Neth. J. Pl. Path. 95 (1989) 79-83

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

Accepted 18 October 1988

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.

Acknowledgement

Cooperative investigation of the Research Institute for Plant Protection (IPO), the Netherlands, and the Agricultural Research Organization (ARO), Israel. Contribution No. 2476-E, 1988 series, from the ARO, Israel.

On request of the Netherlands Minister for Development Cooperation, we state the following:

Responsibility for the contents and for the opinions expressed rests solely with the authors; publication does not constitute an endorsement by the Netherlands Minister for Development Cooperation'.

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.

References

Gassner, G. & Straib, W., 1928. Untersuchungen über die Infektionsbedingungen von *Puccinia glumarum* und *Puccinia graminis*. Arbeiten aus der Biologischen Reichsanstalt 16: 609-629.

Gassner, G. & Straib, W., 1932. Die Bestimmung der biologischen Rassen des Weizengelbrostes (*Puccinia glumarum* f.sp. *tritici* (Schmidt) Erikss. et Henn.). Arbeiten aus der Biologischen Reichsanstalt 20: 141-163.

Gerechter-Amitai, Z.K., 1967. Wild grasses as sources of resistance to the wheat rusts. Summaries First Israel Congress of Plant Pathology, Ministry of Agriculture, Rehovot, State of Israel, p. 43.

Gerechter-Amitai, Z.K. & Grama, A., 1974. Inheritance of resistance to stripe rust (*Puccinia striiformis*) in crosses between wild emmer (*Triticum dicoccoides*) and cultivated tetraploid and hexaploid wheats. I. *Triticum durum*. Euphytica 23: 387-392.

Gerechter-Amitai, Z.K. & Stubbs, R.W., 1970. A valuable source of yellow rust resistance in Israeli populations of wild emmer, *Triticum dicoccoides* Körn. Euphytica 19: 12-21.

Silfhout, C.H. van, Grama, A., Gerechter-Amitai, Z.K., & Kleitman, F., 1989. Resistance to yellow rust in *Triticum dicoccoides*. I. Crosses with susceptible *Triticum durum*. Netherlands Journal of Plant Pathology 95: 73-78