Postulated Genes for Resistance to Stripe Rust in Selected CIMMYT and Related Wheats

H. J. DUBIN, International Maize and Wheat Improvement Center (CIMMYT), Box 1336, Kathmandu, Nepal; R. JOHNSON, Plant Breeding Institute, Cambridge, United Kingdom CB2 2LQ; and R. W. STUBBS, Research Institute for Plant Protection, Box 9060, Wageningen, The Netherlands

ABSTRACT

Dubin, H. J., Johnson, R., and Stubbs, R. W. 1989. Postulated genes for resistance to stripe rust in selected CIMMYT and related wheats. Plant Disease 73:472-475.

Infection type data were used to postulate the presence of stripe rust seedling resistance genes in 20 CIMMYT and related bread wheats. *Yr*3, *Yr*6, *Yr*7, and *Yr*9 were hypothesized to be present in 12 lines, either alone or in combination. The isolates used also indicated the presence of additional unknown seedling resistance genes. Four lines produced only low seedling infection types. Four other lines displayed adult plant resistance.

Additional keywords: *Puccinia striiformis*, *Triticum aestivum*, yellow rust

High-yielding wheat cultivars derived from CIMMYT germ plasm are planted on more than 51 million hectares in the world (2). A key breeding strategy, multilocation testing, is used with the intention of broadening the genetic basis of resistance in this germ plasm (3). However, little information about the resistance to the causal organism of stripe rust (*Puccinia striiformis* f. sp. *tritici* Westend.) in these genotypes is known.

Several researchers have shown the utility of using selected cultures of rust causal organisms to hypothesize the presence of specific rust resistance genes in wheats (1,8,9). The data generated have been of use in breeding programs (18) and in studies on distribution of pathogen races (16). The methods are based on the gene-for-gene relationship (9,21) and, although they are not proofs, they help to develop information to be further confirmed, if necessary, by genetic analysis.

The objective of this study was to obtain information on the presence of resistance genes to stripe rust in selected CIMMYT and related wheat germ plasm.

Accepted for publication 21 November 1988.

MATERIALS AND METHODS

Table 1 lists the bread wheat (*Triticum aestivum* L.) genotypes tested and their origin and pedigrees. The lines originating from the 18th International Bread Wheat Screening Nursery represented almost 50% of the crosses in the nursery. The entry numbers in Table 1 correspond to those used later in the text and in Table 2. Names, standard abbreviations, and genealogy of CIMMYT crosses, as noted in the text, were listed by Villareal and Rajaram (19). Three lines were from the Latin American Disease and Observation Nursery and had shown excellent resistance to stripe rust in the Andean countries of South America. Bobwhite "S" and Teeter "S" were of interest because sibs of these lines had been released in various countries.

Two series of seedling inoculations with selected isolates of *P. striiformis* were made (Table 2). One set was done at the Plant Breeding Institute, Cambridge, U.K., with British cultures during late April to early May 1985. Another set was done during May 1985 at the Research Institute for Plant Protection, Wageningen, The Netherlands, with non-European as well as Dutch cultures.

Methods of inoculation and incubation were different at each institution. At the Plant Breeding Institute, seedlings were grown in greenhouses and inoculated with uredospores of each culture diluted in talc when plants were at the first leaf stage. They were then placed in dew chambers in the dark at 10 C for approximately 24 hr. Thereafter, they were moved to a greenhouse and kept

Table 1. Cultivars or crosses of wheat used in inoculation tests

Entry	Cultivar or cross[a]	Pedigree	Seed Source
1	Pak 81	...	CIMMYT
2	Pavon 76	...	18th IBWSN[b]
3	CD-Pchu × Tes	SWM 6966-6Y-1Y-2Y-4Y-OY	18th IBWSN
4	NS 732-Pima	SWM 6976-9Y-1Y-2Y-OY	18th IBWSN
5	Ttr "S"-Jun "S"	CM 59123-3M-1Y-1M-1Y-1M-OY	18th IBWSN
6	Tan "S"/Ti-Tob × Ald "S"	CM 64340-4M-1Y-1M-2Y-OM	18th IBWSN
7	Bow "S"-Vee "S"	CM 64693-3M-1Y-1M-1Y-OM	18th IBWSN
8	(4777(2) × FKN-Gb/Vee "S") Buc "S"-Pvn "S"	CM 66684-B-1M-6Y-1M-2Y-1M-OY	18th IBWSN
9	PF70354-Yaco "S"	CM 67911-4Y-1M-1Y-OZ-1Y-OM	18th IBWSN
10	INIAP-Altar 82	...	8th VEOLA[c]
11	Hys-Pvn "S"	SWM 4249-1Y-6M-5Y-OM	8th VEOLA
12	Bow "S"	CM 33203-K-10M-7Y-3M-2Y-1M-OY	CIMMYT
13	Ttr "S"	CM 37987-I-1Y-2M-2Y-OM	CIMMYT
14	Genaro 81	...	18th IBWSN
15	Mrl "S"-Buc "S"	CM 61949-3M-4Y-1M-1Y-1M-OY	18th IBWSN
16	[(Kvz/Tob-Ctfn × Bb)Blo "S"] Snb "S"	CM 67982-2M-1Y-1M-1Y-OM	18th IBWSN
17	Bucky × Tob-Cno "S"	E-II.72-8691-1e-1e-1E	8th VEOLA
18	Bza-Afm (2) × WW15/Cndr "S"	CM 41336-A-1M-4Y-3M-1Y-OM	18th IBWSN
19	Dove "S"-Buc "S"	CM 58808-27Y-2M-6Y-1M-OY	18th IBWSN
20	(RRV-WW15/Bj "S"-On(2) × Bon) Nac "S"	CM 65202-3M-2Y-1M-1Y-OM	18th IBWSN

[a] See Villareal and Rajaram (19) for meaning of abbreviations.
[b] IBWSN = International Bread Wheat Screening Nursery.
[c] VEOLA = Latin American Disease and Observation Nursery.

under natural light. Temperatures ranged from a minimum of about 8 C at night to a maximum of about 25 C during bright sunny days. Infection type (IT) was noted at about 14 days and was scored on the 00–4 scale, as described by Taylor et al (18). However, in this paper the designation ";" is written in place of "00" because it is briefer. A score of 3– or lower was generally considered a low infection type (LIT). Typical LITs for important *Yr* genes are as follows: *Yr*1 = ;, *Yr*2 = 0 to 2, *Yr*3 = 0 to 1+, *Yr*4 = ; to 1+, *Yr*6 = 0 to 2+, *Yr*7 = 0N to 1+N, *Yr*8 = ;, and *Yr*9 = ; to 0. Heines Kolben, the *Yr*6 differential, tended to give ITs up to 3+ with avirulent cultures when greenhouse temperatures rose above approximately 20 C. A full set of the stripe rust differentials (6), plus cultivar Clement, was used in the British test.

Seedlings at the Research Institute for Plant Protection were grown under controlled conditions in a growth chamber at about 15 C with 16 hr of light (about 22,000 lx) and were inoculated at the first leaf stage with uredospores suspended in nontoxic light mineral oil. The plants were then incubated at 9 C for 24 hr in a dew chamber and later were transferred to a growth chamber with a diurnal cycle of 16 hr of light (about 26,000 lx) at 18 C and 8 hr of dark at 15 C. The light sources were high-pressure sodium and mercury lamps. Infection types were read at about 14 days using both the ; to 4 scale (18) and the 0 to 9 scale (12,16). A 0–6 rating was normally considered LIT on the 0 to 9 scale (16).

In both series of inoculations, cultures were selected to maximize the possibilities of postulating known resistance genes. A brief set of steps for postulating the presence of resistance genes follows. 1) If the differential and the tested line give the same pattern of high infection type (HIT) and LIT with a series of cultures, the tested line is postulated to contain the same gene or genes as the differential. 2) If a culture is known to condition an LIT with a recognized resistance gene and gives an HIT on the tested line, then this line is postulated not to contain the recognized gene. 3) If a culture produces

Table 2. Infection types and postulated resistance genes of wheat seedlings tested with races of *Puccinia striiformis*[a]

Race[b]	Origin of culture[c]	Virulence on *Yr* genes[d]	Entry[e] 1	2	3	4	5	6	7	8	9	10	11	12	13
Plant Breeding Institute tests															
40E8	GB	3[f]	0N	0N	0N	0N	0N	0N	;0N[g]	*[h]	0N	1–N3–N	1+2N	0	0N
104E9	GB	3,4[i]	;,3–[j]	;	;	0;	0	0	0N	0N;	0N	0,22+	0	;	0;
108E9	GB	3,4,6	;	0N1	0N1–N	0N1N	0N	0	0N	0,3–	0	44–	44–	;0N	0N;
104E137	GB	2,3,4	0N,3+	0N	0N	0N	0N	0N	0N	0N	0N,3	01+	1+3–	0N	0N
41E136(3)	GB	1,2,3	0N	0N	0N	0N	0N	0N	0N	0N	0N	2N,1+2+	1N2N	0	01N
41E136(4)	GB	1,2,3	;,3	0N	0N	0N	0N	0N	0N	*	0N1–N	2N,1+2+	0N1+	0N	0N;
37E132	GB	1,2,6	;	0N	;	0	;	0	0N	;	0;	;	43+	;0	0
105E137	GB	1,2,3,4	0N	0N	0N;	0N	0N	0N;	0N	0N	0N	0N2	21+	;0N	0N;
45E140	GB	1,2,3,6	;	01–N,4	0	0	;	0	0N	;0	0	33–	43+	;	0;
109E9	GB	1,3,4,6	;0N	0N	;0N	0N	0N	0	0N	0N	0N	43	4	;	01–N
108E141	GB	2,3,4,6	0	0N1–	0N1N	1–N1	0N	0N	0N	0N	02–	4,2–	4	0N	0N
106E139	GB	2,3,4,7	0	0N2+	02	32,3+	0	;	0N	0	4–	0;	0N2	;1–	0;
39E134	GB	1,2,6,7	;	4	3+4	4	0N	0N	0N	;	43+,0	;	43–	0;	0N;
43E138	GB	1,2,3,7	00N	2+1–	03	03–	0	0	0N	;	4	0,3–	1–2,0	0;	0
108E25	GB	3,4,6,8	;	1N	0N	0N	0N	0N	0N	0N	0N	4	4	0;	0N
232E137	GB	2,3,4,9	01+,3	0N	0N	0N	0N	33–	33+,0N	43,0N	0N1N	23	3+3	4	3+0
169E136(82/4)	GB	1,2,3,9	0N1N	0N	0N	0N	0N	2N1N	3–N2N,3	0;	0N;	23–N	2–N2+N	3N,3+	3+2+
169E136(83/8)	GB	1,2,3,9	0,3	0	;	;0N	0N	3–0	0–2+	0;	0;	0,12	20	1–2–,3	0,3
171E138	GB	1,2,3,7,9	44–	23+,1	32	3+3	3+3	33+,2	3+3	0N1–	4	34–,0	4–	4	31–
109E141	GB	1,2,3,4,6	;0	0N	0	0N	0	;1N	0N	;0	0;	3,3N1–	3–3	;	0;
Research Institute for Plant Protection tests															
0E0	BO	none[d]	;	0;	0	0	;	;	;	;	0	;	00N	;	;,2–N
66E0	EC	7	0;	0;	02+	20	0	;	;	0	2+3	;	0	;	;
104E9	NL	3,4	;	0	;	;	;	0	;	*	0N	*	;	;	;
6E16	AF	6,7,8	;	2+3–	33+	3–3	;	0;	;	;0	3–3,0	;	1+2	;	;
38E16	TZ	6,7,8	;	*	3	3	;	;	0	;	3	*	3	;	;,3
140E12	CO	3,6,9	0	0	0	01N	0N	33–	0N	0	0N	*	3+	3	3–3
38E22(150)	KE	2,6,7,8	;0	33+,0	4	3+4	;	0N	;0	0	3+	0	3+	;	0
106E139	NL	2,3,4,7	;	02+	3–0	22+	0;	;	;	0;	32–	1	01	;	;
134E150	KE	2,6,7,8,9	33+	*	3	01	3–3	33+,0	3–N,0	0	3–3	*	3	12N	3–3
234E139	NL	3,4,6,7,9	0,3	;0	12+	0	3+	02,3	02+	*	33+,0	12–	2+3–	3+	0
236E141	CL	2,3,4,6,9	0	0N	0N	00N	0N	0;,3	0,3	0,3–	0N	01+	3+,2+0	33–,0	1–1
110E143	CL	2,3,4,6,7	0	3+4,1+	33+,;	33+	;	0;	;	;0	;,3–3	*	3+	;0	;0
7(15)E150	PK	1,2,3,6,7,8	;	3+3–	3	33+,;	;	0	0	;	03	;	3–3+	;	0
Postulated *Yr* genes[k]			7,9,+	6,7,+	6,7	6,7,+	7,9	9,+	9,+	9,+	7	3,6	6	9,+	9,+

[a] Tests with Great Britain cultures were done at the Plant Breeding Institute, Cambridge, England. Tests with cultures from other countries were done concurrently at the Research Institute for Plant Protection, Wageningen, The Netherlands.

[b] Race nomenclature refers to formulae as published (6).

[c] GB = Great Britain, BO = Bolivia, EC = Ecuador, NL = Netherlands, AF = Afghanistan, TZ = Tanzania, CO = Colombia, KE = Kenya, CL = Chile, PK = Pakistan.

[d] Denotes known stripe rust resistance genes overcome by these cultures. Race 0E0 not virulent on any known resistance genes.

[e] Germ plasm entry numbers in Table 1 and text.

[f] *Yr*3 represents *Yr*3a + *Yr*4a, as described in cultivar Cappelle Desprez (10).

[g] Where a range of infection types occurred on different plants without clear segregation into discrete classes the different infection types are written without a gap, with the most common infection type written first. High infection type was generally considered to be 3 or higher. A minus (–) indicates uredia somewhat smaller than normal; a plus (+) indicates uredia somewhat larger than normal.

[h] * = No data.

[i] *Yr*4 represents *Yr*4b + *Yr*3b, as described in cultivar Hybrid 46 (10).

[j] Comma (,) indicates segregation between plants for infection type, with most common first.

[k] See text for discussion. Plus (+) in this row indicates additional genes detected.

different LITs on a differential and a test line it may indicate the presence of different genes. However, it must be remembered that the expression of resistance genes may be influenced by the genetic background and caution must therefore be used in the interpretation of such data. 4) If the tested line gives a lower LIT than the differential, it may still possess the same gene as the differential but, in addition, possess another gene that conditions a lower LIT that is epistatic to the known one. 5) If a known gene produces a characteristic LIT in appropriate environments, this can be used to support the postulated presence of that gene. 6) The postulation of a known gene in a test line can also be supported by evidence that the line has in its pedigree parents that are known to carry the gene, or parents in common with cultivars known to carry the gene. Further details of the method are given by Modawi et al (13) and others (9).

Inoculations of adult plants in isolated field nurseries with several Dutch cultures of *P. striiformis* were made during 1985, according to methods described by Zadoks (21). The nurseries were located in the polders of southern Flevoland, The Netherlands.

RESULTS AND DISCUSSION

Seedling ITs given by each wheat line are presented in Table 2. Where a range of ITs was recorded on different plants, the different ITs are written without a gap to save space (Table 2). Postulation of the presence of recognized resistance genes is based principally on the ITs given by the British isolates because the results were less variable than the other series. However, data from The Netherlands were used to support the hypotheses and were used in attempts to detect resistance due to unknown genes.

Because lines in CIMMYT breeding programs were commonly bulked in F_5 and F_6 and the lines were not selected for resistance to these *P. striiformis* cultures, the seedlings could be heterogeneous for IT in some cases. Also, greenhouse temperatures at the Plant Breeding Institute occasionally rose to around 25 C and thus caused some variability in ITs, especially those due to *Yr6*. These points must be considered in interpretation of the data of Table 2.

We hypothesize that Pak 81 (entry 1) has *Yr7* and *Yr9*, based on the HIT given by race 171E138 and the LITs with races avirulent for *Yr7* and *Yr9* (Table 2). The seed source used was heterogeneous for plants with these genes, but off-types were few. Kenya race 134E150, virulent for *Yr7* and *Yr9*, was the only one of the series from The Netherlands to give an HIT on Pak 81, whereas the others with avirulence to either gene produced LITs. Dutch isolate 234E139 gave an LIT on most of the plants of Pak 81, indicating a possible additional unknown gene present.

The presence of *Yr7* and *Yr9* in Pak 81 = Vee No. 5 confirms the work of Perwaiz and Johnson (14), whereas Hussain et al (5) could only postulate *Yr9* with the cultures they used. Because the Vee "S" lines contain the 1B/1R translocation (7,11,22), most sibs should possess *Yr9* from cultivar Petkus rye. Some lines of Cno 67 have *Yr6* and *Yr7* (R. Johnson, *unpublished*). It is probable that *Yr7* comes from Cno 67, which is in the parentage of Pak 81 as part of Kal-Bb.

It is postulated that Pavon 76 (entry 2) has *Yr6* and *Yr7*. All British isolates avirulent to these genes gave LITs to Pavon 76 and only 39E134, with virulence to both, gave clear HIT (Table 2). The conclusions are supported by the tests in The Netherlands. Races 38E22(150), 110E143, and 7(15)E150 gave HITs, and cultures avirulent to either *Yr6* or *Yr7* gave LITs. However, the LIT with 6E16 may indicate the presence of an additional gene or variability due to environment. Where cultures had virulence for *Yr7* only (e.g., 106E139, 43E138, and 171E138), fluctuating temperatures probably influenced the effectiveness of *Yr6*, giving variable ITs (Table 2). Notwithstanding the variability with respect to *Yr6*, Pavon 76 appears to possess *Yr6* and *Yr7*. The parentage of Pavon 76 includes Cno 67 and is consistent with the presence of *Yr6* and *Yr7*. This confirms the work of Perwaiz and Johnson (14) and of Wellings (20). Hussain et al (5) were able to discern *Yr7* only because of limited variation in their pathogen cultures.

The ITs of CD-Pchu × Tes (entry 3) and NS 732-Pima (entry 4) were very similar to Pavon 76, and we conclude that they have *Yr6* and *Yr7*. In the former, resistant plants were present when the line was tested with cultures virulent on *Yr6* and *Yr7* indicating additional resistance, but these were at low frequency (Table 2). Responses in tests in The Netherlands clearly support the British data. In the latter line, the ITs were similar except that race 134E150, which is virulent for *Yr6* and *Yr7*, gave an LIT. Thus, it can be postulated that NS 732-Pima has an additional unknown resistance gene or genes. The parentages of these two entries do not include lines that are known to have *Yr6* and *Yr7*.

Ttr "S"-Jun "S" (entry 5) had ITs similar to those of Pak 81, and therefore we conclude that *Yr7* and *Yr9* were present (Table 2). An HIT with 234E139 supports this conclusion but 134E150, with virulence for *Yr7* and *Yr9*, gave a 3–3 reaction, indicating intermediate resistance (Table 2). This may have been due to another gene or environmental variability. Parents of Ttr "S"-Jun "S" include Pvn "S" and Bb, which supports the probable presence of *Yr7*. Recent glucose phosphate isomerase tests with Ttr "S" lines indicated the presence of the 1B/1R translocation that carries *Yr9* (J. Bingham, *unpublished*). This probably comes from the Fundulea parent.

In Tan "S"/Ti-Tob × Ald "S" (entry 6), HITs with 171E138 and 232E137 and LITs with all cultures avirulent for *Yr9* indicate that *Yr9* was present (Table 2). LITs with both cultures of 169E136, virulent for *Yr9*, provided evidence that another gene was effective. The data from The Netherlands support the presence of *Yr9* and implicate *Yr6* as the additional gene because the ITs with races 140E12 and 134E150 that combined virulences for *Yr6* and *Yr9* were higher than those for other races. However, this was not clear because race 236E141 gave LIT on most of the plants, but the results were rather variable and no fully susceptible reactions were observed, indicating the possible presence of other resistance genes. The parentage of Tan "S"/Ti-Tob × Ald "S" is consistent with the presence of *Yr9* because Ald "S" possesses the 1B/1R translocation (7,11,22). Although ITs for postulating *Yr6* in this line were variable, the parents in the cross suggest that it is plausible. Both Ti and Ald "S" have Cno "S" in their pedigrees, and Tan "S" has Pvn "S", all hypothesized to have *Yr6*. Nonetheless, additional work is necessary to conclude the presence of *Yr6*.

Bow "S"-Vee "S" (entry 7) and Bow "S" (entry 12) both appear to have *Yr9* and unidentified resistance (Table 2). The data from The Netherlands supports the hypothesis of *Yr9*. The reactions with 140E12 and 134E150 show additional resistance in Bow "S"-Vee "S" and the LIT with 134E150 indicates another resistance gene in Bow "S". It is possible that the additional gene was *Yr6*, although the reactions were rather variable (Table 2). *Yr9* was postulated to be present in the Pakistan cultivar Sarhad 82, which is a Bow "S" (5). The premise that *Yr9* conditioned the LIT in these lines is supported by the presence of the 1B/1R translocation (7). It should be noted that entries 6, 7, and 12 have similar responses to the cultures tested.

Yr9 was postulated for (4777 (2) × FKN-Gb/Vee "S") Buc "S"-Pvn "S" (entry 8) based on the HITs with race 232E137. The presence of Vee "S" in the pedigree supports this. All other isolates gave LITs indicating the presence of at least one other gene (Table 2).

HITs with races 106E139, 39E134, 43E138, 171E138, and LITs with races avirulent for *Yr7* indicated that PF70354-Yaco "S" (entry 9) had *Yr7* (Table 2). The data from The Netherlands supported this, but the somewhat more incompatible ITs, with races such as 66E0, 6E16, 106E139, and others may be due to additional genes or environmental influence. *Yr7* is not traceable in the parentage of this line.

INIAP-Altar 82 (entry 10), postulated

to have *Yr3* and *Yr6*, gave consistent LITs with all races lacking virulence for *Yr3*. HITs occurred with races possessing virulence for both *Yr3* and *Yr6*, and some also occurred with cultures having virulence for *Yr3* but not for *Yr6*, such as 171E138. This could indicate either variable expression of *Yr6* in different environments or, possibly, that some plants lacked *Yr6*. In all tests, there were some plants with intermediate or LITs, indicating additional resistance (Table 2). INIAP-Altar 82 is a cross of Tob "S" × Desc-Frocor. As noted previously, Tob "S" has *Yr6* and Frontana contains *Yr6* (R. A. McIntosh, *unpublished*) and is a parent of Frocor. *Yr3* is more difficult to trace. However, because *Yr3* is common in European winter wheats (16) it is possible that it was derived from such wheats used in the pedigree of Mentana, which is in Frontana.

The evidence generally supports the presence of *Yr6* in Hys-Pvn "S" (entry 11), but races 232E137 and 171E138, avirulent on *Yr6*, gave HITs (Table 2). The fact that *Yr6* gave variable ITs, apparently due to high temperatures, allows more weight to be placed on the evidence implicating *Yr6*. Data from The Netherlands supported the conclusion.

Ttr "S" (entry 13) appeared to carry *Yr9* based on the LITs with cultures avirulent to *Yr9* and HITs with 232E137 and 169E136(82/4). The range of ITs with other cultures indicated interactions with different genes, but these could not be identified.

The following lines gave only LITs with all cultures: Genaro 81 = Vee No. 3 (entry 14), Mrl "S"-Buc "S" (entry 15), [(Kvz/Tob-Ctfn × Bb)Blo "S"]Snb "S" (entry 16), and Bucky × Tob-Cno "S" (entry 17). The seedling resistances present in these lines may be due to unknown genes, a combination of known genes not overcome by our cultures, or both.

Inoculations in field nurseries of the lines in Table 1 yielded additional information on the adult stripe rust resistances in four genotypes (entries 12, 18, 19, and 20). No known seedling genes could be discerned in three of the four lines (entries 18, 19, and 20). Responses of all other entries coincided with their seedling tests or, in a few cases, responses were intermediate, making a conclusion on the presence of adult plant resistance difficult. Only two cultures that were used in the growth chamber were also used in the field in inoculations in The Netherlands (i.e., 106E139 and 234E139). Spreader rows were highly infected with their respective races and each series of inoculations was well isolated, as described by Zadoks (21). In Bza-Afm (2) × WW15/Cndr "S" (entry 18), seedling ITs were 2+ for the two races, and field ITs were very resistant. (RRV-WW15/Bj "S"-On (2) × Bon)Nac "S"(entry 20) showed identical reactions to entry 18 in the adult stage, but had somewhat higher ITs in the seedling stage (i.e., 33− for 106E139 and 3 for 234E139). These lines possibly carry the adult resistance present in WW15 and its derivatives (*unpublished*). In the seedling stage, Dove "S"-Buc "S" (entry 19) conditioned IT 3−3+,0 with race 106E139 and IT 3,0 with 234E139. In the adult plant stage, the ITs were very resistant. This cross has Mildress in its parentage and Mildress is known to carry adult plant resistance (17). Entry 12, Bow "S", gave a seedling IT of 3+ with 234E139 and a low-percentage moderately resistant reaction at the adult plant stage. This adult resistance could be derived from Aurora.

Four known stripe rust resistance genes were detected from IT data in 12 of the 20 lines noted. Thus, *Yr3*, *Yr6*, *Yr7*, *Yr9*, or combinations of these were postulated. Additional unidentified resistance genes, or combinations of genes, were effective in seedlings, and four of the lines gave LITs with all cultures, precluding the gene identification by this method. Lastly, four lines appeared to carry adult plant resistance to stripe rust.

The power of this method to permit identification of resistance genes is limited by certain factors. Cultures used in these tests can only interact with a portion of the resistance genotype and, therefore, only discriminate certain resistance genes. Heterogeneity of the genotypes, although expected, contributed to difficulties in determining gene interactions. Interactions between resistance genes may occur and could have affected some ITs (4,9,15). Temperature influenced the ITs, especially those due to *Yr6*, which seemed to be less effective at higher greenhouse temperatures. In contrast to this suggestion, Wellings (20) reported that *Yr6* was less effective at lower temperatures in controlled environments. It is possible that *Yr6* has optimal expression at intermediate temperatures and that light intensity may also influence its expression. Nonetheless, even with these limitations it was possible to make preliminary postulations about some of the genes controlling the observed resistance.

ACKNOWLEDGMENTS

The first author sincerely thanks the Plant Breeding Institute, Cambridge, U.K., for hosting him while these studies were done. We wish to thank A. J. Taylor and G. M. B. Smith for their excellent technical assistance. Appreciation is given to R. A. McIntosh for helpful comments on the manuscript.

LITERATURE CITED

1. Browder, L. E., and Eversmeyer, M. G. 1980. Sorting of *Puccinia recondita*:*Triticum* infection-type data sets toward the gene-for-gene model. Phytopathology 70:666-670.
2. Dalrymple, D. 1986. Development and spread of high yielding wheat varieties in developing countries. Bureau of Science and Technology. USAID, Washington, DC. 99 pp.
3. Dubin, H. J., and Rajaram, S. 1982. The CIMMYT's international approach to breeding disease-resistant wheat. Plant Dis. 66:967-971.
4. Dyck, P. L., and Samborski, D. J. 1982. The inheritance of resistance to *Puccinia recondita* in a group of common wheat cultivars. Can. J. Genet. Cytol. 24:273-283.
5. Hussain, M., Gordon-Werner, E., Hetherington, S., and McIntosh, R. A. 1986. Seedling responses of Pakistani wheats to Australian rust pathotypes. Pages 68-74 in: Proc. Assem. Wheat Breed. Soc. Aust. 5th., Perth. R. McLean, ed.
6. Johnson, R., Stubbs, R. W., Fuchs, E., and Chamberlain, N. H. 1972. Nomenclature for physiological races of *Puccinia striiformis* infecting wheats. Trans. Br. Mycol. Soc. 58:475-480.
7. Kazi, A. M. 1984. Wide crosses. CIMMYT Report on Wheat Improvement 1982. Int. Maize Wheat Improv. Cent., Mexico. 185 pp.
8. Knott, D. R., and Johnson, R. 1983. Some additional comments on sorting infection-type data sets. Phytopathology 73:514-515.
9. Loegering, W. Q., and Burton, C. H. 1974. Computer-generated hypothetical genotypes for reaction and pathogenicity of wheat cultivars and cultures of *Puccinia graminis tritici*. Phytopathology 64:1380-1384.
10. Lupton, F. G. H., and Macer, R. C. F. 1962. Inheritance of resistance to yellow rust *Puccinia glumarum* Erikss. and Henn. in seven varieties of wheat. Trans. Br. Mycol. Soc. 45:21-45.
11. Macer, R. C. F. 1975. Presidential address. Plant pathology in a changing world. Trans. Br. Mycol. Soc. 65:351-374.
12. McNeal, F. H., Konzak, C. S., Smith, E. P., Tate, W. S., and Russel, T. S. 1971. A uniform system for recording and processing cereal data. U.S. Dep. Agric., Agric. Res. Serv. Bull. 34-121. 42 pp.
13. Modawi, R. S., Browder, L. E., and Heyne, E. G. 1985. Use of infection-type data to identify genes for low reaction to *Puccinia recondita* in several winter wheat cultivars. Crop Sci. 25:9-13.
14. Perwaiz, M. S., and Johnson, R. 1986. Genes for resistance to yellow rust in seedlings of wheat cultivars from Pakistan tested with British isolates of *Puccinia striiformis*. Plant Breed. 97:289-296.
15. Roelfs, A. P. 1984. Race specificity and methods of study. Pages 131-164 in: The Cereal Rusts. Vol. 1. W. R. Bushnell and A. P. Roelfs, eds. Academic Press, Orlando.
16. Stubbs, R. W. 1985. Stripe rust. Pages 61-101 in: The Cereal Rusts. Vol. II. A. P. Roelfs and W. R. Bushnell, eds. Academic Press, Orlando.
17. Stubbs, R. W., Slovencikova, V., and Bartos, P. 1977. Yellow rust resistance of some European wheat cultivars derived from rye. Cereal Rusts Bull. 5:44-47.
18. Taylor, A. J., Smith, G. M. B., and Johnson, R. 1981. Race-specific genetic factors for resistance to *Puccinia striiformis* in wheat cultivars from the Plant Breeding Institute. Cereal Rusts Bull. 9:33-45.
19. Villareal, R., and Rajaram, S. 1984. Semidwarf bread wheat. Names;Parentage;Pedigrees;Origin. Int. Maize Wheat Improv. Cent., Mexico. 37 pp.
20. Wellings, C. W. 1986. Host:pathogen studies of wheat stripe rust in Australia. Ph.D thesis. Univ. Sydney. 237 pp.
21. Zadoks, J. C. 1961. Yellow rust on wheat: Studies in epidemiology and physiologic specialization. T. Pl. Ziekten 67:69-256.
22. Zeller, F. J. 1973. 1B/1R wheat-rye chromosome substitutions and translocations. Pages 209-221 in: Proc. Int. Wheat Genet. Symp. 4th. E. R. Sears and L. M. S. Sears, eds. Columbia, MO.