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SENSITIVITY OF LEAF RUST RESISTANCE GENES TO TEMPERATURE IN WHEAT*

BY

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SUMMARY

The host pathogen interaction (HPI) of genes Lr2a, Lr10, Lr17 and Lr18 are both temperature dependent and virulence specific. Pathogen isolates 12(5R5), 12A(5R13), 104(17R23), 104B(29R23) and 162A(93R15) interacted variably with Lr2a in cv. Webster depending on temperature while 77(45R31) and 162(93R7) were consistant at all temperatures. A similar trend in HPI was observed with genes Lr10, Lr17 and Lr18 with regard to some specific virulences.

INTRODUCTION

Temperature plays an important role in the outcome of the host pathogen interaction (HPI) and is of interest to pathologists and geneticists. Temperature sensitivity of differential cultivars makes reliable identification of virulence difficult, and because of this, removal of some differentials was suggested (Basile, 1957). The new system of virulence identification in Puccinia recondita f.sp. tritici Rob. ex. Desm. which is based on near-isogenic lines or cultivars of wheat with known genes becomes less dependable if the genes are temperature sensitive. A study on the effects of temperature on HPI in four wheat lines possessing known Lr genes was therefore initiated.

MATERIALS AND METHODS

In addition to Agra local that served as a check, four lines namely; Webster (Lr2a), Thatcher^6 x Lee (Lr10), Thatcher^6 x K. Lucero (Lr17) and Timvera (Lr18) which are differentials in the proposed brown rust identification system (Nagarajan et al., 1983) were included in the study. One week old seedlings were hand inoculated with brown rust (P. recondita) isolates and placed in a humidity chamber for 48 hours and then transferred to BOD incubators (York Sci. Co., New Delhi, INDIA). Eight isolates were used, they being: 12(5R5), 12-A(5R13), 77(45R31), 77-A(109R31), 104(17R23), 104B(29R23), 162(93R7) and 162-A(93R15). Codes given in parenthesis

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* Part of Ph.D dissertation submitted by senior author to the H.P.K.V.V., Solan, H.P. (INDIA).

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are virulence designations following Nagarajan et al., (1983).

The BOD incubators were run at 10, 15, 20 and 25°C ± 1°C, with 12 hr photoperiod. Artificial light up to 2450 lux was provided. All the experiments were conducted twice. Observations on infection types were taken after 15 to 20 days and HPI was recorded a 0,0;,1,2,3 and 4; + or - signs are added to indicate minor levels of variation in the reaction.

RESULTS

The HPI produced by these lines against different pathotypes of P. recondita tritici at different temperatures are given in Table 1.

Gene Lr2a (Webster) was temperature sensitive against certain specific virulences. Varying expression of the HPI was observed with isolates of 12(5R5), 12-A(5R13), 104(17R23), 104B(29R23) and 162-A(93R15) while HPI was stable with 77(45R31) and 162(93R7). Even the degree of sensitivity to these isolates varied greatly. The IT with isolates 12(5R5), 12-A(5R13) and 104(17R23) had a marginal difference varying from 0; to 2^+ reaction. However, with isolates 104B(29R23) and 162-A(93R15) there was a change from resistant to susceptible with increase in temperature.

The HPI of Thatcher[6] x Lee (Lr10) was temperature independent against isolate 77(45R31) whereas, with 77-A(109R31), susceptibility increased with temperature.

The HPI on Thatcher[6] x K. Lucerno (Lr17) involving isolate 12(5R5) was temperature independent and always produced a 0; reaction, while that of isolate 77(45R31) tended to be towards susceptibility at increased temperatures. In the latter case, the HPI was of the type 2^+ at 10°C and 15°C and type 4 at 20°C and 25°C.

Timvera (Lr18) showed increased susceptibility with increase in temperature against isolates 77(45R31) and 162(93R7) and the shift was greatest with isolate 77(45R31).

DISCUSSION

The HPI of four Lr genes viz. Lr2a, Lr10, Lr17 and Lr18 showed temperature sensitivity against certain virulences. It was observed that it is not the gene per se which is sensitive to temperature but it is the HPI which showed temperature sensitivity. Browder (1980) classified Lr2a and Lr10 as moderately sensitive, Lr17 as insensitive and Lr18 as highly sensitive to temperature. Contrary to this classification Lr2a was temperature insensitive against isolates 77(45R31) and 162(93R7) P. recondita tritici and considerably sensitive to isolates 104B(29R23) and 162-A(93R15) and moderately so to three others. This virulence-dependent sensitivity of Lr genes was also reported by Dyck and Johnson (1983). The present results do not support van der Planks (1978) hypothesis of increased susceptibility with higher temperature.

In the interactions between Lr2a and isolates 12(5R5), 12-A(5R13), 104(17R239 and 104B(29R23), although there was a general tendency of increased susceptibility with an increase in temperature, this trend was noticeable only up to 20°C. Further increase to 25°C reverted to HPI towards resistance. This response to temperature is not common although the reverse of increased resistance with

temperature up to 20°C and a return susceptibility at 25°C has been reported in the case of the wheat line RL 6057 against WBR 78/1 isolate of P. recondita tritici by Dyck and Johnson (1983).

In most cases we observed a positive correlation between increase in temperature and susceptibility, an observation made by various workers (Anderson, 1963; Ellingboe, 1982 and Vanderplank, 1978). We further support the views of Dyck and Johnson (1983), that the HPI of genes Lr10, Lr17 and Lr18 are temperature sensitive and are virulence dependent. It is therefore imperative that the reaction on these Lr lines should be determined at different temperatures, say 10°C and 21°C, to allow full interpretation of the interaction.

ACKNOWLEDGEMENT

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TABLE 1 Reaction types produced by Webster (Lr2a), Lr10, Lr17 and Lr18 at different temperatures against isolates of Puccinia recondita tritici carrying specific virulences.

Lines and Isolates	Temperatures °C			
Lr2a (Webster)	10°	15°	20°	25°
12(5R5)	0;	2^-	2^+	1
12-A(5R13)	2	2^+	1^+	1^+
77(45R31)	4	4	4	4
104(17R23)	1^+	1^+	2^+	2^-
104B(29R23)	1	3^-	3	2^-
162(93R7)	3	3	3	4
162-A(93R15)	1	3^-	4	3
Lr10 (Thatcher[6] x Lee)				
77(45R31)	0;	0;	0;	0;
77-A(109R3)	2^+	3^-	4	4
Lr17 (Thatcher[6] x K. Lucero)				
12(5R5)	0;	0;	0;	0
77(45R31)	2^+	2^+	4	4
Lr 18 (Timvera)				
77(45R31)	1	0;	3^-	3
162(93R7	0;	0;	1	2^+

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AVIRULENCE/VIRULENCE COMBINATIONS OF OAT STEM RUST IN CENTRAL, CENTRAL WEST AND CENTRAL EAST EUROPE AND THEIR COMPARISON WITH NORTH AMERICAN EQUIVALENTS.

BY

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SUMMARY

The data of oat stem rust incidence on the European Continent, found in the European Oat Disease Nursery, indicate the potential importance of this pathogen in some regions of Europe.

Avirulence/virulence combinations of isolates of P. graminis f.sp. avenae from Austria, Czechoslovakia, the Federal Republic of Germany, Poland and Switzerland indicate the distribution of virulence of stem rust on oats in these countries and the potential effectiveness of known stem rust resistance genes for European breeding programs.

The European combinations E 3 - E 9 correspond to North American combinations NA 5, NA 50, Na23, NA 21, NA 18, NA 27 and NA 22, respectively. The finding of virulence combination E 10 (= P 1), possessing the virulence on Pg 13 and Pg 16 genes, is of great potential importance.

In Europe and in western Canada, Pg a, Pg 13, Pg 16 appear to be the most effective genes, followed by Pg 9 and Pg 4 in Europe. Breeding oats for multigenic resistance to stem rust, similar to that for crown rust, is proposed.

INTRODUCTION

Adequate information on the virulence spectra of cereal rust populations in the region for which varieties are bred, as well as in neighbouring regions, which comprise a relatively large geographically isolated areas, is one of the fundamental criteria for effective resistance breeding against these pathogens. Data from Cereal Disease Nuseries and analysis of pathogen virulence on a defined genetic basis, allows for the pathologic detection of resistance genes either singly or combined in multigenic genotypes.

The aim of this study was, on the basis of single resistance genes transferred into relevant derivatives, to determine existing virulence combinations in uredial samples of Puccinia graminis f.sp. avenae from Austria, Czechoslovakia, the Federal republic of Germany, Poland and Switzerland, and to assess the potential importance of corresponding resistance genes for resistance breeding of oats to stem rust in these countries.

MATERIALS AND METHODS

Uredial samples of oat stem rust were obtained from the European Oat Disease Nursery (Sebesta and Zwatz, 1980), commercial oat fields and wild oats (Avena fatua L.), collected mainly in Czechoslovakia and Austria and to a limited extent in the Federal republic of Germany, Switzerland and Poland between 1978 and 1985. Isolates were differentiated according to avirulence/virulence combinations (Green, 1965) and compared with their North American equivalents (Martens, et al., 1979).

Percentage of the number of isolates from each country with virulence for each Pg line was taken as a criterion for the evaluation of importance for resistance breeding.

RESULTS AND DISCUSSION

Incidence of stem rust on oats in Europe between 1978-1984.

A high level of occurrence of oat stem rust was recorded in Austria at Drauhofen (1978) and St. Donat (1979, 1981) and in Yugoslavia (Kragujevac) in 1979.

Moderate levels of stem rust occurred in Austria in 1980 (St. Donat), in Czechoslovakia in 1978, 1980 and 1984 (Bystrice n. P.), 1980 (Pstrusa-Viglas and Brezova), in Hungary in 1979 (Szeged), in Spain (Madrid) in 1978 and 1984 and in Yugoslavia in 1978 and 1980.

Low levels of stem rust were recorded in Austria in 1978 (Petzenkirchen, St. Donat), 1979 (Fuchsenbigl, Petzenkirchen, Drauhofen), 1980 (Fuchsenbigl, Petzenkirchen), 1982 (Fuchsenbigl, Petzenkirche, Drauhofen, St. Donat), 1983 (Drauhofen) and 1984 (Fuchsenbigl, Petzenkirchen). In Czechoslovakia, a low level of stem rust occurred in 1978 (Viglas), 1979 (Bystrice n. P.), 1981 (Bystrice, Viglas). A low level of stem rust was recorded in the German Democratic Republic in 1980 (Petkus) and in 1981 and 1984 (Salzmunde) and in Poland, in 1979, 1982 and 1984 (Wielopole). In Spain low stem rust levels occurred in 1980 and 1982 and in Yugoslavia (Kragujuvac) in 1981, 1982 and 1983 (Sebesta, 1985).

Avirulence/virulence combinations of P. graminis f.sp. avenae identified in 1978-85

Analyses of the reactions of ten oat lines with single genes for resistance to stem rust indicated that in Czechoslovakia, Austria and Poland isolates with virulence for the resistance genes Pg 1, Pg 2, Pg 3, Pg 4, Pg 8, Pg 9 and Pg 15 occurred. In Poland in 1978, a race, preliminarily designated P 1 (= E 10) with virulence for Pg 1, Pg 8, Pg 9, Pg 13 and Pg 16 was identified. Isolates with virulence for Pg 1, Pg 2, Pg 3, Pg 8 and Pg 15 and less on Pg 9 occurred in the Federal republic of Germany and in Switzerland. No isolates with virulence for Pg 4, Pg 13 and Pg 16 were found in these two countries.

Between 1978-1985 in Czechoslovakia, Austria, FRG, Switzerland and Poland, ten avirulence/virulence combinations were isolated (Table 1). However, the limited numbers of isolates from the FRG, Switzerland and Poland in particular, were not adequate to make any conclusions about the distribution of these combinations of virulence or about the mean virulence capability (Sebesta and Harder, 1983).

The European avirulence/virulence combinations of P. graminis f.sp. avenae and their North American equivalents are also shown in Table 1. European combinations E 3 to E 9 correspond to North American (NA) combinations, NA 5, NA 50, NA 23, NA 21, NA 18, Na 27 and NA 22, respectively. Of these, race NA 5 is common in the Pacific and far western regions of North America, and race NA 27 is the overwhelmingly predominant race in the prairie region (Harder, 1984).

From the stem rust resistance breeding point of view the detection of isolates with virulence for Pg 13 and Pg 16 is of great importance, because genes Pg 13 and Pg 16 have been the most effective in both North American (Brown et al., 1985) and European continents (Sebesta et al., 1985).

In Europe, as in western Canada, Pg a, Pg 13 and Pg 16 are the most effective genes, followed by Pg 9. Gene Pg 4 is also highly effective in Europe but is ineffective in Canada (Table 2). Breeding of oats for multigenic resistance to stem rust is proposed similar to that for crown rust (Sebesta and Harder, 1983).

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TABLE 1 - Avirulence/virulence combinations of isolates of Puccinia graminis f.sp. avenae from Austria (A), Czechoslovakia (CS), Federal republic of Germany (D), Poland (P) and Switzerland (CH) in 1978-1985.

European Nos. (correspondence to NA Nos.)	Avirulence/virulence combination of Pg genes	Number of isolates				
		A	CS	D	CH	P
E 1	1,2,3,4,8,9,13,15,16,a/	2	2	-	-	-
E 2	1,2,4,8,13,15,16,a/3,9	1	1	2	-	-
E 3 (=NA 5)	1,2,4,8,9,13,16,a/3,15	-	6	-	-	-
E 4 (=NA 50)	3,4,9,13,15,16,a/1,2,8	3	2	-	-	1
E 5 (=NA 23)	4,9,13,15,16,a/1,2,3,8	5+4 1/	22+8 1/	4	1	-
E 6 (=NA 21)	3,9,13,15,16,a/1,2,4,8	2+1 2/	7	-	-	-
E 7 (=NA 18)	2,4,9,13,16,a/1,3,8,15	1 2/	1	-	-	-
E 8 (=NA 27)	9,13,15,16,a/1,2,3,4,8	-	8	-	-	-
E 9 (=NA 22)	4,9,13,16,a/1,2,3,8,15	1	3	7	1	-
E10 (=P 1)	2,3,4,15,a/1,8,9,13,16	-	-	-	-	1
Total number of isolates		20	60	13	2	2
Mean virulence capability		3,4	3,8	4,2	4,5	4,0

Mean virulence capability = (a x b)/c, where a = no. of isolates of virulence combination, b = no. of ineffective host (Pg) genes in that combination and c = total no. of isolates.

1/ used a reduced set of differentials (1,2,3,4,6,8,13), also E9 (=NA 22) cannot be excluded,

2/ used a reduced set of differentials

TABLE 2 - Percentage of isolates of Puccinia graminis f.sp. avenae with virulence on Pg resistance genes in 1978-1985.

Pg resistance gene	Country[a/] A	CS	D	CH	P	CDN [e/]
Avirulent[b/]	10.0	3.3	0.0	0.0	0.0	0.0
Pg 1	85.0	85.0	84.6	100.0	100.0	99.6
Pg 2	80.0	83.3	84.6	100.0	50.0	99.6
Pg 3	60.0	81.7	100.0	100.0	0.0	100.0
Pg 4	15.0[c/]	25.0[c/]	0.0	0.0	0.0	98.9
Pg 8	85.0	85.0	84.6	100.0	100.0	99.6
Pg 9	5.0[d/]	1.7[d/]	15.4	0.0	50.0	4.3
Pg 13	0.0	0.0	0.0	0.0	50.0	0.0
Pg 15	20.0	16.7	53.8	50.0	0.0	12.4
Pg 16	0.0	0.0	0.0	0.0	50.0	0.0
Pg a	0.0	0.0	0.0	0.0	0.0	0.0

a/ A = Austria, CS = Czechoslovakia, D = Federal republic of Germany, CH = Switzerland, P = Poland.

b/ Refers to percentage of isolates avirulent on all of the Pg-gene lines tested.

c/ Virulence on Pg 4 occurred also earlier in isolates of races 22 (8 A) (Sebesta and Zwatz, 1977), 24, A 2, A 3, A 4 and CS 1 (Sebesta, 1984).

d/ Virulence on Pg 9 occurred earlier in isolates of races 76 and 77 (A, CS) (Sebesta, 1984).

e/ Prairie region of Canada.

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MONOSOMIC ANALYSIS OF RESISTANCE TO STEM RUST RACE 11 IN THE SPRING WHEAT CULTIVAR SYLVA.

BY

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INTRODUCTION

Cereal rusts are important wheat diseases which can be controlled or reduced by resistance breeding. With knowledge of the genes for resistance in the breeding lines permits the production of optimal combinations of genes for resistance and other agronomically important traits and qualities. This contribution presents results of the monosomic analysis of stem rust resistance in the Czechoslovak spring wheat cultivar Sylva.

MATERIAL AND METHODS

The cultivar Sylva is a high yielding, medium early spring wheat with lower bread-making quality originating from the cross Praga x Siete Cerros. The cultivar Praga (released in 1968) was derived from the cross Capega x Wheat/Agropyron hybrid 22850. Since 1982, when Sylva was released, it has been resistant to all stem rust races found in Czechoslovakia. It is also resistant to most leaf rust isolates but virulent isolates were found sporadically in the last 3 years. In the field it is also usually resistant to yellow rust and powdery mildew.

For the location of stem rust resistance, standard monosomic analysis using monosomic series of the cultivar Zlatka was applied. Single progenies of F_2 generation of mono-Zlatka x Sylva were tested in the greenhouse at the seedling stage. Greenhouse temperature varied between 18 and 22°C, additional illumination of fluorescent tubes for 18 hours per day was used.

Inoculation was carried out by rubbing the wetted first leaf with urediospores of stem rust race 11 (isolate G 425). Inoculated plants were sprayed with tap water and kept under covered glass cylinders for 48 hours. Infection types were classified after Stakman et al. (1962).

RESULTS AND DISCUSSION

The F_2 generation of the crosses mono-Zlatka x Sylva segregated into two classes: resistant, infection type 0; and susceptible, infection type 3-4. When data for all progenies were summarized, the frequency of resistant plants was 76.6%. This indicates segregation for one dominant gene, 3:1. Segregation of single progenies in F_2 generation and the corresponding χ^2 values are summarized in Table. 1 The data confirmed the segregation ratio 3:1

except in the progeny from the monosomic 6B with 93.5% of resistant plants (X^2 = 11.386). This leads to the conclusion that stem rust resistance to race 11 of the cultivar Sylva is governed by one dominant gene on chromosome 6B. The four susceptible plants of this critical progeny are assumed to be nullisomic. The conclusion about monogenic resistance was also confirmed by a χ^2 test of homogeneity. When all lines were tested the value of this test was 29.464. This indicates (at P = 0.01 - 0.05) that a certain progeny does not belong to the analyzed set. When 6B progeny was excluded, the value of χ^2 test of homogeneity decreased to 18.719 (f=18, $P_{0.01}$ = 34.805) which proves that the remaining progenies have the same segregation; i.e. 6B progeny is the critical one. The evaluation of the segregation of all progenies except 6B also shows a better coincidence with 3:1 ratio than the total sum (Table 1). The value of χ^2 test is 0.598 with the total number of 1456 plants.

Location of the resistance gene on chromosome 6B as well as the low infection type suggest that the gene for stem rust resistance is Sr 11. It is possessed by Siete Cerros (Luig, 1983), one of the parents of Sylva.

Besides the critical progeny 6B, increased numbers of resistant plants (over 80%) were observed in the progenies of the 5th homoeologous group, particularly in the progeny 5B. The effect of chromosomes of the fifth homoeologous group on increased stem rust resistance was also found in the cultivar Slavia when monosomic analysis of stem rust resistance was carried out using monosomic series of Chinese Spring, both in seedlings inoculated with race 11 (Kosner and Bartos, 1983a) and in adult plants inoculated with race 21 (Kosner and Bartos, 1983b).

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TABLE 1 - Segregation of F_2 generation progenies of the crosses mono - Zlatka x Sylva after inoculation with stem rust race 11.

Progeny from monosomic	Total number of plants	Resistant IT 0,; number of plants	%	Susceptible IT 3,4 number of plants	%	χ^2 3 : 1
1A	75	52	69.3	23	30.7	1.284
1B	74	53	71.6	21	28.4	0.450
1D	72	52	72.2	20	27.8	0.296
2A	75	54	72.0	21	28.0	0.360
2B	64	44	68.8	20	31.2	1.333
2D	69	62	78.5	17	21.5	0.511
3A	71	55	77.5	16	22.5	0.230
3B	76	58	76.3	18	23.7	0.070
3D	60	48	80.0	12	20.0	0.800
4A	75	57	76.0	18	24.0	0.040
4B	73	52	71.2	21	28.8	0.553
4D	66	50	75.8	16	24.2	0.020
5A	73	60	82.2	13	17.8	2.014
5B	74	63	85.1	11	14.9	4.054 *
5D	70	59	84.3	11	15.7	3.219
6A	73	57	78.1	16	21.9	0.370
6B	62	58	93.5	4	6.5	11.376 **
6D	72	58	80.5	14	19.4	1.185
7A	-	-	-	-	-	-
7B	76	58	76.3	18	23.7	0.070
7D	96	66	68.3	30	31.3	2.000
Σ	1456	1116	76.6	340	23.4	2.110
Σ - 6B	1394	1058	75.9	336	24.1	0.598

Significance: P 0.01 = 6.635
P 0.05 = 3.842

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DISCUSSION OF A METHOD RESULTING IN ERRONEOUS POSTULATION OF THE GENE Yr8 FOR RESISTANCE TO PUCCINIA STRIIFORMIS IN PAKISTANI WHEAT CULTIVARS

BY

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Several methods have been proposed for using the gene-for-gene hypothesis to postulate genes for resistance to rust diseases in wheat cultivars (eg Loegering, McIntosh & Burton, 1971; Dinoor & Peleg, 1972; Browder & Eversmeyer, 1980). The methods are based on tests of a set of cultivars with a series of pathogen cultures to assess the infection types resulting from the interaction between host and pathogen. When the resulting data are set out in an array of cultivars versus pathogen cultures, patterns of high and low infection types are often observed. Cultivars that differ in their patterns of high and low infection types are assumed to differ in their resistance genotypes. The comparisons that can be made, to assess whether resistance genes are present, were discussed by Loegering and Burton (1974). Cultivars that show identical patterns of infection types may be assumed to carry identical genes for resistance. It is widely appreciated that such postulations do not constitute definitive proof of the presence of particular resistance genes, but can provide useful working hypotheses for further investigations.

Data produced during tests of many cultivars with many pathogen isolates are extensive and they are rarely presented in full. Various methods have been used to condense them, among which is a method for comparing reactions of two cultivars. This was first used by Browder (1973) who stated that it was based on a suggestion by J.F. Schafer. In this method, criteria are chosen for deciding in each test whether the infection type is high (HIT) or low (LIT). A table is constructed in which a control cultivar (C), for example a line with a single identified resistance gene, is compared with a test cultivar (T). The data are divided into four classes viz: 1) C-LIT:T-LIT, 2) C-LIT:T-HIT, 3) C-HIT:T-LIT, 4) C-HIT:T-HIT. The number in each class is the number of pathogen isolates giving the appropriate reactions on the two lines being compared. A hypothetical example is given in Table 1.

Table 1 shows that 20 pathogen cultures were used and it is

Table 1 - Numbers of pathogen cultures giving low infection types (L) or high infection types (H) on pairs of host lines. Hypothetical data.

	L:L	L:H	H:L	H:H
Control 1: Test Cultivar A	10	0	0	10
Control 2: Test Cultivar A	7	3	3	7
Control 1: Test Cultivar B	10	0	5	5
Control 2: Test Cultivar C	10	0	10	0

assumed that they were the same 20 in each of the tests. The total numbers of cultures giving LIT on each Control cultivar is the sum of classes L:L and L:H in the corresponding row and of those giving HIT is the sum of classes H:L and H:H. The total numbers of cultures giving LIT for each Test cultivar is the sum of classes L:L and H:L in the corresponding row and of those giving HIT is the sum of classes L:H and H:H. The data indicate that Control 1, Control 2 and Test cultivar A each gave LITs with 10 pathogen cultures and HITs with 10 whereas Test cultivar B gave LITs with 15 and HITs with 5 and Test cultivar C gave LITs with all 20 cultures. In any extension of the table, using the same pathogen cultures, each cultivar or control should give the same numbers of low and high infection types wherever it appears. In published tables this condition is sometimes not fulfilled. This must presumably be due to an error of calculation or a misprint but it leads to some doubt about the interpretation. For example in data of Rizvi & Statler (1982) for a line possessing for gene Lr10 (Table 2), in most rows a total of 8 cultures gave LITs for Lr10 but for the comparison with cultivar Ellar, 12 cultures were listed as giving LITs with Lr10. No explanation was offered for this variation.

Table 1 also shows that all the cultures that gave LITs on Control 1 also gave LITs on Test cultivar A and all cultures that gave HITs on Control 1 also gave HITs on Test cultivar A and that there were no cultures in the L:H and H:L classes. The reactions of the two lines are thus exactly parallel and the simplest hypothesis is that they contain the same gene or genes. Less likely but not impossibly, a similar pattern could arise where the pathogen cultures were interacting with a different gene in the test cultivar but variation for pathogenicity for the second gene was parallel to that for the first gene.

In the next line of Table 1 there are 3 cultures in the L:H class, giving LITs on Control 2 but HITs on Test cultivar A. This indicates that Test cultivar A does not possess the same resistance gene or genes as are present in Control 2. However, a similar result could be obtained if there was a previously unrecognised resistance gene in the control cultivar in addition to the recognised gene. Pathogen cultures could then exist that possessed matching pathogenicity for the recognised gene, which could also be present in the test cultivar, but lack pathogenicity for the unrecognised extra gene in the control. Such cultures would fall into the L:H class instead of the H:H class despite the presence of a common resistance gene between the control and test cultivars.

Either of the secondary hypotheses proposed in the previous two paragraphs could be the cause of doubts about the identification of the resistance gene Yr2 in some Pakistani wheat cultivars discussed by Perwaiz & Johnson (1986).

In the third line of Table 1 there were no cultures in the L:H class, and the reactions of Control 1 and Test cultivar B were parallel for 15 of the 20 cultures, giving either LIT on both or HIT on both. Such a pattern is often taken to indicate that the two lines possess a gene in common. However, there were 5 cultures that gave HIT on Control 2 but LIT on Test cultivar B. Where it is assumed that the lines have a gene in common this class (H:L) can be taken to indicate that the test cultivar also contains an additional gene or genes. It is important to note that this is not the only possible explanation. An alternative possibility is that the two lines do not have a gene in common but possess different genes. The data could indicate that, among the pathogen cultures used there were 10 that lack pathogenicity for both genes and 5 that possess pathogenicity for both genes. However, 5 cultures possess pathogenicity for the gene or genes in Control 1 but not for the gene or genes in Test cultivar B. By chance, or due perhaps to an aspect of the population genetics of the pathogen, no cultures possessed the opposite pattern, of pathogenicity for the gene(s) in cultivar B but not for those in Control 1. The implied non-random association of genes for pathogenicity, so that no cultures occurred in the L:H class, is not uncommon in populations of rust pathogens and collections of cultures held in laboratories (see Wolfe & Knott, 1982).

In the fourth line of Table 1, none of the cultures gave HITs on the Test cultivar C. This pattern has also been used to infer that the Test cultivar and Control cultivar possess a gene or genes in common but that the Test cultivar also contains additional genes (Rizvi & Statler, 1982). This interpretation may not be correct and alternative explanations may be more likely. Any single gene, or combination of genes, giving resistance to all pathogen cultures in a collection could give an identical pattern but such genes need not be similar in any way to the gene or genes in the Control cultivar (Statler, 1984).

One further aspect of the presentation of data in the form of Table 1 is that the identity of individual pathogen cultures is lost as a result of condensation of the data. Thus the table does not show how the particular cultures that gave LITs or HITs on Test cultivar A relate to those that gave LITs or HITs on Test Cultivar B, although this must be known from the original data. Models of gene-for-gene interactions, such as that of Person (1959), show that the same numbers of HITs and LITs can occur in rows or columns of the model due to different combinations of genes. It is the precise position of LITs in the model that relates them to particular genes. In the condensation of data into the four classes this information is suppressed.

In Table 2, data extracted from Rizvi & Statler (1982) are presented. These authors used the data to postulate the presence of Lr10 in all the cultivars, supported by data showing that Lr10 was present in their pedigrees. Without such support, the postulation of Lr10 for Red River 68 and Era, which were resistant to all cultures, would not be very secure. For these cultivars it is inevitable, as argued above, that classes L:H and H:H will be zero. If a zero in the

L:H class is taken to be indicative of a gene in common with the control it will be deduced that the cultivar possesses the same gene(s) as in each of the controls. Thus the test has no resolving power and is likely to lead not only to an overestimate of the number of genes in the test cultivar, as noted by Rizvi & Statler (1982), but also to completely wrong diagnoses. Although Rizvi & Statler (1982) postulated several possible genes in Red River 68, they only indicated the possibility of Lr10 in the cultivar Era. They gave no explanation for the different treatment of the two cultivars although it could have been due to knowledge of genes in the pedigrees of these cultivars.

Table 2 - Paired line:cultivar comparisons showing number of cultures of Puccinia recondita Rob. ex. Desm. f. sp. tritici that produced specified combinations of infection types. (Risvi & Statler, 1982).

Line/Cultivar comparison	Numbers of cultures with infection type combinations			
	L:L	L:H	H:L	H:H
Lr10: Butte	8	0	2	10
Lr10: Ellar	12	0	6	2
Lr10: Era	8	0	12	0
Lr10: Kenya Farmer	8	0	4	8
Lr10: Kitt	8	0	11	1
Lr10: Len	8	0	6	6
Lr10: Olaf	8	0	6	6
Lr10: Red River 68	8	0	12	0
Lr10: Waldron	8	0	7	5

In Table 2 there are variations in the distribution of cultures between the H:L and H:H classes. As noted above, it would be of interest to know whether the two cultures that are classified in H:L for Butte are the same as two of the cultures in the H:L class for all the remaining cultivars. Similarly it could be helpful to know which particular cultures fall into each of the H:L and H:H classes for each of the cultivars, but the method of classification conceals this.

Using the method of classification into four classes discussed above, Kirmani et al (1984) produced Table 3. These data are similar to those shown in line 3 of Table 1. Because there were no cultures in the L:H category the authors concluded that all the cultivars possessed the gene Yr8. The risk of making a wrong postulation with this pattern of data, as outlined above, is illustrated in this example. Although the two senior authors were not aware of it when they prepared the paper, it was known to the third author that data at the Research Institute for Plant Protection (IPO), Wageningen showed many cultures of P. striiformis to give LITs on Compair, which possesses Yr8, but to give HITs on the cultivars of Table 3. Such cultivars would fall into the L:H class, indicating that the test

Table 3 - Paired line:cultivar comparisons showing number of cultures of Puccinia striiformis West. that produced specified combinations of Low infection type (L) and High infection type. (Kirmani et al. 1984).

Line/Cultivar comparison	Numbers of pathogen cultures with L:L	L:H	H:L	H:H
Yr8: Sandal	7	0	13	20
Yr8: Lyallpur 73	7	0	11	22
Yr8: SA 75	7	0	11	22
Yr8: Nuri 75	7	0	15	18
Yr8: Yecora 70	7	0	11	22
Yr8: Indus 79	7	0	14	19
Yr8: PARI 73	7	0	20	13
Yr8: Blue Silver	7	0	12	21
Yr8: Bahawalpur 79	7	0	14	19
Yr8: Sonalika	7	0	10	23
Yr8: Pavon 76	7	0	6	27

cultivars do not carry the resistance of the control cultivar. The alternative possibility that Compair contains another previously undetected resistance gene in addition to Yr8 is unlikely because the cultures lacking pathogenicity for Compair but possessing pathogenicity for the test cultivars are of very diverse origin and would not be expected to be uniform with respect to pathogenicity for any hypothetical extra gene in Compair. The data of Table 3 show that 40 cultures were used in the tests, of which only 7 lacked pathogenicity for Yr8. By chance, none of these 7 possessed pathogenicity for any of the cultivars in Table 3. Among the 33 cultures that possessed pathogenicity for Yr8 there was considerable variation in the numbers of cultures giving LITs and HITs on the cultivars, indicating diversity in their resistance.

In conclusion, the authors wish to record that the designation of the gene Yr8 in the cultivars in Table 3 (Kirmani et al, 1984) was shown by data already existing at the IPO to be highly improbable. This conclusion is supported by knowledge of the pedigrees of the cultivars which do not include a source of the gene Yr8. This paper is intended to record the error and to illustrate how it could arise with limited data and insufficient circumspection in the application of the method discussed. Unfortunately, although additional data were available with the third author of Kirmani et al. (1984) that would have indicated the improbability of the postulation, geographical separation of the authors inhibited the transmission of the information in time to prevent publication.

The comparison of further data from IPO (Wageningen), PBI (Cambridge) (Perwiaz & Johnson, 1986) and from the Plant Breeding Institute at Castle Hill, University of Sydney (Hussain et al, 1986) has permitted preliminary agreements on some of the possible genes for resistance to yellow rust in the cultivars listed in Table 3 and the present information is summarized in Table 4. Finally, it should again be emphasized that these are postulations and that confirmation

Table 4 - Summary of postulated genes for resistance to P. striiformis in Pakistani wheat cultivars.

Cultivar	Postulated Yr genes
Bahawalpur 79	?
Blue Silver	"A"*, 2?**
Indus 79	7
Lyallpur 73	"A", 6
Nuri 70	"A", 6
PARI 73	"A", 6
Pavon 76	6, 7
SA 75	2?
Sandal	"A", 2?
Sonalika	"A", 2?
Yecora 70	"A", 2?, 6

*"A" gene(s) first detected in the Australian cultivar Avocet (see Hussain et al, 1986)
**2? possible presence of Yr2 requires further study (see Perwaiz & Johnson, 1986).

will require further work. Also, other genes for resistance to P. striiformis are likely to be discovered in the future in some of these cultivars.

Some of the difficulties referred to here are also relevant to the interpretation of data of the same type set out in other ways. As pointed out by Browder (1973) the resolving power of such analyses is limited by the range of variation represented in the set of pathogen isolates used in the tests. In addition, as noted by Browder (1973), the difficulties of undetected variation in pathogen isolates can also lead to misinterpretation of data from standard genetical studies involving the assessment of reactions in F_2, F_3 and other generations from crosses between cultivars.

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INHERITANCE OF RESISTANCE TO PUCCINIA HORDEI OTTH. IN THE SPRING BARLEY VARIETY TRUMPF

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SUMMARY

Genetics of resistance of the spring barley variety Trumpf to leaf rust was studied by testing the F_2 generations in the seedling stage. Tests were made with races avirulent and virulent for Trumpf. When testing with avirulent races, two dominant to semi-dominant, complementarily or additive genes were detected in Trumpf. In addition, a third gene acting complementarily or additively with these genes was found to avirulent races. None of the dominant genes was identical with gene Pa9 (HOR 2596).

INTRODUCTION

By using barley genotypes of Ethiopian origin it was possible, with the varieties Trumpf and Nadja to create for the first time varieties which, in addition to being resistant to mildew, were also resistant to leaf rust races frequent between 1974 to 1976. Trumpf virulent rust races emerged quickly and were first detected in 1977 due to the cultivation of these varieties. Seedlings of both varieties were susceptible. Resistance in the field declined over several years to a moderate level. The aim of this study was to determine the genetic basis in the variety Trumpf.

MATERIALS AND METHODS

The evaluation of inheritance of the resistance was carried out by testing the seedlings of the F_2 generation in the glasshouse, where some environmental control was possible. The following combinations were included in the tests:

HOR 2596 (CI 1243) and Trumpf x susceptible variety (Bigo and/or L94)
Trumpf x HOR 2596(CI 1243 = Pa9)
Trumpf x Pa3-donors (HOR 2476)
Trumpf x Pa7-donors (HOR 4519), HOR 1884, HOR 2564)

A total of 500 F_2 plants per combination and 20 plants of each parent were cultivated and tested with Trumpf-virulent and avirulent leaf rust races (Walther, 1979). The fully developed primary leaf of the test plants was inoculated by shaking well infected spreader plants over them. Following the work of Levine and Cherewick (1952) and Parlevliet (1976), the reaction types were assessed on a 1-4 scale. In the medium range of this scale, class 2 was subdivided into classes

2^- and 2^+ to facilitate the classification of the F_2 plants which occurred very frequently in this class. For statistical evaluation of the segregation results the χ^2 test was used. It was presumed, that the genes act dominantly or recessively, independently or complementarily and can be transmitted independently of each other. The classes 0 - 2^- were considered as resistant, classes 2^+ - 4 as susceptible but this sometimes varied depending on the parental reaction.

RESULTS AND DISCUSSION

Table 1 shows the results of testing F_2 plants of Trumpf and HOR 2596 crossed with a susceptible variety. When testing with avirulent races, gene Pa9 established by Tan (1977) was confirmed for HOR 2596. For Trumpf two dominant genes acting complementarily can be assumed. This was affirmed by another test when, analogously to the reaction of Trumpf, classes R and MR were combined. However, when considering class MR separately, the ratio 5:4:7 suggests an additive gene effect with semidominance of both genes. Full resistance would be reached if one semidominant gene would be homozygous and the other one at least heterozygous. When testing with Trumpf-virulent races the dominant gene Pa9 was no longer detectable for HOR 2596. Two recessive genes acting complementarily could be ascertained against the highly virulent race UN 23-1 + HOR 4280, whereby the high number of plants in the range suggests an additive effect of the recessive factors. In Trumpf another recessive gene may be assumed against races UN 23-1 + HOR 4280 and UN 30-1 + HOR 4280 in addition to the two dominant genes already detected against the avirulent races.

These genes act either complementarily or additively. Depending on the environment, this combined action varied although the dominance relations remained constant. The number of F_2-plants in the "MR" class varied from 0 to 10%.

Assuming two dominant or partially dominant genes for Trumpf acting complementarily or additively proved also to be correct when testing F_2 populations from combinations with Pa7 donors (Table 2). For both HOR 2596 and Trumpf, in addition to the dominant gene Pa7 of the respective partner, the expected number of genes against avirulent and virulent races and respective dominance relations were revealed. Against Trumpf races, for HOR 2596 also two recessive, complementary genes were found while for Trumpf two dominant genes with one further recessive, complementary gene were detected. Analogous results were produced when testing combinations with HOR 2476 (Pa3) with avirulent races (Table 3).

Dominance variations of HOR 2596 genes in contrast to those of Trumpf, when testing with virulent races allow the conclusion that despite the similar seedling reaction, different genes are present. Testing F_2 populations from the combination HOR 2596 x Trumpf and reciprocally confirmed this finding (Table 4). The genes in HOR 2596 were detected against races that were either avirulent or virulent for Trumpf, thus indicating that none of the dominant genes of Trumpf is identical with gene Pa9 in HOR 2596.

Honecker (1943) showed that field resistance was due to intensification of a moderate seedling resistance influenced by different growth conditions and could already be detected in the varying type of infection on seedlings. The results produced by

by Hartleb and Gerlach (1983) confirmed the significance of the characteristic pustule type. Therefore, based on six years of field results, the assumption seems justified, that the different level of field resistance of Trumpf and HOR 2596 is due to the above mentioned genes identified in the seedlings. The disease intensity of HOR 2596 varied in the course of six years' observations from 50 to 100 per cent, in the case of Trumpf from 10 to 70 per cent.

The high percentage of resistant plants in crosses of both varieties with each other as compared with combinations with the susceptible parent, show that the genes can be freely combined and their effect is additive. This fact allows the conclusion that the addition of genes from barleys of only medium resistance seems to be promising breeding work to improve the level of resistance.

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TABLE 1 - Inheritance of leaf rust resistance of Trumpf and HOR 2596 in crosses with susceptible varieties when testing with avirulent and virulent races

Cross	races			n	observed R	MR/MS	S	ratio	expected R	S	χ^2	genes
1. avirulent races												
Bigo x HOR 2596	23-1	S	R	216	161	-	55	3:1	162	: 54	0.025	1d(Pa9)
HOR 2596 x Bigo	23-1	R	S	338	266	-	72	3:1	253.5	: 84.5	2.465	1d(Pa9)
Trumpf x Bigo	23-1	R	S	600	433	-	167	3:1	450	: 150	2.569	1d
								9:7	337.5	: 262.5	1.620	2dk
Trumpf x Bigo	23-3	R-MR	S	1153	356	:314	:483	(5:4:7)	360.3	: 288.3	3.263	2dk
								9:7		: 504.4		
2. virulent races												
HOR 2596 x Bigo	23-1 +4280	S	S	817	61	:380	:376	1:15	51.1	: 765.9	2.062	2rk
Bigo x HOR 2596	23-1 +4280	S	S	377	30	: 94	:253	1:15	23.6	: 353.4	1.876	2rk
Bigo x Trumpf	23-1 +4280	S	R-S	246	66	: 25	:155	15:49	57.7	: 188.3	1.576	(2du+1r)k
Trumpf x Bigo	23-1 +4280	S-MS	S	660	94	: 76	:490	9:55	92.8	: 567.2	0.018	(2d+1r)k
L94 x Trumpf	30-1 +4280	S	S	609	89		:520	9:55	85.6	: 523.4	0.153	(2d+1r)k

S = susceptible
MS = moderately susceptible
MR = moderately resistant
R = resistant

d = dominant
r = recessive
k = complementary
u = independent

$\chi^2 = 3.481$ ($\alpha = 0.05$, 1DF)

$\chi^2 = 5.991$ ($\alpha = 0.05$, 2 DF)

TABLE 2 - Inheritance of leaf rust resistance of Trumpf and HOR 2596 in crosses with Pa7-donors when tested with avirulent and virulent races

Crosses	races			n	observed R MR/MS S	ratio	expected R S	χ^2	genes
1. avirulent races									
HOR 4519 x HOR 2596	23-1	R	R	507	480 - :27	15:1	475.3: 31.7	0.740	2du
HOR 2596 x HOR 1884	"	R	R	359	337 - :22	15:1	336.6: 22.4	0.009	2du
Trumpf x HOR 1884	"	R	R	222	145:13: 64	45:19	156.1: 65.9	0.078	(2du+1d)k
HOR 1884 x Trumpf	"	R	R	428	315:113:0	45:19	300.9:127.1	2.214	"
Trumpf x HOR 4519	"	R	R	289	249 - :40	54:10	243.8: 45.2	0.692	(2 from 3d)k
HOR 4519 x Trumpf	"	R	R	640	534: 21:85	54:10	540.0:100.0	0.467	"
HOR 2564 x Trumpf	"	R	R	693	611 - :82	57:7	617.2: 75.8	0.570	(2dk+1d)u
Trumpf x HOR 2564	"	R	R	394	346 - :48	57:7	350.9: 43.1	0.627	"
2. virulent races									
HOR 1884 x HOR 2596	23-1 +4280	R	MS	1259	978:105:176	55:9	1082.0:177.0	0.007	(1d+2r)u
HOR 2596 x HOR 1884	"	MS	R	482	358:43:81	55:9	414.2: 67.8	2.999	" (2d+1r)k+1du
Trumpf x HOR 1884	"	MS-S	R	492	388 - :104	201:55	386.3:105.7	0.035	"
HOR 1884 x Trumpf	"	R	MS-S	596	396:82:118	201:55	468.0:128.0	1.004	"
Trumpf x HOR 4519	"	S	R	479	371 - :108	201:55	376.1:102.9	0.321	"
HOR 4519 x Trumpf	"	R	S	884	487:230:167	201:55	694.1:189.9	3.523	"
Trumpf x HOR 2564	"	S	R	424	334 - :90	201:55	332.9: 91.1	0.017	"
HOR 2564 x Trumpf	"	R	MS-S	257	141:51:65	201:55	201.8: 55.2	2.211	"

χ^2 = 3.841
= 0.05/DF = 1

TABLE 3 - Inheritance of leaf rust resistance of Trumpf and HOR 2596 in crosses with HOR 2476 (Pa3) when testing with avirulent races

Crosses	races			n	observed R MR/MS S	ratio	expected R	S	χ^2	genes
1. avirulent races										
HOR 2596 x HOR 2476	8-2	R	S	361	206:18:137	9:7	203.1	157.9	0.097	2dk
HOR 2476 x Trumpf	23-1	R	R	483	204:279: -	27:37	203.8	279.2	0.001	3dk
HOR 2476 x Trumpf	8-2	S	S	629	493:35: 101	193:63	474.2	154.8	3.026	(2dk+2r)u
HOR 2476 x Trumpf	23-2	MS	MR	288	48:136:104	43:21	193.5	94.5	1.421	(2dk+1r)u

HOR 2476, when testing with avirulent races = 1 gene = Pa3 (χ^2 = 3.841)
HOR 2476, when testing with UN 8-2 1-2 recessively = 1 rez. Gen (= 0.05/DF=1)
HOR 2476, when testing with UN 23-3 = no gene for resistance
(Walther 1983)

TABLE 4 - Inheritance of leaf rust resistance of Trumpf and HOR 2596 in crosses with one another when testing with avirulent and virulent races

Crosses	races			n	observed R	MR/MS	S	ratio	expected R	S	χ^2	genes
1. avirulent races												
Trumpf x HOR 2596	23-1	R/MR	R/MR	417	276	98	43	57:7	371.4	45.6	0.168	(2dk+1d)u
HOR 2596 x Trumpf	23-3	R/MR	R/MR	254	209	18	27	57:7	226.2	27.8	0.025	(2dk+1d)u
Trumpf x HOR 2596	23-3	S/MR	R/MR	199	140	42	17	57:7	177.2	21.8	1.171	(2dk+1d)u
				247	88	56	103	39:25	150.5	96.5	0.723	(1d+1r)u+1d)k
2. virulent races												
Trumpf x HOR 2596	23-1 +4280	MS	MS	710	150:433:127			199:825	138.0:572.0		1.300	[(2d+1r)k+2rk]u
HOR 2596 x Trumpf	"	S	MS	137	65		72	529:495	70.8:	66.2	0.974	[(2d+1r)k+2r]u

$\chi^2 = 3.841$
($\alpha = 0.05$, DF 1)

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IS TALL OATGRASS AN ACCESSORY HOST OF THE BROWN RUST FUNGUS (PUCCINIA RECONDITA) OF RYE?

BY

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SUMMARY

Evidence is presented that tall oatgrass (Arrhenatherum elatius) may be an accessory host of the brown rust fungus (Puccinia recondita) of rye. This finding is interesting, since host range studies reported in literature suggest that the fungus is largely confined to rye (Secale cereale) and S. montanum. An isolate A collected from tall oatgrass combined pathogenicity to at least two graminaceous species, rye and tall oatgrass, that belong to two tribes, Triticeae and Aveneae, respectively. Two isolates B and C collected from rye were not pathogenic to the tall oatgrass plant on which isolate A had been found. The mycelial morphology of isolate A was very similar to that of the rye brown rust fungus.

INTRODUCTION

In September 1986 two large (diameter about 2 m) foci of 'brown rust' were found on tall oatgrass (Arrhenatherum elatius) growing along the highway between Wageningen and Rhenen, The Netherlands. The foci were about 1 km apart. The identity of the rust occurring in one focus was studied, and evidence was obtained that the rust was closely related to or belonged to the brown rust fungus of rye (Puccinia recondita, syn. P. dispersa).

MATERIALS AND METHODS

One rusted tall oatgrass plant was transplanted to a bucket and placed in the greenhouse. The same day, urediospores (isolate A) from this plant were applied to a seedling of barley line L94. About 40 hours after inoculation the seedling leaf was collected and stained according to previously described methods (Niks, 1986). By phase contrast microscope (1000 x), the mycelial morphology of 30 sporelings was studied for the criteria mentioned in Niks (1986).

Meanwhile the rust was multiplied on the host-plant by shaking of the plant to spread the spores followed by incubation in a greenhouse compartment at 100% relative humidity for one night. This multiplication procedure was carried out once a week, for 1 month. Spores were collected and applied to primary leaves of 10 barley, 10 wheat, 9 rye and 10 oat lines to study aspects of nonhost resistance, on which I hope to report in a separate paper.

Since all rye accessions proved to be very susceptible (IT 9 on the 0-9 scale) (MacNeal et al., 1971), the brown rust isolate from rye used at IVP was tested for pathogenicity on the collected tall

oatgrass plant. This plant was cut back, to remove the rust growing on it. The plant was divided into four parts that were planted in separate pots. After regrowth one part was inoculated with the brown rust isolate B, which was collected from rye several years ago in Wageningen (Niks, 1986). The second part was inoculated with isolate C, which was collected in 1986 from rye at a distance of less than 100 m from the place where isolate A was found. Before that time the focus in tall oatgrass had been mown away. The remaining two parts were not inoculated and served as control to check whether elimination of isolate A by cutting back had been effective. All four plants were incubated. The IT resulting from this inoculation was determined 14 days after inoculation. Parts of inoculated leaves were collected and processed for UV microscopy, according to the methods described before (Niks, 1982).

A specimen of isolate A on tall oatgrass has been sent to the Rijksherbarium, Leiden, The Netherlands.

RESULTS

Morphology of isolate A

In neither of the foci of the tall oatgrass vegetation were telia observed. The uredia from the focus from which isolate A was collected did not contain paraphyses, which rules out the possibility that the rust fungus was *P. brachypodii* var. *arrhenatheri*. The germpores of the urediospores were scattered.

The morphology of the primary infection structures (Table 1) resembled that of the rye brown rust isolate B, described before (Niks, 1986). The longitudinal orientation of the substomatal vesicles (SSVs) and primary infection hyphae (PIHs), the eccentric position of the septum in the SSV and the shape of the haustorial mother cells were among the common features. The differences between the two isolates were quantitative. The isolate from tall oatgrass had a higher frequency of septate SSV, the septa were less obscure, the frequency of sporelings with one of the PIHs reduced to a pointed protrusion was nil, and the PIHs were shorter and less straight.

Inoculation experiments

Isolate A was highly compatible with all nine pure lines of rye. Isolates B and C did not produce any symptoms on the tall oatgrass plant. The histological observations indicated that the two rye brown rust isolates were arrested in early stages of the infection process. A large number of infection units had formed appressoria elsewhere than over stomata. Many appressoria had not penetrated the stomata. The remainder of the infection units were arrested before they had formed more than two primary infection hyphae (early abortion, *sensu* Niks, (1982)).

DISCUSSION

Literature suggests that the host range of the brown leaf rust fungus of rye is very limited. Anikster and Wahl (1979) give as host range rye and "to a limited extent" *Bromus*, *Hordeum*, and *Elymus*. Wilson and Henderson (1966) consider rye the only host species. Extensive tests by Mains (1933) in which many grass species from many genera were included, but not *Arrhenatherum*, suggested that the brown

rust was only pathogenic on Secale cereale and S. montanum. Hassebrauk (1932) and Waterhouse (1929) did include Arrhenatherum elatius (syn. Avena elatior) in their tests, and found no symptoms on this species. In view of these reports the presently reported non-pathogenicity of rye brown rust isolates B and C to the tall oatgrass plant is not surprising.

Identification of rust fungi is often not easy, especially when telia are lacking. The absence of paraphyses, pathogenicity on seedlings of rye and similarity of the mycelial morphology with that of the rye brown rust fungus strongly point to the conclusion that isolate A belongs to the Puccinia recondita complex. Neither Straib (1952) nor Wilson and Henderson (1966) mention occurrence of a P. recondita forma on tall oatgrass. The small differences in mycelial morphology between isolate A and B and non-pathogenicity of isolates B and C on tall oatgrass suggest that isolate A is a different forma of P. recondita than the regular brown rust fungus of rye, but very closely related to it.

It remains to be investigated to what extent tall oatgrass and rye play a part in the epidemiology of their respective brown rust fungi.

The frequency of P. recondita of tall oatgrass is unknown. A possible explanation that the tall oatgrass population consisted of abnormally susceptible plants ('occasional host' sensu Niks (1987)) with regard to the rye leaf rust is refuted by the observation that isolates B and C were non-pathogenic on the tall oatgrass plant. It is more likely that the all oatgrass 'P. recondita' represents a (relatively recent ?) jump by the rye attacking form to tall oatgrass. This is remarkable, since rye and tall oatgrass belong to two different tribes (Triticeae and Aveneae, respectively). The fact that single isolates of specialized biotrophic pathogens may be pathogenic to grass species belonging to different tribes has been reported before, e.g. in P. coronata (Eshed and Dinoor, 1981) and in Erysiphe graminis (Wahl et al., 1978). For the highly specialized brown rust of rye, such a jump for oat grass is expected to be more difficult than a jump to a species such as wheat. Until now, there is no evidence of occurrence of rye brown rust on wheat. Wheat brown rust can be considered a distinct rust species, that differs in many respects from the rye brown rust (Niks and Dekens, 1987).

The present report illustrates that host range studies of highly specialized biotrophic fungi should not be restricted to plant species that are closely related to the known host. It also demonstrated that mycelial morphology has its value in the identification of rust fungi. In this case the identification of isolate A was confirmed by its pathogenicity for rye seedlings.

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TABLE 1 - Morphological features of the 'brown rust' isolate from tall oatgrass as compared to those of a brown rust isolate from rye.

Rust isolate from:	Diameter of PIH		Percentage of sporelings with		Predominant shape of HMC
	Average	range	septum in SSV	two poles	
tall oatgrass	4.6	3.5-6	63	97	lobed
rye	5.1	4-6	27	83	lobed

PIH Primary infection hypha
HMC Haustorial mother cell
SSV Sub-stomatal vesicle

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PROBABLE LEAF RUST RESISTANCE GENES AND FIELD REACTIONS OF BACK CROSS LINES OF TWO INDIAN WHEAT CULTIVARS[1]

BY

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INTRODUCTION

In India, leaf rust of wheat (Puccinia recondita f.sp. tritici) can cause damage in all wheat growing areas of the country. Breeding rust resistant varieties is a most economical and practical solution. To develop leaf rust resistant lines, two Indian standard commercial varieties, Kalyansona and Sonalika, were backcrossed with several donor parents resistant to leaf rust at the Wheat Project Directorate, Indian Agricultural Research Institute (IARI), New Delhi. Here we present information on probable genes for leaf rust resistance carried by 17 backcross lines developed under this program. The Area Under the Disease Progress Curve (AUDPC) was recorded on eight of these lines for two seasons and seedling and adult plant reactions were compared.

MATERIALS AND METHODS

The pedigree, height and days to flowering of the 17 lines studied are given in Table 1. These lines were tested for seedling reaction to six Indian leaf rust races viz., Race 11, Race 12, Race 77, Race 104, Race 107 and Race 162. Seedlings of twenty Lr isogenic lines, namely Lr 1, Lr 2a, Lr 2b, Lr 2c, Lr 2d, Lr 3, Lr 3 ka, Lr 10, Lr 11, Lr 12, Lr 13, Lr 14a, Lr 14b, Lr 14ab, Lr 15, Lr 16, Lr 17, Lr 18, Lr 20 and Lr 23 were also tested with the above-mentioned six races. The first leaf of 7-8 days old seedlings was inoculated following the standard procedures for inoculation of seedlings. Two weeks after inoculation, seedling reactions were recorded according to the key developed by Mains and Jackson (1926).

The Matching technique was used to speculate the probable presence of Lr gene or gene combinations in the 17 lines. This technique involves the matching of the infection type of the test lines with that of the standard Lr isogenic lines for each of the races of the pathogen (Table 2).

Eight lines (S. nos. 10 to 17 in Table 1) were grown in replicated experiments in a field at the Indian Agricultural Research Institute, New Delhi, India in Rabi 1979-80 and Rabi 1980-81. Mean

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Coefficient of Infection (Loegering, 1959) for leaf rust was recorded at weekly intervals at 9 widely spaced points (five tillers per point) in each experimental plot. The rate of rust spread ("r") was estimated for each plot by the regression method suggested by Vanderplank (1963) which gives equal weight to all the points on the disease progress curve. The Area Under the Disease Progress Curve (AUDPC) was calculated for each plot as suggested by Wilcoxson et al. (1975).

To identify the races/biotypes of P. recondita present on the experimental material in the field, leaf rust samples were collected in both the seasons. Single rows of the eight lines were also grown in Summer, 1980 at Lahaul (Northern hills zone) and Wellington (Southern hills zone) representing the two foci of infection of leaf rust in India. Leaf samples infected with leaf rust were collected from the experimental material at both the locations. The leaf rust samples were analysed for race composition as suggested by Johnston and Mains (1932) based on the reactions on the standard differential set and ten other additional differentials (Thew, IWP 94, NI 5439, HD 4502, Moti, Kalyansona, Sonalika, Sharbati Sonora, UP 319 and KLM-mutant) used to identify Indian leaf rust races/biotypes.

RESULTS AND DISCUSSION

The seedling reactions of the seventeen backcross lines and Lr isogenic lines to the six races of leaf rust used in this investigation and probable Lr gene(s) in the backcross lines are given in Table 1 and Table 2.

79D-2301 was resistant to all the six races of leaf rust (Table 2). The seedling resistance of this line could be due to one of the five alternative combinations of Lr genes which are: Lr 1 + Lr 10 + Lr 17, Lr 3ka + Lr 10 + Lr 17, Lr 1 + Lr 10 + Lr 16, Lr 10 + Lr 15 or Lr 10 + Lr 20. However, its donor parent, J-85, has Sonora 64 (Lr 1; McIntosh, 1983) and Klein Lucero (Lr 17; McIntosh, 1983) in its pedigree. This suggests that the most probable combination of Lr genes possessed by this line is Lr 1 + Lr 10 + Lr 17. Similarly, the presence of Kenya (Kenya W 1483 is reported to possess Lr 15; McIntosh, 1983) in the pedigree of Timgalen (Zeven and Zenen-Hissink, 1976) which is the donor parent of 78D-1615 and 78D-1716, supports the presence of Lr 15 in these two lines. Lr 15 provides resistance to biotypes 12B and 104B (Nagarajan et al., 1981) which are now widely prevalent in India (Nayar et al., 1985). Therefore the five backcross lines possessing Lr 15 (Table 1 and Table 2) may be useful in the Indian wheat program.

The Mean Coefficient of Infection (MCI), Area Under the Disease Progress Curve (AUDPC) and rate of rust spread ("r") of four backcross lines each of Kalyansona and Sonalika and the two recurrent parents are given in Table 4a and Table 4b. Kalyansona had the highest MCI (at all the stages), AUDPC and "r" in both the seasons. But one of its backcross lines, 79D 2257, had negligible infection in both the seasons. Since race 77 was dominant during the period when the field tests were conduced (Table 5a and Table 5b) and Lr 10 is effective against race 77 (Table 2), the field resistance of this line may be attributed to presence of the gene Lr 10 in it. Two other backcross lines of Kalyansona, 79D-2254 and 79D-2256, which do not possess seedling resistance to race 77 and do not appear to possess Lr 10, also had low disease in both the seasons. The highest rate of

rust spread of these two lines (0.1190 and 0.1454, respectively) was also almost half of that of their recurrent parent, Kalyansona (r = 0.2579). The analyses of leaf rust samples collected from these two lines at different locations suggest that they are susceptible to race 77 at the adult plant stage. Since race 77 was dominant in the field, the lower disease level of these two lines cannot be attributed to their resistance to other races of leaf rust. This may indicate that 79D-2254 and 79D-2256 possess slow-rusting resistance.

In contrast to the above observation, two backcross lines of Sonalika, 79D-2301 and 79D-2305, which were resistant to race 77 at the seedling stage, had high infection at adult plant stage. The analyses of samples from these two lines suggest that these two lines were susceptible to race 77 (the dominant race) at adult plant stage. One simple explanation could be that the lack of correspondence between seedling and adult plant reactions in this case was due to the presence of temperature sensitive Lr gene(s). As discussed earlier, 79D-2301 may have received Lr 17 from Klein Lucero in its pedigree and Lr 17 has been reported to be temperature sensitive (Dyck and Samborski, 1968). Similarly, the unidentified gene(s) in 79D-2305 (Table 3) may be temperature sensitive. However, another possible explanation could be that another biotype of race 77, to which the two lines were susceptible, was prevalent in the field but could not be distinguished with the differentials used to analyse the rust samples.

In spite of the possibility of the presence of a new biotype of race 77 in the field, lines 79D-2254 and 79D-2256 still displayed slow rusting resistance. These lines may be useful in Indian breeding programmes to develop slow-rusting cultivars which are particularly desirable for the two foci of infection in India (Himalayas in the north and Nilgiri and Pulney hills in the south). However, it should be noted that slow-rusting resistance cannot be guaranteed to be race non-specific (Johnson, 1984). Therefore, this resistance should be used with caution.

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TABLE 1 - Pedigree, height and days to flowering of 17 Indian lines of wheat.

S. No.	Line	Pedigree	Height (cm)	Days to flowering
1.	IWP-19	E6254 x Kalyansona^2	92-106	103
2.	78D-1533	CPAN493 x Kalyansona^2	97-104	108
3.	78D-1535	E5788-3 x Kalyansona^4	99-105	108
4.	78D-1549	E5788-3 x Kalyansona^4	94-100	106
5.	78D-1615	Timgalen x HD1553^2	92-97	94
6.	78D-1641	(PPSN972 x HD1553)^2 x HD1553	102-103	95
7.	78D-1716	Timgalen x HD 1553^2	94-98	94
8.	78D-1718	Tr260 x Sonalika^2	88-104	94
9.	78D-1725	(IRN68-22 x HD1553) x HD1553	102-105	96
10.	79D-2226	Bulgaria 88 x Kalyansona^2	93-106	108
11.	79D-2254	E4667 x Kalyansona^2	91-97	104
12.	79D-2256	E5868 x Kalyansona^4	96-100	104
13.	79D-2257	(Tr260 x Kalyansona) x Kalyansona^2	94-95	103
14.	79D-2301	(J85 x HD1553) x HD1553^2	98-108	97
15.	79D-2305	(HS19 x HD1553) x HD1553^2	99-104	96
16.	79D-2313	(Timgalen x HD1553) x HD1553^2	97-104	94
17.	79D-2323	(HS19 x HD1553) x HD1553^2	94-110	93

TABLE 2 - Seedling reactions of 12 Indian wheat lines and 4 Lr isogenic lines to 6 Indian races of leaf rust and probable Lr gene(s) in the Indian lines.

Line	Reaction to leaf rust						Probable Lr gene(s) in the test line
	Race 11	Race 12	Race 77	Race 104	Race 107	Race 162	
Lr 3ka	R	S	S	S	R	R	
78D-1535	R	S	S	S	R	R	Lr 3ka
79D-2256	R	S	S	S	R	R	Lr 3ka
Lr 15	R	R	S	R	S	R	
78D-1615	R	R	S	R	S	R	Lr 15
78D-1716	R	R	S	R	S	R	Lr 15
78D-1718	R	R	S	R	S	R	Lr 15
79D-2226	R	R	S	R	S	R	Lr 15
Lr 16	R	S	S	R	R	S	
79D-2323	R	S	S	R	R	S	Lr 16
IWP-19	R	S	S	R	R	R	Lr 3ka + Lr 16
78D-1549	R	S	S	R	R	R	Lr 3ka + Lr 16
79D-2254	R	S	S	R	R	R	Lr 3ka + Lr 16
Lr 10	R	S	R	S	R	S	
78D-1533	R	S	R	R	R	R	Lr 3ka + Lr 10 + Lr 16
79D-2257	R	S	R	R	R	R	Lr 3ka + Lr 10 + Lr 16

S in Shimla table

Nayar says he can't identify Lr16 with confidence It is not in the differential set.

By definition Lr10 is low with Race 12 = 5R5

ditto race 104 = 17R23 in Shimla diff. data reaction is given as 3/2+

TABLE 3 - Seedling reactions of 5 Indian lines and 4 Lr isogenic lines to 6 Indian races of leaf rust and probable Lr gene(s) in the Indian lines.

Line	Reaction to leaf rust						Probable Lr gene(s) in the test line
	Race 11	Race 12	Race 77	Race 104	Race 107	Race 162	
Lr 2a	R	R	S	S	S	S	
78D-1725	R	R	S	S	S	R	Lr 2a + additional gene(s) for resistance to race 162
78D-1641	R	R	S	R	S	S	Lr 2a + another gene(s) for resistance to race 104
79D-2313	R	R	S	R	S	S	
Lr 15	R	R	S	R	S	R	
79D-2305	R	R	R	R	S	R	Lr 15 + additional gene(s) for resistance to race 77
Lr 1	R	R	S	S	R	R	
Lr 10	R	S	R	S	R	S	
Lr 17	S	R	S	R	R	S	
79D-2301	R	R	R	R	R	R	Lr 1* + Lr 10 + Lr 17*

* Based on pedigree

TABLE 4a - Mean Coefficient of Infection, Area Under the Disease Progress Curve (AUDPC) and rate of rust spread ("r") on eight backcross lines and two recurrent parents (Kalyansona and Sonalika) in Rabi 1979-80.

Line/ Variety	Mean Coefficient of Infection				AUDPC (unit.day)	"r" (per unit per day)
	20 March	27 March	3 April	10 April		
Backcross lines of Kalyansona						
79D-2226	0.03	0.23	0.57	0.90	8.81	0.0998
79D-2254	0.11	0.32	0.51	0.68	8.59	0.0647
79D-2256	0.06	0.18	0.78	1.30	11.52	0.0967
79D-2257	0.00	0.00	0.00	0.001	0.007	0.0004
Kalyansona	5.00	18.00	70.74	77.11	908.57	0.2095
S.E.±	0.08	0.71	0.73	0.51	7.01	0.0129
C.D. (5%)	0.23	2.01	2.07	1.46	19.95	0.0366
Backcross lines of Sonalika						
79D-2301	0.49	2.40	5.62	6.47	80.53	0.1181
79D-2305	0.47	1.11	2.82	3.01	39.66	0.0862
79D-2313	0.99	1.99	4.67	4.67	66.40	0.0744
79D-2323	0.64	1.64	3.31	3.25	47.79	0.0742
Sonalika	0.73	1.82	4.22	4.33	59.93	0.0854
S.E.±	0.08	0.71	0.73	0.51	7.01	0.0129
C.D. (5%)	0.23	2.01	2.07	1.46	19.95	0.0366

TABLE 4b - Mean Coefficient of Infection, Area Under the Disease Progress Curve (AUDPC) and rate of rust spread ("r") on eight backcross lines and two recurrent parents (Kalyansona and Sonalika) in Rabi 1980-81.

Line/ Variety	Mean Coefficient of Infection					AUDPC (unit.day)	"r" (per unit per day)
	9 March	16 March	23 March	30 March	6 April		
Backcross lines of Kalyansona							
79D-2226	0.01	0.11	0.50	9.71	18.38	136.60	0.2088
79D-2254	0.04	0.33	0.71	2.61	3.02	36.28	0.1190
79D-2256	0.04	0.26	0.56	4.88	5.81	60.41	0.1454
79D-2257	0.00	0.00	0.001	0.02	0.02	0.17	0.0054
Kalyansona	0.36	1.19	20.92	66.67	71.44	872.73	0.2579
S.E.±	0.04	0.12	1.25	1.57	1.43	11.35	0.0150
C.D. (5%)	0.12	0.33	3.61	4.53	4.14	32.79	0.0433
Backcross lines of Sonalika							
79D-2301	0.06	0.61	5.32	25.94	26.07	314.59	0.2057
79D-2305	0.03	0.41	2.83	18.38	20.32	229.57	0.2037
79D-2313	0.12	0.83	4.44	21.88	22.55	269.44	0.1855
79D-2323	0.08	0.42	2.45	19.77	20.95	232.79	0.1978
Sonalika	0.05	0.39	3.27	23.67	25.30	280.01	0.2144
S.E.±	0.03	0.11	0.72	4.30	4.26	44.07	0.0174
C.D. (5%)	-	0.31	2.08	12.50	12.39	128.13	0.0505

TABLE 5a - Leaf rust virulences observed on eight back cross lines and two recurrent parents

Line	Races/biotype(s) observed on the line		
	Delhi (1980-81)	Lahaul (Summer, 1980)	Wellington (Summer, 1980)
79D-2226	77(2)*	77(1)	77(1)
79D-2254	77(2)	-	-
79D-2256	77(1)	77(1)	77(1)
79D-2257	77(1)	-	-
Kalyansona	104B	77(1)	-
79D-2301	77(1)	77(1)	-
79D-2305	-	77(1)	77A,77A-1(1)**
79D-2313	-	-	-
79D-2323	-	-	-
Sonalika	77(1)	77(1)	77(1),77A-1(1)

* Figures in parenthesis indicate the number of samples analysed.
** Two virulences detected in one sample.

TABLE 5b - Leaf rust races/biotypes prevalent at Delhi (1979-80 and 1980-81), Lahaul and Wellington

Location	Year	Number of samples					
		12B	77	77A	77A-1	104B	Total
Delhi	1979-80	1	1	0	0	2	4
	1980-81	0	50	7	0	1	58
Lahaul	Summer, 1980	1	43	0	0	0	44
Wellington	Summer, 1980	0	11	5	2	1	19
Total		2	105	12	2	4	125