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CHANGE OF EDITOR

The distribution of this issue of the Cereal Rusts Bulletin marks the retirement of Dr. J.G. Manners from his position as Editor of the Bulletin. Dr. Manners shepherded the Bulletin from the stage when it was only an idea through the first few volumes, to its present status as a journal appearing regularly.

This obviously required great energy and enthusiasm and I am sure I speak for all members of the "Great Rust Family" (Feekes, 1976 pers. comm.) in expressing our sincere thanks to Dr. Manners.

Our new Editor will be Professor E. Russell, Department of Agricultural Biology, University of Newcastle, NE1 7RU, England. We welcome him to this position and wish him success in it. He will need plenty of co-operation from all of us in continuing to send in papers.

R. Johnson

Secretary

MONITORING VIRULENCE IN *PUCCINIA HORDEI*: A PROPOSAL FOR THE CHOICE OF HOST GENOTYPES AND SURVEY PROCEDURES

BY BRIAN C. CLIFFORD

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In February 1975, the author circulated a proposal to colleagues actively engaged in studies of variation in *Puccinia hordei* Otth concerning the choice of host genotypes and technical procedures best suited to monitor virulence genes in populations of *P.hordei*. It was suggested that a meeting of interested workers could be held at the next European and Mediterranean Cereal Rusts Conference in Interlaken, Switzerland, and this meeting was held on 7 September, 1976. It was agreed that the results of the meeting should be published as a proposal in the Cereal Rusts Bulletin for the consideration of other barley brown rust workers who were unable to attend the meeting and the following report summarizes the proposals agreed by the participants.

The traditional aim of physiologic race surveys has been the early detection of important virulence genes and assessments of their frequencies in the pathogen population. Modern concepts in the rational use of race specific resistance relate to management of such resistances through their planned release in specified regions or in particular carriers as, for example, in multigene or multiline varieties. Concomitant with these latter aims is a need to monitor the pathogen population for virulence genes and virulence gene combinations relevant to the resistance genes being deployed.

The host resistance 'gene pool' is available internationally through various formal and informal co-operative schemes and so it seems desirable that surveys of corresponding virulences in the pathogen population should have a similar breadth to assist in the conservative use of gene resources.

COMPOSITION OF DIFFERENTIAL SERIES

The basis for any virulence survey is a set of host genotypes carrying specific resistance genes capable of resolving the pathogen population into virulence components. These genotypes should be relevant to regional breeding programmes and also allow comparative analysis of geographically separated pathogen populations. Any set of genotypes should be flexible enough to allow for evolving breeding programmes and yet sufficiently conservative to allow temporal as well as spacial analysis of population dynamics of the pathogens. To meet these somewhat conflicting demands, two components to the differential series are proposed:

(1) A Standard Set of International differential genotypes for comparative studies of virulence gene frequencies and associations on a world-wide basis.

(2) Regional Sets of supplementary differentials relevant to the breeding and research interests of individual workers.

The Standard Set of genotypes is given in Table 1 and comprises the most desirable known carriers of the specific resistances designated. The Pa_3 carrier is a line from a cross Rika x F_1 (Baladi 16 x Rika No. 7) studied by Bruckner (1971) and later by Parlevliet (1976) who abbreviated the designation to Ri x (Ba x Ri). A further condensation to Ribari seems appropriate and this name is therefore proposed. Nucleus seed stocks of the resistant genotypes from Dr C.W. Roane have been supplied to the author by Dr B.H. Tan. Stocks of the varieties will be maintained at the Welsh Plant Breeding Station and should be available for release in Autumn 1977.

It is proposed to backcross the resistance genes into Sultan, the universal suscept, to obtain a near-isogenic series differing only at the Pa locus. As a step in this direction Tan has succeeded in isolating Pa_5 from Quinn and Pa_6 from Bolivia. This isogenic series should take 3 years to develop.

No proposals were made regarding Regional Sets of differentials. These will be assembled by individual workers for specific research investigations and regional breeding requirements.

Table 1. Standard set of differential barley genotypes for analysis of virulence in *Puccinia hordei* Otth.

Genotype	C.I. Number	Gene	Reference
Sudan	6489	Pa	Roane and Starling (1967)
Peruvian	935	Pa_2	Starling (1956)
Ribari	-	Pa_3	Bruckner (1971)
Gold	1145	Pa_4	Roane and Starling (1967)
Quinn	1024	$Pa_5(+Pa_2)$	Roane and Starling (1967)
Bolivia	1257	$Pa_6(+Pa_2)$	Roane and Starling (1970)
Cebada Capa	6193	Pa_7	Parlevliet (1976)
Egypt 4	6481	Pa_8	Tan, pers.comm.
Sultan		Universal suscept.	

TESTING PROCEDURES

Optimum conditions for growing test plants prior to and after inoculation are considered to be 20°C for a 16 h photoperiod (artificially supplemented when necessary) followed by an 8 h period at 10°C. Seedlings should be inoculated with fresh urediospores when the second leaf is beginning to emerge and carriers such as talc or mineral oil may be used if desired. Plants when inoculated should be either sprayed with water droplets or placed in a dew simulation chamber. Free moisture should be maintained on the plants for a 16 h dark period at 17°C. Plants should then be returned to their growing environment and assessments made 3 days after the eruption of uredia. Plants should be grown in isolation where possible and chemical control of other pests and diseases should not be employed unless absolutely necessary.

Initial field sampling should be carried out on a random basis, if practicable, to allow interpretation of relative frequencies of virulences to be made. In this regard, an alternative to collecting rust samples may be the use of mobile nurseries of the differential varieties to directly assess field populations of the pathogen (Eyal *et al.*, 1973).

RACE NOMENCLATURE

No firm decision was reached but it is considered that a formal system of nomenclature should be employed that is short, simple, logical, flexible and informative. Some of these requirements are mutually exclusive but the best compromise appears to be the octal/binary notation system proposal by Gilmour (1973). The differential genotypes would be arranged with Pa on the right, grading to Pa_8 on the left. Additional resistance genes could be numbered consecutively and added to the left. The system is particularly useful to designate virulence genotypes which is one of the prime functions of virulence gene surveys.

CO-ORDINATION AND COMMUNICATION OF RESULTS

Those workers listed in Table 2 have already expressed interest in the above proposals and will therefore receive further communications, but the author would be pleased to hear from other *P.hordei* workers who wish to participate in the scheme.

Co-operators wishing for a reciprocal exchange of virulence genes survey data should send their survey results to the author. A compilation of survey data could be circulated annually. Individual publication of regional surveys is also to be encouraged.

It is hoped that a review of the proposed procedures can be made at the next International Phytopathology Congress in München, W. Germany in 1978 and thereafter at European and Mediterranean Cereal Rusts Conferences.

The author would like to thank those colleagues who responded to the earlier circulars and attended the Interlaken meeting for their help and enthusiasm.

Table 2. List of co-operators in the proposed virulence gene surveys of *P.hordei* Otth.

Dr Anneke Balkema	Netherlands
Fr. Dr Eva Fuchs	West Germany
Fr. Gisla Grunewaldt	West Germany
Mrs Mahrokh Rastegar	United Kingdom
Fr. Dr Ursula Walthur	East Germany
Dr B. Arnst	New Zealand
Dr A.R. Brown	United States of America
Dr F. Bruckner	Czechoslovakia
Dr B.C. Clifford	United Kingdom
Dr J. Grunewaldt	West Germany
Dr J.E. Hermansen	Denmark
Dr R. Johnson	United Kingdom
Dr J.H. Jorgensen	Denmark
Dr J. Moseman	United States of America
Dr J.E. Parlevliet	Netherlands
Dr R. Priestley	United Kingdom
Dr J. Rintelen	West Germany
Dr C.W. Roane	United States of America
Dr B.H. Tan	West Germany
Prof. I. Wahl	Israel

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A NEW GENE FOR RESISTANCE TO *PUCCINIA HORDEI* IN CERTAIN ETHIOPIAN BARLEYS

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A new gene for resistance to barley leaf rust (*Puccinia hordei* Otth) appears to be common to several collections of barley (*Hordeum vulgare* L.) originating in Ethiopia, viz. Abyssinian Schwarz (A.S.), Uadera, Ab 14 and C.I. 1243. Tentative evidence in support of this assumption is given in the correlated responses of these barleys to a number of diverse strains of the pathogen, which collectively also differentiate them from known genotypes (see Table 1). Clifford and Udeogalanya (1976) have independently observed similar differential responses given by C.I. 1243 and lines carrying either gene Pa 3 or Pa 7 to a strain virulent on C.I. 1243.

Two of the above-mentioned Ethiopian barleys, viz. A.S. and Uadera, were subjected to a further genetic analysis in which two contrasting strains of the pathogen were employed. These were (1) culture 75.27 of race UN 4-0 (Roane's (1962) culture 57.19) which is avirulent to all presently known Pa genes with the exception of Pa8(Tan, 1977) and, as such, is regarded as the reference culture for the recognition of genes Pa to Pa 7; and (2) culture 76.51 of race Un 23-3 (Nover and Lehmann, 1974) which, by contrast, is virulent to all known Pa genes except Pa 3 and Pa 7. Resistance to the latter strain can therefore be ascribed to Pa 3, Pa 7, one or more genes hitherto unrecognized, or to a combination of these genes. These are precisely the considerations to be resolved with respect to these Ethiopian barleys.

The characteristic infection type (IT) which these barleys exhibit is a legitimate criterion for the recognition of the gene(s) they carry, but because of variation under normal test conditions, it is taken to supplement rather than substitute for the necessary genetic evidence. As shown in Table 1, these Ethiopian barleys typically give chlorotic flecks when tested with various cultures, but under certain test conditions and delayed scoring, they can yield an IT as high as 1 + 3-c, and to one strain at least, an IT 3c is consistently elicited. The latter is believed to reflect heterozygosity of the parasite's corresponding gene for avirulence (cf. Samborski, 1963). Intermediate reactions to at least one strain have also been reported on Ab 14 and C.I. 1243 (Nover and Lehmann, 1974). The variation nothwithstanding, their reactions are invariably associated with chlorosis and, as such, are normally distinguishable from those conditioned by Pa 3 (IT 0) or Pa 7 (IT 0;n) and, for that matter, those typical to Pa, Pa 2, Pa 4, Pa 2 + Pa 5, Pa 2 + Pa 6 and Pa 8.

Table 1. Infection types exhibited by seedlings of standard differentials and four Ethiopian barleys at 20C

Culture Race (UN-S)[a]	75.27 4-0	75.22 2-0	75.5 ?-0	76.49 14-0	76.51 23-3	76.29 30-1	76.39 19-1[b]
Sudan C.I.6489 (Pa)[1]	;n	4	4	4	4	4	4
Peruvian C.I.935 (Pa2)[2]	X2-n	X2	X2	X2	3+	4	4
Estate CI.3410 (Pa3)[1]	0	0	0	3	0	0	0
Gold C.I.1145 (Pa4)[1]	;	;	4	4	4	4	4
Cebada Capa (Pa7)[3] CI.6193	0;n	0;n	0;n	0;n	0;n	0;n	0;n
Egypt 4 C.I.6481 (Pa8)[4]	4	4	;n	4	4	4	4
Quinn C.I.1024 (Pa2+Pa5)[1]	0;	0;	0;	0;	4	0;	;
Bolivia C.I.1257 (Pa2+Pa6)[1]	0;	0;	;1-2n	X2	3+	4	4
A.S. H899[c]	;c	;c	;c	;c	;c	3+	3+
Uadera H3620	;c	;c	;c	;c	;c	3+	3+
Ab14[d] HOR 1859	;c	;c	;c	;c	;c	3+	3+
C.I.1243 HOR 2596	;c	;c	;c	;c	;c	3+	3+

[1] Roane and Starling (1967)

[2] Clifford (1974)

[3] Nover and Lehmann (1974)

[4] Tan (1977)

[a] Unified Numeration (Levine and Cherewick, 1952) and Supplemental Designation based on virulence on HOR 500 (1), HOR679 (2) and HOR 1132 (3) (Nover and Lehmann, 1974).

[b] resembles race 30-1, except for its avirulence to Reka 1 C.I.5051.

[c] 'H' refers to our barley accession and 'HOR' to Gatersleben.

[d] carries a recessive gene (yr) for resistance to *P. striiformis* (Nover and Scholz, 1969).

F_2 data (Table 2) indicate, firstly, that A.S. and Uadera each possesses a partially dominant gene (IT's ;c and 3c). When F_2 seedlings from the crosses Moore x A.S. or Uadera were tested with a mixture of both strains, the responses were completely correlated, suggesting that the same gene was responsible in each instance. Secondly, the results demonstrate independence between the respective gene on the one hand, and Pa 3, Pa 4 and Pa 7, on the other, although one test involving Estate (Pa 3) gave a result differing significantly from the expected ratio. Such genetic data are crucial for establishing non-identity with Pa 3 and Pa 7 in the absence of strains capable of differentiating them. Thirdly, within the limits of the F_2 population tested (viz. 292), the single genes present in A.S. and Uadera appeared to be identical; if they are in fact distinct, their upper limit of recombination should not exceed 20.2%.

There is little doubt, despite the lack of further data involving other known Pa genes, that the gene conditioning resistance in A.S. and Uadera, as well as in Ab 14 and C.I. 1243 by inference, is new and has not been described previously. Logically it should be designated Pa 9; its presence in Ab 14 and C.I. 1243 nevertheless remains to be confirmed, and its relationships with Pa, Pa 2, Pa 5, Pa 6 and Pa 8 elucidated.

A subsequent survey of other Ethiopian barleys (ex Gatersleben) with the same differentiating strains led the author to conjecture that the new gene may be present in collections Ab 15 (HOR 1643), Ab 1113 (HOR 2927), Ab 1118 (HOR 2926), EP 73 (HOR 1633) and EP 74 (HOR 4280), but as in the cases of Ab 14 and C.I. 1243, their genotypes await genetic confirmation.

Pa 9 appears to be a valuable gene for differentiating certain isolates collected in Britain (Clifford and Udeogalanya, 1976) and Germany (Tan, unpubl.). In glasshouse tests it was found to be effective against a wide spectrum of strains originating from USA, Britain, Denmark, The Netherlands, Germany, DDR, USSR and Australia (unpublished data) and compares with Pa 3 in this respect, but it is by no means as universally effective as Pa 7 (see Parlevliet, 1976; and Golan *et al.*, 1977). In the breeding context its potential is perhaps best viewed in conjunction with other known effective genes or sources, in the development, for instance, of varieties with multigenic resistance, or components of multi-line varieties.

ACKNOWLEDGEMENTS

I am indebted to Dr C.O. Lehmann (Gatersleben) for making available cultures 76.49 and 76.51, and Ethiopian barley lines bearing 'HOR' designations; and to Dr J.G. Moseman (Beltsville) for providing culture 75.27. Technical assistance was given by Misses Brigitte Merkel and Sieglinde Frey.

Table 2. F_2 data on crosses between (a) known genotypes and A.S.; (b) known genotypes and Uadera; and (c) A.S. and Uadera.

Cross	Culture(s)	IT	No.	IT	No.	IT	No.	Ratio	P
(a)									
Moore (susc.)[1] x A.S.	75.27	;c	39	3c	94	4	47	1:2:1	N.S.
.....".....	76.51	;c	62	3c	148	4	63	1:2:1	N.S.
.....".....	75.27 + 76.51	;c	64	3c	165	4	73	1:2:1	N.S.
Gold (Pa 4)[1] x A.S.	75.27	;,;c,1±,3c	336			4	16	15:1	N.S.
A.S. x Estate (Pa 3)[1]	75.27	0,0;	197	;c,3c	48	4	7	12:3:1	N.S.
.....".....	76.51	0,0;	56	;c,3c	24	4	1	12:3:1	*
A.S. x Rika x F_1 (Baladi x Rika) (Pa3)[2]	75.27	0,0;	154	;c,3c	45	4	14	12:3:1	N.S.
.....".....	76.51	0 -;c,3c	318			4	18	15:1	N.S.
Cebada Capa (Pa 7)[3] x A.S.	75.27	0 -;cn	600	3c	96	4	44	13:2:1	N.S.
A.S. x Dabat (Pa 7)[4]	75.27	0 -;cn,3c	149			4	13	15:1	N.S.
.....".....	76.51	0 -;cn,3c	291			4	19	15:1	N.S.
(b)									
Moore x Uadera	75.27	;c	34	3c	76	4	35	1:2:1	N.S.
.....".....	76.51	;c	33	3c	67	4	31	1:2:1	N.S.
.....".....	75.27 + 76.51	;c	41	3c	103	4	35	1:2:1	N.S.
Gold x Uadera	75.27	0 - 1+,3c	352			4	23	15:1	N.S.
Estate x Uadera	75.27	0 - 1c,3c	218			4	10	15:1	N.S.
.....".....	76.51	0 - 1c	129	3c	18	4	8	13:2:1	N.S.
Cebada Capa x Uadera	75.27	0 - 1cn,3c	117			4	9	15:1	N.S.
.....".....	76.51	0 - 1cn	142	3c	24	4	14	13:2:1	N.S.
(c)									
A.S. x Uadera	76.51	;c	292	-		-			

[1] Roane and Starling (1967):
[2] Brueckner (1971);
[3] Nover and Lehmann (1974);
[4] Parlevliet (1976)

N.S. = Not significant at the P = 0.05 level;
* = 0.01 < P < 0.05

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YELLOW RUST RESISTANCE OF SOME EUROPEAN WHEAT CULTIVARS DERIVED FROM RYE

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Many European wheat cultivars developed from crosses with various Salzmünde lines, e.g. Riebesel 47-51, Neuzucht and similar material, possess rust resistance derived from a rye chromosome (Zeller, 1973, Bartoš *et al.* 1973). Growing some of these cultivars on extensive scale resulted in the appearance of virulent races of brown rust (*Puccinia recondita* Rob. et Desm. f.sp. *recondita* Rob. et Desm.) (Lesovoi and Panteleev, 1973; Bartoš, 1975) and recently the resistance to yellow rust *Puccinia striiformis* West f.sp. *tritici* has also broken down. Virulent yellow rust races have been described in the USSR (Matveyenko, 1973) and in the Netherlands (Stubbs, 1974) where the newly introduced cultivar Clement deriving its resistance from 'Riebesel 47-51' was severely attacked by yellow rust in 1974 locally and in 1975 throughout the country.

MATERIALS AND METHODS

Race 232 E 137 virulent to 'Clement' and previously named 104 E 137 E has been used to study the reaction of some cultivars in which resistance from rye was identified or assumed. The seedling tests were made in growth chambers in Wageningen, The Netherlands, at constant temperatures of 15°C, 17°C and 22°C under a light intensity of 22.000 lux during 16 hours per day. The adult plant reaction to race 232 E 137 has been studied in race nurseries in the IJsselmeerpolders, The Netherlands. The tests with races 0 E 8, 36 E 132, 37 E 132, 40 E 8 and 41 E 136 were carried out in Prague, Czechoslovakia in the greenhouse at a temperature of about 15°C. The genetic studies were also performed in Prague. The infection types were scored on a 0-9 scale and the severity of attack in the field on a 0-100% scale.

RESULTS AND DISCUSSION

All cultivars were highly resistant to races 0 E 8, 36 E 132, 37 E 132, 40 E 8 and 41 E 136 in the seedling stage (Table 1). However, with race 232 E 137 only the cultivars Orlando, Saladin and Winnetou exhibited resistance. In repeated tests 'Riebesel 47-51', as a seedling, was moderately susceptible to moderately resistant to race 232 E 137. The seedling reactions were similar at temperatures of 15°C and 17°C. At 22°C only chloroses with sporadic sporulation appeared on the susceptible cultivars. The field data show varietal differences in the level of severity of infection with race 232 E 137. However, these differences are not entirely in accordance with the differences in the seedling stage. Susceptibility in the seedling stage changed into a varying degree of adult plant resistance, possibly also influenced by temperature as demonstrated for 'Winnetou'. 'Riebesel 47-51' in the adult plant stage exhibited high resistance to race 232 E 137, a phenomenon which was also observed in yellow rust trials located in the Netherlands and naturally infected with that race. Obviously only a part of the resistance of 'Riebesel 47-51' has been transferred to 'Clement'.

Table 1. Seedling[1] and adult plant reaction of cultivars with resistance derived from rye to several races of yellow rust.

	Seedling						Adult plant	
Race / Cultivar	232E137	0E8	36E132	37E132	40E8	41E136	40E8	232E137
	Infection type						Severity	
Aurora	8-9	0	0	0	0	0	tr.	50
Bezostaya 2	8-9	0	0	0	0	0	tr.	25
Clement	8-9	0	0	0	0	0	0	50
Kavkaz	7-9	0	0	0	0	0	tr.	15
Lovrin 13	8-9	0	0	0	0	0	tr.	25
Mildress	7-8	0	0	0	0	0	0	15
Orlando	1-4	0	0	0	0	0	0	0
Riebesel 47-51	4-6	0	0	0	0	0	0	0-5
Saladin	1	0	0	0	0	0	0	0
Salz.Bartweizen	7-9	0	0	0	0	0	1-5	25
Skorospelka 35	7-8	0	0	0	0	0	tr.	25
Weique	8	0	0	0	0	0	0	10
Winnetou	2-4(8)[2]	0	0	0	0	0	0	0
Zorba	8	0	0	0	0	0	0	-

1) at 15°C day and night

2) (8) at 18°C day and 4°C night.

Table 2. Reaction of parents, F1, and F2 generation to yellow rust race 40 E 8 in the greenhouse and in the field.

Parents, F1, F2	Infection type and number of plants										
	0	1	2	3	4	5	6	7	8	9	Total
Greenhouse											
F1 Weique x Orlando	5										5
F2 " "	264										264
Weique	10										10
Orlando	10										10
Field											
F1 Weique x Orlando	5										5
F2 " "	128			3			7				139
Greenhouse											
F1 Salz.Bartw. x Orlando	6										6
F2 " "	334										334
Salz.Bartw.	10										10
Orlando	10										10
Field											
F1 Salz.Bartw. x Orlando	26										26
F2 " "	153						14		5		172
Salz.Bartw.	26										26
Orlando	26										26
Greenhouse											
F1 Weique x Salz.Bartw.	5										5
F2 " "	358										358
Weique	10										10
Salz.Bartw.	15										15
Field											
F1 Weique x Salz.Bartw.	30										30
F2 " "	188	7	21				5		2		223
Greenhouse											
F1 Weique x Zorba	5										5
F2 " "	272										272
Weique	10										
Zorba	6										6
Field											
F1 Weique x Zorba	39										39
F2 " "	138		3								141
Weique	13										13
Zorba	13										13
Greenhouse											
F1 Orlando x Zorba	12										12
F2 " "	277			1							278
Orlando	10										10
Zorba	10										10
Field											
F1 Orlando x Zorba	26										26
F2 " "	158										158
Orlando	13										13
Zorba	13										13

Differences between seedling (greenhouse) and adult plant (field) reactions were also observed when the F_2 progenies of crosses of a few of these cultivars were inoculated with race 40 E 8 (Table 2). Segregation for susceptibility in the F_2 progeny did not occur in the seedling stage. However, in the field susceptible plants were found in the F_2 population of the crosses Weique x Orlando, Salzmünder Bartweizen x Orlando, and Weique x Salzmünder Bartweizen. The adult plant reaction may have been influenced by some factor(s) which can suppress the expression of gene(s) active in the seedling stage. It is also possible that one or more additional genes are necessary for the expression of adult plant resistance. These may not be identical in the cultivars analysed in the tests.

The results obtained so far do not allow the presentation of definite conclusions regarding the genetic background of resistance. However, they indicate that the studied cultivars with resistance derived from rye have one or more genes in common as well as different genes governing their reaction to yellow rust in the seedling and adult plant stage.

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