts of opposite direction occur with different races, is difficult to explain, unless two different genes are involved in the same entry.

The present study is part of a more extensive investigation on the existence of temperature-sensitive resistance genes in wild emmer, including a comparison of the characteristics of these genes found in wild emmer and those described in cultivated wheat. As mentioned in the introduction, the genes we studied resemble those in cultivated wheat both with respect to temperature-sensitivity (Gerechter-Amitai et al., 1984) and additivity (Grama et al., 1984; Reinhold et al., 1982). In the present study of the temperature-sensitive 'minor-effect' genes in wild emmer, we showed that these genes are race-specific. Thus, with respect to this characteristic, the temperature-sensitive genes in wild emmer might differ from those in cultivated wheat. However, as indicated in this study, the occurrence of race-specific interactions can be demonstrated also by taking into consideration the deviations from expected shifts. The data obtained by Sharp & Fuchs (1982) show that, approached in this way, some variation was displayed in tests with different isolates, which may indicate that also in cultivated wheat lines possessing temperature-sensitive genes, race-specific effects do occur. Therefore it may well be that also with respect to race-specificity, the temperature-sensitive genes in wild emmer are comparable to those in cultivated wheat.

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# RESISTANCE TO POWDERY MILDEW IN WILD EMMER (*TRITICUM DICOCCOIDES* KÖRN.)

Z. K. GERECHTER-AMITAI[1] and C. H. VAN SILFHOUT[2]

[1]Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan, Israel
[2]Research Institute for Plant Protection, P.O. Box 9060, 6700 GW Wageningen, the Netherlands

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INDEX WORDS

*Erysiphe graminis tritici*, powdery mildew, *Triticum dicoccoides*, wild emmer, wheat.

SUMMARY

Wild emmer from 73 collection sites, including 107 accessions from Israel, two from Lebanon and one from Turkey, were evaluated for resistance to powdery mildew in field nurseries in Israel and the Netherlands.

The wild emmer entries displayed a diversity of responses to powdery mildew infection, ranging from high resistance to complete susceptibility. Most entries were resistant in at least one of the nurseries; several entries proved to be resistant in all the tests.

Comparing the reactions of 47 wild emmer accessions tested in six nurseries, 11 markedly different patterns were discerned, indicating the probable presence of several different resistance genes.

Genes for resistance to powdery mildew appear to be very common in wild emmer indigenous to Israel. Resistance was found in accessions from most collection sites, in all the geographic regions represented in the collection.

The common occurrence of resistance and the apparent diversity of genotypes makes wild emmer a rich gene-pool for resistance to powdery mildew. Since genes for resistance to wheat pathogens can be quite readily transferred to cultivated wheat, wild emmer may be utilized as a valuable source of powdery mildew resistance in wheat breeding.

## INTRODUCTION

Powdery mildew of wheat, which is caused by the fungus *Erysiphe graminis* f.sp. *tritici*, has become an increasing problem in recent years. One of the reasons for this is the more dense stands of modern wheat crops.

Before the 1960's, in most breeding programmes in Europe, no special effort was made to incorporate mildew resistance into wheat cultivars. Selection against susceptible plant types led to varieties which were moderately resistant and did not carry any identified race-specific resistance-genes to powdery mildew (LEIJERSTAM, 1965; BENNETT, 1979). When work on wheat mildew resistance was intensified, the choice lay between concentrating on 'field' resistance and on race-specific resistance. At that time, as now, there were no known methods of how 'field' resistance could be utilized in a breeding programme (LEIJERSTAM, 1972c).

The early literature on the genetics of powdery mildew, which came mainly from Australia and the United States, has been reviewed by MOSEMAN (1966). At present ten identified genes have been described as conditioning resistance to powdery mildew

(BENNETT & VAN KINTS, 1982; MCINTOSH, 1978; WOLFE & SCHWARZBACH, 1978). Most of these genes were already matched by virulence genes in the pathogen when one started to use those genes in breeding for resistance (LEIJERSTAM, 1965). Combinations of resistance genes are thus necessary to give protection (LEIJERSTAM, 1972a; WOLFE & SCHWARZBACH, 1978).

To reduce the risk of the breakdown of such resistance, genes should be used of which the frequency of the matching individual virulence genes is low or zero (WOLFE & SCHWARZBACH, 1978; LEIJERSTAM, 1972b). Increase in virulence can also be partly controlled by using a number of varieties with different resistance genes in variety mixtures or diversification schemes (LEIJERSTAM, 1982; BENNETT & VAN KINTS, 1981a). However, the possibility of finding resistance genes in hexaploid wheats, of which the frequency of the matching virulence genes is low or zero, seems to be limited (MEIJER, 1982; BENNETT & VAN KINTS, 1981b; LEIJERSTAM, 1972b).

The literature on resistance to powdery mildew in wild relatives of wheat was reviewed by WAHL et al. (1978). Resistance is found in several species of *Triticum, Aegilops* and *Agropyron*. Within *Triticum* several workers (FITALENKO, 1969; KRIVCHENKO et al., 1979; LEIJERSTAM, 1972b; LEHMAN, 1968) reported the resistance of *T. dicoccoides* KÖRN. to powdery mildew. LEIJERSTAM and LEHMANN probably used the same three accessions from the colllection of the Zentral Institut für Kulturpflanzenforschung, Gatersleben, GDR. These accessions were resistant in Sweden and the GDR. From FITALENKO's paper it is not clear how many accessions of *T. dicoccoides* were tested, but most of them were susceptible. KRIVCHENKO et al. (1979) reported that in field experiments with 29 accessions of *T. dicoccoides*, 86.3% did not show symptoms of infection, 10.3% were resistant and only 3.4% were susceptible.

In the present study, an extensive collection of *T. dicoccoides* was tested under field conditions in Israel, where this species is indigenous, and also in the Netherlands, where powdery mildew is a problem in wheat production.

## MATERIALS AND METHODS

The seed samples used in this study are part of an extensive collection of wild emmer sampled and maintained by the first author. This collection was originally made to study wild emmer for stripe rust resistance and the test material consists, in part, of selections which had been made with regard to stripe rust performance.

The present study included 110 accessions of wild emmer, originating from 73 collection sites (Figs. 1 and 2). Nearly all of these, 107 accessions, were indigenous to Israel and represented the major distribution area of *T. dicoccoides* in this country. Two of the seed samples were obtained from Lebanon and one from Turkey. The collection included several morphological forms of wild emmer – mainly var. *kotschyanum* PERC., var. *spontaneonigrum* PERC. and intermediates between these two forms, and a few samples belonging to var. *aaronsohni* PERC. and *fulvovillosum* PERC. The collection sites range from sea level near the Mount of Beatitudes to about 1400 m on Mt. Hermon.

The entries were evaluated for powdery mildew resistance in field nurseries under conditions of natural infection.

Many of the entries were tested at first during one growing season in Israel, where

powdery mildew on wheat is not common but in 1978 reached a severity high enough for disease evaluation. The observations were made in two nurseries where the same wild emmer entries were grown – at the Bet Dagan Experiment Farm in the central coastal plain, and at the En Dor Experiment Station, about 100 km to the north, in the mountains of Lower Galilee. At both locations, powdery mildew ratings were made on leaf infection in April–May.

In the Netherlands, most of the wild emmer entries that had been tested before in Israel, and many additional ones, were evaluated at three locations – IPO, Zelder and Bouwing – in 1981, and at one location, IPO, in 1982. In order to enhance good disease development at least at one of the locations, the nursery at IPO was grown in both years in plastic tunnels. Owing to favorable climatic conditions, severity of infection in the controls reached 100% in all the nurseries. Readings were taken on both the leaves and the heads several times during the growing season. The highest readings on the leaves were used for evaluation.

In disease evaluation in Israel and the Netherlands entries with no visible infection were rated very resistant (O); low severity of disease, resistant (R); intermediate levels, moderately resistant to moderately susceptible (M); and high severity, susceptible (S).

## RESULTS

In Israel as well as in the Netherlands the wild emmer entries displayed a diversity of responses to powdery mildew infection, ranging from high resistance to complete susceptibility. In each country many of the entries displayed distinctly differential interactions. Most significantly, several wild emmer entries proved to be very resistant, remaining completely free of powdery mildew infection in each of the test years in all nurseries.

Reading of head infection fell, in most instances, in the same category as those of leaf infection. However, in a number of instances the leaves appeared completely resistant, while the heads showed a considerable degree of infection. In several other entries the reverse was observed.

A summary of the reactions of 110 wild emmer accessions to natural infection with powdery mildew, in Israel and the Netherlands, is given in Table 1. More than half of the 52 entries tested in two nurseries in Israel were resistant, whereas the remainder, in about equal numbers, were susceptible, displayed a differential reaction or could not be assigned (due to impurity or incomplete testing) to any of these reaction groups. Similarly, in the Netherlands, nearly half of the 105 entries tested in four nurseries were classified as resistant; of the remainder, nearly two-thirds were susceptible, one-third reacted differentially, and a few entries could not be assigned to either category. Evaluating the 47 entries tested in both Israel and the Netherlands, about one-third were resistant in all the nurseries whereas two-thirds reacted differentially; remarkably, only very few entries were susceptible at all the locations.

Comparing the reactions of the 47 wild emmer accessions, tested in both countries, 21 different patterns can be discerned (Table 2). Each of these reaction patterns, illustrated in the table by one selected wild emmer accession, differed from all others in at least one of the nurseries by at least one step in the classification system; very resistant (O) → resistant (R) → intermediate (M) → susceptible (S). While this grouping

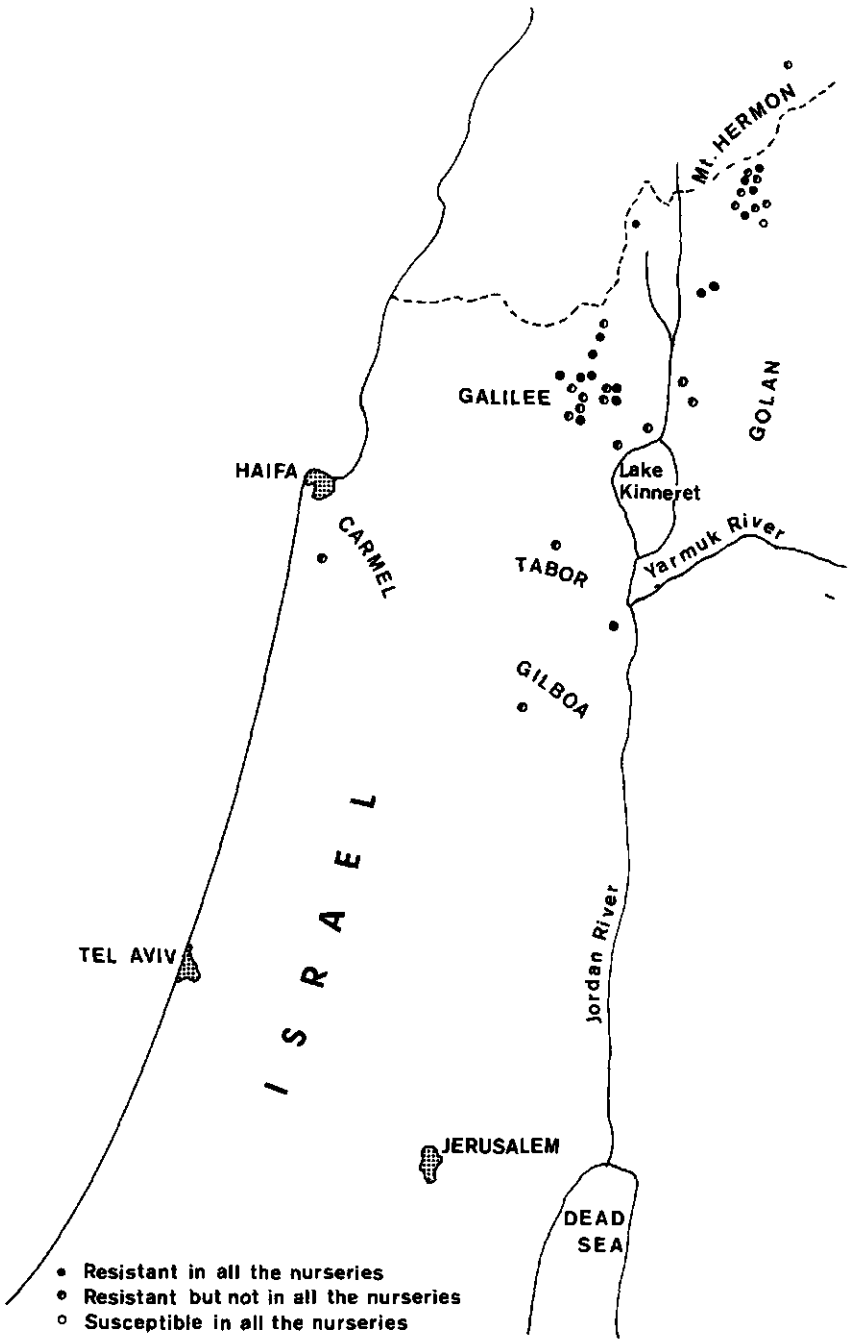


Fig. 1. The geographic distribution of 38 collection sites of wild emmer sampled for powdery mildew evaluation in six field nurseries in Israel and the Netherlands.

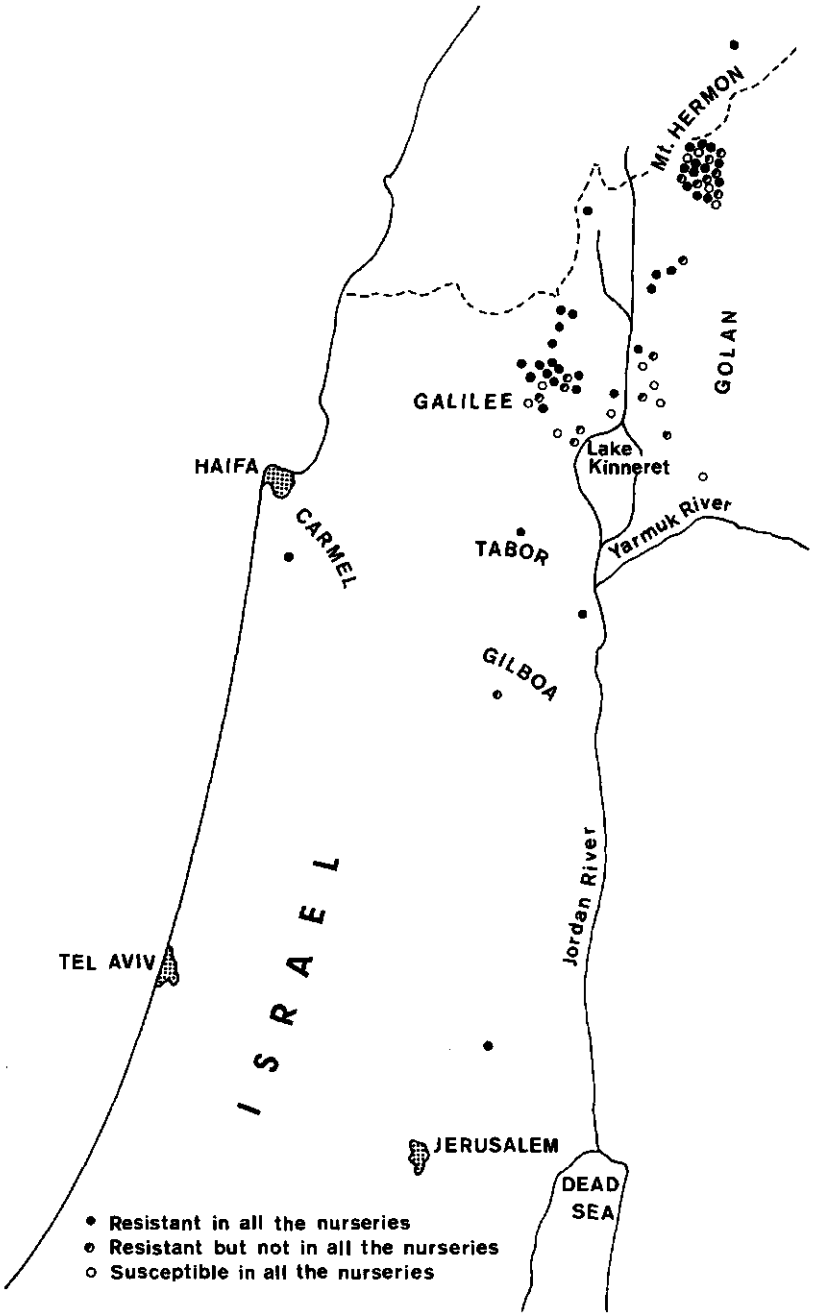


Fig. 2. The geographic distribution of 69 collection sites of wild emmer sampled for powdery mildew evaluation in four field nurseries in the Netherlands.

Table 1. Performance of 110 *T. dicoccoides* accessions to natural infection with powdery mildew in field nurseries in Israel and the Netherlands.

| Reaction group | Number of accessions in indicated country with specified reaction | | |
|---|---|---|---|
| | Israel | Netherlands | Israel and Netherlands |
| Resistant | 32 | 50 | 17 |
| Differential | 5 | 17 | 26 |
| Susceptible | 7 | 33 | 2 |
| Other | 8 | 5 | 2 |
| Total number tested | 52 | 105 | 47 |

Table 2. Reaction patterns of selected wild emmer accessions to powdery mildew infection in field nurseries in Israel and the Netherlands (significantly different patterns are shown in italics).

| Wild emmer selection | Israel 1978 | | Netherlands | | | |
|---|---|---|---|---|---|---|
| | | | 1981 | | | 1982 |
| | En Dor | Bet Dagan | IPO | Zelder | Bouwing | IPO |
| *G 360* | *O* | *O* | *O* | *O* | *O* | *O* |
| G 90-1 | R | O | O | O | O | O |
| G 305-3 | O | O | O | O | O | R |
| G 387 | R | O | O | O | | R |
| *G 239* | *S* | *O* | *O* | *O* | *O* | *O* |
| *G 313* | *O* | *O* | *M* | *O* | | *O* |
| *G 314* | *O* | *O* | *O* | *O* | *O* | *M* |
| G 194-3 | M | R | O | | | O |
| G 323 | M | R | O | O | O | R |
| *G 303-3* | *S* | *S* | *O* | *O* | | *O* |
| *G 288* | *S* | *O* | *O* | | | *M* |
| *G 340* | *R* | *O* | *M* | *R* | *O* | *M* |
| G 25 | M | R | M | R | R | S |
| G 315-a | R | O | S | M | | S |
| G 148-1 | R | R | S | M | O | S |
| *G 280-1* | *O* | *O* | *M* | *M* | *M* | *S* |
| G 306 | R | O | M | M | M | S |
| G 341 | R | O | M | M | M | M |
| *G 348* | *S* | *O* | *M* | *S* | | *S* |
| *G 7-2* | *S* | *S* | *M* | *M* | *O* | *S* |
| *G 281-3* | *M* | *M* | *S* | *M* | *S* | *S* |

is based, in part, on minor variations in reaction (one step), it includes 11 patterns distinguishable by major differences (two or three steps) in reaction (Table 2, entries printed in italics).

The 47 wild emmer accessions which were studied in both Israel and the Netherlands originated from 38 collection sites; the geographic distribution of these sites, and the

reaction to powdery midew infection of the accessions obtained from these collection sites, are shown in Figure 1. Accessions from 16 sites were recorded as resistant in all the tests, from 21 sites as variable (resistant in some nurseries but not in others), and from one site as susceptible (or intermediate in reaction) at all the locations.

In the Netherlands, as part of a more extensive study, altogether 105 accessions of wild emmer, originating from 69 collection sites, were evaluated for powdery mildew resistance. The geographic distribution of these collection sites and the performance of the entries from these collection sites are shown in Figure 2. Accessions from 37 collection sites proved to be resistant to powdery mildew in all the nurseries in the Netherlands, accessions from 20 sites were found to be variable, whereas accessions from 12 sites were susceptible at all the locations.

In the nursery trials performed in the Netherlands, resistance to powdery mildew was found in wild emmer accessions from all the geographic regions represented in the collection (Fig. 2). In those areas in which many collections had been made, in Upper Galilee and on Mt. Hermon, most sites furnished selections which proved to be resistant in all the nurseries. However, good resistance was found also in other geographic regions where only sporadic collections had been made, such as Mt. Carmel not far from the Mediterranean Sea, and southern Samaria near Jericho, at the outskirts of the Judean Desert.

Resistance to powdery mildew was observed in all the morphological forms of wild emmer that were included in the tests.

## DISCUSSION

In this work it was shown that wild emmer can serve as an effective source for powdery mildew resistance.

The wild emmer entries which were grown in six nurseries, in Israel and the Netherlands displayed a variety of reaction patterns to powdery mildew infection (Table 2). By classification based on major variations in reaction in the various nurseries, 11 different reaction patterns could be discerned; if minor variations in reaction were taken into account, as many as 21 reaction patterns could be detected. Assuming that the gene-for-gene hypothesis is applicable to wild emmer/powdery mildew interactions, this profusion of reaction patterns would mean that the wild emmer entries were exposed in different nurseries to powdery mildew pathogens of different virulence formulae and, similarly, that various wild emmer entries possess different genotypes for powdery mildew resistance. Since the number of reaction patterns is quite considerable, it may be assumed that many different genes for resistance are involved. The fact that so many entries (17 out of 47 – Table 1) were resistant in all the nurseries in both Israel and the Netherlands, indicates that the wild emmer populations native to the Middle East possess resistance genes effective over wide geographic areas.

Remarkably, genes for resistance to powdery mildew appear to be very common in wild emmer indigenous to Israel. This became evident both in the wide geographic distribution of resistance over the entire area of sampling and, also, in the large percentage of collection sites which furnished resistant plants.

The diversity of genotypes for resistance and the common occurrence of resistance in the natural populations makes *Triticum dicoccoides* a rich gene-pool for resistance

to powdery mildew. Since genes for resistance to wheat diseases are transferable from wild emmer to both *T. durum* (GERECHTER-AMITAI & GRAMA, 1974) and *T. aestivum* (GRAMA & GERECHTER-AMITAI, 1974), *T. dicoccoides* may be utilized as a valuable source of powdery mildew resistance in wheat breeding.

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# A comparative study of resistance to powdery mildew in wild emmer wheat in the seedling and adult plant stage

C.H. VAN SILFHOUT[1] and Z.K. GERECHTER-AMITAI[2]

[1] Research Institute for Plant Protection (IPO), P.O. Box 9060, 6700 GW Wageningen, the Netherlands
[2] Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan, Israel

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**Abstract**

In a comparative study of 272 entries of wild emmer (*Triticum dicoccoides*), resistance to powdery mildew (*Erysiphe graminis* f.sp. *tritici*) was determined both in the seedling and the adult plant stage. The performance of the entries was evaluated with two methods, scoring of infection types and noting the uppermost leaf which had become affected. With the first method, 133 entries were resistant both in the seedling stage and the adult plant stage, while with the second method 134 entries were resistant in the seedling stage and free of infection or affected only on the lower leaves in the adult plant stage. Moreover, 26 entries which were susceptible in the seedling stage, became moderately resistant or resistant in the adult plant stage. In general, there was good agreement between seedling and adult plant reactions. Screening for resistance to powdery mildew in the seedling stage was shown to be a reliable method to secure selections for wheat breeding programmes. By screening in the adult plant stage and comparing with the seedling reaction, four different types of resistance were found, namely true seedling resistance, overall resistance, adult plant resistance and partial resistance.

*Additional keywords: Erysiphe graminis* f.sp. *tritici, Triticum dicoccoides.*

## Introduction

The importance of finding new sources of resistance to the powdery mildew fungus of wheat, *Erysiphe graminis* DC. ex Merat f.sp. *tritici* [E. Marchal], has been stressed by various workers. Already two decades ago it was realized that in cultivated wheat the few known genes for resistance successively were matched by virulence genes in the pathogen when cultivars with those genes were grown commercially (Leijerstam, 1965). Therefore, the search was extended to the wild and semi-wild species related to cultivated wheat, and in particular to wild emmer, *Triticum dicoccoides* Körn., a new potential source of resistance (Lehmann, 1968; Filatenko, 1969; Leijerstam, 1972; Krivchenko et al., 1979; Moseman et al., 1984; Gerechter-Amitai and Van Silfhout, 1984).

In a collection of wild emmer from ten sites in Israel, Moseman et al. (1984) found that 49% of the entries were resistant to powdery mildew when they were inoculated in the seedling stage with American cultures of the pathogen. Studying a different collection of wild emmer from 73 sites in Israel and other Middle East countries, Gerechter-Amitai and Van Silfhout (1984) showed that in field nurseries 62% and 48% of the

entries were resistant in the adult plant stage to the local populations of powdery mildew in Israel and in the Netherlands, respectively. Comparing the performance of 47 wild emmer entries which were tested in all six nurseries, they discerned 11 markedly different reaction patterns, indicating the presence of several different resistance genes.

Thus, it has been established by two teams, studying two collections of wild emmer with powdery mildew of different origin, that resistance to the pathogen is rather common in the seedling stage (Moseman et al., 1984) and in the adult plant stage (Gerechter-Amitai and Van Silfhout, 1984). However, it is not known whether the same entry which is resistant to the local powdery mildew population in the seedling stage, will also be resistant to this inoculum in the adult plant stage. In wild cereals, it is often found that seedling resistance is not effective in the adult plant stage. In studies by the second author (unpublished data), seedlings of *Avena barbata* and *A. sterilis* resistant to *Puccinia graminis* f.sp. *avenae* and of *Hordeum spontaneum* resistant to *P. graminis* f.sp. *tritici* and to *P. graminis* f.sp. *secalis*, as well as juvenile plants of *Triticum dicoccoides* resistant to *P. recondita*, were found to be susceptible to the respective pathogens in the adult plant stage.

The main objective of the present investigation is to study the relation between seedling and adult plant resistance to powdery mildew in wild emmer wheat.

## Materials and methods

The wild emmer material used in the present study consisted of 272 entries, collected by the second author. This collection included for the greater part the same entries which were placed at the disposal of Moseman et al. (1984) for their seedling experiments, but differed from that used in our previous adult plant studies (Gerechter-Amitai and Van Silfhout, 1984). For the present investigation, seeds were obtained from 158 collection sites representing the major distribution area of *T. dicoccoides* in Israel, both geographically and ecologically. The sites range from −180 m alt. near Lake Kinneret (Sea of Galilee) to approximately 1500 m alt. on Mt. Hermon. The entries showed a wide range of variation for morphological traits and colour characteristics.

The experiment was carried out at the Zelder Breeding Station, Ottersum, the Netherlands. A local field collection of powdery mildew, isolated from wheat, served as inoculum. The inoculum was multiplied in a greenhouse at 20-25 °C, protected from direct sunlight by a cheese cloth screen. During cloudy days, light was supplemented by SONT lamps supplying approximately 400 Wm $^{-2}$. The same field collection was used both in the seedling tests and in the field trials.

For seedling tests, about 12 seeds were sown in 5 × 5 cm plastic pots, the seedlings were inoculated ten days after sowing, when the second leaf had appeared. Inoculations were made by brushing the seedlings evenly with sporulating plants. The inoculated seedlings were maintained under the same conditions as used for the multiplication of the inoculum. Infection types were scored ten days after inoculation, using a scale from 0 (immunity) to 9 (complete susceptibility), (Moseman et al., 1984).

For the field trial, plants were sown in 4 × 4 cm pots, one seed per pot. For germination of the seeds, the pots were placed in a heated greenhouse. After germination they were kept out of doors and protected from frost at night, for 3 weeks during February. Finally they were planted out in the field in rows of one meter length, ten plants per row, spacing between the rows 40 cm. When the plants were in the tillering stage, pots

with sporulating seedlings were transplanted into the field to serve as spreaders of the inoculum. In order to ensure good infection, the plants in the trial were also inoculated by brushing them with sporulating leaves from the seedling experiment approximately three weeks after the spreaders were set out.

In the adult plant stage, ratings for powdery mildew reaction were made twice, first on June 18 when most entries were in the boot to heading stages and the disease had reached the highest leaf layer, and secondly 15 days later, when the plants were in the heading to milky ripe stages and the disease had developed optimally, with up to 50% severity on the flag leaves. In field readings, reactions were recorded as O = very resistant (no sporulation), R = resistant (small sporulating areas), M = moderately resistant (intermediate size sporulating areas) and S = susceptible (large sporulating areas). In order to facilitate a comparison between seedling and adult plant reactions, the infection types (IT) recorded in the seedling tests were grouped into the same four categories: O (IT 0-2), R (IT 3), M (IT 4-6) and S (IT 7-9).

In the present study, a simplified version of the scale devised by Saari and Prescott (1975) was also used for scoring powdery mildew infection. In this scale, which is based on the position of the highest diseased leaf, a plant is considered more susceptible when higher leaf layers are affected. In this version, disease development was assessed as absent (O), low (L), intermediate (I) or high (H), when no symptoms were visible or when the symptoms were visible on the lower, middle or upper leaves, respectively.

## Results

The reactions of the 272 wild emmer entries tested for powdery mildew resistance are summarized in Table 1a, listing the observed reaction patterns and the number of entries displaying each of these patterns.

In the seedling tests, 136 of the entries (50.0%) were non-sporulating (highly resistant), 19 (7.0%) were resistant and 54 (19.8%) were moderately resistant whereas 63 (23.2%) proved to be susceptible (Table 1b). In the adult plant stage, considering the higher of the two readings only, 138 entries (50.7%) were non-sporulating, 54 (19.8%) were resistant, 35 (12.9%) were moderately resistant and 45 (16.6%) were susceptible (Table 1a). The combined results of the seedling and adult plant tests showed that 151 of the 272 entries were immune or resistant (O or R) in all stages of growth (Table 1c).

Comparing the highest reactions of the adult plants with those of the seedlings (Table 1a), more than half of the entries (173 out of 272) displayed reactions which were similar in both growth stages. In the others, a shift towards resistance was observed in 68 entries and towards susceptibility in 31 entries. Most of these shifts were of minor magnitude, major shifts towards susceptibility occurred in three entries and major shifts towards resistance in 41 entries (Table 1c).

To evaluate resistance in the adult plant stage, a comparison was made between the data obtained on reaction to infection and on the highest leaf layer affected (Table 2). On the basis of this table, the Spearman rank correlation coefficient between the two methods was calculated to be 0.72 for the first adult plant scoring. The correlation coefficient for the second scoring appeared to be slightly higher at 0.76. The high correlation indicates that both methods will generally lead to the same conclusions. However the differences between the two methods indicate cases of special interest. The 14 entries with a resistant reaction and symptoms on intermediate leaf layers (Table 2) and

Table 1a. Comparison of seedling and adult plant reaction to powdery mildew in 272 entries of wild emmer and their distribution according to reaction pattern.

| Seedling reaction* | Adult plant reaction | | | Number of entries |
|---|---|---|---|---|
| | first scoring | second scoring | highest score | |
| O | O | O | O | 113 |
| O | O | R | R | 1 |
| O | R | O | R | 12 |
| O | R | R | R | 7 |
| O | R | M | M | 1 |
| O | M | O | M | 1 |
| O | S | S | S | 1 |
| R | O | O | O | 14 |
| R | R | O | R | 3 |
| R | R | R | R | 1 |
| R | M | O | M | 1 |
| M | O | O | O | 10 |
| M | O | R | R | 1 |
| M | O | S | S | 1 |
| M | R | O | R | 7 |
| M | R | R | R | 10 |
| M | R | M | M | 1 |
| M | M | O | M | 3 |
| M | M | R | M | 6 |
| M | M | M | M | 9 |
| M | M | S | S | 1 |
| M | S | M | S | 3 |
| M | S | S | S | 2 |
| S | O | O | O | 1 |
| S | O | R | R | 1 |
| S | R | O | R | 3 |
| S | R | R | R | 8 |
| S | R | M | M | 2 |
| S | M | R | M | 4 |
| S | M | M | M | 7 |
| S | M | S | S | 4 |
| S | S | R | S | 1 |
| S | S | M | S | 6 |
| S | S | S | S | 26 |
| Total | | | | 272 |

* Reaction classes: O = no visible infection or immune; R = resistant; M = moderately resistant; S = susceptible.

the 12 entries which were moderately resistant and showed symptoms on the higher leaf layers, are considered more susceptible when evaluated with the modified Saari-Prescott scale (fast disease development). Even more interesting are the six entries with

Table 1b. Number of wild emmer entries which were classified as immune (O), resistant (R), moderately resistant (M) or susceptible (S) to powdery mildew at three growth stages.

| Growth stage | (DC*) | O | R | M | S | Total |
|---|---|---|---|---|---|---|
| Seedling | (11) | 136 | 19 | 54 | 63 | 272 |
| Boot - heading | (45-55) | 142 | 55 | 36 | 39 | 272 |
| Water ripe - early milk | (71-73) | 168 | 40 | 29 | 35 | 272 |

* DC = Decimal Code for growth stages (Zadoks et al., 1974).

Table 1c. Seedling reaction and highest adult plant reaction to powdery mildew with postulated type of resistance in 201 wild emmer entries.

| Seedling reaction | Adult plant reaction | Type of resistance | Number of entries |
|---|---|---|---|
| O | M or S | seedling | 3 |
| O or R | O or R | overall | 151 |
| M or S | O or R | adult | 41 |
| S | S | partial | 26* |

* See Table 4.

Table 2. Relation between reaction to powdery mildew and uppermost leaf layer at which symptoms were visible in 130 entries of wild emmer at boot to heading stage.

| Leaf layer | Number of entries in reaction class | | | |
|---|---|---|---|---|
| | R | M | S | total |
| L* | 41 | 14 | 2 | 57 |
| I | 14 | 10 | 4 | 28 |
| H | 0 | 12 | 33 | 45 |
| Total | 55 | 36 | 39 | 130** |

Spearmans $r$ = 0.72.
* L = lower; I = intermediate; H = higher leaf layers.
** Immune plants were excluded.

a susceptible reaction and symptoms on lower or intermediate leaf layers which are considered more resistant when evaluated with the modified Saari-Prescott scale (Table 2 and 4), indicating a slow disease development.

Since in the nursery considerable variation in phenology was observed, notes were taken also on the developmental stage (DC) of each entry (Zadoks et al., 1974). At the time of the first disease reading in the adult plant stage (Table 3), ten entries were in the elongation stage (DC 37), 21 in the flag leaf stage (DC 41), 48 in the boot stage (DC 45), 81 in the awn-peeping stage (DC 49) and 112 in the heading stage (DC 55).

Table 3. Relation between reaction to powdery mildew and earliness in 272 wild emmer entries at the first observation in the adult plant stage.

| Earliness DC* | O | R | M | S | Total |
|---|---|---|---|---|---|
| 37 | 8 | 1 | 1 | 0 | 10 |
| 41 | 16 | 2 | 3 | 0 | 21 |
| 45 | 26 | 7 | 8 | 7 | 48 |
| 49 | 35 | 17 | 12 | 17 | 81 |
| 55 | 57 | 28 | 12 | 15 | 112 |
| | | | | | 272 |

Spearmans $r = 0.07$.
* DC = Decimal Code for growth stages (Zadoks et al., 1974).

Table 4. Level of partial resistance to powdery mildew estimated by percentage leaf coverage at the flag leaf in 26 wild emmer entries which were susceptible in all growth stages.

| Number of entries | Percent coverage | Resistance level |
|---|---|---|
| 6 | 0 | high |
| 2 | < 1 | moderate |
| 4 | 1 | moderate |
| 3 | 5 | low |
| 2 | 10 | low |
| 7 | 25 | low |
| 2 | 50 | check |

From a cross-tabulation of the frequencies of the five developmental stages and the four reaction classes, a Spearman rank correlation coefficient was calculated. This correlation coefficient was not significantly different from zero, indicating that the differences in earliness of the wild emmer entries at the recording date are not correlated with the level of resistance of the entries.

Although in our studies the resistance of wild emmer was evaluated on the basis of reaction to infection or highest leaf layer affected, notes were taken also on the severity of disease. Differences in severity became most evident in the susceptible group, ranging from mere traces of infection to 50% coverage on the flag leaves (Table 4).

## Discussion and conclusions

There was generally a good agreement between seedling resistance and adult plant performance; likely this resistance is based on genes which are effective in all growth stages. This type of resistance is often referred to as 'seedling' resistance, but might better be called 'overall' resistance (Zadoks, 1961). However, there were two groups of entries, in which seedling reaction was not in conformity with adult plant reaction.

In one of these groups, the plants were more resistant in the adult plant stage than in the seedling stage. This shift can be explained by the presence of one or more genes active only in the adult plant stage ('adult plant' resistance). In the other group, in which the plants were more susceptible in the adult plant stage than in the seedling stage, most entries showed only a minor shift in reaction. In a few entries a major shift towards susceptibility was observed; the resistance of these entries could be due to true 'seedling' resistance. In these entries also another mechanism of resistance became evident. Whereas these plants displayed a high infection type in the adult plant stage, and therefore would be classified as susceptible, the severity of infection was low. This was also observed in plants which had a high infection type in all growth stages. This phenomenon is called 'partial' resistance (Parlevliet, 1975) or is sometimes referred to as 'slow mildewing', in conformity with 'slow rusting'.

The fact that resistance to powdery mildew found in the seedling stage of wild emmer is expressed nearly always also in the adult plant stage is of great practical importance. Unlike in cultivated cereals, this is not a general feature in wild cereals, as said before.

In conclusion, for the utilization of wild emmer in wheat breeding programmes, screening for resistance to powdery mildew in the seedling stage was proved to be a reliable method. By additional screening in the adult plant stage, four different types of resistance can be detected, namely 'overall' resistance, 'adult plant' resistance, true 'seedling' resistance and 'partial' resistance.

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**Samenvatting**

*Vergelijkend onderzoek over resistentie tegen meeldauw van wilde emmer tarwe in het kiemplantstadium en volwassenplantstadium*

Van 272 wilde emmer (*Triticum dicoccoides*) herkomsten werd de resistentie tegen meeldauw (*Erysiphe graminis* f.sp. *tritici*) bepaald. Waarnemingen werden gedaan in het kiemplantstadium en tweemaal in het volwassenplantstadium. Voor de evaluatie van

de resistentie in het volwassenplantstadium werden twee methoden vergeleken. Bij de ene methode werd gelet op mate van chlorose en puistgrootte, terwijl bij de tweede methode het hoogste bladniveau werd bepaald waarop meeldauwpuistjes werden waargenomen.

Met de eerste methode bleken 133 herkomsten zowel in het kiemplantstadium als in het volwassenplantstadium resistent te zijn, terwijl met de tweede methode 134 herkomsten in het kiemplantstadium resistent waren en in het volwassenplantstadium geen sporulatie vertoonden of slechts op de onderste bladeren.

Bij vergelijking van de kiemplantreactie met de reactie in het volwassenplantstadium kon onderscheid gemaakt worden tussen resistentie die alleen werkzaam is in het kiemplantstadium (echte kiemplantresistentie), resistentie die alleen in het volwassenplantstadium werkt (volwassenplantresistentie) en resistentie die in alle groeistadia bescherming geeft ('overall' resistentie).

Binnen de groep van 26 herkomsten, die zowel in het volwassenplantstadium als in het kiemplantstadium een vatbaar infectietype vertoonden, bleken 6 herkomsten een lage aantastingsgraad te hebben. Dit duidt op het voorkomen van partiële resistentie.

Concluderend kan gesteld worden dat resistentie tegen meeldauw veelvuldig voorkomt in herkomsten van wilde emmer tarwe en dat deze resistentie wordt veroorzaakt door genen met verschillende werkingsmechanismen.

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# The use of *T. dicoccoides* in winter wheat breeding

C.H. van SILFHOUT, L.J.M. GROENEWEGEN

*Research Institute for Plant Protection, Wageningen, the Netherlands*
*Breeding station Zelder B.V., Ottersum, the Netherlands*

During the last 2-3 decades most known resistance genes to *Puccinia striiformis* have been broken down and even most of the two-gene combinations are not functional anymore. Since the beginning of the sixties attention was drawn to possible new resistance genes (Gerechter-Amitai et al., 1970) in *Triticum dicoccoides* (wild emmer wheat), being the donor of the AA and BB genomes of *T. aestivum* (AABBDD). During the seventies a Dutch-Israeli team detected a number of new genes in *T. dicoccoides* by means of different techniques.

1. Testing for resistance numerous accesions and selections of wild emmer with 20 isolates of *P.striiformis* from all over the world (van Silfhout et al., submitted).
2. Genetical analysis of testcrosses between resistant wild emmer accessions and a susceptible *T. durum* (AABB) cultivar.
3. Genetical analysis of a diallel crossing scheme with accessions resistant to all test isolates.

To transfer resistance genes from *T. dicoccoides* into practically usable bread wheats, bridge crosses using *T. durum* followed by some backcrosses to *T. aestivum* proved already succesful (Grama, 1982). Our paper deals with straight crosses between wild emmer and North-West European winterwheats. This approach opens the possibility to transfer not only monogenic resistances but also polygenic inherited characters like high protein content, high seed weight and others. These characters make *T. dicoccoides* an even more interesting species to use in wheat breeding.

Nineteen accessions of wild emmer were selected to be crossed with eighteen bread wheats. Since we expected very low seedset in the crosses the initial choice of bread wheat parents were two locally adapted breeding lines (Ze 2215 and Ze 19987) known to have the kr-genes for easy crossability with rye and other wheat related species. Several other cultivars however were also crossed with wild emmer for comparison. In total 44 crosses were made in a heated and illuminated greenhouse in early spring. To check whether crossability with rye has an important influence on the crossing-barrier between bread wheat and *T. dicoccoides*, 17 of the used wheat lines were also crossed with rye, mostly on the same plant. $F_1$-pentaploid plants were grown next winter in the same greenhouse and -without emasculation- pollinated with bread wheat pollen from several cultivars in order to obtain a $BC_1$ generation. In total 96 $BC_1$ populations were made on 594 ears.

From each of the eighteen original wild emmer selections one $BC_1$ population was chosen to be tested with the appropriate *P. striiformis* race in order to select the plants with *T. dicoccoides* resistance genes for the next backcross. The races used had virulence for the genes already present in at least the backcross parent and mostly also for the initial bread wheat parent (Table 3). Sixty seedlings of each $BC_1$ population were grown individually in small peat pots and inoculated just before the second leaf appeared. Incubation was done in a controlled environment chamber at L/D: 12/12 hours, RH: 100%, temperature 9° C for 24 hours. Before and after incubation plants were kept in climate rooms at L/D: 16/8 hours, RH: 70%, temperature 18/12° C, light intensity 25000 lux. Scoring of the infection types, using the 0-9 scale (McNeal et al., 1971) was done 17 days after inoculation. The initial 44 crosses yielded in total 1028 $F_1$ seeds, that is about 23 seeds per pollinated ear. The 17 test crosses with rye yielded 154 seeds, nine seeds per ear. Table 1 shows the influence of rye crossability on the seedset obtained after pollinating different bread wheat groups with rye and wild emmer pollen.

Table 1. Comparison of seedset between rye-crossable lines hybridized with rye and *T. dicoccoides*.

| Rye crossability of bread wheat parents | Mean seedset (%) | |
|---|---|---|
| | Rye pollen | *T. dicoccoides* pollen |
| Good | 84 | 87 |
| Fair | 27 | 52 |
| Low | 7 | 45 |

Many of the 1028 $F_1$ plants showed hybrid necrosis, the others were very vigourous but semi-sterile at flowering. With the remaining plants 96 back crosses were made on 594 heads, using 10 different bread wheat pollinators In total 15000 $BC_1$ seeds were obtained, that is 26 seeds per ear. To check the procedure of pollinating without emasculating, several ears of four selected crosses were bagged to avoid crosspollination. In Table 2 the resulting seedset is compared with the seedset after pollination with bread wheat pollen.

Table 2. Comparison of seedset between selfed (aneuploid pollen) and hand-pollinated (euploid -ABD- pollen) $F_1$ plants of four selected crosses.

| Cross | Selfed | | Hand-pollinated | |
|---|---|---|---|---|
| | Number of ears | Grains/ear | Number of ears | Grains/ear |
| 1 | 3 | 10 | 18 | 32 |
| 2 | 8 | 7 | 18 | 25 |
| 3 | 13 | 15 | 22 | 25 |
| 4 | 4 | 15 | 14 | 45 |

The offspring of cross 1 was grown in the field to check the number of selfed plants. Both bread wheat parents are awnless while *T. dicoccoides* is awned. Since formation of awns is a monogenic recessive inherited character, 25% of the plants are expected to be awned if there was only selfing. On the basis of the results of cross 1 in Table 2, it would be expected that 1/3 of the seeds is a result of selfing. However in the field out of 270 plants only 2 awned plants were found, while 31 were expected.

The reaction of the 18 $BC_1$ populations and the segregation ratio's are

shown in Table 3. If both bread wheat parents are susceptible to the test-isolate, and the wild emmer parent has one dominant gene, the expected ratio is 1:1. Those conditions were observed in the first eight crosses shown in Table 1. In these cases the resistant plants have the resistance genes of wild emmer in heterozygous condition. In entries 9, 10 and 11 also a 1:1 segregation was observed but the inheritance of the wild emmer gene was not completely dominant. In entries 12-16 the first bread wheat parent was resistant. In this case a 3:1 segregation ratio is expected. Three of those five crosses fitted a 3:1 ratio, if only the susceptible plants are put in the susceptible class. However if the border-line is drawn according to the infection types of the parents a 1:1 segregation fits the observations, which probably means that the most resistant plants inherited the wild emmer gene. The other two did not fit a 3:1 ratio indicating that either the gene in the bread wheat parent or the gene in the *T. dicoccoides* parent behaves recessive with this isolate. The small number of resistant plants in the next cross could be explained by heterozygoty of the wild emmer parent. In the last cross the expected ratio is 1:0 because the backcross parent was resistant. The observed 1:1 ratio could be explained by a recessive inheritance of the gene in the backcross parent.

Table 3 : Parentage and infectiontypes of $BC_1$ populations and the respective parents with segregation ratio's

| Entry | Pedigree | IT parents | | | IT $BC_1$ | | | | | | | | | | Segregation | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_3$ | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | Exp. | Obs. | p[1] |
| 1 | Ze 2215-4/G25-4M//Obelisk | 8 | 1 | 4 | | 13 | | 9 | | | | | 26 | | 1:1 | 22:26 | ** |
| 2 | Ze 19987-4/G90-1-18M//Obelisk | 8 | 1 | 4 | | 23 | | | | | | | 19 | | 1:1 | 23:19 | ** |
| 3 | Ze 2215-4/G148-1-2M//Obelisk | 9 | 1 | 5 | | 18 | | 8 | | | 6 | | | 17 | 1:1 | 26:23 | ** |
| 4 | Granada/G213-2M//Donjon | 6 | 2 | 7 | | 13 | 28 | 13 | | 4 | 9 | | 28 | | 1:1 | 54:41 | ** |
| 5 | Ze 19987-3/G342-2-2-2M//Granta | 9 | 1 | 5 | 5 | 13 | | | | | 13 | | 12 | | 1:1 | 18:25 | ** |
| 6 | Mission/G387-2M//Donjon | 8 | 2 | 7 | | 14 | 14 | 9 | | | | | 22 | | 1:1 | 28:31 | ** |
| 7 | Ze 19987-1/G487-2M//Donjon | 7 | 2 | 6-7 | | 12 | 11 | 3 | | | | | 17 | | 1:1 | 26:17 | ** |
| 8 | Ze 19987-3/G168-1-2-4BM//Donjon | 8 | 1 | 6 | | 12 | 4 | 15 | | | 8 | | | 17 | 1:1 | 31:25 | ** |
| 9 | Ze 2215-4/G303-3M//Kanzler | 9 | 1 | 8 | | | | 10 | | | 11 | | | 24 | 1:1 | 21:24 | ** |
| 10 | Ze 2215-4/G306-3M//Kanzler | 9 | 1-2 | 8, | | | | 14 | | | | | 16 | | 1:1 | 14:16 | ** |
| 11 | Ze 2215-4/G327-2BM//Kanzler | 9 | 2 | 8 | | | | | 7 | | | | 16 | | 1:1 | 7:16 | * |
| 12 | Ze 2215-4/G7-2-4M//Donjon | 2-4 | 1 | 5 | | 16 | 12 | 15 | 5 | | | | 10 | | 3:1 | 48:10 | ** |
| 13 | Ze 2215-4/G194-3M-GM//Citadel | 2-3 | 1 | 5 | | 13 | 14 | 12 | 6 | | | | 15 | | 3:1 | 45:15 | ** |
| 14 | Ze 19987-7/G395-7-1M//Donjon | 1 | 0 | 7 | | 17 | 11 | | | | | | 11 | | 3:1 | 28:11 | ** |
| 15 | Ze 19987-7/G4-1M//Donjon | 1 | 1 | 7 | | 10 | 12 | 11 | | | | | 29 | | 3:1 | 33:29 | - |
| 16 | Ze 2215-4/G315[a]-1M//Donjon | 1-2 | 2 | 6 | | 19 | | | 4 | | | | 14 | | 3:1 | 19:18 | - |
| 17 | Daws/G7-2M//Daws | 8 | 2 | 8 | | 4 | | 8 | | 8 | | | 39 | | 1:1 | 12:47 | - |
| 18 | Ze 19987-4/G363-4-4B//Obelisk | 9 | 1 | 2 | | 23 | 8 | 4 | | | | | 21 | | 1:0 | 35:21 | - |

1) - = non fitting
* = fitting
** = good fitting

Under our greenhouse conditions and with our rye crossable lines we found a normal seedset. With the commercial cultivars there is a weak crossing barrier. A strong crossing barrier as reported earlier (Lange and Jochemsen, 1979; Grama and Gerechter-Amitai, 1974) was not found. Conditions and techniques of crossing seem to play an important role.

From the results of the selfing check it is clear that emasculation of the pentaploid $F_1$'s is not necessary. Apparently there is preferential

fertilization by the euploid bread wheat pollen. Although a few selfed plants will occur in $BC_1$ populations this approach offers the possibility to create easily a very large $BC_1$ generation.

In our opinion there are two main ways to use *T. dicoccoides* in winter wheat breeding. (1) Transferring resistance genes from wild emmer into bread wheat by back crossing about five times. (2) Performing only two back crosses and start selection from thereon.

The first method is a rapid way to produce agronomic well adapted lines enriched with resistance genes which can serve as a breeding stock to produce new varieties or which can be used straight as (multiline) varieties. The choice of the backcross parents, with regard to their resistance genes, is essential to be able to find suitable races to check the transfer of the wild emmer resistance genes.

The second method will not lead quickly to agronomic adapted lines, but has the advantage that also selection for polygenical inherited characters can be carried out in material containing a high percentage of wild emmer genes.

Experience with the use of *T. dicoccoides* shows that many problems can be solved and that useful characters can be transferred to cultivated wheat. Indeed it is well known that the detected resistance genes, which are probably race specific, will not always give a durable protection. However the risk of a breakdown has quite been decreased since integrated control based on intelligent use of resistance and fungicides, has been accepted in agricultural practice.

Résumé

Le but de la présente recherche était de transférer la résistance à *Puccinia striiformis* de *Triticum dicoccoides* à *T. aestivum*. Dans l'exposé la méthode utilisée pour l'exécution des croisements est indiquée. On décrit comment de transfert des gènes de résistance de *T. dicoccoides* est vérifié. Les résultats montrent que des croisements directs, suivis de rétrocroissements sans émasculation de la génération $F_1$ sont prometteurs. Dans la majorité des cas le transfert des gènes de résistance de *Triticum dicoccoides* à la $BC_1$ est réalisable.

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# THE USE OF WILD EMMER IN PRACTICAL WHEAT BREEDING

L.J.M. Groenwegen, Zelder B.V. Plant Breeding Station,
6595 NW Ottersum, The Netherlands
C.H. van Silfhout, Research Institute for Plant Protection (IPO),
Box 9060, 6700 GW Wageningen, The Netherlands

## Summary

Since 1983 a group of four private dutch wheat breeding companies and several governmental institutes co-operate in performing a breeding and research project with wild emmer wheat (*Triticum dicoccoides*). Over 300 interspecific crosses gave rise to more than 700 backcross populations with West European winter bread wheats (*T. aestivum*). The crossing barrier between the two wheat species is not serious; mean seedsets are about half of normal. Genetic material of about 50 wild emmer acccesions was succesfully transferred to hexaploid winter wheat. A growing number of wild emmer derived lines, up to now over 5000, are under evaluation. Synthetic hexaploids recently developed from crossing wild emmer (AABB) with *Triticum tauschii* (DD) offer special possibilities. Attention is mainly paid to yellow rust (*Puccinia striiformis*) and mildew (*Erysiphe graminis tritici*) but also to other disease resistances and valuable crop and quality characters.

## Introduction

Wild emmer wheat is certainly the most important ancestor of bread wheat. Two of the three wheat genomes are derived from this wild species, which was discovered by Aaronsohn (1910). Its possible importance for wheat improvement was indicated a few years later (Aaronsohn, 1913). It is therefore astonishing how little attention we, wheat-breeders, paid to wild emmer. Only about 15 years ago extensive collecting was started, mainly by Israelian scientists and no sooner than 1965 (Grama & Gerechter-Amitai, 1974) the first attempts at the use of wild emmer in bread-wheat breeding were made. In the last decade, however, wild emmer was rediscovered as a major source of useful variation.

Deterred by the reported crossing barrier we have made our first bread-wheat-emmer cross in 1981, soon followed by a series of crosses which proved that this threshold was much lower than expected (van Silfhout & Groenewegen, 1984). The variation created was so exciting and the possibilities were so numerous that we decided for a broader approach. In 1983 IPO, Foundation for Agricultural Plant Breeding (SVP) and Zelder brought the private dutch wheat breeders together in a working party aiming at basic research by the institutes and breeding efforts by the firms. This paper reports on five years activity of this "Research and breeding group wild emmer".

## Materials

Wild emmer wheats were obtained by IPO through a cooperative project with the Volcani Institute in Israel. Bread wheats of course were abundantly available in the collections of the wheat breeders. *Triticum tauschii*, used for synthetic hexaploids by SVP were obtained from Gatersleben and PBI Cambridge.

**Methods**

IPO tested the material for several diseases. First priority was given to yellow rust resistance, second to mildew resistance, third to the ear and leaf didseases *Septoria nodorum*, *S. tritici* and *Fusarium culmorum*. Disease tests were carried out in greenhouse and field in seedling as well as adult-plant stage. IPO provided infection material for the breeders and supported tests of segregating backcross material. Many populations and breeders lines were completely tested and selected at IPO in order to analyse the inheritance of emmer genes. Programs for the creation of isogenic lines have been started. SVP resynthesised hexaploids by crossing wild emmer wheats with *T. tauschii* followed by embryoculture and colchicine-doubling. Research on protein content in wild emmer derivatives has been started.

Four private breeding companies, Cebeco, Semundo, Van der Have and Zelder/Wiersum performed crosses, backcrosses and second backcrosses between wild emmer wheats and bread wheats. Backcross in this report is used in the broad sense: For cross and backcross mostly different bread wheats were used. Lines from these crosses are exchanged within the group, as well as experience. The goal is to create a gene-pool of wild emmer characters in a well adapted European winter wheat background.

The University Plant Breeding Department (IVP) finally provided the group with any significant literature. In later stages other institutes may join the group.

**Results - 1. Practical breeding work**

The ease of crossing and the virtual absence of a serious crossing barrier between *T. dicoccoides* and *T. aestivum* was confirmed by our group.

Table 1. Seedset in crosses and backcrosses between *Triticum dicoccoides* and *Triticum aestivum*

| Cross | v.d.Have | Zelder | Cebeco | IPO | Mean |
|---|---|---|---|---|---|
| T.aest. x T.dic. | 8.1 (69)[1] | 13.4 (55) | 20.2 (30) | 8.3 (53) | 11.3 (207) |
| T.dic. x T.aest. | 8.1 (30) | | 7.0 (37) | | 7.5 ( 67) |
| Pentapl. x T.aest.[2] | 10.2 (78) | 23.9 (104) | | | |
| | | 25.7 (594) | | | |
| T.aest. x Pentapl. | 2.6 (8) | | | | |
| Check;aest. x aest. | 16.7 | 26.2 (78) | | | 21.5 |

[1] in grains per ear () number of ears

[2] v.d.Have crosses emasculated, Zelder crosses only pollinated

Wheat x emmer crosses give about half the normal seedset. The reciprocal cross also gives a fair number of seeds but most seeds do not germinate. Backcrosses on the pentaploid are not problematic, its use as pollen parent, however is not recommended. Hybrid necrosis was in many combinations a serious problem which can only be avoided by matching combinations and the use of non Ne-gene carriers.

In Table 2 a summary is given of the numbers of crosses and backcrosses realized by the four breeding teams, while in Table 3 the number of "lines" is given which are selected from the backcross populations for evaluation. "Lines" is used in the broad sense; up to now the lines are mainly $F_2$ or $F_3$ plant progenies.

Table 2. Number of crosses and backcrosses between wild emmer and bread wheat

| | Zelder | | | v.d.Have | | | Cebeco | | | Semundo | | Total | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Year | F1 | BC1 | BC2 | F1 | BC1 | BC2 | F1 | BC1 | BC2 | F1 | BC1 | F1 | BC1 | BC2 |
| 1981 | 1 | | | | | | | | | | | 1 | | |
| 1982 | 44 | 6 | | | | | | | | | | 44 | 6 | |
| 1983 | 3 | 99 | 35 | 21 | | | 45 | | | 36 | | 105 | 99 | 35 |
| 1984 | | 6 | 102 | 22 | 29 | | 10 | 33 | | 15 | 43 | 47 | 111 | 102 |
| 1985 | 8 | | | 7 | 21 | 29 | | | 16 | 43 | 78 | 58 | 99 | 45 |
| 1986 | | 23 | | | 7 | 20 | 31 | | | 23 | 39 | 54 | 69 | 20 |
| 1987 | 55 | 29 | | | | 39 | 11 | 25 | 8 | | 40 | 66 | 94 | 47 |
| | | | | | | | | | | | | 375 | 478 | 249 |

Table 3. Number of wild emmer derived lines evaluated in the field

| Year | Tested material | | Zelder | v.d.Have | Cebeco | Semundo |
|---|---|---|---|---|---|---|
| 1986 | Observation rows | BC1 | 1800 | | | |
| 1987 | Observation rows | BC1 | | 348 | 650 | |
| | | BC2 | 810 | 327 | | |
| | Yield test | BC1 | 111 | | | |
| 1988 | Observation rows | BC1 | 224 | 446 | 413 | 63 |
| | | BC2 | | 480 | 20 | |
| | Yield test | BC1 | 26 [1] | 35 | 19 | |
| | | BC2 | 32 | 20 | | |

Totals: lines row-evaluated 5581, lines yield-tested 217
[1] tested 2nd year on four locations

In seven years 375 interspecific crosses were realized and used for the creation of over 700 backcross populations. More than 5 thousand plant progenies were selected from these populations and evaluated. This work is still continuing. In Table 4, 31 wild emmer accessions are listed mainly used for transferring yellow rust resistance genes and in Table 5 we have listed 21 accessions mainly used for mildew resistance genes.

Table 4. Wild emmer accesions crossed for yellow rust resistance

| Accession | ZEL | VDH | CEB | SEM | Accession | ZEL | VDH | CEB | SEM |
|---|---|---|---|---|---|---|---|---|---|
| G4-1M | x | x | + | x | G213-2M | x | x | | |
| G7-9499 | | x | | | G280-1BM | x | + | x | |
| G7-2M | x | | | | G297-3M | | x | | + |
| G7-2-4M | x | x | x | + | G303-3M | x | | x | x |
| G7-2-6B-3M | | + | x | + | G306-3M | x | | | |
| G25-4M | x | x | x | | G306-12M | | + | + | + |
| G25-78-27 | | + | x | x | G315a-1M | x | + | + | x |
| G29-1M-8-2M | | x | | | G326-1-4-5-3M | | x | x | x |
| G90-1-1BM | x | + | | x | G327-2BM | + | x | + | x |
| G148-1-2M | x | x | x | x | G330-1-6B-1M | | x | x | |
| G156-3M | | x | x | x | G332-1-3-1M | | x | | x |
| G168-1-2-4BM | + | + | | + | G342-2-2M | x | | | |
| G193-1M | | | x | | G363-4-4BM | x | x | | |
| G194-3M-6M | x | + | x | x | G387-2M | x | x | x | x |
| G197-2-1M-4M | | | + | | G395-7-1M | x | x | x | |
| | | | | | G487-2M | x | | | |

+ = crosses made, x = crosses made and lines in evaluation

Table 5. Wild emmer accesions used for mildew resistance

| Accession | ZEL | CEB | VDH | Accession | ZEL | CEB | VDH |
|---|---|---|---|---|---|---|---|
| G314-7M | + | | | G600 | + | | |
| G542 | | + | | G606 | + | + | |
| G543 | + | | | G609-1B | + | | |
| G551 | + | | | G695 | + | | |
| G552 | + | + | | G789-1 | x | | |
| G554 | | + | | G797-M | + | + | |
| G560 | + | + | | G802 | + | | |
| G569 | + | | | G815 | + | | |
| G584 | + | + | | TTD09 | + | | + |
| G591 | x | + | | TTD12 | | | + |
| | | | | TTD15 | + | | + |

+, x see Table 4.

In conclusion genetic material of 52 wild emmer accessions was succesfully transferred to hexaploid wheat. Stable hexaploid lines derived from 30 wild emmer accessions are under evaluation now.

Also from the bread wheat side the program is very broad. This is illustrated by Table 6.

Table 6. Number of different bread wheat parents used in crosses and as pollinators in first backcrosses

| | Zelder | v.d.Have | Cebeco | Semundo | Total[1] |
|---|---|---|---|---|---|
| F1 parents | 24 | 29 | 13 | 13 | 72 |
| BC1 pollinators | 17 | 34 | 3 [2] | 18 | 64 |

[1] 7 resp. 8 varieties were used by more than one breeder for $F_1$ resp. $BC_1$ pollination

[2] Apart from 3 named varieties Cebeco used pollen mixtures of unnamed bread wheat material

Altogether about 120 different winter wheat varieties were used by the four breeding teams for the creation of the wild emmer/bread wheat genepool. There was surprisingly little overlap in the use of bread wheat parents by the four breeding teams.

From 1987 on wild emmer derived backcross lines (see Table 3) are evaluated in yield and performance trials. Table 7 gives a summary of the first replicated yield trial with 111 Zelder lines in 1987. The "lines" are $BC_1$ plant progeny bulks from $F_2$ or $F_3$ plants.

Table 7. Relative yield[1] and grain weight of 111 wild emmer derived $BC_1$ lines in 1987

| | | Yield % | Range | TGW | Range |
|---|---|---|---|---|---|
| Check | Obelisk | 102 | 98-107 | 44.0 | 42-45 |
| | Okapi | 95 | 90-99 | | |
| Mean of all WE-lines | | 76 | 54-96 | | |
| | 26 WE-lines | 85 | 66-96 | 44.9 | 36-51 |

[1] 100 = 7323 kg/ha

The yield of the 26 best performing lines of this material is quite surprising, taking into account that 25% of the A and B genomes should be "wild" genes. Above all, the variation in grain weight and in healthiness of the leaves was impressive. Our conclusion: Either wild emmer wheat is not so wild or wheat is not so domesticated as it seems.

Results - 2. Research

In this "breeders" paper a brief review of the institute research results of our group is added. The SVP succesfully resynthesised 26 hexaploid lines from crosses of 9 wild emmers with 6 *T. tauschii*. In 1987 the synthetic hexaploids were handed to the breeders and used in crosses as summarized in Table 8.

Table 8. Crosses between synthetic hexaploids and winter wheat

| Synthetic[1] | ZEL | VDH | CEB | SEM | Synthetic | ZEL | VDH | CEB | SEM |
|---|---|---|---|---|---|---|---|---|---|
| SVP G004/33 | + | + | + | + | SVP G168/G | + | + | + | + |
| SVP G004/473 | + | | | | SVP G168/L | + | + | | + |
| SVP G004/525 | | + | + | | SVP G193/G | + | | + | |
| SVP G025/L | | + | + | + | SVP G193/L | + | + | | |
| SVP G090/G | + | | | | SVP G315a/33 | + | + | | |
| SVP G148/33 | + | | + | + | SVP G315a/L | | | + | |
| SVP G148/473 | + | + | | | SVP G342/33 | + | + | | + |
| SVP G148/525 | + | + | + | | SVP G342/143 | + | | | |
| SVP G148/L | | + | | | SVP G342/L | + | + | + | + |
| ABD Berlin | + | | + | | SVP G363/L | | + | + | |

[1] Wild emmer line / *T. tauschii* line

From cytological investigations at the SVP with this material we know already that in most cases the fertility of these crosses is normal (and not disturbed by aneuploidy as is the case with the bread wheat x emmer crosses). Thus a broad material with 50% wild emmer genes can be created.

Some research on possible high quality was done by SVP. Hexaploid derivatives from a few crosses only gave an indication that significant progress in protein quantity per unit area is not easily obtainable from middle high protein wild emmer. Israelian high protein derivatives under our conditions neither gave better protein yields than adapted local spring wheats.

The main contribution of IPO has been disease testing. Table 9 gives a summary of 5 years.

Table 9. Number of entries tested for disease resistance at IPO

| Year | Yellow rust | | | Mildew | | *Septoria nodorum* | *Fusarium culmorum* | *Septoria tritici* |
|---|---|---|---|---|---|---|---|---|
| | wild[1] | pop. | der. | wild | der. | wild | wild | wild |
| 1983 | 90 | 19 | | | | | | |
| 1984 | | 35 | | 272 | | | | |
| 1985 | | 34 | | | | 27 | 27 | |
| 1986 | | 27 | | | | 27 | 27 | 27 |
| 1987 | 67[2] | | 134 | 43 | 111 | | | 51 |

[1] wild = wild emmer accessions, pop. = populations with wild emmer parents, der. = wild emmer derived lines

[2] accesions of *T. tauschii*

Most emphasis was given to yellow rust and powdery mildew resistance. In many wild emmer derived lines the presence of resistance genes of the wild parents was demonstrated. Preliminary research on ear diseases showed that in wild emmer also interesting variation exists. For *S. nodorum* most entries were more resistant than the best check, while some were very resistant. For *F. culmorum* good levels of resistance were demonstrated under field conditions. In greenhouse conditions, however, results were less promising. For *S. tritici* the pycnidial density on wild emmer leaves was found to be generally lower than on leaves of bread wheat check varieties.

**Conclusions and discussion**

The cooperation between private companies and governmental institutes has resulted in well organised effective transfer of genes from wild emmer to bread wheat. Large scale crossing and selection work by for breeding teams each with its own philosophy and breeding stock will increase chances of promising combinations. Both guidance on the transfer of resistance genes as well as better understanding of chromosome and gene interactions promoted this result. A hesitating begin aimed at a few exotic disease resistance genes can grow now to a significant effort in broadening the genetic base of the bread wheat crop. Realizing, however, that over 2000 accessions of wild emmer are available in the world collection (Chapman, 1985) we may just make the first steps on a long way. For a broad exploitation of the variation in wild emmer wheat, we therefore recommend any wheat breeder not (only) to use the accessions listed in our tables.

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## GENERAL DISCUSSION AND CONCLUSIONS

### *Major-gene resistance to yellow rust*

At the beginning of this study, research was mainly concentrated on the search for yet unused major genes for hypersensitivity resistance to yellow rust (*Puccinia striiformis*) in wild emmer wheat (*Triticum dicoccoides*). This search was started because the known major genes for resistance in bread wheat to this pathogen had become ineffective in many wheat-growing areas of the world. The known major genes are characterized by large gene effects, mainly dominant inheritance and race-specificity. Most of these genes are effective at all growth stages of the plant. Therefore they are often screened for in the seedling stage by which they are sometimes called 'seedling' resistance genes. A better term is 'overall' resistance genes (Zadoks, 1961). Though it was generally accepted, at the start of this study, that resistance caused by overall major genes is in most cases not durable (Johnson and Law, 1975; Johnson, 1979, 1981, 1984), and therefore not desirable, several reasons advocate the use of this type of resistance.

In the first place a solution was needed on a short-term until practical methods would be developed to select for more durable types of resistance. Breeding procedures to introduce overall resistance are well known, by which new sources of overall resistance can be quickly introduced in breeding programmes.

Secondly, overall major genes for resistance are easily recognizable and selection for these genes is simple, because they usually are stable under different environmental conditions, and the resistance caused by these genes often has a high heritability. In contrast, other mechanisms for resistance to air-borne obligate parasites, which are shown or claimed to be more durable, are generally less easy to recognize and selection for this type of resistance requires special knowledge and refined methods.

Thirdly, although more genes may be involved in a resistant genotype, resistance caused by overall major genes is in many cases effectively inherited in a monogenic way, which makes it easier to combine with a number of characters which are inherited in a polygenic way (e.g. yield, baking-quality, adaptation etc.). Moreover a breeder has often to combine resistance genes to more than one pathogen which is easier when using monogenic overall resistance. If the resistance to these pathogens is inherited in a oligogenic or polygenic way, the chance to find genotypes which combine all these genes will be rather low. Such resistance has been demonstrated in several host-pathogen relations. For instance, partial or slow-rusting resistance of wheat to leaf rust has an oligogenic inheritance (Bjarko and Line, 1988; Broers and Jacobs, 1989; Lee and Shaner, 1985), temperature-sensitive resistance to stripe rust of wheat has an oligogenic or polygenic inheritance (Milus and Line, 1986; Sharp, 1982), while partial resistance to leaf rust of barley (Parlevliet, 1978a) and partial resistance to stem rust of wheat (Knott, 1988) are inherited in a polygenic way.

Fourthly, by resistance management (Mundt and Browning, 1985) the usefulness of the major-gene resistance can be extended. These methods and strategies include the use of different genes in succeeding zones of rust-pathways (Vanderplank, 1968; Knott, 1972; Reddy and Rao, 1979; Nagarajan and Joshi, 1980, 1985), cultivation on the same farm of more than one variety with different genes for resistance and belonging to different varietal groups with regard to the prevailing races (Priestley, 1981; Priest-

ley and Bayles, 1982; Anonymus, 1989), and the use of multilines (Jensen, 1952; Borlaug, 1959; Browning and Frey, 1981; Groenewegen and Zadoks, 1979). Another possibility to prolong the usefulness of major-gene resistance is the accumulation of several major genes in one genotype (pyramiding). Pyramiding should be performed in such a way that at least two genes or two different combinations of major genes are still effective against the race population in the epidemiological unit. It has been shown (Drijfhout, 1987) that this is possible with conventional breeding methods although it is laborious and requires a special breeding programme. However, if biotechnological methods become available for the detection of resistance genes in wheat (e.g. with RFLP-markers), this option may become more attractive. By screening the offspring of a cross between parents carrying different major genes with such a method, plants which have inherited the resistance genes from both parents could be detected. In the more distant future, when direct gene transfer would be possible, pyramiding resistance genes may become an important method to protect wheat against yellow rust.

A prerequisite for breeding for overall resistance is the availability of major genes, preferably yet unused and for which virulence has not yet been observed. The number of major genes for overall resistance in bread wheat is limited and most of these genes have become ineffective (Stubbs, 1988). In wild emmer wheat at least eight major genes have been found (chapter 3 and 4) which differ from the identified genes in bread wheat (chapter 2), while in the latter study at least eleven different resistance factors were indicated. The resistance genes from wild emmer can be used for a quick introduction of new resistance in bread wheat breeding programmes. When used in a sophisticated way, the expected short durability of these resistance genes can be prolonged by proper resistance management. The large number of genes which were found in wild emmer creates many possibilities for diversification and for pyramiding these yet unused genes.

*Minor-gene resistance to yellow rust*

In addition to the above-mentioned major-gene resistance, another kind of hypersensitivity resistance is also found in wild emmer wheat. This kind of resistance has been detected by testing seedlings at higher temperatures (15/24 °C) than commonly used (15/17 °C) in screening for resistance to yellow rust. In wild emmer wheat, the genes which induce this type of resistance are characterized by temperature-sensitivity, additive gene action, mainly dominant inheritance, small gene-effects and race-specificity (chapter 7 and 8).

Although hypersensitivity resistance is considered to be not very durable, this generalisation has to be used with care. The lack of durability, which is often reported when this type of resistance is used, may not be inherent to the mechanism, but due to the fact that this kind of resistance is mostly inherited in a monogenic way (i.e. based on one effective gene). In the case of additive minor genes, complete (hypersensitivity) resistance can be obtained which has an oligogenic or polygenic inheritance and is supposed to be more durable.

Race-specificity of resistance is often considered to be a negative trait as well. This may be true for the character 'resistance' but is not necessarily true for individual resistance genes. Parlevliet and Zadoks

(1977) concluded, from a model study, that race-specific minor genes with additive effects confer on average a higher level of resistance and a higher degree of durability than race-non-specific minor genes. However it is often difficult to distinguish between race-specificity and race-non-specificity of minor genes. If the effects of the individual genes are very small, the differential interaction will be covered up by the trial error and the conclusion would be that the resistance is race-non-specific (Parlevliet, 1983). In addition to the expected effect on the average level of resistance and the durability of the resistance, race-specificity is a trait which can be used for the recognition of resistance genes. It is an advantage to be able to recognize different genes (chapter 8), because if two entries carry different minor genes, it can be predicted that transgressive segregation for higher levels of resistance will occur in the offspring of a cross between these entries (chapter 7).

In contrast to hypersensitivity inducing major genes, which are epistatic to each other, the minor genes described above can be easily pyramidised, because visual selection for higher numbers of these resistance genes is possible (chapter 7). Selection for this trait can be done in the seedling stage as well as in the adult-plant stage (chapter 6). In the seedling stage, first a negative selection can be done at common screening temperatures (17/15 °C) against major genes, followed by a positive selection at higher temperatures (24/15 °C) for temperature-sensitive minor genes. If several minor genes are combined in one genotype, the resistance is also expressed at common screening temperatures (chapter 7). Hence selection, for still higher levels of resistance, can be continued at common screening temperatures. In the adult-plant stage, selection for minor genes is more difficult because the phenotypic expression of minor genes can be similar to that of 'overall' major genes and to that of 'adult-plant' major genes. Testing the offspring of a cross with an isolate of the fungus to which the parents were susceptible will help to avoid the selection of undesirable new effective combinations of overall major genes or adult-plant major genes.

The number of different minor genes in wild emmer wheat seems to be large (chapter 7 and 8). Additivity of the gene-action does not only occur among the minor genes in wild emmer but also between minor genes from wild emmer and from bread wheat (Reinhold et al., 1983). Thus it seems that there is a large gene-pool of still unexploited resistance genes available. This large number of genes will enable breeders to diversify their germplasm with respect to resistance against yellow rust. This may also prolong the durability of the resistance of a cultivar as explained above.

Although it can be argued that resistance based on several genes (oligogenic or polygenic inheritance) would in general be more durable than resistance based on a single gene, this is of course no proof that this will be true in practice. Multilocational testing of lines which carry one or more temperature-sensitive minor resistance genes has shown (Stubbs, 1977) that this kind of resistance is stable in different environments, but this does not prove that the resistance is durable. However, several varieties like 'Nugaines', 'Crest' and others, with resistance based on temperature-sensitive genes, have been cultivated in the north-western part of the United States of America for about 20 years and are still resistant to yellow rust (Milus and Line, 1986; Qayoum and Line 1985). This indicates that this kind of resistance is indeed more durable than resistance based on major genes.

*Adult-plant resistance to yellow rust*

Selection for adult-plant resistance is usually performed in the field. This may cause some difficulties in the recognition of the type of resistance. As described above 'overall' major genes as well as temperature-sensitive minor genes are effective in the adult-plant stage, the latter especially when temperatures are increasing at the end of the growing season. Additional seedling tests under controlled environmental conditions are necessary to distinguish between these types of resistance and true adult-plant resistance.

In wild emmer wheat also true adult-plant resistance was detected (chapter 6). At least four sources of adult-plant resistance were found. Adult-plant resistance can be race-specific such as the resistance induced by the resistance genes Yr11 to Yr14 (Johnson et al., 1987) and others (Stubbs, 1985 and 1988; Zadoks, 1961), and can also be durable (Johnson, 1988). Although the inheritance of the adult-plant resistance in wild emmer was not studied, van Silfhout and Drenth (1988) did study the inheritance of adult-plant resistance in a large number of bread wheat lines resistant to all used isolates. They showed that this kind of resistance is based on two or more genes which often singly induce incomplete resistance. Johnson (1988) stated that durable resistance in bread wheat to yellow rust is often based on incomplete adult-plant resistance but that the reverse is not true. It was shown that the durable resistance of the variety Capelle Desprez (Johnson, 1978) is inherited in a complex way (Law et al., 1978; Worland and Law, 1986) consisting, at least, of two factors which are additively inherited. It may be a coincidence that the adult-plant resistance in both studies seems to be based on genes with additive effects but it may indicate that durability of adult-plant resistance is dependent on the number of effective genes which is involved in the resistance. Whether the adult-plant resistance in wild emmer will be of the durable type can not be predicted before the background of the mechanism of durable resistance is known. However, it may contribute to the genetic variability of resistance to yellow rust in wheat.

*Resistance to powdery mildew*

The study on disease resistance in wild emmer wheat also revealed that there are many accessions which exhibit resistance to powdery mildew. In total, 379 entries were tested in the adult-plant stage (chapter 9 and 10) of which 272 also in the seedling stage (chapter 10). In the seedling stage about half of the number of entries showed a low infection type, probably due to the presence of major genes, after inoculation with conidiospores from a field population of *Erysiphe graminis* collected in the Netherlands. Using 217 entries of the same collection of wild emmer, Moseman et al. (1984a) found about the same percentage of resistant entries in tests with two composites of two cultures of *E. graminis* collected in the United States of America. The four cultures were selected becuase of their virulence on known genes for resistance in wheat, indicating the presence of yet undescribed resistance genes in those wild emmer entries which were resistant to both composites. Using the same two composite cultures, these authors found that also among another 233 wild emmer entries about 50 % exhibited resistance. They concluded that the highest level of resistance to powdery mildew, measured as the average infection type of all entries from a certain collection site, is obtained in habitats where *Triticum*

*dicoccoides* is well adapted. However this is not the main point for a breeder, for whom variability is of more importance. Collecting a high percentage of resistant entries from a certain site does not mean that there will be a large number of different genes in these entries. On the other hand, the observation that highly resistant entries were found in most collection sites (chapter 9; Moseman et al., 1984a), might be a better indication for the chance to find different genes for resistance. Moreover, the fact that several entries showed a differential interaction with different mildew populations (chapter 9) indicates that at least in these entries different resistance genes do occur. In addition, this differential interaction may provide an indication that different genes can also be found in those entries which were resistant in all tests. Experience has learned that the use of hypersensitivity resistance against powdery mildew has at least the same risk as with this kind of resistance against yellow rust. The risk may even be higher because the virulence genes in *Erysiphe graminis* can recombine during the generative phase. Nevertheless the remarks made above on the possible reduction of this risk in the case of resistance to yellow rust are valid here as well.

In addition to the type of hypersensitivity resistance which gives protection in all growth stages, hypersensitivity resistance has also been found which is effective only in the seedling stage (true seedling resistance) as well as resistance which is effective only in the adult-plant stage (adult-plant resistance) (chapter 10). These genes are in any case different from the overall resistance genes mentioned above and can help to increase the diversity of resistance genes. True seedling resistance can be combined with adult-plant resistance without complicated tests to ensure that at least two different genes will protect the variety during the cropping season.

The presence of partial resistance to powdery mildew has been shown in a number of entries, even though the effect of partial resistance has probably been underestimated because of interplot interference (chapter 10). Partial resistance can only be shown in those entries in which genes for hypersensitivity resistance are not expressed. In this relative small group of entries about half of the entries showed a moderate to high level of partial resistance. Therefore it may be expected that several of the entries, which exhibit hypersensitivity resistance, also carry genes for partial resistance. If a breeder wants to use the partial resistance found in wild emmer, special precaution should be taken to eliminate the hypersensitivity resistance genes, because these genes for hypersensitivity resistance occur in most entries and because they often cause a resistance reaction without any visible symptoms.

*Aspects of resistance in natural host-pathogen systems*

The frequency of plants of wild emmer in which complete hypersensitivity resistance was found, has been highest for powdery mildew, moderate for yellow rust and low for leaf rust. Data on the frequency of occurence of other resistance mechanisms, like partial resistance and minor-gene resistance to these pathogens are less abundant. At present times the frequency of occurrence of these pathogens in the Middle East is lowest for powdery mildew and highest for leaf rust. Several questions may arise from these observations, including the questions whether the importance of these pathogens has been the same during the evolution of wild emmer and what the role of the different resistance mechanisms in these host-

pathogen systems is or has been. To answer these questions, if this is anyhow possible, the difference between these pathogens in selection pressure on the resistance should also be taken into account.

Furthermore these host-pathogen systems cannot be compared with the host-pathogen systems of commercial wheat fields, because wild emmer is always growing in mixed stands of plants of different species, and in most cases the distance between different subpopulations of wild emmer is several hunderds of meters to many kilometers.

With respect to the above-mentioned questions we can be sure of only a few facts. Wild emmer is found in the Middle East and from historical, archaeological and botanical evidence it seems obvious that this species evolved in this area. Concerning the pathogens less evidence is available, but from their specialisation on wheat it can be deduced that they have co-evolved with their host. It is stated that the climatic conditions in the Middle East have been more or less similar to the present conditions over the last few thousand years of the evolutionary period of wild emmer (Bruins, 1989), but it is questionable whether from this evidence the conclusion can be drawn that the frequency and intensity of occurrence of the pathogens has also been similar over a long period of time.

In the case of leaf rust, the most frequent occurring of these pathogens, part of the plants is protected in the seedling stage by hypersensitivety resistance, but most of these plants show already in the early tillering stage a fully susceptible infection type, indicating as with powdery mildew a true seedling resistance. During the later stages of development, when the disease pressure is increasing, only partial resistance combined with the scattered growing of the plants seems to give enough protection against this pathogen. Serious epidemics of leaf rust in populations of wild emmer have not been observed in present times (Z.K. Gerechter-Amitai, pers. comm.). In the case of powdery mildew, the least frequent occurring of these pathogens, about half of the plants are protected by hypersensitivity resistance, although this seems not necessary to keep the pathogen at such a level that it does not eradicate the host, as suggested by Parlevliet (1983). Moreover other protective mechanisms to this pathogen exist as well. It seems that the genetical protection to this infrequently occurring pathogen is more complex than to the frequently occurring leaf rust, while one would expect the reverse. Several factors may explain these differences, such as difference in selection pressure, influence of other host populations, deviations from the present situation in earlier times as mentioned above and probably some more. However our knowledge about these factors is limited.

In conclusion, it seems to be rather hypothetical to speculate about the role of resistance mechanisms in natural host-pathogen systems and even more to transfer these hypotheses to cultivated wheat.

*Transfer of resistance and other characters to Triticum aestivum*

Besides tests for resistance to yellow rust and powdery mildew, wild emmer was also tested for resistance to leaf rust (Moseman, 1984b; van Silfhout, unpublished), *Septoria tritici* (Yechilevich-Auster et al., 1983; Kema and van Silfhout, unpublished), *Septoria nodorum*, and *Fusarium culmorum* (chapter 12; van Silfhout and Kema, unpublished).

Although Moseman (1984b) has found resistance to leaf rust in the seedling stage, many entries of wild emmer exhibit a high susceptibility to this pathogen. Hypersensitivity resistance to leaf rust seems to be mainly

based on genes for true seedling resistance, while in the adult-plant stage hardly any hypersensitivity resistance seems to exist. In a monocyclic test in the field at flowering stage, several entries showed a low infection type at the lowest leaf, while the higher leaves showed increasingly higher infection types, culminating in a fully susceptible infection type at the flag leaf (van Silfhout, unpublished). However in the adult-plant stage variation for partial resistance has been observed.

Resistance to *Septoria tritici*, *Septoria nodorum* and *Fusarium culmorum* has also been found, while in the crosses with bread wheat transgressive segregation for resistance to these pathogens was observed (Groenewegen, personal communication). Possibly with the exception of resistance to leaf rust, wild emmer wheat is a rich source of resistance to several pathogens. With respect to leaf rust, sometimes the reverse may be true.

Among the many other agronomic and quality characters for which large variation has been observed in wild emmer wheat; kernel size, protein content and protein quality have been studied in more detail within the framework of the cooperative project mentioned in the acknowledgement (Gerechter-Amitai and Grama, 1977; Grama et al., 1987a, 1987b) and by other researchers (Avivi, 1979; Avivi et al. 1983; Kushnir and Halloran, 1982, 1984). Several entries of wild emmer exhibit a very large kernel size which may be of importance to enlarge the yield potential of bread wheat. In wild emmer entries the grain protein content is considerably higher than in bread wheats, often ammounting to twice as much. Although in bread wheat a negative correlation has been found between grain yield and protein content, results obtained with high protein lines derived from wild emmer wheat showed that the derivatives of wild emmer have a stronger capacity to accumulate nitrogen in the plant, combined with a more efficient translocation of nitrogen into the grain (Grama et al., 1984a; Grama et al., 1987b; Levy and Feldman, 1987), while preliminary data indicate that the higher protein content (1-2 % more than the control) can be combined with equal or better yields than the control (7000 kg/ha) (Grama et al., 1984b). Protein content and protein quality are both important characters for baking quality. A higher protein content may also increase the nutritional value of bread or other food products made of wheat, which is considered to be important in certain areas (e.g. CIMMYT, 1989, p. 11).

Although all these characters are of much interest for wheat breeders, the problem remained to transfer the genes, conferring these characters, from tetraploid wild emmer wheat (AABB) to hexaploid bread wheat (AABBDD).
There are various strategies to transfer genes from wild emmer wheat to bread wheat. In general they can be grouped in three categories: a) the reconstitution of hexaploid wheat from *Triticum dicoccoides* and *Triticum tauschii* (Lange, 1986), b) using a bridge-cross with *T. durum* and c) straight crosses between bread wheat and wild emmer wheat. The advantages and disadvantages of the different strategies have been discussed by Lange and Balkema-Boomstra (1988). From early reports (Grama and Gerechter-Amitai, 1974; Lange and Jochemsen, 1979) it was deduced that a strong crossing barrier exists between wild emmer wheat and bread wheat. Therefore a crossing experiment was carried out between some wild emmer entries and two winter wheat lines carying the *kr*-genes for crossability between bread wheat and other related species (chapter 11). This experiment included also some north-west European winter wheat varieties which did not carry the *kr*-genes. Surprisingly the seedset in all crosses was about 50 % or more, although the experimental lines carrying the *kr*-genes had a better seed set than the varieties used in this experiment. In further work

(chapter 12) it was shown that no serious crossing barrier does exist, and it was indicated that the main factors which influence the crossability are time of fertilization and environmental conditions.

Hybrid necrosis however, may cause some problems in the early phases of a breeding programme in which straight crosses are used between wild emmer and bread wheat. Many wild emmer entries carry *Ne*1 while many bread wheats carry *Ne*2. The problem can be reduced by selecting bread wheat parents which carry *Ne*1 or which are non-carriers of these necrosis alleles.

The incorporation of genes from wild emmer into spring wheats, which are adapted to the conditions in many developing countries, has been performed through straight crosses between wild emmer and bread wheat. In this programmme 28 wild emmer entries and ten bread wheat varieties, including six varieties from the CIMMYT-programme and four recommended by the cooperators from Nepal. The most advanced material is in BC2-F2 and is being evaluated in Israel and Nepal (Z.K. Gerechter-Amitai and A. Grama, pers. comm.). At present also a substantial part of the breeding programmes in the Netherlands is devoted to the incorporation of valuable characters from wild emmer in hexaploid winter wheat and has reached the stage of BC2-BC5 and F2-F5 in which yield trials and quality tests can be performed (L.J.M. Groenewegen and M.E. Roothaan, pers. comm.).

From the results of the above-mentioned studies and breeding programmes, it can be concluded that the valuable traits of wild emmer can be transferred to bread wheat and that the negative traits, such as brittle rachis and hard chaff, can be overcome in shorter time than expected when these studies were commenced. The yield potential of the most advanced material equals the yields in commercial varieties or is even higher (A. Grama, L.J.M. Groenewegen, pers. comm.). The question whether the often reported negative correlation between grain yield and protein yield also exists in wild emmer derivatives has not yet been fully solved, but the first results indicate that wild emmer derivatives can be selected which outyield the commercial standards, both in grain yield and protein content, while the protein quality for dough making and baking seems to be excellent.

*Choices in breeding for resistance*

As explained and discussed above, there seem to be at least three different types of genes inducing hypersensitivity resistance to yellow rust (i.e. overall resistance, adult-plant resistance and minor-gene resistance) and also three different types to powdery mildew (i.e. overall resistance, seedling resistance and adult-plant resistance). Besides the hypersensitivity reaction also other resistance mechanisms are reported; often referred to as slow rusting in cereal rusts pathosystems or more general as partial resistance. Partial resistance *sensu* Parlevliet (Parlevliet and van Ommeren, 1975) has been found in wild emmer wheat against powdery mildew (chapter 10), and variability for this mechanism with respect to yellow rust has also been observed in wild emmer (unpublished results). In experiments with durable-resistant bread wheats, using a method developed by van Silfhout and Kema for the detection of the above mentioned four types of resistance to yellow rust in race-nurseries and seedling tests (Kema and Stubbs, 1983), it was found that the durable resistance to yellow rust in the older wheat varieties seems largely due to the collective effects of partial resistance factors although some adult-

plant resistance cannot be excluded (van Dijk et al., 1988). Partial resistance can only be recognized in the absence of a hypersensitivity reaction by its influence on different components of the infection process. Resistance to each of these components (e.g. infection frequency, latent period, spore production etc.) may be based on a different kind of genes. Direct selection for a combination of most of these mechanismes is in general not possible, because of epistatic effects. However, in a backcross programme using a recurrent parent carrying adult-plant resistance, overall resistance may be combined with adult-plant resistance in an indirect way. Selection for overall resistance can be carried out in the seedling stage, while with every backcross the chance will increase to find in the offspring plants which combine the selected overall resistance with the adult-plant resistance of the recurrent parent. This procedure has already been carried out with several selections of wild emmer wheat, carrying genes for overall resistance, and the recurrent parent cv. Arminda, a winter bread wheat with adult-plant resistance (Kema and van Silfhout, unpublished).

Thus there are different options to choose in breeding for resistance to yellow rust. The choice for one or more of these options depends on biological as well as technical factors.

Among the factors which are important in the stategy of a breeding programme with respect to disease resistance the following could be mentioned:

Firstly, the frequency of occurrence of the pathogen has a strong influence on the durability of the resistance to that pathogen. If the pathogen is not endemic and it is not of yearly occurrence, the probability that resistance genes become ineffective (through a mutation in the fungus) will be lower. Therefore, it is essential to know the dynamics of the epidemics in the region for which the new resistant varieties are intended.

Secondly, knowledge about the variability of the pathogen, with respect to virulence genes, is essential for the application of diversification schemes, either on regional scale, on farm scale or field scale. If this knowledge is not available, the use of race-specific overall hypersensitivity resistance is hazardous. For the use of partial resistance, temperature-sensitive resistance or durable adult-plant resistance this may seem of less importance although also here race-specific effects do occur. Examples are resistance of barley to leaf rust (Parlevliet, 1978b, 1983), of wheat to leaf rust (van der Putten and Mesdag, 1987) and of wheat to stripe rust (chapter 8; Kema and Stubbs, 1983). Johnson (1988) also states that some forms of incomplete adult-plant resistance have proven to be durable, but that this does not guarantee that this type of resistance is race-non-specific or permanent. Therefore it is essential to test new sources of resistance, breeding material and advanced lines with different isolates or populations of the pathogen and to have a good knowledge about the variability of these isolates or populations. Moreover, even if no interactions can be detected, some isolates of the pathogen may have a higher general pathogenicity. Knowledge about the variability in the populations of the pathogen is necessary to establish the minimum level of partial or incomplete resistance.

Thirdly, the organisation of the seed supply, including the possibilities for a quick change of varieties and the supply of varietal mixtures in combination with a disease monitoring programme and a well functioning extension service will reduce the risk that involves the use of (possible) race-specific resistance. If one or more of these conditions are not ful-

filled, more effort should be made to obtain durable resistance.

Fourthly, the possibility or impossibility of the use of chemicals for disease control plays a role in the choice of the breeding strategy. If chemicals are available, the risk of severe losses is reduced, but even more important, the risk of the development of new races can be reduced by eradication of early foci which will keep the population of the pathogen at a low level. The possibility to apply a chemical treatment is also important when using partial or incomplete resistance as the basic protection measure. This kind of resistance may not give sufficient protection under conditions which are very favourable for the pathogen. In such circumstances, a chemical treatment can complement the genetical protection. On the other hand if chemicals can not be applied, for economic or other reasons, the breeder has to take care that the level of partial or incomplete resistance is high enough to give sufficient protection even under such conditions.

Every stategy will have its price in terms of research to investigate the nature of the sources of resistance, time to develop new varieties with complex resistance to several pathogens, size of the breeding programme in manpower as well as size and number of test plots and other facilities, insurance of food supply, and environmental pollution. Therefore it will depend on local conditions and also on the priorities set by governmental policy makers which strategy should be chosen.

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SUMMARY

The depletion of genes for resistance to yellow rust (*Puccinia striiformis*) in bread wheat (*Triticum aestivum*) has caused concern among many wheat breeders all over the world who have to breed varieties for areas which are prone to this pathogen. Therefore it was considered necessary to search for new sources of resistance. In wild emmer wheat (*Triticum dicoccoides*), the most important ancestor of bread wheat, a new source of resistance to yellow rust was found. The aim of the present study was to elucidate whether there exist more sources of resistance to yellow rust or other pathogens in wild emmer wheat, and if so to identify these sources, to investigate the nature of this resistance, its effectiveness, its inheritance, and the possibility of transfer to bread wheat.

From about 850 accessions of wild emmer wheat which were collected in Israel, circa 10% of the accessions proved to be resistant to Israeli isolates of yellow rust. Sixty-eight of these accessions were further tested with 28 isolates collected in different areas, especially in developing countries. The resistance of the accessions showed different levels of effectiveness against these isolates; 30 were resistant to all isolates for which conclusive results were obtained, while the others varied from being resistant against all but one of the isolates to being resistant against only a few isolates. The variation in reaction to the various isolates could be explained by the assumption that eleven different resistant factors are present in the wild emmer accessions.

The most promising of these accessions were further evaluated by analysing the $F_2$-populations of crosses with the susceptible *Triticum durum* cv. D447 and the resistant wild emmer sel. G-25. It was shown that in most cases (67%) the resistance is based on one or more dominant genes, while in the remaining cases the resistance is conferred by one or more recessive genes or a combination of recessive and dominant genes. In the majority of these accessions the gene(s) were shown to be different from the previously found dominant gene in wild emmer sel. G-25. At least eight different genes have to be involved to account for our findings.

In a study, involving comparison of reaction patterns with the already described Yr-genes for resistance to yellow rust and analysis of $F_1$-, $F_2$- and $F_3$-data of a cross with *Triticum spelta album* (*Yr5*), the dominant gene in wild emmer sel. G-25 was found to be different from all described genes and therefore it was suggested to assign the symbol *Yr15* to this gene.

Besides the above mentioned overall resistance, adult-plant resistance occurs in wild emmer as well. In field and growth room experiments, it was shown that about 25 % of the tested accessions, which were susceptible in the seedling stage, were resistant in the adult-plant stage. In 16 of these accessions this resistance appeared to be based on temperature-sensitive genes, while the remaining four appeared to have true host-stage-bound adult-plant resistance.

The temperature-sensitive resistance, which was detected in many accessions in the seedling stage at high temperatures, was further characterized in several genetical experiments, including crossing experiments and testing with isolates from different parts of the world. It was shown that there are many different genes involved in this kind of resistance in wild emmer, which interact differentially with the isolates used. These genes seem to be mainly dominantly inherited while the gene action is additive. When several of these genes are combined, the resistance is also expressed at low temperatures.

Resistance to powdery mildew in wild emmer wheat was detected in about half of the several hundreds of accessions which were tested. By analysing the results of different test-locations, it was shown that differential interactions do occur, indicating the presence of different genes for resistance. In a comparison between seedling and adult-plant reaction it was found that in wild emmer at least four different types of resistance to powdery mildew are operating, namely overall resistance, true seedling resistance, adult-plant resistance and partial resistance.

In crossing experiments between hexaploid bread wheat and tetraploid wild emmer it was revealed that no serious crossing barrier between the two species exists. When using bread wheat lines, carrying the *kr*-genes for easy crossability with rye and other wheat-related species, a normal seedset was obtained while wheat lines without these genes gave about half of the normal seedset. Reciprocal crosses using wild emmer as the female parent also gave a fair number of seeds but the germination percentage of these seeds was low. The pentaploid $F_1$-hybrids, although partial male-fertile, may be pollinated with bread wheat pollen without emasculation because there seems to be preferential fertilization with this pollen. Hybrid necrosis however was a serious problem in several combinations which can only be avoided by matching combinations and the use of non-carriers of Ne-alleles.

In four separate breeding programmes the resistance to yellow rust and powdery mildew of about 50 wild emmer accessions was successfully transferred to winter bread wheat. Over 300 interspecific crosses gave rise to more than 700 backcross populations, of which up to now about 5000 lines are under evaluation. In the first replicated yield trial, the grain yields of 111 plant progeny bulks of $BC_1$-$F_2$ or $BC_1$-$F_3$ plants ranged from 54 to 96 percent of the standard commercial variety, indicating that the yield potential of wild emmer derivatives can match or even surpass the potential of commercial varieties.

The study has been carried out mainly in a cooperation between the Research Institute for Plant Protection, The Netherlands and the Agricultural Research Organization, Israel and was financially supported by the Netherlands Minister for Development Cooperation. The solution of practical problems and the development of methods for the application of the obtained results in bread wheat breeding has been carried out in a cooperation with the dutch wheat breeding companies.

## SAMENVATTING

Het geleidelijk ineffectief worden van de resistentiegenen in broodtarwe (*Triticum aestivum*) tegen gele roest (*Puccinia striiformis*) heeft bezorgdheid veroorzaakt bij veel tarweveredelaars die betrokken zijn bij de ontwikkeling van nieuwe tarwerassen voor gebieden waar dit pathogeen een probleem vormt. Daarom werd het noodzakelijk geacht om te zoeken naar nieuwe bronnen van resistentie. Er was toen reeds bekend dat in wilde emmer tarwe (*Triticum dicoccoides*),de belangrijkste voorouder van broodtarwe, een nieuwe zeer effectieve bron van resistentie te vinden was. Het doel van het in dit proefschrift beschreven onderzoek was na te gaan of er meer bronnen van resistentie tegen gele roest of andere pathogenen in wilde emmer voorkomen. Vervolgens dienden deze nieuwe resistenties nader te worden geanalyseerd met betrekking tot hun effectiviteit, de aard van de resistentie en de overerving daarvan, en diende nagegaan te worden of de resistentie kon worden overgebracht naar broodtarwe en op welke wijze dit het meest efficient gedaan kon worden.

Van israelische wilde emmer populaties werden gedurende de afgelopen jaren ongeveer 850 zaadmonsters verzameld. Na toetsing met enkele Israelische isolaten van gele roest, en vaak enkele cycli van selectie en herselectie op resistentie, werden er circa 90 herkomsten van wilde emmer verkregen die homogeen reageerden na toetsing met de israelische isolaten. Achtenzestig van deze herkomsten werden nader onderzocht op de effectiviteit van hun resistentie door toetsing met 28 isolaten van gele roest voornamelijk afkomstig uit (sub)tropische gebieden. Hierbij bleken 30 herkomsten resistent te zijn tegen alle isolaten waarmee consistente gegevens werden verkregen. De effectiviteit van de resistentie in de overige 38 herkomsten varieerde; de besten waren resistent tegen alle isolaten op één na terwijl sommigen slechts resistent waren tegen enkele isolaten. De waargenomen variatie in reactie op de gebruikte isolaten kon worden verklaard door aan te nemen dat er elf resistentiefactoren aanwezig zijn in de onderzochte herkomsten.

De meest belovende van deze herkomsten werden gekruist met de vatbare *Triticum durum* cv. D447 en de resistente wilde emmer sel. G-25, zowel om de overerving van de resistentie te bepalen als om de resistentiegenen uit wilde emmer tarwe over te brengen naar *Triticum durum*. Van deze tarwesoort is namelijk bekend dat de kruisbaarheid met broodtarwe goed is. Het bleek dat de resistentie in de meeste gevallen (67%) berust op één of meer dominante genen. In de overige gevallen bleek de resistentie te berusten op één of meer recessieve genen of een combinatie van recessieve en dominante genen. In de meeste herkomsten bleek de resistentie op andere genen te berusten dan het reeds eerder gevonden dominante gen in sel. G-25. Op basis van het aantal genen in bepaalde herkomsten en de verschillen in genwerking moeten er in deze herkomsten tenminste acht genen bij de resistentie betrokken zijn.

Door het vergelijken van de reactiepatronen van rassen met bekende Yr-genen voor resistentie tegen gele roest met het reactiepatroon van wilde emmer sel. G-25 na toetsing met 24 isolaten van gele roest, bleek alleen het reactiepatroon van *Triticum spelta album* (*Yr5*) met dat van sel. G-25 overeen te komen. Hieruit en uit de analyse van de $F_1$-, $F_2$- en $F_3$-gegevens van een kruising tussen deze twee herkomsten, bleek dat het gen in sel. G-25 verschillend is van alle tot nu toe beschreven genen. Daarom wordt voorgesteld om dit gen het symbool *Yr15* toe te kennen.

Behalve de bovengenoemde 'overall' resistentie, die in alle groeistadia van de plant effectief is, komt er in wilde emmer ook volwassenplant-

resistentie voor die alleen in het volwassenplantstadium tot expressie komt. In veld- en klimaatkast-proeven bleek dat ongeveer een kwart van de getoetste wilde emmer herkomsten, die in het kiemplantstadium vatbaar waren voor fysio 39E134, in het volwassenplantstadium resistent waren. Van deze 20 herkomsten bleken er 4 echte groeistadiumgebonden volwassenplantresistentie te vertonen, terwijl de resistentie in de overige 16 door temperatuurgevoelige genen werd veroorzaakt.

De temperatuurgevoelige resistentie, die in vele herkomsten van wilde emmer in het kiemplantstadium is gevonden na toetsing bij hogere temperatuur, werd nader gekarakteriseerd in kruisingsexperimenten zowel als toetsexperimenten met 15 isolaten uit verschillende delen van de wereld. Hierbij bleek dat er vele genen in wilde emmer tarwe betrokken zijn bij dit type resistentie en dat deze genen een fysio-specifieke interactie vertonen met de gebruikte isolaten. In het algemeen lijken deze resistentiegenen dominant over te erven en vertonen zij duidelijk additieve effecten. Indien verschillende van deze genen gecombineerd worden, komt de resistentie ook bij lage temperatuur tot expressie in latere generaties. Alhoewel de onvolledige resistentie, die door individuele 'minor-effect' genen wordt veroorzaakt, fysiospecifiek blijkt te zijn, kan aannemelijk gemaakt worden dat volledige resistentie berustend op een combinatie van deze genen een duurzaam karakter zal vertonen.

Resistentie tegen meeldauw (*Erysiphe graminis*) kwam in ongeveer de helft van de enkele honderden getoetste wilde emmer herkomsten voor. Bij analyse van de resultaten uit experimenten die uitgevoerd werden op verschillende plaatsen in Nederland en Israel, bleek dat er differentiële interacties optreden die erop duiden dat bij de waargenomen resistentie verschillende genen betrokken zijn. In een vergelijkend onderzoek tussen kiemplant- en volwassenplant-resistentie bleek dat er in wilde emmer tarwe tenminste vier verschillende resistentiemechanismen tegen meeldauw voorkomen, namelijk 'overall' resistentie, echte kiemplant-resistentie, volwassenplant-resistentie, en partiële resistentie.

Alhoewel genen uit wilde emmer overgebracht kunnen worden naar broodtarwe via de brugkruising met *Triticum durum*, werd toch nagegaan of ook de rechtstreekse kruising tussen wilde emmer en broodtarwe op praktische schaal uitvoerbaar was, omdat eerstgenoemde methode een aantal bezwaren heeft. Hierbij bleek dat er onder bepaalde condities geen problematische kruisingsbarrière is. Bij het gebruik van tarwelijnen met de *kr*-genen voor kruisbaarheid met rogge en andere verwante soorten bleek de zaadzetting normaal te zijn, terwijl bij tarwelijnen die deze genen niet bezitten ongeveer de helft van de normale zaadzetting werd verkregen. De pentaploide $F_1$-hybriden, alhoewel gedeeltelijk mannelijk fertiel, kunnen eventueel zonder emasculatie worden bestoven met broodtarwepollen omdat er kennelijk preferentiële bevruchting optreedt met dit pollen. Hybride necrose is echter wel een belangrijk probleem in veel combinaties en kan alleen vermeden worden door compatibele combinaties te maken of door het gebruik van broodtarwerassen die geen Ne-allelen dragen.

Dat de verkregen resultaten en methoden praktisch bruikbaar zijn mag blijken uit het feit dat in vier onafhankelijke veredelingsprogramma's de resistentie tegen gele roest en meeldauw van ongeveer 50 wilde emmer herkomsten is overgebracht naar broodtarwe. Er zijn meer dan 300 interspecifieke kruisingen gemaakt die ongeveer 700 terugkruisingspopulaties hebben opgeleverd. Hiervan worden nu ongeveer 5000 lijnen geevalueerd. In de eer-

ste opbrengstproef bleek dat de opbrengst van 111 plantnakomelingschappen van $BC_1$-$F_2$ of $BC_1$-$F_3$ planten varieerde van 54 tot 96 procent van het standaardras. Dit geeft aan dat het opbrengstvermogen van de wilde emmerderivaten, na verdere selectie, de opbrengst van de huidige rassen kan evenaren of zelfs daarboven uit kan gaan.

Het hier beschreven onderzoek werd in belangrijke mate uitgevoerd in een samenwerkingsverband tussen het Instituut voor Plantenziektenkundig Onderzoek in Nederland en de Landbouwkundige Onderzoeksorganisatie in Israel, mede gefinancierd door het Ministerie voor Ontwikkelingssamenwerking. Het oplossen van de praktische problemen, en het uitwerken van methoden voor de toepassing van de verkregen resultaten in de veredeling van broodtarwe werd in belangrijke mate uitgevoerd in een samenwerking met de Stichting voor Plantenveredeling en met verscheidene nederlandse tarweveredelingsbedrijven.

## CURRICULUM VITAE

Cornelis Herman van Silfhout werd geboren op 2 juni 1946 te Wolfheze. Na in 1964 het diploma ULO-B te hebben behaald aan de Chr. ULO te Oosterbeek, genoot hij voorbereidend wetenschappelijk onderwijs aan het Marnixcollege te Ede waar hij in 1966 het diploma HBS-B behaalde. Na het vervullen van zijn militaire dienstplicht begon hij in 1968 zijn studie Planteziektenkunde aan de toenmalige Landbouw Hogeschool te Wageningen met als hoofdvak Entomologie, welke studie later uitgebreid werd met Plantenveredeling. Het doctoraalexamen werd in 1974 behaald (*cum laude*).

In 1974 trad hij in dienst bij het Instituut voor Plantenziektenkundig Onderzoek (IPO) te Wageningen. Tot 1979 op contract basis, betaald door het Ministerie van Buitenlandse Zaken, om onderzoek te doen over de fysiologische specialisatie van gele roest van tarwe in ontwikkelingslanden, over resistentie in de daar geteelde of te telen tarwerassen en over het daarmee samenhangende resistentie-management.

Daarna trad hij in vaste dienst bij het IPO met als onderzoeksopdracht de bestudering van resistentie in tarwe tegen pathogene schimmels.

Naast deze onderzoeksactiviteit was hij in de periode 1980 tot 1987 nauw betrokken bij de opzet en begeleiding van de automatisering in bovengenoemd instituut. Tevens maakte hij deel uit van het driemanschap dat in 1986 de reorganisatie van het instituut voorbereidde door de hoofdlijnen voor een nieuw onderzoeksplan en een nieuwe organisatiestructuur te schrijven.

Vanaf 1987 is hij hoofd van de sectie Resistentie van bovengenoemd instituut.