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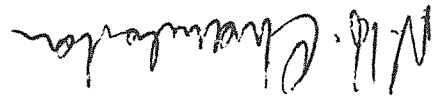
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CEREAL RUSTS BULLETIN

K.O. M. 1985
Liberty

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Yours sincerely

At present there are no papers to continue publication. Therefore if subscribers wish that the Rust Bulletin continues I urgently ask you to send me your contributions on any aspect of cereal rust research such as race surveys, resistance gene analysis, techniques, breeding.

I would appreciate receiving comments from subscribers concerning the Bulletin. What is your opinion, do you wish the Bulletin to continue in its present form?

Firstly very many apologies for the lateness of this part of the Cereal Rusts Bulletin. This is because of a severe shortage of contributions. Without papers, short articles or brief notes on all aspects of cereal rust research the Bulletin cannot be published.

Dear Subscribers

From the Editor

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LATELAT PERIOD AND SPORE PRODUCTION OF FOUR CULTIVARS OF BARLEY HEAVILY
INFECTED BY TWO RACES OF Puccinia hordei

BY

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The latent period and spore production of two isolates of *Puccinia hordei* were measured on the second leaf of four barley cultivars. Greater spore production and shorter latent period were observed with race 673 than with race 11. The cultivars Sonja and Triumph were more resistant than Midas and Golden Promise having a longer latent period and lower spore production. However, significant differences in latent period and spore production were observed between the tip and the base of the leaves in each cultivar. In addition, production of teliospores was noticed on the cultivars when infected by race 673 but not when infected by race 11; differences in the percentage of unicellular teliospores were observed between the cultivars.

INTRODUCTION

Brown rust, caused by *Puccinia hordei* Otth, is one of the most important foliar diseases of barley. In the establishment of the disease, and its epidemiology, environmental factors and the characteristics of the host are important. In the barley - leaf rust relation, apart from the hypersensitive reaction, another type of resistance is known that has been designated partial resistance or slow leaf rusting. Latent period (LP) and spore production have been considered as the most important components of this resistance (Zadoks, 1972; Cliford, 1972; Parlevliet, 1975; Johnson and Wilcoxson, 1978). The objective of the work described here was to investigate differences in length of LP, spore production, spores per uredinium, size of uredinia and uredinia per square centimetre of leaf, between 4 different cultivars of barley infected heavily with two isolates of *P. hordei*.

MATERIALS AND METHODS

One winter (Sonja) and three spring barley cultivars (Midas, Golden Promise and Triumph) and two isolates of *P. hordei*, race 11 and 673 respectively, (supplied by Dr B.C. Cliford from the Welsh Plant Breeding Station, Aberystwyth) were used. A monospore culture of each isolate was made and multiplied before the experiments started. Single spore cultures were obtained using a Singer micromanipulator fitted with a fine tungsten needle. The plants were grown in the glasshouse, in soil (John Innes No 2) in pots of 10 cm diameter and inoculated eleven days after sowing, when the second leaf was fully expanded. For each cultivar, 10 plants were infected with fresh urediniospores of race 11, and 10 with race 673. The second leaf of each plant was infected using a paint brush to distribute the urediniospores uniformly over the upper surface of the leaf. The density of inoculum was about 4,000 spores/cm² and the germinability of the urediniospores was 66% for race 11 and 85% for race 673. The infected plants were covered by plastic bags and incubated at 20°C for 48 hours. They were then uncovered and maintained in the glasshouse at 20°C (±2) and 60-80% relative humidity.

Using a magnifying lens (10x), data were taken daily from when the first uredinia were seen until no more developed. The total number and the number of newly erupted uredinia were counted at the base, in the middle and at the tip of the upper surface of each leaf. The LP was estimated as the time interval between inoculation and that at which 50% of the uredinia were erupted (Shaner, 1980).

To estimate spore production, urediniospores were collected at 3-day intervals, from the time when sporulation began until the leaves had senesced by micro 'cyclone' spore collectors (Tervet and Cassell, 1951). To prevent spores scattering, a V-shaped aluminium foil tray was placed immediately below the second leaf. The harvested spores were dried and then counted using a Coulter Counter model ZB (Coulter Electronics Ltd, Harpenden, Herts, England).

After the first collection of spores, the numbers of uredinia on both the leaf surfaces was counted. The size of uredinia was determined by measuring the diameters, both parallel and perpendicular to the leaf veins. Nine pustules from every leaf: 3 on the basal third, 3 on the middle third and 3 on the tip third, were measured in μm using a stereobinocular microscope. The area (A) of each uredinium was measured using the formula $A = 11.3 \cdot b^2/4$ where a and b are the diameters. The leaf area was calculated using Ondok's method (Ondok, 1968). The data were statistically analysed.

The experiments were done between August and November 1984.

RESULTS

All the cultivar-isolate combinations produced a type 4 infection with the exception of Triumph, infected with race 11, which gave a hypersensitive (type 0) reaction.

On all the cultivars, the LP of race 11 was longer than that of race 673. Among the cultivars infected with race 11 there were no significant differences in LP but differences were obtained between the cultivars infected with race 673 (Table 1).

The shortest LP was observed on Golden Promise infected with race 673 and the longest on Sonja infected with race 11.

Significant differences were found in Golden Promise between infections of race 11 and those of race 673 but no such significant differences occurred in Sonja and Midas infected by the same races. Observations on the different parts of the leaf indicated a significant difference between the tip and the base of the leaf with the exception of Sonja infected with race 11 (Table 2). On Triumph, Sonja and particularly on Midas, infected with race 673, significant differences were noted also between the middle and the base of the leaf.

In all cultivars the eruption of uredinia began earlier with race 673 but proceeded more slowly than with race 11 (Fig 1). In consequence, on each cultivar, the percentage of erupted uredinia of race 11 soon overtook that of race 673.

The number of spores produced per square centimetre varied principally between the cultivars infected by a given race but also between these two races infected by a given race but also between these two races infected by a given race but also between these two single cultivar (Table 1). A substantial difference was observed between the different parts of the leaf in the amount and/or the duration of spore production. The tip always produced few spores for a short period (3-6 days) while the base produced a great number of spores and for a longer period (Table 3).

The total spore production and the spore production per square centimetre of leaf varied because of differences between cultivars and between races in the number of uredinia per square centimetre of leaf, in uredinal size and in spore production per uredinium (Table 1).

In the experiments described here, the LP with both the isolates was relatively short (less than 7 days) and between the cultivars the differences, even if significant, amounted to only a few hours. The small sizes of the differences were due not only to characteristics of the host and of the strains of the pathogen but possibly also to the experiments being carried out at the optimum temperature for the pathogen. At lower temperatures the LP would be longer and the differences between the cultivars would probably be more evident as demonstrated by Eversmeyer et al. (1980) with *P. recondita*. Furthermore the LP can vary with the stage of development of the plant (Parlevliet, 1975).

From preliminary tests, with light infection the LP seems to be longer than with heavy infections and similar in different parts of the leaf. Also the spore production pattern is rather complex and can depend on many factors. In these tests the spore production per square centimetre depended principally on the spore production per ureidium (Midas and Triumph infected by race 673) or on the spore production per ureidium and size of ureidium (Golden Promise infected by race 11) or to the spore production per ureidium and number of uredia per square centimetre of leaf (Sonja infected by race 11).

The same race produced different sizes of ureidia on different cultivars but there was no correlation between size of ureidium and spore production. In fact ureidia of small and equal size (Midas and Sonja infected by race 673 and Golden Promise and Midas infected by race 11) produced different amounts of spores; furthermore the biggest ureidia (Triumph and Sonja infected by race 11) produced few spores. There seems to be a better correlation between size of ureidium and number of ureidia per square centimetre.

The small number of ureidia per square centimetre in Sonja could indicate a low infectibility of this cultivar by race 11, probably caused by 'early abortion' (Niks, 1982).

The spore production, and the duration of spore production, varied between the different parts of the leaf because in some cultivars, and particularly with race 11, the tip and the middle of the leaf dried early (ie Golden Promise and Sonja), while the base remained alive for a long time and produced more spores.

DISCUSSION

The number of ureidia per square centimetre differed significantly between Golden Promise and Midas with race 673 but not between the same cultivars infected with race 11. Race 11 produced very few ureidia on Sonja.

The size of ureidia of the two races, on the same cultivar, did not differ much. Also between cultivars, with the exception of Triumph, the size of ureidia did not differ greatly.

Greater difference occurred in spore production per ureidium both between the cultivars and in the same cultivar infected with the two races. Race 673 always produced considerably more spores than race 11.

After 7 days from inoculation, the production of teliospores began on all the tested cultivars infected with race 673 of *P. hordei*. No telia were observed on the same cultivars infected by race 11.

The number of telia per square centimetre varied between the cultivars, the parts of the leaf and on any one leaf between the upper and the lower surface (Fig 2). The cultivar that produced most telia on all parts of the leaf and for longest was Sonja, while Golden Promise produced very few telia, just at the tip of the leaf and for a short time.

The percentage of unicellular teliospores varied considerably. On Midas and Golden Promise there were many more unicellular than bicellular teliospores, while in Sonja and Triumph there were nearly equal numbers of the two types (Table 4).

Why race 673 but not race 11 produced teliospores and why their production varied cultivar by cultivar and on each cultivar between the different parts of the leaf, was not investigated.

However, the formation of teliospores may represent another component of resistance which interrupts the multiplication of the agamic stage of the pathogen and consequently reduces its spread, and delays the epidemic. But where the alternate hosts of *P. hordei* (*Ornithogalum* spp., *Dipcadi erythraeum*, *Leopoldia eburnea*) (Anikster, 1982) are present, the formation of the sexual stage might be particularly important for the variability and increase in virulence of the pathogen.

The measurement of spore production is a metrical character that avoids the subjectivity that may arise when estimates of pathogenicity of the pathogen and the resistance of the host are made visually (Johnson and Taylor, 1976). The spore production is also an important component for the equilibrium of the races in the field (Kastegar, 1976).

Race 673, having a short LP, a heavy production of spores for a long period and an ability to attack Triumph, is more highly competitive than race 11. Probably this is the reason why race 673 has recently become very common in England (Clifford and Jones, 1984). Among the cultivars, Sonja and Triumph are of interest for their long LP, low spore production and resistance to race 11. These results seem to be related to the known degree of susceptibility of these cultivars in the field (Clifford and Jones, 1984; Anon. 1984).

These results may not be applicable to different temperatures or different stages of plant development or with different amounts of inoculum. However, the resistance of the host and the virulence of the pathogen are not static in space or time, therefore it is important to see not only the degree of quantitative resistance but also its durability.

ACKNOWLEDGEMENTS

I wish to thank the Italian 'Consiglio Nazionale delle Ricerche' for its financial support. I am grateful to Professor J.L. Hall for the hospitality extended by the Department of Biology, University of Southampton, England and to Dr J.G. Manners, Reader in Biology of that University, who supervised the work, for his help and advice.

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TABLE 1 - Latent period, uredinia per square centimetre, size of uredinium, urediniospores produced per uredinium and number of urediniospores for each square centimetre of the second leaf of 4 cultivars of barley infected by races 673 and 11 of *Puccinia hordei*

Race	Cultivar	L.P. (a) (hours)	Uredinia/ cm ² leaf (No)	Size of uredinium (mm ²)	Spores/ uredinium (No)	Spores/ cm ² leaf (No)
673	Golden Promise	153.9 D (b)	337.6 A	.019 BC	53.5 C	17,498 B
	Midas	160.6 BC	247.4 BC	.016 C	119.5 A	28,818 A
	Sonja	166.8 AB	326.5 AB	.020 BC	41.3 C	13,326 B
	Triumph	156.3 CD	265.9 ABC	.030 A	19.2 D	5,014 C
	Golden Promise	161.8 ABC	343.0 A	.016 C	32.6 CD	11,261 BC
	Midas	166.0 AB	313.7 AB	.020 BC	84.9 B	26,794 A
	Sonja	167.8 A	183.8 C	.024 AB	11.9 D	2,195 C
	Triumph	---	---	---	---	---
11	Golden Promise	161.8 ABC	343.0 A	.016 C	32.6 CD	11,261 BC
	Triumph	156.3 CD	265.9 ABC	.030 A	19.2 D	5,014 C
	Sonja	166.8 AB	326.5 AB	.020 BC	41.3 C	13,326 B
	Midas	160.6 BC	247.4 BC	.016 C	119.5 A	28,818 A
	Golden Promise	153.9 D (b)	337.6 A	.019 BC	53.5 C	17,498 B
	Midas	166.0 AB	313.7 AB	.020 BC	84.9 B	26,794 A
	Sonja	167.8 A	183.8 C	.024 AB	11.9 D	2,195 C
	Triumph	---	---	---	---	---

(a) : Hours between inoculation and the moment when 50% of the uredinia were erupted.
 (b) : Values with same letter are not significantly different at P = 0.01.
 (c) : Hypersensitive reaction.

TABLE 2 - Latent period (1) between different regions of the second leaf of 4 cultivars of barley infected by two races of *Puccinia hordei*

Race	Region of leaf	Cultivar			
		Triumph	Golden Promise	Midas	Sonja
673	Tip	148.5 de CD (*)	144.4 e D	148.9 de CD	160.5 bc B
	Middle	155.5 cd BC	156.4 bcd BC	157.9 bc BC	165.1 b AB
	Base	164.8 b AB	161.0 bc B	174.9 a A	174.8 a A
	Mean of the above	156.3	153.9	160.6	166.8
	Tip	-	153.2 c B	155.5 c B	164.8 b A
	Middle	-	165.3 b A	169.0 ab A	168.0 ab A
	Base	-	167.0 ab A	173.3 a A	170.6 ab A
	Mean of the above	-	161.8	166.0	167.8
11	Tip	-	153.2 c B	155.5 c B	164.8 b A
	Middle	-	165.3 b A	169.0 ab A	168.0 ab A
	Base	-	167.0 ab A	173.3 a A	170.6 ab A
	Mean of the above	156.3	153.9	160.6	166.8
	Tip	-	153.2 c B	155.5 c B	164.8 b A
	Middle	-	165.3 b A	169.0 ab A	168.0 ab A
	Base	-	167.0 ab A	173.3 a A	170.6 ab A
	Mean of the above	-	161.8	166.0	167.8

(1) : Hours between inoculation and the moment when 50% of the pustules were erupted
 (*) : Significance with $P = 0.05$ (small letter) and $P = 0.01$ (capital letter). Both refer to LP differences between the cultivars and the regions of the leaf infected by the same race
 (-) : Hypersensitive reaction

TABLE 3 - Regional and total spore production (1) of 4 cultivars of barley infected by two races of Puccinia hordei

Race	Region of the leaf	Cultivar			
		Triumph	Sonja	Golden Promise	Midas
673	Tip	42,500 (6)	360,800 (9)	198,400 (6)	391,700 (6)
	Middle	101,500 (6)	464,700 (9)	375,300 (6)	742,800 (9)
	Base	263,200 (12)	800,000 (15)	676,200 (12)	1,091,800 (15)
	TOTAL	407,200	1,625,500	1,249,900	2,226,300
	Tip	-	12,000 (3)	59,200 (3)	285,200 (6)
	Middle	-	78,000 (6)	172,700 (6)	601,500 (6)
Base	-	179,800 (9)	450,500 (12)	1,118,200 (15)	
TOTAL	-	269,800	682,400	2,004,900	

(1) : Between brackets duration of spore production (days) - : Hypersensitive reaction

TABLE 4 - Percentage of unicellular teliospores produced by Puccinia hordei, race 673, on four cultivars of barley

Cultivar	% of unicellular teliospores			
	Midas	Golden Promise	Sonja	Triumph
	78.8	76.8	59.9	51.6

The percentages are based on counts of about 550 spores for each cultivar

..... Sonja.
 -.-.-.-.- Golden Promise.
 - - - - - Triumph.

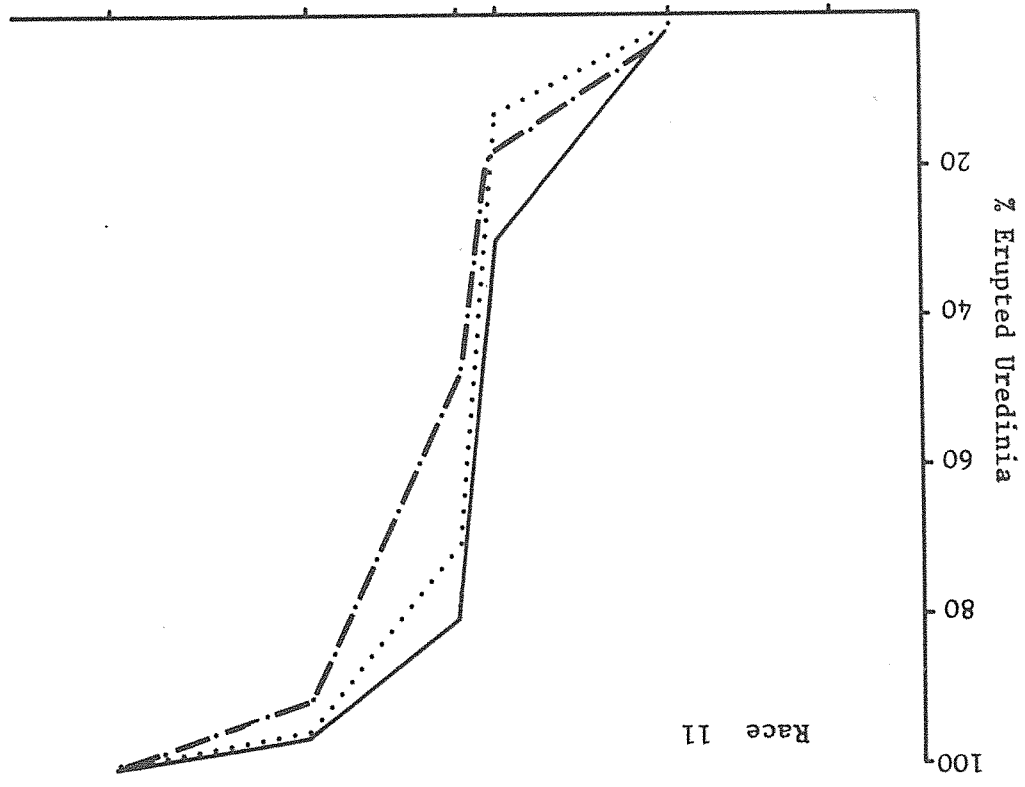
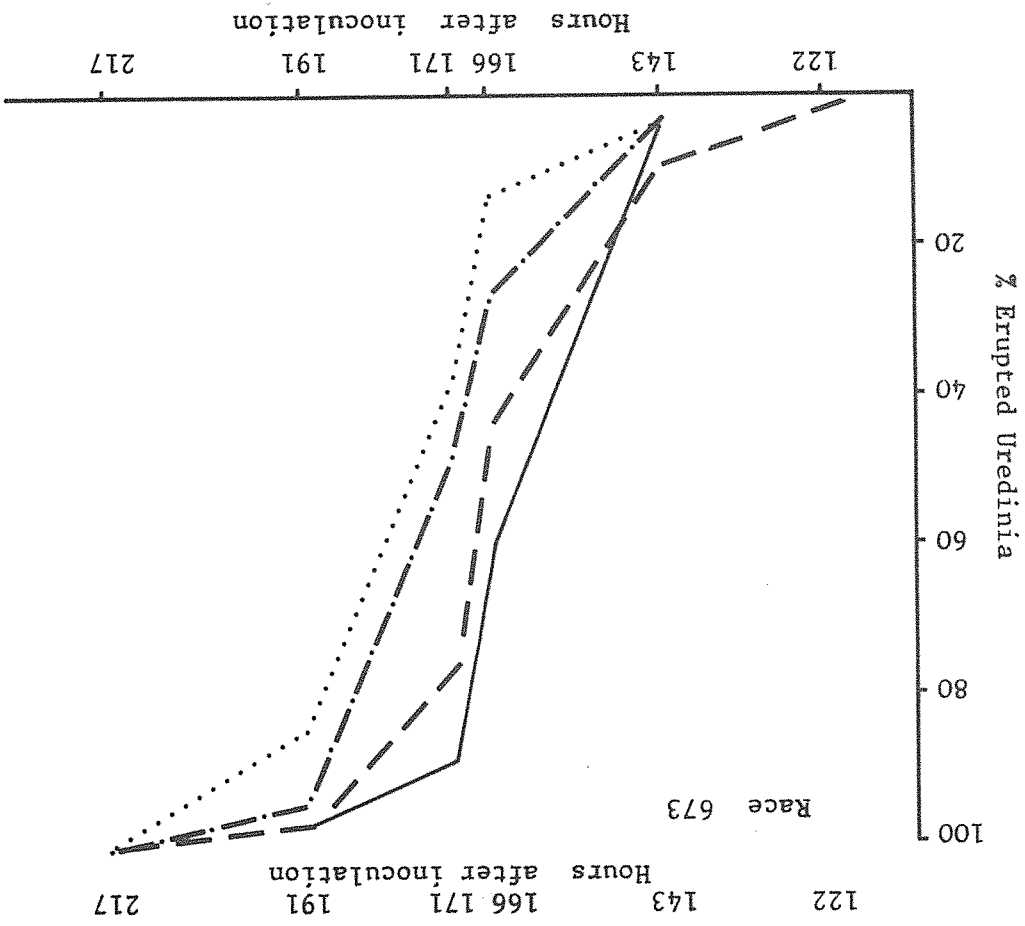
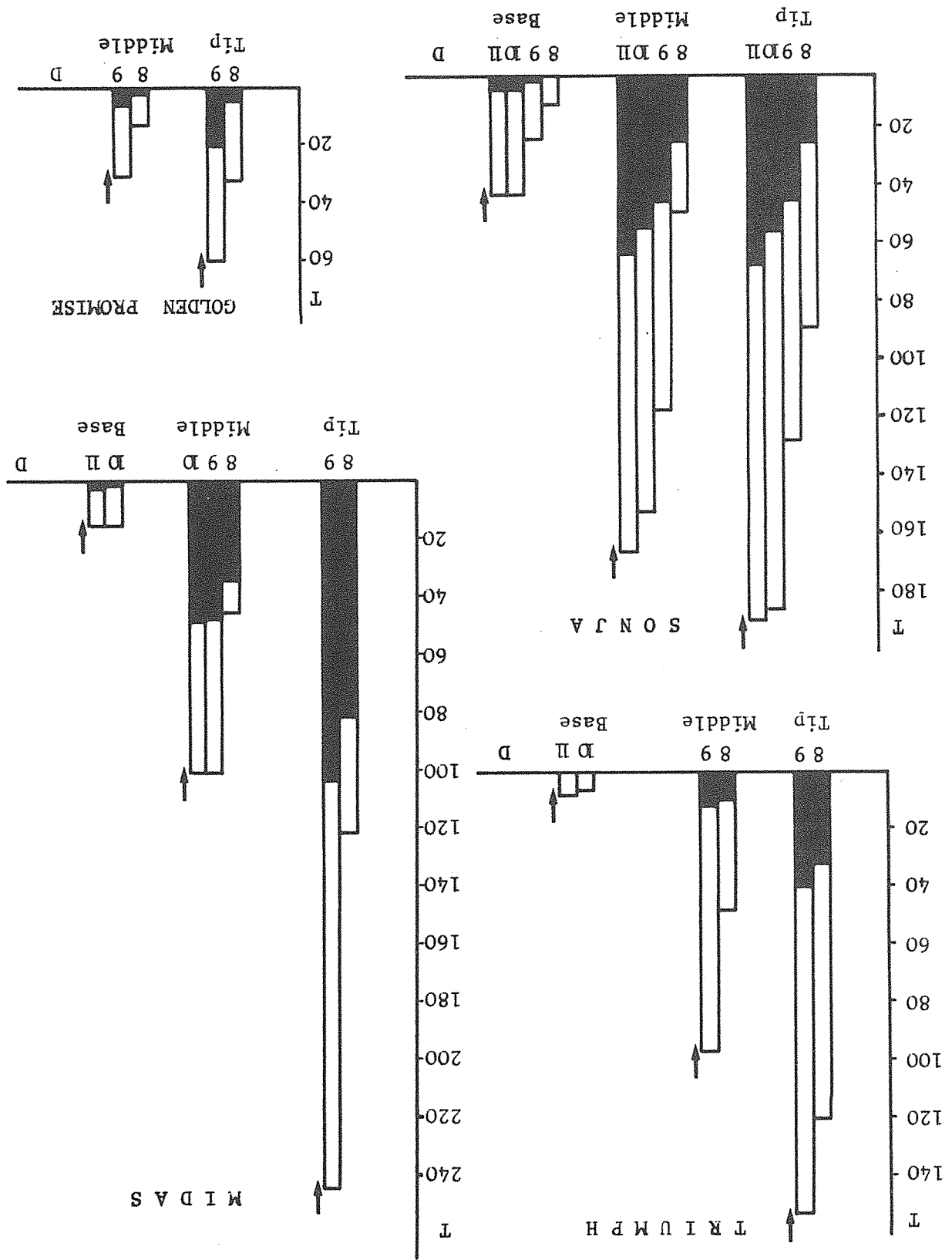


Fig. 1. Course of eruption of uredinia in the basal region of the second leaf of four cultivars of barley infected by race 673 of *Puccinia hordei*.

Fig. 2. Production of telia on different parts of the second leaf of four cultivars of barley infected by *Puccinia hordei* race 673.



VIRULENCE ANALYSIS OF LOCAL POPULATIONS OF Puccinia striiformis f. sp.

TRITICI

BY

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INTRODUCTION

Race surveys on a large scale, involving different locations,

different cultivars or different times, indicate that populations of rust

fungi are polymorphic for virulence genes. In such surveys, single isolates

are often collected at each site visited, and sites may be distant from each

other. While such data give a broad picture of the race structure of the

pathogen population they give little information on the variability of local

populations, such as within a single focus of infection or infected field.

The occasional reports of race mixtures in isolates collected in surveys

(Dinooor, 1973; Johnson and Taylor, 1975; Priestley and Byford, 1980) indicate

the possibility of greater variability in local populations than might be

inferred from large scale surveys. This study was designed to provide

information on the composition of local populations of Puccinia striiformis

Westend. f. sp. tritici ERIKSS (yellow rust of wheat) by taking several

samples within small infected areas at each location sampled.

METHODS

Single wheat leaves naturally infected with yellow rust were collected

in 1979 and 1980 from five locations in East Anglia and one in Scotland

(Table 1). Each location consisted of a field of one cultivar or a group of

trial plots of several different cultivars. The samples within a location

included separate leaves from the same plant, separate plants from the same

infection focus or plot and separate infection foci or plots from the same

location.

Spores from the leaves sampled were transferred to seedlings of the

susceptible cultivar Lemhi using alcohol-sterilised fine hair brushes. The

seedlings were enclosed in plastic bags and incubated at 8°C for 36 to 48

hours before being transferred to an isolated plant propagator in a cooled

glasshouse (Jenkyu, Hirst and King, 1973). Seedlings infected with spores

from different leaves were kept separate throughout. Spores were harvested

from these seedlings using an alcohol-sterilised cyclone spore collector

(Terwet and Cassell, 1951) and were further multiplied by reinfection of

fresh seedlings. For storage, freshly produced spores were dried for 24

hours over 50% (v/v) sulphuric acid and then placed in gelatin capsules in a

vapour phase liquid nitrogen refrigerator (Johnson and Taylor, 1974). A

batch of spores derived from a single leaf collected in the field constituted

an isolate.

Isolates were each used to inoculate seedlings of the 15 differential

host cultivars of Johnson, et al. (1972), plus the additional differentials

Kiebesel 47/51 and Triticum spelta. All seedlings were grown in a

spore-proof glasshouse before inoculation and placed on an isolated plant

propagator after inoculation. Four seedlings of each of eight differentials

Isolates were not established from all leaves collected and consequently the number of isolates tested varied from location to location and from plot to plot within a location. Altogether 88 isolates were tested with the number from each location varying from 6 to 41 (Table 1). For each location, however, the isolates tested represented all levels of sampling. All isolates from locations 1, 3, 4 and 6 were classified as race 41 E 136 (Johnson et al., 1972), suggesting not only homogeneity within each location but also between these locations (Table 1). This uniformity is not simply explained by 41 E 136 being the only race capable of attacking the cultivars sampled; other races virulent on these cultivars occur in the U.K. (Taylor, Smith and Johnson, 1981). Domination of the wheat yellow rust population in the U.K. by race 41 E 136 is also apparent from the cereal pathogen virulence surveys conducted around the same period (Priestley and Byford, 1979, 1980; Bayles and Priestley, 1983). The identification of the three isolates from Argent (Location 4) as race 41 E 136 was unexpected since this race does not possess virulence to the resistance gene Yr6 carried by Argent. However, the race classification was clear, with these isolates avirulent in the seedling tests on Heines Kolben which differentiates Yr6 virulence. This isolation of a non-matching race could be explained by Yr6 not being strongly expressed either in the Argent background or under the environmental conditions at the time of sampling or by impurity of the Argent seed.

The samples from the other two locations indicated heterogeneity of the local populations. Two races, 41 E 136 and 108 E 141, were recovered at location 5. However, their distribution was clearly related to the cultivar sampled and independent isolates from the same cultivar were always homogeneous (Table 1). This non-random distribution of the two races can be explained by race-specific resistance factors carried by the four cultivars. Virulence carries the resistance gene Yr1 to which 41 E 136 is virulent but 108 E 141 is not, while Kinsman carries Yr6 for which the reverse applies (Taylor et al., 1981). Hobbit and Kador are both susceptible to both races at the seedling stage (Taylor et al., 1981 and unpublished). Johnson and Taylor (1976) reported that Hobbit possessed race-specific adult plant resistance due to a component subsequently designated R14 (Priestley, 1978). Unpublished data from field trials in 1978 suggested that Kador also possessed R14 and this is supported by data of Priestley and Byford (1979). At the time of sampling, pathogenicity for R14 was detected in isolates of race 41 E 136 (the virulence combination WYV 1, 2, 3) but not in isolates of race 108 E 141 (the virulence combination WYV 2, 3, 4, 6) (Priestley and Byford, 1980).

The evidence for heterogeneity within location 2 is of a different nature. All eight isolates from this location gave type 4 reactions on the differentials susceptible to race 41 E 136 but differed from this race in producing intermediate reactions (type 2 or 3) or variable reactions within single leaves on Suwon 92 x Omar, Heines Kolben, Lee, Heines Peko, Reichenberg 42 and Hybrid 46, which are resistant to 41 E 136. This reaction pattern did not correspond to any of the common U.K. races and it

RESULTS AND DISCUSSION

were arranged in a single pot and another eight similarly arranged in a second pot. Seedlings of Nord Desprez were grown in a third pot to serve as both a differential test and to multiply the spores; all isolates common in the U.K. at the time of sampling were pathogenic on this cultivar. The seedlings were all inoculated simultaneously with the same isolate on a laminar flow bench. The reaction types developed on the seedlings were scored on a 0 to 4 scale with 0, 1, 2 indicating avirulence and 3, 4 indicating virulence (Stakman and Levine, 1922). From the pattern of virulent and avirulent reactions on these differentials the isolates were classified into races (Johnson et al., 1972).

REFERENCES

We thank the staff of the Plant Breeding Institute Rust Laboratory for much practical assistance, especially in multiplying up some of the field isolates. One of us (A.C.N.) gratefully acknowledges receipt of a CASR studentship from the Science and Engineering Research Council.

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Seemingly possible that the isolates from location 2 were mixtures of two or more races. To test this hypothesis, 60 single uredospore isolations were attempted from one of the original isolates following the method of Johnson, Taylor and Smith (1981). Unfortunately, the majority of these isolations failed and only three single spore isolations were established. However, when tested on the differential series these three isolates gave the characteristic reactions of race 41 E 136 with no sign of the intermediate reactions shown by the original isolates on the resistant differentials. This indicates that race 41 E 136 was present and that the unusual reactions on cultivars resistant to this race were probably due to its mixture with at least one other race in the original isolates. Natural mixed infections of wheat yellow rust have been previously reported and in all cases race 41 E 136 was a component (Johnson and Taylor, 1975; Priestley and Byford 1980). Considering the general predominance of this race in the U.K. population at the time (Priestley and Byford, 1980), no special significance can be attached to its involvement in all mixed infections described. Assuming that the population of yellow rust at location 2 was polymorphic, it is surprising that all eight single leaf isolates appeared mixed. The absence of pure isolates of the components, especially of the dominant race 41 E 136, indicates an intimate mixture. Brown and Sharp (1970) found that experimental mixtures of races of *P. striiformis* persisted through several generations with one race maintained at a much lower level than the other. Despite the attempt at intensive sampling, practical constraints restricted the number of isolates that could be tested and this will have limited the detection of less common races. For example, if ten isolates are collected at a site and all classified the same, the 95 percent confidence interval for the frequency of other undetected races lies between 0 and 30 percent (see Campbell, 1974). Clearly this allows considerable scope for undetected variation. However, if we consider the 72 isolates that were collected from sites at which only race 41 E 136 was found, we can say, with 95 percent confidence, that the population could not have contained more than 5 percent of other unobserved races. Thus, taken together, the results suggest that the local populations of *P. striiformis* on wheat sampled were generally dominated by one race and may often have been monomorphic. Seeding virulence is only one aspect of the pathogen's genotype and it is possible that the populations examined were polymorphic for some other characters, or for virulence genes not included in these particular tests. However, a more diverse collection of isolates of wheat yellow rust than those used in the present study all possessed the same isozyme and double-stranded RNA phenotypes as revealed by gel electrophoresis (Newton, Caten and Johnson, 1985). Together these studies suggest that populations of *P. striiformis* f. sp. *tritici* possess a high degree of genetic uniformity both within and between locations. Polymorphism can be detected only in those genes which interact directly with the host and even here the variation is limited, as might be expected from the organism's asexual mode of reproduction.

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TABLE 1 - Origin and race classification of field isolates of Puccinia striiformis f. sp. tritici

Location	Host			Year	Number of isolates	Race classification				
	Cultivar	Yr genes	R. factors			41 E	136 E	108 E	141 Other	Mixture
1. Newton, Cambs.	Kador		14	1979	8	8	-	-	-	-
2. Royston, Herts.	Hobbit	3a,4a	14	1979	8	-	-	-	-	8
3. Stowmarket, Cambs.	Score		14	1979	6	6	-	-	-	-
4. Trumpington, Cambs.	Argent	1,3a,4a,6		1980	3	3	-	-	-	-
	Maris Templar	1,3a,4a		1980	12	12	-	-	-	-
5. Terrington St.	Virtue	1,3a,4a	13	1980	4	4	-	-	-	-
Clements, Norfolk	Hobbit	3a,4a	14	1980	19	19	-	-	-	-
	Kinsman	3a,4a,6	13	1980	8	-	8	-	-	-
	Kador		14	1980	10	10	-	-	-	-
6. Pentlandfield, Midlothian	Maris Huntsman	2,3a,4a	13	1979	10	10	-	-	-	-

PATTERN OF VIRULENCE IN Puccinia Graminis f. sp. tritici IN INDIA

BY

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Virulence surveys of wheat rusts in India have been carried out annually since 1931 (Mehta, 1940) and became more intensive in 1967 after the introduction of dwarf wheat (Joshi, 1976). Stem rust urediniospores move from Nilgiri and Pulney hills with cyclonic disturbances along the Puccinia path (Nagarajan and Singh, 1974) and damage the wheat crop every year. The analysis of virulence survey data for the past 50 years revealed that virulence for the stem rust resistance gene Sr9e had been common since the initiation of virulence survey in India (Bahadur, 1984) although gene Sr9e was not in commercial cultivars. After the cultivation of dwarf wheat, races virulent for Sr9e acquired additional virulence for gene Sr11t in 1974 and 1977 (Bahadur et al., 1982).

Wheat is primarily a rabi crop (winter) and is grown between October to June. The duration of crop becomes longer as one moves from south to north. Since races were adapted in certain parts of the country, India was divided into 4 distinct ecological areas. Race 21A-2 (75G5) had been prevalent for the last 15 years in Northern and Central parts of India; race 40A (62G29) for 8 years in Nilgiris and Central India; and virulences 117A (36G2) and 117A-1 (38G18) for 7 years in Karnataka. The available information also shows that genes Sr5, Sr6, Sr7, Sr8, Sr9b, Sr11 and Sr12 are present in Indian cultivars (Sawhney, 1982, Nagarajan et al., 1984).

Vanderplank (1982) suggested that the composition of the fungus population was influenced by the host and temperature. He also suggested that virulence for some resistance genes associates and for other resistance genes dissociates. Based on this information the pattern of variation in the population of Puccinia oraminis tritic in India between 1975-83 was analysed (Table 1). The data revealed that virulence for all the stem rust resistance genes (virulence/virulence combination of Sr5, Sr6, Sr7, Sr8, Sr9b, Sr9e, Sr12) was prevalent in 89% of the pathogen population in Nilgiris, the source area for stem rust spread, while in Northern India virulence/virulence combination Sr5, Sr7, Sr8, Sr9e, Sr11 / Sr6, Sr9b, Sr12 was prevalent (87%) during the same period. After 1982, the number of races had further declined to 4 and 94% of isolates belong to Sr5, Sr7, Sr8, Sr9e, Sr11 / Sr6, Sr9b, Sr12 avirulence/virulence combination in Northern India. The data revealed that virulence remains associated in the larger population of P. graminis tritic in Nilgiris and gets dissociated in Northern India. The pattern of variation may be due to host resistance and agroecological conditions. Vanderplank (1982) classified genes Sr6, Sr9a, Sr9b and Sr15 in ABC group and genes Sr7b, Sr9d, Sr9e, Sr10, Sr11, Sr11t-1, Sr11t-2 in XYZ group based on their effectiveness and also concluded that other stem rust resistance genes belong to the neutral group. The analysis in India revealed that genes Sr5 and Sr8 are effective and gene Sr12 ineffective in Northern India and does not belong to the category of neutral genes. Based on rust survey data of United States for 1975 to 1980, Knott (1984) also pointed out that genes Sr5, Sr8 and Sr13 are not neutral and almost all races were virulent on Sr5 and Sr8 while none was virulent on Sr13. Roelfs and Martens (1984) also concluded that the concept of matching genes (Vanderplank, 1982) may have some merit and in Avena-Puccinia graminis system and some helpful inferences might be made.

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TABLE 1 - Reaction of isolates of *Puccinia graminis tritici* for wheat stem rust resistance genes Sr5, Sr6, Sr7, Sr8, Sr9b, Sr9e, Sr11 and Sr12 in Northern India, Central India, Karnataka and Nilgiris in 1975-1983

Effective genes	Ineffective genes	Number of isolates	Percentage of total isolates
Northern India			
Sr5, 7, 8, 9e, 11	Sr6, 9b, 12	415a	86.8
Sr5, 7, 8, 9b, 9e, 11, 12	Sr6, 12	21b	4.3
Sr5, 7, 8, 11	Sr5, 6, 7, 8, 9b, 12	10c	2.1
Sr5, 8, 9b, 12	Sr6, 7, 9e, 11	11d	2.3
Others	-	21	4.5
Central India			
Sr5, 7, 8, 9e, 11	Sr6, 9b, 12	359	67.7
Sr5, 8, 5b, 12	Sr6, 7, 9e, 11	53	10.0
Sr5, 7, 8	Sr6, 9b, 9e, 11, 12	30e	5.6
Sr5, 7, 5, 9b, 9e, 11, 12	Sr6, 12	26	4.9
Others	AI1	25f	4.3
Sr9e, 11	Sr5, 7, 6, 8, 9b, 12	16	3.0
Others	-	23	4.5
Karnataka			
Sr5, 7, 8	Sr6, 9b, 9e, 11, 12	101	44.2
Sr5, 8, 9b, 12	Sr6, 7, 9e, 11	81	35.5
Sr5, 7, 8, 9e, 11	Sr6, 9b, 12	35	15.3
Others	AI1	9	3.9
Others	-	2	1.1
Nilgiris*			
Sr5, 6, 7, 8, 9b, 9e, 11, 12	Sr5, 6, 7, 8, 9b, 12	280	88.8
Sr5, 8, 9b, 12	Sr6, 7, 9e, 11	22	6.9
Sr9e, 11	Sr5, 6, 7, 8, 9b, 12	5	1.5
Others	-	8	2.8

* Isolates from Summer Nursery (Old race numbers are given in parenthesis)

- a - race 75G5 (21A-2)
- b - race 73G7 (17)
- c - race 26G13 (34)
- d - race 36G2 (117A)
- e - race 38G18 (117A-1)
- f - race 62G29 (40A)

SOURCES OF RESISTANCE TO STEM RUST OF WHEAT (*Puccinia graminis* f. sp.

tritici)

BY

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SUMMARY

The study showed that entries CPAN-1946, CPAN-1989, CPAN-1994, CPAN-2005, HD-2278, HW-741 and VL-614 were good sources of resistance against stem rust (*Puccinia graminis* f. sp. tritici) of wheat as they conferred resistance in seedlings as well as adult plants. Their use in breeding programmes for disease resistance is recommended.

INTRODUCTION

Stem rust of wheat (*Puccinia graminis* Pers f. sp. tritici Erikss and Hen) is potentially an important disease in India particularly in the Peninsular wheat growing zone. There is evidence of the occurrence of epidemics of stem rust as early as 1786 which destroyed the wheat crop on thousands of hectares in India (Nagarajan and Joshi 1975). Recently the incidence has declined due to extensive cultivation of rust resistant varieties. Breeding for disease resistance cannot be pursued efficiently without knowledge of the genetic basis of resistance and availability of effective sources of resistance. Screening of genetic resources is therefore an important aspect in developing new varieties possessing good agronomic characters combined with good disease resistance.

MATERIALS AND METHODS

Twenty five entries of wheat were sown in the field at the wheat Rust Research Station, Mahabaleshwar during Rabi 1984-85; each entry was represented by one row of 1 m length. The inoculum of a mixture of races of black rust was sprayed onto the plants to create artificial epiphytotic. The rust reactions were recorded at late boot stage using the modified Cobbs scale. The average temperature during the crop growth was between Min 8.7°C, Max 26.6°C with 57% relative humidity. The seedlings of the entries were grown in 4" diameter earthen pots in the glass-house. Seven day old seedlings were inoculated with the inoculum of individual races and biotypes of stem rust (*Puccinia graminis* f. sp. tritici). After inoculation the plants were exposed to high humidity in a moist chamber for 24 hrs and then transferred to the glass-house for disease development. The temperatures and humidity prevailing during the test period in the glass-house were Min 18.7°C, Max 37.4°C and 65% relative humidity. Rustule type was recorded using the key developed by Stakman and Levine (1922).

RESULTS AND DISCUSSION

The seedling and adult plant reactions are presented in Table 1. The percentage of the entries tested is listed in Table 2.

The entries CPAN-1889, CPAN-1946, CPAN-1994, CPAN-2005, HD-2278, HW-741, VL-614 have shown resistance to all the races and biotypes in seedling and adult plant stages.

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The entries CC-493, CPAN-1933, CPAN-1976, HUW-206 and HW-971 although susceptible to a few races as seedlings exhibited adult plant resistance. It is interesting to note that the entries CPAN-1830, 1884, 1885, 1922, 1929, 1967, 1973, HD-2315, VL-490 though resistant to all the races and biotypes in the seedling stage were susceptible in the adult plant stage indicating that entries having resistance at seedling stage to all races do not necessarily possess adult plant resistance and vice versa and therefore, it may be concluded that seedling resistance and adult plant resistance are quite distinct. Golden et al. (1928) showed that adult plant resistance was inherited independently of seedling resistance.

The entries CPAN-1869, 1951, and HW-840 were susceptible at seedling and adult plant stages.

TABLE 1 - Seedling and adult plant reactions of the varieties against stem rust of wheat

Varieties	11	14	15	15C	21	21A1	24	Races				117A	122	184	295	Adult Plant reactions against mixture of races
								24A	34	40	40A					
1 CPAN 1830	0,	0	0	2	0	0	0	0	0	0	2	2	0	2	TrS	
2 CPAN 1929	2++	1	2	2	0,	0	0	0	1-2	0	0	2	0	2	30S	
3 CPAN 1933	0	2	0	2++	0	0	2	0	0	1	2	1-2	3	2	0	
4 CPAN 1946	0	0	0	0	0	0	0	0	0	0	0	1	0,	0	0	
5 CPAN 1967	0,	0	0	0	0	0	0,	1	0,-1	0,	0	2	0	0	TrS	
6 CPAN 1869	1	0	2	1	2	1-2	0	0	0,	0,	1-2	2	3	1	15S	
7 CPAN 1884	0	0,	0	0,	0	0	0	0	0	0	2+	1-2	0	0	10S	
8 CPAN 1885	0	0	0	0,	0	0	0,-1	0	0	2	1-2	2+	0	2	10MS	
9 CPAN 1922	1	0	0,	1	2	0,-1	2	-	0,	1	1	2	0	1	40X	
10 CPAN 1951	0	2	0	2	1	0,-1	2	0	0	2	2	2	2++	3	20MS	
11 CPAN 1973	1	0	1-2	1-2	0,	0,	0	2	2	1	0,	2	0	2	5MS	
12 CPAN 1994	1	0,	0	2	0,	0,	0	2	2+	1	0	1-2	0	2	0	
13 CPAN 2005	1	2	2	2	0,-1	0	2+	0	0	1	2	2	1	2	20MR	
14 CPAN 1676	3	2	0	-	3	0	-	2	2-3	3	2	1	1	4	30MR	
15 CPAN 1889	2	0	-	-	0,	0	1	0	0	0	0	0	0	0	0	
16 HW 741	0	0	0	2+	0,	0,	1	1-2	0	1	2	0,-1	0	1	15MR	
17 HW 840	4	0	3-4	2	0	0	0	4	4	4	0	0	0	3	50S	
18 CC 493	2	2+	1	2	2	0	2	1	1-2	2	0	2	3	2	10MR	
19 HD 2278	0	0	0	2	0	0	2	0	0	0	2	2	0,	2	0	
20 VL 490	1	0	2	1+	2	1	2	2	2	2	1-2	2	0	1	30S	
21 HD 2315	1	1	0	2++	2	0,-1	1-2	1	1	0	0	2	1	1	30S	
22 VL 614	0	0,	2	-	1	2	-	0	0	2	2	2	-	-	5MR	
23 HUW 206	0	2	0	1	-	2	2	2	2	2	2	0	3+	2	0	
24 HW 971	0	2	4	2	0	0,	2	1	0	1-2	2	2	0	0,	10MR	
25 WG 2109	0	1	0	1	1	0	2	0	0	0	2	2	1	0	20MS	

1	CPAN 1830	Tob-Era/Tob x Gno
2	CPAN 1929	Jup 73-Zp "S" x Coc 75
3	CPAN 1933	Patu/Son 64-Pdue x Gno-India) HD 832-Bb
4	CPAN 1946	Kawko "S"
5	CPAN 1967	(Bb-Gno x India-Saty/Sparrow"S") Pavon "S"
6	CPAN 1869	(Mo/N-K 117axFr-KAD/Gb)Fr-KADxGb)Yr)Bb-Ron x Maya 74
7	CPAN 1884	Polk/Tob 66/2Fletcher, Mn 70113
8	CPAN 1885	Era/Chris Mutant (ECM 403)
9	CPAN 1922	Ore Fl 158-Fdl x Mexifen "S"-Tib 632/Coc75
10	CPAN 1951	Forlant-Acciaio x An 75, SWM 4575
11	CPAN 1973	Sunbird "S"
12	CPAN 1994	Sunbird "S"
13	CPAN 2005	Rsk - Mo x Emu "S"
14	CPAN 1676	Bon x Gno-Son64/Kal-Bb
15	CPAN 1889	Timagalen/2/Bajio/Kastell, W 5864-4m-2-Lm-W131
16	WG 2109	Ravi 43 x HD 2177
17	HW 741	Bb-cc/Gno-No.66 x Pl 62
18	HW 840	Tob 66 x TR 260
19	HW 971	Pl18 x HW 517
20	HUW 206	(KVZ x Bu40) x (Kal x Bb)
21	VL 490	NS 879/4 x Girlja
22	VL 614	HD 2009 x CPAN 1283
23	HD 2315	HD 2160 x Tob-Gno-23584/Na1 60TT-Son 64/HD 1954
24	HD 2278	HD 2119 x 247
25	CC 493	Pato (B)-7c x Gno "S" - Gallow, 2D-OW

TABLE 2 - Percentage of the entries tested against stem rust of wheat

VARIETAL REACTIONS OF RYE TO WHEAT STEM RUST, RYE STEM RUST, WHEAT LEAF RUST AND RYE LEAF RUST

BY

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SUMMARY

The reaction of the rye cultivars 'Dankovske Nove', 'Danae', 'Breno', 'Kustro' to wheat stem rust, wheat leaf rust, rye stem rust and rye leaf rust was studied.

All cultivars contained a certain proportion of plants susceptible to wheat stem rust, wheat leaf rust and were susceptible to rye stem rust and rye leaf rust. In the glasshouse three plants of the cultivar 'Dankovske Nove' resistant to the rye stem rust were selected.

INTRODUCTION

In Czechoslovakia, rye is affected by rye stem rust (*Puccinia graminis* Pers., subsp. *graminis* f. sp. *secalis*), rye leaf rust (*Puccinia recondita* Pers., subsp. *graminis* f. sp. *tritici*) and wheat leaf rust (*Puccinia graminis* Pers., subsp. *graminis* f. sp. *tritici* var. *triticina*/Eriks./Urban et Markova).
Tan, Luig and Watson (1976) mentioned infection of some rye plants by wheat stem rust and explained it by the heterogeneity of rye cultivars given by cross pollination of this crop.

During the field infection trials with wheat stem rust, some rye plants affected by this rust were often detected in the neighbourhood. Isolates of this rust were tested on the standard differentials for testing wheat stem rust and in all cases the race identified was always the same as that used for wheat inoculation in the field trial.

The aim of all trials described in this paper was to determine in different rye cultivars the proportion of plants susceptible to wheat stem rust and wheat leaf rust, which can also affect some rye plants, as well as the reactions of the cultivars to rye stem rust and rye leaf rust.

MATERIALS AND METHODS

The rye cultivars used in these trials were as follows: 'Dankovske Nove' (Poland), 'Breno' (Czechoslovak Socialist Republic), 'Kustro' (German Democratic Republic) and 'Danae' (German Democratic Republic). Wheat stem rust, race 34 (isolate 334) and wheat leaf rust, races 61 and 61 Saba, were propagated on the wheat cultivar 'Little Club'. Rye stem rust was obtained either from couch grass or from rye and propagated on the rye cultivar 'Lankovske Nove'. Rye leaf rust was collected in a rye field. No detailed determination of these isolates was performed.

In the field trials a strong infection pressure was produced by inoculating a susceptible cultivar planted as spreader rows in the field. In the glasshouse trials the moistened first leaf was infected by the application of uredospores with talc and subsequent incubation under high atmospheric humidity in glass cylinders for 48 hrs. The reaction was evaluated by means of infection types according to Stakman et al. (1962). The seed material of the rye cultivars originated from the rye collection of

the Research Institute of Crop Production, Praha - Kuzyne, previously obtained from the Central Agricultural Control and Testing Institute where it was used for state yield tests.

RESULTS

Wheat Stem Rust
In the glasshouse trial with the cultivars 'Breno', 'Kustro' and 'Danae', the proportion of susceptible plants was approximately 7% (Table 1). The proportion of susceptible plants in the progenies of healthy and susceptible plants was studied both in the field and glasshouse trials. In the cultivar 'Dankovske Nove', tested in the glasshouse, the open pollinated progeny of healthy plants contained 1% of susceptible plants, whereas the open pollinated progeny of susceptible plants was 12.6%. For the cultivar 'Breno', tested in the glasshouse, the open pollinated progeny of the healthy ears contained 10.9% of susceptible plants, whereas the open pollinated progeny of susceptible ears 40.6%. Similar results, suggesting considerable variability in the proportion of susceptible plants at free open pollination, were obtained with adult plants tested under field conditions (Table 2). In most cases, the differentiation of resistant and susceptible plants was easy because of the prevailing segregation into IT (infection type) 0;1 and 3. Only in the progeny of the cultivar 'Breno' there were about 20% plants with intermediate reaction of IT 2CH and 2-3.

Wheat Leaf Rust
The seedling reactions to wheat leaf rust, races 61 and 61 Saba, of the cultivars 'Dankovske Nove' and 'Breno' are given in Table 3. Most plants were resistant but susceptible plants with IT 3 were also observed. In contrast to wheat stem rust, a whole range of infection types was detected. With respect to spontaneous occurrence of rye leaf rust and its similarity to wheat leaf rust the field trials with adult plants were not feasible.

Rye Leaf Rust
All plants of the tested cultivars ('Dankovske Nove' : 537 plants, 'Breno' : 190 plants, 'Danae' : 62 plants) inoculated with the rye leaf rust in the seedling stage were susceptible.

Rye Stem Rust
All studied cultivars were susceptible to rye stem rust originating from couch grass. In the glasshouse test only three out of 404 plants of the cultivar 'Dankovske Nove' (less than 1%, had a resistant reaction (IT;1-2). In rye breeding, these plants could serve as a source of resistance to rye stem rust and therefore they were vernalized and sown out in the field to obtain the progeny. They were not isolated from other rye plots, so that pollination with susceptible plants was probable. In the progeny of these three plants there were 52.3%, 50.9% and 37.2% of plants resistant (IT;1) to rye stem rust.

DISCUSSION

As shown by the trials, the rye cultivars grown in Czechoslovakia contain a certain proportion of individuals susceptible to wheat stem rust. The progenies of susceptible plants (ears) had always a higher proportion of susceptible plants. However, because of free pollination it was difficult to judge the genetic base of resistance to wheat stem rust. The prevailing infection types 0;1 and 3, detected mostly in the segregating progenies, suggest that the resistance is based monogenically or oligogenically. This observation is in accordance with the data given by Tan, Luig and Watson

(1977), who proved the oligogenic inheritance of the studied rye species to wheat stem rust and described eight different genes for resistance. As proved by the tests with wheat leaf rust, a certain proportion of rye plants showed susceptibility to wheat leaf rust as well. In contrast to wheat stem rust, the inoculated samples were characterized by continuous variation of infection types from resistant to susceptible. This indicates that resistance is obviously not controlled only by one dominant gene. Musa, Dyck and Samborski (1984) found with several inbred rye lines that their resistance to wheat leaf rust was controlled by one to three genes. Nevertheless, even in the trials performed by these authors segregation included a wide range of infection types. A parallel test (Table 3) with race 61, avirulent to gene Lr26 (transferred to wheat from rye chromosome 1R) and its biotype 61Saba, virulent to this gene, revealed that the cultivars 'Dankovske Nove' and 'Breno' either do not contain this gene or contain some additional gene (genes) effective also to the isolate virulent to the gene Lr26.

The test carried out simultaneously with stem rust and leaf rust on the same plants showed that genes in rye for wheat stem rust resistance and wheat leaf rust resistance were not linked.

The trials with rye stem rust showed the susceptibility of all tested cultivars to this type of rust, but also the possibility of selection of the resistant plants, at least in the case of the cultivar 'Dankovske Nove'. A clearcut segregation into resistant and susceptible plants in the progenies of rye plants resistant to rye stem rust suggests major gene(s) effect. Tan, Luig and Watson (1976) detected in four inbred rye lines originating from the USA six different genes for resistance to rye stem rust. A comparison with the resistance gene(s) of plants originating from the cultivar 'Dankovske Nove' would require the creation of inbred lines from these plants.

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TABLE 1 - The proportion of plants susceptible to wheat stem rust race 34, in three rye cultivars in the 1st leaf stage

Cultivar	Number of plants		% of susceptible plants
	Resistant	Susceptible	
Breno	255	18	6,6
Kustro	76	6	7,3
Danae	54	4	7,4

TABLE 2 - The proportion of plants susceptible to wheat stem rust, race 11, in four rye cultivars - field trial (R = resistant, S = susceptible)

Cultivar	Progeny plants/ears	Number of plants		% of susceptible plants
		R	S	
Dankovske Nove	R 190 S 252	190	225	0
Breno	R 178 S 146	170	80	4,5
Kustro	R 104 S 52	90	14	13,5
Danae	R 24 S 81	15	17	37,5

TABLE 3 - The reaction of two rye cultivars to two races of wheat leaf rust - glasshouse trial

Cultivar	Race	Total Number of Plants	Number of plants with infection type						
			0	1	1-2	2	2-3		
Dankovske Nove	61Saba	109	50	42	11	3	1	2	0
Breno	61Saba	113	10	15	22	11	26	14	15
Dankovske Nove	61	114	40	33	20	4	8	5	4
Breno	61	106	51	40	10	1	2	1	1

EFFECTIVENESS OF GENES FOR RESISTANCE TO LEAF RUST OF WHEAT IN MOROCCO

BY

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ABSTRACT

Twenty four genes for resistance to wheat leaf rust were evaluated under natural epiphytotic conditions at 3 locations in Morocco. The best genes were Lr19, Lr9 and Lr24, followed by Lr3Ka, Lr18, Lr13, Lr14a, Lr15, Lr20 and Lr29. Genes Lr21, Lr22, Lr23 and Lr12 were intermediate in effectiveness. All other genes (Lr1, Lr2a, Lr2b, Lr2c, Lr3, Lr10, Lr14b, Lr16, Lr17 and Lr25) were more or less ineffective.

INTRODUCTION

Leaf rust, Puccinia recondita f. sp. tritici, is the most important rust species on wheat in Morocco. The pathogen usually infects wheat beginning in early spring and continuing until maturity of the crop. Although leaf rust epidemics are seldom spectacular, the disease causes appreciable damage through the yearly 'erosion' of yield. All popular wheat cultivars in the country, both durum and aestivum, are susceptible to the disease.

The genetics of leaf rust resistance has been extensively studied (1, 2, 7, 8, 9, 11, 18). Knowledge of the genetics of resistance is useful in reducing empiricism involved in rust breeding programs and in hastening the development of resistant cultivars (18). Interactions of wheat and the leaf rust pathogen have been shown to operate in a gene-for-gene relationship. Genes for leaf rust resistance (Lr) can thus protect wheat plants from strains of the pathogen population that lack the corresponding genes for virulence (5). Many Lr genes have been identified (1, 9, 10, 11, 12, 18). Lr genes are furthermore available in 'monogenic' lines (MG), developed by incorporating individual genes into a susceptible background - mostly spring wheat CV Thatcher - (5). MG lines are ideal for testing individual genes in specific geographic areas in order to identify effective genes for use in breeding programs. The objective of the present study was to evaluate the effectiveness of the available genes for leaf rust resistance in Morocco.

MATERIALS AND METHODS

Monogenic lines were planted during the 1983-84 season in 3 locations: Rabat, Sidi Kacem (120 km northeast of Rabat) and Khmis Zmamra (250 km southwest of Rabat). Twentyfour genes were tested. CV Thatcher was used as the susceptible check. Three local cultivars (Fartas 3, Fartas 5, Algerian F) known for their high susceptibility were also included as checks in Rabat. Disease scores were taken at flowering on the basis of infection type and disease severity.

RESULTS AND DISCUSSION

Genes Lr19, Lr9 and Lr24. Lr19 conferred immunity to the disease in all 3 locations. Lr9 conferred immunity in 2 locations and high resistance in the 3rd. Lr24 was immune in one location and highly resistant in 2 others. All

3 genes were derived from intergeneric crosses, Lr19 and 24 originate from Agropyron elongatum and Lr9 from Aegilops umbellulata (5). Other effective genes were Lr3Ka, 18, 13, 14a, 15, 20 and 29. Genes Lr3Ka and 18 conferred immunity in one location and high resistance in a 2nd, genes Lr13 and 14a conferred high resistance in 2 locations whereas genes Lr15, 20 and 29 were evaluated in only one location where they conferred high resistance. Genes Lr21, 22, 23 and 12 were intermediate in effectiveness; all conferred high resistance in one location only and moderate resistance; moderate susceptibility in the others. All other genes - Lr1, 2a, 2b, 2c, 3, 10, 14b, 16, 17 and 25 - were ineffective, although most were less susceptible than the checks. Two of the local cultivars used as susceptible checks were more susceptible than Thatcher. In spite of their high susceptibility, these cultivars are widely grown in the southern 'saharan' parts of the country where leaf rust is not a problem. The genes found most effective in this study - Lr9 and 19 - have been reported as resistant in other parts of the world (13, 14, 17). However, susceptible readings have also been reported (16, 17), thus indicating the existence of races capable of overcoming these genes. The same applies for other genes found resistant here; instances of both resistance (14, 17) and susceptibility (13, 14) have been reported. These differences are due to the occurrence of distinct races of the wheat leaf rust fungus throughout the world (13, 17). Assuming that the most prevalent races of the leaf rust pathogen in Morocco were present in the 3 trial locations, the genes found effective here could be used to control the disease. The best genes - Lr19, 9, 24 - could be used singly, while genes with intermediate effectiveness could be used in combination to confer complete resistance to all prevalent races of the pathogen.

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TABLE 1 - Evaluation of genes for resistance to wheat leaf rust, P. recondita

Resistance genes		MG line		Location*	
Lr 1	TC6 Centenario = RL 6003	15 X	50 S	Sidi	Zmamra
2a	TC6 Webster = RL 6016	20 MS	40 S	40 S	15 MS
2b	TC6 Carina = RL 6019	40 S	50 S	40 S	20 MS
2c	TC6 Loros = RL 6047	20 S	30 S	20 S	10 MS
3	TC6 Democrat = RL 6002	40 S	20 MS	40 S	20 MS
3Ka	TC6 Antiversario = RL 6007	10 MR	0	0	Tr R
9	TC6 Transfer	0	0	0	5 MR
10	TC6 Exchange = RL 6004	40 S	60 S	40 S	25 S
12	TC6 Exchange = RL 6011	5 R	20 X	20 X	25 MS
13	Mantou type Thatcher	Tr R	**	Tr MR	Tr MR
14a	TC6 Selkirk = RL 6013	40 MS	5 R	40 MS	Tr R
14b	TC6 Maria Escobar = RL 6006	15 MS	40 MS	15 MS	30 MS
15	Kenya 1483	Tr R	-	Tr R	-
16	TC6 Exchange = RL 6005	30 S	20 S	30 S	10 MS
17	TC6 Klein Lucero	15 MS	40 MS	15 MS	20 MS
18	TC6 Africa 43 = RL 6009	10 MR	Tr R	10 MR	0
19	TC6-T4 = RL 6040	0	0	0	0
20	Axminster	Tr R	-	Tr R	-
21	TC6-RL5406 = RL 6043	Tr MR	10 MR	10 MR	10 R
22	TC6-RL504 = RL 6044	10 MR	10 MS	10 MR	Tr R
23	TC6-Lee 310 = RL 6012	Tr MR	5 MS	Tr MR	15 MS
24	Agent	Tr R	0	Tr R	5 MR
25	Transec	40 MS	-	40 MS	-
29	70 Ag #11 Sears	Tr MR	-	Tr MR	-
0	Thatcher	60 S	50 S	60 S	50 S
0?	Fartas 3	60 S	-	60 S	-
0?	Fartas 5	70 S	-	70 S	-
0?	Algerian F	80 S	-	80 S	-

* First column = disease severity
 Second column = infection type

** No score taken

GENETIC BASIS OF LEAF RUST RESISTANCE IN WHEAT CULTIVAR MEDITERRANEAN

BY

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SUMMARY

Three Sydney University accessions of the standard leaf rust differential, Mediterranean CI 3332 introduced on different occasions from the USA were genetically heterogeneous in leaf rust response. A stock of Mediterranean W1728 selected for use in local pathogenicity surveys possessed Lr2a and Lr3, whereas a further accession W3732 introduced from Canada, possessed only Lr3. Outcrossing with Webster was suggested as a cause of the heterogeneity. Laboratories continuing to use Mediterranean as a differential in pathogenicity surveys should establish the genetic status of the stocks being used.

INTRODUCTION

The wheat cultivars Mediterranean (CI 3332) and Democrat (CI 3384) were selected as standard testers for the study of pathogenic variation in *P. recondita* Rob. ex. Desm. f. sp. *tritici* Erikss and E Henn, (Johnston and Mains, 1932). Their responses to different races are listed in the key of Johnston and Levine (1955) where they are considered to provide differentiation. Soliman et al. (1964) studied the inheritance of resistance in both Mediterranean and Democrat and found that resistance to race 9 was due to the same single gene, located in chromosome 6B. Hagga and Dyck (1973) investigated the inheritance of resistance in Democrat, Bage and Klein Antversario and concluded that resistance alleles in Bage (Lr3bg) and Klein Antversario (Lr3Ka) were allelic or very closely linked with a gene in Democrat (Lr3), indicating a multiple-allelic series at the Lr3 locus. The accession Mediterranean W1728 (W numbers refer to the University of Sydney Wheat Accession Register), used as a differential in pathogenicity surveys for *P. recondita tritici* at the Plant Breeding Institute, Castle Hill, displays low infection types (IT) when inoculated with cultures of races '76' and '104' in contrast to the high ITs of Democrat W977. This difference in response led to a presumption by Plant Breeding Institute staff that Mediterranean possessed an allele of Lr3 differing from that present in Democrat. The present work examined that presumption more closely.

MATERIALS AND METHODS

Various host materials are listed in Table 1. Three different accessions of Mediterranean were available in the wheat collection, viz, W584 (CI 3332-2) introduced from Indiana in 1924, and W974 (CI 3332-3-1) and W1728 (CI 3332) introduced from Kansas in 1932 and 1947, respectively. For recent pathogenicity surveys, W1728 has been used, but seed-lots for survey use have been increased separately from that in the cultivar collection, hence both sources were investigated. Seed of Mediterranean W3732 was supplied in 1980 by Dr P.L. Dyck, Agriculture Canada, Winnipeg, Manitoba, Canada. Mentana (W1124) and Festigay (W2706), which possess Lr3 or an allele, and Lr2a, respectively, and Chinese Spring (Timstein 6B), a seedling susceptible parent, were used in the crossing program.

The *P. recondita* tritic pathotypes (and culture numbers) used in

these studies were:

Democrat avirulent: 10-1,2,3,4 [72469] and 26-1,3 [67028]

Democrat virulent: 76-0 [63666], 104-2,3,6,(7) [76694] and 162-1,2,3,6 [70201]

In addition, two *P. graminis* tritic pathotypes were used, viz.,

34-1,2,3,4,5,6,7 [74-L-1] and 34-1,2,3,6,7,8,9 [76-L-7]. These were chosen

for avirulence and virulence, respectively, on seedlings of Festigay and

Webster with Sr30. The Australian pathotype classification systems are based

on the classical differential set with additional supplementary

differentials. Supplementary differentials for *P. recondita* tritic were

described in Watson and Lutz (1961); in addition, -6 and -7 indicate

virulence on seedlings of 'Gatcher' (Lr27 + Lr31) and Songlen (Lr17),

respectively. The system of pathotype designation for *P. graminis* tritic

was given in McIntosh et al. (1981).

Seedlings were grown in 9 cm pots at the rate of 20-25 per pot for F2

populations and as two clumps per pot for F3 lines. Uredospores suspended in

light mineral oil were atomized over seedlings at the one-to-two leaf stage

which were then incubated for 18-24 hrs in misting chambers, and later

transferred to greenhouse benches. The disease responses were scored on the

basis of infection type, 10-12 days after inoculation.

RESULTS

The infection type responses of the various accessions of

Mediterranean and other wheats, tested with two Democrat-avirulent and three

Democrat-avirulent pathotypes are presented in Table 1. The response pattern

for Mediterranean W3732 was similar to that of Democrat which was adopted as

a standard with Lr3. The selected Mediterranean W1728 used in pathogenicity

surveys displayed IT '1'; with both cultures avirulent on Democrat; however,

with two Democrat-avirulent cultures viz., pathotypes 76-0 and 104-2,3,6,(7),

low ITS '1-' and '1' respectively, were recorded. The third culture was

virulent. On the other hand, the presumably unselected W1728 from wheat

collection and accessions W584 and W975, were heterogeneous in response.

All wheats listed in Table 1 gave high infection types with pathotype

162-1,2,3,6. The response of W1728 appeared to combine those of Democrat

with Lr3 and Webster with Lr2a. Moreover, the genetic heterogeneity that

occurred in W584, W975 and W1728 was consistent with such a postulation.

W584 was uniformly susceptible with pathotype 10-1,2,3,4 and thus appeared to

be heterogeneous for only Lr2a. On the other hand, W975 and W1728 possessed

plants with either and/or both Lr3 and Lr2a. The results with pathotype

26-1,3 which was avirulent for both Lr3 and Lr2a indicated that some plants

of W975 possessed neither gene.

Until recently, Mentana was assumed to have the same Lr3 allele as

Democrat. It is now believed to carry the allele known as Lr3bg present in

cultivar Bage (Lutz, personal communication). The distinctive effect of the

Mentana gene is shown by the IT 'X++3' obtained with pathotype 76-0.

Seedling segregation results for F2 and F3 populations from various

crosses involving Mediterranean W1728 (pathogenicity survey source) are

summarized in Table 2. When tested with the Lr2a-avirulent pathotype

10-1,2,3,4, segregations in the cross Chinese Spring (Timstein 68) /

Mediterranean were in accordance with monogenic ratios. When the same F3

lines were tested with the Lr3-avirulent pathotype 76-0, the line distribution

again conformed with a 1:2:1 ratio. Assuming that the alleles conferring

resistance to 10-1,2,3,4 and 76-0, were Lr3 and Lr2a respectively, the

following genotypic distribution was obtained:

The two genes segregated independently ($\chi^2_{1:2:1:2:4:2:1:2:1} = 7.48, p > 0.3$)

$\frac{Lr3Lr3 \ Lr2Lr2}{Lr3Lr3 \ Lr2Lr2} = 2$	$\frac{Lr2Lr2}{Lr2Lr2} = 4$	$\frac{Lr2Lr2}{Lr2Lr2} = 4$
$\frac{Lr3Lr3 \ Lr2Lr2}{Lr3Lr3 \ Lr2Lr2} = 2$	$\frac{Lr2Lr2}{Lr2Lr2} = 4$	$\frac{Lr2Lr2}{Lr2Lr2} = 4$
$\frac{Lr3Lr3 \ Lr2Lr2}{Lr3Lr3 \ Lr2Lr2} = 6$	$\frac{Lr2Lr2}{Lr2Lr2} = 20$	$\frac{Lr2Lr2}{Lr2Lr2} = 4$
$\frac{Lr3Lr3 \ Lr2Lr2}{Lr3Lr3 \ Lr2Lr2} = 6$	$\frac{Lr2Lr2}{Lr2Lr2} = 9$	$\frac{Lr2Lr2}{Lr2Lr2} = 2$
$\frac{Lr3Lr3 \ Lr2Lr2}{Lr3Lr3 \ Lr2Lr2} = 3$	$\frac{Lr2Lr2}{Lr2Lr2} = 4$	$\frac{Lr2Lr2}{Lr2Lr2} = 2$

With the Lr3-avirulent strain, 10-1,2,3,4 Mediterranean/Mentana gave only resistant F2 seedlings with ITS ranging from '1' to '1-'. However, with the Lr3-virulent pathotype 76-0, this cross segregated, the infection types on susceptible segregates being similar to those displayed by Mentana with IT 'X+3'. In contrast, Mediterranean/Festigny segregated for a single gene with pathotype 10-1,2,3,4, and all F2 seedlings were resistant with pathotype 76-0. These results could be obtained only if the Mediterranean parent possessed both Lr3 and Lr2a.

When tested with *P. graminis* tritic pathotype 34-1,2,3,4,5,6,7, the Mediterranean W1728 line displayed IT '2', compared to ITS '3+', '2-', and '22+' for Mediterranean W3732, Festigny and Webster, respectively. These results indicated that the Mediterranean W1728 line also differed from the Canadian source in respect of stem rust reaction. When tested with pathotype 34-1,2,3,6,7,8,9, chosen for virulence for Sr30, all three wheats displayed IT '3+' leading to the postulation that the allele conferring resistance to pathotype 34-1,2,3,4,5,6,7 was Sr30. All 105 F2 seedlings from Mediterranean W1728/Festigny were scored as resistant with ITS '2-' to '23+'. Since Festigny was known to possess Sr30 (Knot and McIntosh, 1978), the presence of this gene in Mediterranean W1728 was confirmed.

DISCUSSION

The results showed that a selected line of Mediterranean being used as a differential for pathogenicity surveys by the University of Sydney possessed Lr3 and Lr2a. This contrasted with a recently introduced Canadian source, W3732, with only Lr3. From 1924, Mediterranean was imported from the USA to the University of Sydney wheat collection on three different occasions. Because present holdings of all three accessions were genetically heterogeneous, it appeared that the original source of CI3332 was also heterogeneous. The selected stock of W1728 also possessed Sr30. As Webster, 1964) and Sr30 (Knot and McIntosh, 1978), it is suggested that CI 3332 was contaminated, at an early stage, by outcrossing with Webster. Mediterranean W584 lacked seedlings with Lr3, probably a consequence of genetic drift occurring in the original heterogeneous population. The other two accessions of Mediterranean were heterogeneous for both Lr2a and Lr3. The responses given for physiologic races listed in the key of Johnston and Levine (1955) fail to indicate a genotype for Mediterranean. Most random combinations of the alternative high and low responses for the testers Democrat, Webster and Mediterranean were present. Such results thus indicated that Mediterranean stocks used by different laboratories were different. Indeed, the responses that are listed could indicate that particular Mediterranean stocks: have no gene for resistance or have no resistance gene in common with Webster or Democrat, have Lr2a, Lr3 or both genes, have a resistance gene that is different from Lr2a and Lr3 as suggested from response patterns listed for races 41, 82, 88, 99, 112, 117, 152 and probably 94. Given the heterogeneity found in Australian stocks of CI 3332, lines of Mediterranean could be selected to satisfy any of these possibilities except the last.

Research workers still using Mediterranean as a differential for pathotype surveys should evaluate their current sources of this wheat. Laboratories having obtained seed from the University of Sydney will have the selected WI728 with both Lr2a and Lr3; those with seed from other sources may have stocks with only Lr3. Finally, sources of Mediterranean that are resistant to cultures virulent for Lr2a and Lr3 should be further investigated in order to confirm the presence and identity of a further resistance gene.

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TABLE 1 - Infection type responses of various Mediterranean sources and controls when tested with five *P. recondita tritici* pathotypes

WHEAT	ACCESSION NO.	PATHOTYPE						KNOWN RESISTANCE GENE		
		DEMOCRAT-AVIRULENT			DEMOCRAT-VIRULENT					
		10-1,2,3,4	26-1,3	76-0	104-2,3,6(7)	162-1,2,3,6				
Mediterranean	W1728 ¹	;	-	;1-	-	;1	-	33+		
	W584	-	3+ (59p) ³	;1-	3+ (12p)	;1-	3+ (42p)	;1- 3+ (56p)	3+ (40p)	
	W975	;	3+ (17p)	to;1	3+ (59p)	;1-	3+ (8p)	;1 3+ (45p)	3+ (37p)	
	W1728 ²	;	3+ (10p)	to;1-	- (42p)	;1-	3+ (4p)	;1 3+ (105p)	3+ (43p)	
	W3732	;	-	;	-	-	3+	-	33+	
Democrat	W977	;	-	;	-	-	3+	-	3+	Lr3
Mentana	W1124	0;	-	;	-	-	X++3	-	33+	Lr3bg
Webster	W973	-	3+	;1-	-	;1	-	;1	-1	Lr2a
Festiguay	W2706	-	3+	;1-	-	;1	-	;1	-	Lr2a
CS(Tim.6B)	-	-	3+	-	3+	-	3+	-	3+	-

¹ Used in pathogenicity surveys, and for inheritance studies

² University of Sydney wheat collection

³ Numbers in parenthesis refers to number of seedlings with designated IT

TABLE 2 - Responses of F₁, F₂ and F₃ progenies from three Mediterranean crosses tested with two *P. recondita tritici* pathotypes

Mediterranean/	Pathotype	F ₁ IT	No.F ₂ seedlings res	sus.	X ² 3:1	No.F ₃ lines HR seg. HS	X ² 1:2:1	IT's for res. plants
CS(Tim.6B)	10-1,2,3,4	X	52	18	0.02	10 35	9	4.78 ;toX
	76-0	-	-	-	-	11 28	15	0.67 ;1-to;23-
Mentana	10-1,2,3,4	0;	210	0	-	-	-	;to;1-
	76-0	-	41	15	0.10	-	-	;1-to;23-
Festiguay	10-1,2,3,4	X	54	16	0.17	-	-	;toX
	76-0	-	181	0	-	-	-	;1-

res. = resistant, sus. = susceptible, HR = homozygous non-segregating resistant,
seg. = segregating, HS = homozygous non-segregating susceptible, IT = infection type

OBITUARY

Dr Ilse Nover, 1915-1985

Dr Ilse Nover was born on 14th October 1915 in Kassel, Germany, and died on 13th February 1985 in Wernigerode. After her university studies in biology and agriculture, completed in 1941 in Halle, she was employed in the Institute of Zoology, Martin-Luther-Universität. Since 1948 Dr Ilse Nover has been engaged first as an assistant and later as a scientist in the Institute of Phytopathology at the Martin-Luther-Universität in Halle/Saale. During her university years, inspired by Professor Dr Theodor Roemer, Dr Ilse Nover focused her interest on the principles of breeding for disease resistance. The research of Dr Nover was devoted to wheat and barley diseases, particularly rusts and mildew, physiological specialization of these pathogens and sources of resistance to them.

Her papers dealing with physiological specialization of rust and powdery mildew belong to the most comprehensive ones in Central Europe. Her studies on sources of disease resistance in the wheat and barley collection of the Zentralinstitut für Genetik und Kulturpflanzenforschung (Central Institute of Genetics and Research of Cultivated Plants), Gatersleben, represent one of the best sources of information on genetic resources for breeding wheat and barley for disease resistance. They were published in 17 contributions in Kulturpflanze.

Later on, Dr Nover, published papers on methods of disease resistance testing, on genetics of yellow rust resistance in wheat and of powdery mildew resistance in barley mutants and contributed to several monographs on cereal diseases.

Dr Ilse Nover lost her husband in the World War II and divided her endless energy and love between her research and her three children. By her selfless dedication to her scientific work and readiness to share all her professional experience with young colleagues also from abroad she contributed significantly to the development of wheat and barley breeding for disease resistance not only in her own country but also in other countries. Her human warmth, personal devotion and independent mind will be remembered by all who have met her.

Pavel Bartos

