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Identification of resistance genes to Puccinia striiformis in seedlings of Ethiopian and CIMMYT bread wheat varieties and lines

AYELE BADEBO[1], RON W. STUBBS[2], MAARTEN VAN GINKEL[3] and GETINET GEBEYEHU[4]

[1] Institute of Agricultural Research (IAR), Kulumsa Research Center, Asella, Ethiopia
[2] Research Institute for Plant Protection (IPO), P.O. Box 9060, 6700 GW Wageningen, the Netherlands
[3] International Maize and Wheat Improvement Center (CIMMYT), El Batan, Mexico
[4] Institute of Agricultural Research (IAR), Holetta Research Center, Holetta, Ethiopia

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Abstract

In a controlled environment, the reaction was observed of 42 bread wheat varieties and lines inoculated with 19 isolates of yellow rust differing in their virulence to 20 differential varieties. Five varieties and lines showed resistance to all isolates. The remaining ones appeared to have the genes *Yr*2, *Yr*3, *Yr*4, *Yr*6, *Yr*7, *Yr*9 and *Yr*A, either singly or in combination. *Yr*9 derived from rye was present in 67% of the varieties and lines. *Yr*4 is the only effective gene in that material as, in Eastern and Central Africa, yellow rust has virulence to the other *Yr* genes. Recognition of virulence to *Yr* genes is enhanced by the use of a supplemental set of differential varieties supposedly carrying a single gene.

Additional keywords: yellow (stripe) rust races, *Triticum aestivum*, *Yr* genes.

Introduction

Both *Triticum aestivum* and *T. durum* are grown in Ethiopia. Bread wheat makes up about 40% of the area and is cultivated in the highlands, above 2000 m.a.s.l. on an area exceeding 250 000 ha (Getinet Gebeyehu, 1985). The areas is expected to increase.

Rusts represent the major disease threat to wheat production. The importance of the three wheat rusts in Ethiopia was described by Dagnatchew Yirgou (1967) and Eshetu Bekele (1985). In recent years, yellow rust (*Puccinia striiformis* f.sp. *tritici*) has come to require careful attention if stable bread wheat production is to be guaranteed.

Since the mid seventies the area grown to bread wheat has steadily increased, especially on the large farms. On these mechanized farms epidemics of yellow rust on bread wheat occurred in 1977, 1980-83 and 1986. Yield losses of 20-30% were noted on some old and new commercial varieties (Mozgovoy, 1987). In 1987 several previously resistant varieties and advanced lines were heavily attacked in hot-spot areas. In 1988 the earlier resistant and high-yielding CIMMYT derived variety, Dashen (= Veery), was attacked with severities reaching 90% in the Gedeb Asasa plain. Concurrent or successive release of varieties with commonalities in their parentage, as occurred in the past, may have

contributed to a stepwise evolution of the rust population (Bonthuis, 1985).

The epidemiology of yellow rust in Ethiopia is not yet well studied (Van Ginkel et al., 1989). Yellow rust race identification has been carried out since 1973 at the Research Institute for Plant Protection (IPO), Wageningen, the Netherlands, and since 1977 at the Scientific Phytopathological Laboratory (SPL) at Ambo, Ethiopia. Stubbs (1988) showed similarities among race patterns within East Africa, especially between Kenya and Ethiopia.

Virulences for the *Yr*2, *Yr*6, *Yr*7 and *Yr*8 genes conditioning 'overall' resistance were observed to be frequent in Ethiopia before the detection in 1986 of a new race, 166E150, which added virulence for *Yr*9.

The main aim in a breeding program for disease resistance is to provide host materials with combinations of effective genes for resistance in relation to the prevalent pathogen population. Usually, breeding and selection for resistance are solely based on field observations and little is known about the exact gene constitution of resistant germplasm.

In the fifties a theory was proposed by H.H. Flor to elucidate host-pathogen genetic relationships (Person, 1959). The concept of complementary genic systems in host and pathogen, known as 'gene-for-gene theory', has in the last thirty years been shown to operate in many host-pathogen systems. Although Zadoks (1961) proposed the application of the gene-for-gene hypothesis on wheat-yellow rust genetic relationships as a support to on-going breeding programs, his advice has seldom been taken up by wheat improvement programs in the developing world.

In this study the existence of certain yellow rust resistance genes in selected wheat varieties of interest to Ethiopia is identified from the reaction of the varieties to genetically defined rust isolates. The information gained is to serve breeding strategies and variety release decisions in the Ethiopian Wheat Program.

Materials and methods

This study was carried out at the Research Institute for Plant Protection (IPO), Wageningen, the Netherlands. A total of 42 commercial bread wheat varieties and advanced lines obtained from the National Wheat Program, IAR, Ethiopia, were included (Table 1). The material was tested against 19 isolates of wheat stripe rust, collected from Ethiopia (9), Kenya (1), Rwanda (1), Zambia (1), the Netherlands (5), France (1) and China (1). The East African isolates were especially studied to determine additional virulences in this region.

Greenhouse seedling tests were conducted as described by Stubbs (1988). Of each entry, 12-15 seeds were sown in a plastic pot (4 × 4 cm) and grown in a regulated growth chamber. The day/night regime was 16 hours of light (22 000 lux) at 18 °C and 8 hours of darkness at 15 °C, with a relative humidity of about 70%. After 10 days the seedlings were inoculated by spraying a suspension of stripe rust spores in mineral oil (Soltrol 170) onto the leaves. The inoculated seedlings were incubated for 24 to 48 hours in plastic cages in a dew chamber at 9 °C and about 100% relative humidity. After incubation the seedlings were transferred to a growth chamber with identical conditions as prior to inoculation. About 6-7 days after inoculation 2 grams of fertilizer (N : P : K = 15 : 20 : 25) in solution was applied to each set of 25 pots. Second leaves were cut once, about 8-10 days after inoculation. Disease observations were made 17-18 days after inoculation using the 0-9 scale (McNeal et al., 1971).

Table 1. The forty two wheat varieties/lines, tested against various stripe rust isolates. Seed source: IAR, Ethiopia.

Entry No.	Variety/line and pedigree	Entry No.	Variety/line and pedigree
1	GONDER 1	24	SERI82 (= HAR 837)
2	LAKETCH	25	GARA (= BOW) (= HAR 404) CM33203-K-9M-19Y-3M-3Y-0M
3	ISRAEL		
4	ENKOY	26	BOW (= HAR 431) CM33203-K-9M-33Y-1M-1Y-1M-0Y
5	K6295-4A		
6	ETX-C-3H-6H-OH (= HAR 733)	27	PAK 81 (= HAR 1058)
7	KENYA KULUNGU (= HAR 472)	28	ULUCAK75 (= HAR 1217)
8	PAVON 76	29	CMT/CDC//PIO (= HAR 712) CM43473-J-1Y-1M-3Y-3M-0Y
9	H/COLL/546 (= HAR 800)		
10	PRL/BOW (= HAR 1023) CM67363-33Y-2M-2Y-2M-0Y	30	MRNG/BUC//BLO/PSN (= HAR 1067) CM69191-A-5Y-1M-1Y-2M-2Y-2M-0Y
11	BOW/BUC (= HAR 908) CM74005-8M-1Y-03M-4Y-0B	31	MRNG/BUC//BLO/PSN (= HAR 1032) CM69191-A-5Y-1M-1Y-2M-2Y-0M
12	BOW (= HAR 421) CM33203-K-10M-7Y-3M-2Y-1M-0Y	32	MRNG/BUC//BLO/PSN (= HAR 1022) CM69191-A-5Y-1M-1Y-2M-2Y-1M-0Y
13	NS732/HER (= HAR 921) SWM11179-2AP-3AP-1AP-0AP	33	MRNG/BUC//BLO/PSN (= HAR 1034) CM69191-A-5Y-1M-1Y-2M-2Y-3M-0Y
14	BOW (= HAR 416) CM33203-K-10M-7Y-3M-1Y-2M-0Y	34	MRNG/BUC//BLO/PSN (= HAR 1035) CM69191-A-5Y-1M-3Y-1M-0Y
15	HAR 857	35	LIRA (= HAR 719) CM43903-H-2Y-1M-5Y-1M-1Y-1M-0Y
16	KVZ/K4500 L6.A.4 (= HAR 423) SWM176-3M-1Y-6Y-1Y-2M-0Y-4KE-0KE	36	ET12D4
17	PRL/BOW (= HAR 884) CM67363-33Y-2M-2Y-2M-0Y	37	BATU (= SNB) (= HAR 403) CM34630
18	KVZ/7C (= HAR 424) SWM4064-6Y-4M-3Y-1M-1Y-3M-0Y-0PTZ-0Y-0PT	38	BOW/VEE (= HAR 743) CM64693-3M-1Y-1M-3Y-0M
19	CHIL (= HAR 723) CM66684-B-1M-6Y-1M-3Y-1M-0Y	39	CARPINTERO (= HAR 720) II30724-1C-4C-0C-7M-0Y-0AP
20	CHIL (= HAR 820) CM66684-B-1M-6Y-2M-1Y-0Y	40	KVZ/3/CTFN//BB/4/BLO/5/TSH (= HAR 1038) CM70294-4M-2Y-1M-1Y-1M-0Y
21	MYNA/VUL (= HAR 845) CM64546-2M-1Y-5Y-1M-2Y-0M	41	ZP/PATO(B)//CHRC/3/ALDAN/BLT (= HAR 1003) CM60482-1B-1Y-2M-2Y-1M-2Y-2M-0Y
22	TJB788.1039/PVN76 (= HAR 934) SWM09766-1Y-2Y-0Y-1AP-0AP	42	2109.36/VEE/4/WRM//KAL/BB/3/KAL/BB//ALD (= HAR 1018) CM66120-D-1M-1Y-1M-2Y-2M-1Y-0M
23	VEE (= HAR 407) CM33027-F-15M-500Y-0M-0PTZ-0Y		

Results

The reaction of the standard and supplemental differential sets with recognized race-specific genes to the 19 isolates of *P. striiformis* f.sp. *tritici* is given in Table 2, together with the race designation. Besides the full set of standard differentials described by Johnson et al. (1972) plus the variety Clement, a supplemental set of four cultivars was included for further differentiation. No virulence was observed for *Triticum spelta album* (*Yr5*) (not presented). The relevant data are also presented in Table 2.

After the virulence/avirulence formulae were established for the isolates, the wheat

Table 2. Reaction of selected stripe rust isolates on standard and supplemental differential sets.

Country	Isolate No.	World set								European set								Supplemental set				Race Code
		Yr gene[1]	Chinese 166	Lee	Heines kolben	Vilmorin 23	Moro	Strubes Dickkopf	Suwon 92/Omar	Clement	Hybrid 46	Reichersberg 42	Heines Peko	Nord Desprez	Compair	Carstens V	Spaldings Prolific	Heines VII	Fed. 4x/Kavkaz	Kalyansona	Anza	Sonalika	
		→	1	7	6	3+[2]	10	−[3]	−	9+	4+	7+	6+	3+	8	−	−	2+	9	2	A+[4]	A+	
1. Ethiopia	73233				+[5]			+					+					+		+	+	+	36E132
Ethiopia	77212			+	+				+						+					+			70E16
Ethiopia	77219			+	+										+					+			6E16
Ethiopia	81038			+	+				+			+	+		+			+		+			70E150
Ethiopia	81092			+	+										+					+	+	+	6E16
Ethiopia	82050	+	+	+	+							+	+	+	+			+		+	+	+	15E158
Ethiopia	86106			+			+		+						+					+	+		82E16
Ethiopia	87082			+			+		+										+				82E0
Ethiopia	87095			+	+			+		+		+	+		+			+	+	+	+	+	166E150
2. Kenya	87087	+	+	+	+			+	+	±[6]		+	+	±	+			+	+	+	+	+	103(231)E150(158)
3. Rwanda	86053			+	+			+				+	+		+			+	+	+	+		38E150
4. Zambia	86115				+									±					+	+	+	+	4E0(8)
5. Netherlands	68012	+	+	+				+				+	+					+		+			39E134
Netherlands	72078			+	+			+	+		+		+	+				+		+			108E141
Netherlands	78627		+		+			+	+	+	+	+		+				+	+	+			234E139
Netherlands	81551				+			+	+	+	+			+				+	+	+			232E137
Netherlands	86517	+			+			+		+				+				+	+	+			169E136
6. France	59375				+			+						+									40E8
7. China	87010	+	+	+	+			+		+		+	+	+				+	+	+	+	+	175E142

[1] Gene number accorsing to Macer (1975).
[2] + : indicates additional gene(s) present.
[3] − : gene(s) unknown.
[4] A : refers to 'Avocet' (Hussain et al., 1986).
[5] + : compatible reaction (infection type: 7-9).
[6] ± : indicates an intermediate reaction.

lines were subjected to these isolates. The resistance genes in the varieties were determined by comparing their reaction across the 19 known isolates, and applying the gene-for-gene hypothesis (Table 3).

1. *GONDER I*'s seed stock was not pure, but most of the seedlings were susceptible to all 19 races. Therefore this variety does not appear to have any known, stripe rust resistance genes.

2. *LAKETCH*'s origin is the cross II8156 (PJ/GB55), and it is therefore a sister line of 'Kalyansona' (Villareal and Rajaram, 1988), which is being used, upon recommendation by R.W. Stubbs, as a supplemental differetial for *Yr*2. High infection types were recorded consistently with the 17 races which possess virulence to *Yr*2, while low infection types were observed with the two races, that lack virulence to *Yr*2.

Gene composition: *Yr*2.

3. *ISRAEL*'s exact origin is unknown. It is susceptible to the 12 races which have virulence to *Yr*2+ and resistant to all that lack virulence to *Yr*2+.

Gene composition: *Yr*2+.

4. *ENKOY* and 5. *K6295-4A* are commercial varieties in Ethiopia, that originated from crosses made in Kenya. They gave high infection types with races having virulence to *Yr*3+, with the exception of race 4EO(8), and low infection types with races lacking virulence for *Yr*3+. The low infection type with race 4EO(8), which gives an intermediate reaction on 'Nord Desprez' (*Yr*3+) could be due to a longer than usual latent period, also implied by other data, or to the presence of additional gene(s). The latter maybe derived from the differential Strubes Dickkopf to which race 4EO(8) is avirulent.

Gene composition: *Yr*3+ (Nord Desprez), plus additional gene(s).

6. *ETX-C-3H-6H-OH (= HAR 733).* Disease reactions were similar to those noted for 'Enkoy'. However, this line was also resistant race 103(231)E150(158). Consequently, it is only susceptible to races carrying virulence to 'Vilmorin 23' (*Yr*3+).

Gene composition: *Yr*3+ (Vilmorin 23).

7. *KENYA KULUNGU (= HAR 472).* This Kenyan variety was only susceptible to races containing virulence to *Yr*4+. The presence of *Yr*2, *Yr*2+ and/or *Yr*3+ could not be unambiguously shown because no races with virulence to *Yr*4+, but lacking virulence to those genes were available.

Gene composition: *Yr*4+ with or without *Yr*2, *Yr*2+ and/or *Yr*3+.

8. *PAVON 76.* This variety consistently gave high infection types to the 10 races carrying virulence for *Yr*6 and *Yr*7, and low infection types when one or both of these genes were absent.

Gene composition: *Yr*6 and *Yr*7, with or without *Yr*2.

9. *H/COLL/546 (= HAR 800).* This line gave high infection types to 8 races having virulence to *Yr*2 and *Yr*9 and low infection types in the absence of one or both virulence genes.

Gene composition: *Yr*2 and *Yr*9.

Table 3. Virulence of 19 stripe rust isolates and disease reaction of 37 varieties/lines to these isolates, and

Race Code	*Yr* genes														Entry No[1]												
	1	2	2+	3+	4+	6	6+	7	7+	8	9	9+	10	A[2]	1	2	3	4	5	6	7	8	9	10	11	12	13
40E8				3+[3]											+[4]			+	+	+							
82E0								7			9		10		+												
6E16		2				6		7		8					+	+						+					
70E16		2				6		7		8					+	+						+					
36E132		2	2+			6	6+							A	+	+	+										
6E16		2				6		7		8				A	+	+						+					
82E16		2						7		8			10	A	+	+											
4E0(8)		2		(3N)[5]		6					9			A	+	+							+	+			+
108E141		2	2+	3+	4+	6	6+								+	+	+	+	+	+	+						
169E136	1	2	2+	3+							9	9+			+	+	+	+	+	+			+	+	+	+	+
232E137		2	2+	3+	4+						9	9+			+	+	+	+	+	+	+		+	+	+	+	+
39E134	1	2	2+			6	6+	7	7+						+	+	+					+					
70E150		2	2+			6	6+	7	7+	8					+	+	+					+					
234E139		2	2+	3+	4+			7	7+		9	9+			+	+	+	+	+	+	+		+	+	+	+	+
38E150		2	2+			6	6+	7	7+	8	9			A	+	+	+					+	+		+		
15E158	1	2	2+	3+		6	6+	7	7+	8				A	+	+	+	+	+	+		+					
166E150		2	2+			6	6+	7	7+	8	9	9+		A	+	+	+					+	+	+	+	+	
103(231)E150(158)	1	2	2+	(3N)		6	6+	7	7+	8	9	(9+)		A	+	+	+	+	+			+	+		+	+	+
175E142	1	2	2+	3+		6	6+	7	7+	8	9	9+		A	+	+	+	+	+	+		+	+	+	+	+	+
Yr genes															−	2	2+	3N	3N	3+	4+	6	2	2	2+	2(+)[7]	3N
																		+[6]	+			7	9	9	9	9+	9
																								+			

[1] Refers to germplasm entry numbers in Table 1.
[2] Refers to 'Anza'.
[3] Race 40E8 carries virulence for *Yr*3 and the additional gene(s) in the differential host.
[4] +: compatible reaction.

10. *PRL/BOW (= HAR 1023).* High infection types were recorded with six of the eight races having virulence to *Yr*2 and *Yr*9. Races 38E150 and 103(231)E150(158) gave low infection types, although they are virulent to *Yr*2 and *Yr*9, indicating the presence of extra genes.

Gene composition: *Yr*2 and *Yr*9, plus additional gene(s).

11. BOW/BUC (= HAR 908). High infection types were recorded with races having virulence to *Yr*2+ and *Yr*9 and low infection types when one or both virulences were absent.

Gene composition: *Yr*2+ and *Yr*9.

12. BOW (= HAR 421). This line was only susceptible to races carrying virulence to *Yr*2, *Yr*2+ and *Yr*9+. In the absence of these virulences the entry was resistant.

Gene composition: *Yr*2 or *Yr*2+ and *Yr*9+.

the derived *Yr* genes.

14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37
									+														
+	+	+	+	+																			
+	+	+	+	+	+	+	+	+															
+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+		
									+	+	+												
									+	+	+	+	+	+	+	+	+	+	+	+		+	
+	+	+	+						+	+	+	+	+	+	+	+	+	+	+	+		+	+
+	+	+	+	+					+	+	+	+	+	+	+						+	+	+
3N	3N	3N	3N	3+	4+	4+	4+	4+	7	2(+)	2(+)	2(+)	2(+)	2(+)	2(+)	2(+)	2(+)	2(+)	2(+)	2(+)	3+	6(+) /[8]	6(+) /
9+	9+	9+	9+	9(+)	9(+)	9(+)	9(+)	9(+)	9	7(+) 9	7(+) 9	7(+) 9+	7(+) 9+	7(+) 9+	7(+) 9+	7(+) 9+ +	7(+) 9+ +	7(+) 9+ +	7(+) 9+ +	7(+) 9+ +	7(+) 9+	A 9+	A 9+

[5] Refers only to 'Nord Desprez' (*Yr*3+), not 'Vilmorin 23' (see Table 2).
[6] +: extra gene(s) present.
[7] (+): with or without the additional gene(s).
[8] /: and/or.

13. NS732/HER (= HAR 921). This line gave high infection types with races carrying virulence to *Yr*2, *Yr*3+ (Nord Desprez) and *Yr*9 and low infection types when these were lacking. No isolate was available with virulence to two of the genes but with avirulence to *Yr*2.

Gene composition: *Yr*3+ (Nord Desprez) and *Yr*9, with or without *Yr*2.

14. *BOW (= HAR 416)*, 15. *HAR 857*, 16. *KVZ/K4500 L6.A.4 (= HAR 423)* and 17. *PRL/BOW (= HAR 884)* reacted as the previous line NS732/HER. However, they were resistant to race 4EO(8) being avirulent to *Yr*9+. No isolate was available carrying virulence only to *Yr*3+ (Nord Desprez) and *Yr*9+ to ascertain the presence of *Yr*2 or *Yr*2+.

Gene composition: *Yr*3+ (Nord Desprez) and *Yr*9+ with or without *Yr*2 or *Yr*2+.

18. *KVZ/7C (= HAR 424).* This variety was susceptible to all races carrying virulence to *Yr*2, *Yr*2+, *Yr*3+ (Vilmorin 23), *Yr*9 and *Yr*9+. When these virulences were absent,

the response was resistant. No isolate was available carrying only virulence to *Yr3+* (Vilmorin 23) and *Yr9* or *Yr9+*.

Gene composition: *Yr3+* (Vilmorin 23) and *Yr9* or *Yr9+*, with or without *Yr2* or *Yr2+*.

19. *CHIL (= HAR 723)*, 20. *CHIL (= HAR 820)*, 21. *MYNA/VUL (= HAR 845)* and 22. *TJB788.1039/PVN76 (= HAR 934)* were susceptible only to two Dutch races, 232E139 and 234E137, which combine virulence to *Yr2*, *Yr2+*, *Yr3+*, *Yr4+*, *Yr9* and *Yr9+*. No isolate with virulence to *Yr4+* and *Yr9* or *Yr9+*, but lacking virulence to *Yr2*, *Yr2+* and *Yr3+* was available. Therefore complete differentiation was not possible.

Gene composition: *Yr4+* and *Yr9* or *Yr9+*, with or without *Yr2*, *Yr2+* and/or *Yr3+*.

23. *VEE (= HAR 407)* showed high infection types to six races with virulence to *Yr7* and *Yr9*, and low infection types with races lacking virulence to one or both these genes.

Gene composition: *Yr7* and *Yr9*.

24. *SERI82 (= HAR 837)*, a Mexican commercial variety, and 25. *GARA (= BOW = HAR 404)* were susceptible to races combining virulence to *Yr2*, *Yr2+*, *Yr7*, *Yr7+* and *Yr9*. Infection types by races lacking this gene combination were low.

Gene composition: *Yr2* or *Yr2+*, *Yr7* or *Yr7+* and *Yr9*.

26. *BOW (= HAR 431)*, 27. *PAK 81 (= HAR 1058)*, 28. ULUCAK75 (= HAR 1217) and 29. *CMT/CDC//PIO (= HAR 712)* gave high infection types with races combining virulence to *Yr2*, *Yr2+*, *Yr7*, *Yr7+* and *Yr9+*.

Gene composition: *Yr2* or *Yr2+*, *Yr7* or *Yr7+* and *Yr9+*.

30, 31, 32, 33 and 34. *MRNG/BUC//BLO/PSN (= AMSEL)*, lines *HAR 1032*, *HAR 1033*, *HAR1034*, *HAR 1035* and *HAR 1067*, respectively, showed high infection types with races combining virulence to *Yr2*, *Yr2+*, *Yr7*, *Yr7+* and *Yr9+*, and low infection types with races lacking virulence to these genes. However, they were also resistant to the Chinese race 175E142 having the same gene combination, which indicate the presence of an additional genes.

Gene composition: *Yr2* or *Yr2+*, *Yr7* or *Yr7+* and *Yr9+*, plus additional gene(s).

35. *LIRA (= HAR 719)* was only susceptible to two races, which combine virulence to *Yr3+* (Vilmorin 23), *Yr7*, *Yr7+* and *Yr9+*.

Gene composition: *Yr3+* (Vilmorin 23), *Yr7* or *Yr7+* and *Yr9+*, with or without *Yr2* or *Yr2+*.

36. *ET12D4* and 37. *BATU (= SNB) (= HAR 403)* gave high infection types with three races combining virulence to *Yr6*, *Yr6+*, *Yr8*, *Yr9+* and *YrA*. *Yr9+* virulence appears necessary for infection in these lines. However, virulence for the remaining genes can not be definitely shown, because races separating them were not available. Since *Yr8* has not been actively used in breeding programs it is, in fact, unlikely that this gene would be present in this material.

Gene composition: *Yr6* or *Yr6+* and/or *YrA* and *Yr9+*.

38. *BOW/VEE (= HAR 743)*, 39. *CARPINTERO (= HAR 720)*, 40. *KVZ/3/TOB/CTFN//BB/4/BLO/5/TSH (= HAR 1038)*, 41. *ZP/PATO(B)//CHRC/3/ALDAN/BLT (= HAR 1003)* and 42. *2109.36/VEE/4/WRM//KAL/BB/3/KAL/BB//ALD (= HAR 1018)* showed resistant reactions to all races tested.

Gene composition: It is not possible to determine if these lines contain unknown, additional resistance genes or such extensive arrays of known genes so as to be resistant to all races tested.

Discussion

Ethiopian races carrying virulence to one or more of the following known yellow rust genes were identified in this study: *Yr*1, *Yr*2, *Yr*3, *Yr*6, *Yr*7, *Yr*8, *Yr*9, *Yr*10 and *Yr*A. In addition, those races often possess virulence to the unknown genes indicated with plus (+) in the differential varieties used for the recognition of virulence to the known *Yr* genes. Virulence was also observed to 'Strubes Dickkopf' and 'Suwon92/Omar', the genes of which are still unknown. Thus only virulence to *Yr*4 in Hybrid 46 and *Yr*5 in *Triticum spelta album* was not detected in the nine Ethiopian isolates studied.

Virulence to *Yr*1, *Yr*2, *Yr*6 and *Yr*7 occurs worldwide, while that to *Yr*3, *Yr*4, *Yr*8 and *Yr*10 is restricted to certain wheat growing regions of the world. Presently, isolates virulent to *Yr*9 are becoming more frequent in most continents. Virulence to *Yr*5 is still very rare (Stubbs, 1985). Quite recently, the latter virulence has also been found in Australia (Wellings, 1988).

The advanced wheat germplasm studied in this report, was selected after the 1987 season on the basis of field resistance in Ethiopia. In addition some commercial varieties were included. The majority (67%) of the advanced lines contain *Yr*9 and many of these presumably also *Yr*2. Those two genes are widely present in CIMMYT derived germplasm. *Yr*9 was introduced through the 1B/1R translocation originating from Russian winter wheats, and *Yr*2 through 'Kalyansona', a much used parent in CIMMYT crosses (Rajaram et al., 1983).

The inclusion of 'Federation 4x/Kavkaz', as a supplemental differential made it better possible to uniquely identify the presence of *Yr*9, as the previous differential, Clement, contains unidentified genes in addition to *Yr*9, as indicated by *Yr*9+. Similarly, the inclusion of 'Kalyansona' itself as a *Yr*2 differential besides 'Heines VII' (*Yr*2+), which also contains extra genes (Singh and Johnson, 1988), increased precision. These extensions of the differentials sets were suggested by Stubbs, but they have not yet been officially included in the standard differentials sets. In addition 'Anza' and 'Sonalika' were added to the supplemental set. Both carry *Yr*A (Hussain et al., 1986). According to Stubbs, they also possess other genes beside *Yr*A and, therefore, their gene composition is indicated by *Yr*A+.

Due to extra genes in the relevant differential varieties, virulence for *Yr*3 alone could not be identified. Only when virulence was also present to those additional genes could virulence for *Yr*3 be noted. In several cases the presence of *Yr*2 and *Yr*3 in addition to *Yr*9, could not be definitely proven, since races separating them were not available for this study. It should be mentioned that *Yr*3 refers to the gene in 'Cappelle Desprez' (*Yr*3a + *Yr*4a) (Lupton and Macer, 1962) reacting similarly as 'Vilmorin 23' and 'Nord Desprez' with European races. According to Stubbs and as shown in Table 2, the two latter varieties react differently with non-European races, and consequently, differ in

their gene composition.

The presence of *Yr*6 and *Yr*7 in 'Pavon 76' confirmed earlier work by Perwaiz and Johnson (1986) and Dubin et al., (1989). These authors also found *Yr*7 and *Yr*9 in 'Pak81' as we did in this study. In addition 'Pak81' possibly also contains *Yr*2, since isolate 82EO, which contains virulence for *Yr*7 and *Yr*9 but lacks virulence for *Yr*2, does not attack 'Pak81'.

Clearly varieties with their resistance based on a single gene will not suffice in the long run. For example in Eastern and Central Africa quite complex races already occur that combine virulence for *Yr*1, *Yr*2, *Yr*3, *Yr*6, *Yr*7, *Yr*8, *Yr*9 and/or *Yr*10. Breeders with the aid of pathologists must attempt to identify germplasm with multiple resistance or durable resistance genes. A relatively quick, but not fool-proof way of determining the genetic constitution of germplasm, is the postulation of genes following inoculation with various known races. This study has taken that approach. Subsequently those varietal candidates can be selected that contain combinations of resistance genes, that are nog yet matched by the pathogen. The more unmatched resistance genes a variety contains the more mutational steps are required of the pathogen to overcome the combined resistance. Such multiple resistances seem to express increased durability (Schafer and Roelfs, 1985). Adult plant resistance genes, that were possibly present in our material, were not studied.

Information about the genetic consistution of promising germplasm, coupled with disease surveys, regular race/virulence analyses of the pathogen population, multilocation evaluation of differential varieties, and field tests for adult plant resistance will enable wheat researchers to make well-founded decisions regarding future variety releases and breeding strategies.

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Samenvatting

Resistentiegenen tegen Puccinia striiformis in Ethiopische en CIMMYT broodtarwerassen en -lijnen in het zaailingstadium

Onder geconditioneerde klimaatsomstandigheden zijn 42 Ethiopische en CIMMYT rassen en lijnen van broodtarwe (*Triticum aestivum*) in het kiemplantstadium geïnoculeerd met 19 isolaten van gele roest die onderling verschilden in hun pathogeniteit voor 20 differentiërende tarwerassen waarvan de resistentie-achtergrond bekend is. De genom-gen relatie is toegepast om resistentiegenen te identificeren. Vier rassen en lijnen bleken resistent te zijn tegen alle isolaten. Verondersteld wordt dat hun resistentie berust op genen die niet eerder herkend waren of op een combinatie van bekende genen die niet compatibel was met de gebruikte isolaten. In het overige tarwemateriaal kon de aanwezigheid worden aangegeven van de resistentiegenen *Yr*2, *Yr*3, *Yr*4, *Yr*6, *Yr*7, *Yr*9 en *Yr*A. Het van rogge afkomstige en door het CIMMYT veel gebruikte resistentiegen *Yr*9 was in 28 rassen en lijnen (67%) aanwezig. In het onderzochte tarwemateriaal

is *Yr*4 het enige voor Oost en Centraal Afrika effectieve resistentiegen omdat de daar voorkomende gele roest pathogeniteit bezit voor de overige genen. Het herkennen van pathogeniteit van gele roest voor bepaalde resistentiegenen is verbeterd door het toevoegen van tarwerassen met monogene resistentie aan het internatinale gebruikte tarwesortiment voor de determinatie van gele-roestfysio's.

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