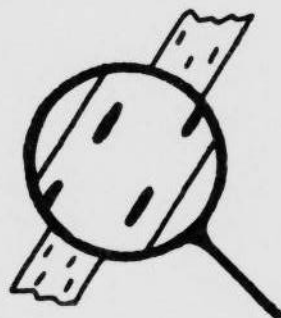


U.K. CEREAL PATHOGEN VIRULENCE SURVEY



1979 Annual Report

UNITED KINGDOM CEREAL PATHOGEN VIRULENCE SURVEY

Chairman: Dr G D H Bell CBE FRS

Secretary: Dr R H Priestley, National Institute of Agricultural Botany,
Huntingdon Road, Cambridge CB3 0LE

Tel: Cambridge (0223) 76381

1979 Annual Report

Published by the United Kingdom Cereal Pathogen Virulence Survey Committee
Cambridge, England

May 1980

Price £1

© United Kingdom Cereal Pathogen Virulence Survey Committee 1980

Printed by the National Institute of Agricultural Botany, Cambridge

MEMBERS OF THE U.K. CEREAL PATHOGEN VIRULENCE SURVEY COMMITTEE, 1979-80

Dr R A Bayles	National Institute of Agricultural Botany, Cambridge
Dr G D H Bell	formerly Plant Breeding Institute, Cambridge
Mrs F G A Bennett	Plant Breeding Institute, Cambridge
Mr P Byford	National Institute of Agricultural Botany, Cambridge
Dr A J Carr	Welsh Plant Breeding Station, Aberystwyth
Dr N H Chamberlain	British Association of Plant Breeders
Dr B C Clifford	Welsh Plant Breeding Station, Aberystwyth
Dr D A Doling	RHM Research Ltd, High Wycombe
Dr J K Doodson	National Institute of Agricultural Botany, Cambridge
Mr C S Elliott	National Institute of Agricultural Botany, Cambridge
Dr J Gilmour	East of Scotland College of Agriculture, Edinburgh
Mr J E E Jenkins	Agricultural Development & Advisory Service
Dr R Johnson	Plant Breeding Institute, Cambridge
Mr E L Jones	Welsh Plant Breeding Station, Aberystwyth
Mr I T Jones	Welsh Plant Breeding Station, Aberystwyth
Dr J G Kay	British Association of Plant Breeders
Mr E Lester	Rothamsted Experimental Station, Harpenden
Professor R K McKee	Department of Agriculture, Northern Ireland
Dr R H Priestley	National Institute of Agricultural Botany, Cambridge
Mrs S E Slater	Plant Breeding Institute, Cambridge
Mr M J Richardson	Department of Agriculture & Fisheries for Scotland
Dr P S Wellington	National Institute of Agricultural Botany, Cambridge
Dr M S Wolfe	Plant Breeding Institute, Cambridge

CONTENTS

	page
THE UK CEREAL PATHOGEN VIRULENCE SURVEY	1
MILDEW OF WHEAT	
Fiona G A Bennett	3
YELLOW RUST OF WHEAT	
R H Priestley & P Byford	15
BROWN RUST OF WHEAT	
B C Clifford, E R L Jones & R H Priestley	24
MILDEW OF BARLEY	
M S Wolfe & Susan E Slater	29
YELLOW RUST OF BARLEY	
R H Priestley & P Byford	50
BROWN RUST OF BARLEY	
B C Clifford, E R L Jones & R H Priestley	55
RHYNCHOSPORIUM OF BARLEY	
E R L Jones & B C Clifford	60
MILDEW OF OATS	
I T Jones & E R L Jones	64
EVIDENCE FOR THE EFFECTIVENESS OF CULTIVAR DIVERSIFICATION IN REDUCING THE SPREAD OF YELLOW RUST AND MILDEW IN CEREALS	
R H Priestley & M S Wolfe	71
CULTIVAR DIVERSIFICATION SCHEMES FOR 1980	
Wheat yellow rust	76
Barley mildew	77
Wheat combined yellow rust and mildew	78

The yellow rust of wheat paper includes an appendix giving the results of the UK and Eire section of the International Survey of Factors of Virulence of Puccinia striiformis.

THE UNITED KINGDOM CEREAL PATHOGEN VIRULENCE SURVEY

The Survey, formerly the Physiologic Race Survey of Cereal Pathogens, commenced in 1967 following an unexpected epidemic of wheat yellow rust (Puccinia striiformis) that caused severe yield losses in the then recently introduced but widely grown variety Rothwell Perdix. The epidemic was the result of the development of increased virulence for this previously resistant variety.

The Survey is supported financially by the Ministry of Agriculture, Fisheries and Food and the Agricultural Research Council.

OBJECTIVES

The principal objective is the early detection of increased virulence compatible with those resistances currently being exploited in commercial varieties and breeding programmes.

Secondary objectives include providing information for varietal diversification schemes, monitoring the frequency of virulences and virulence combinations, evaluating the compatibility of virulences with one another and measuring the effect of changes in variety on the pathogen population.

OPERATION

The Survey is carried out on an annual basis. In April, a list of cereal varieties from which disease samples are requested is sent to about 100 pathologists and agronomists within the United Kingdom. They collect samples of infected leaves from field crops and cultivar trials and send them by post to the three testing centres:

National Institute of Agricultural Botany, Cambridge, for
rust of wheat and barley.

Plant Breeding Institute, Cambridge, for mildew of wheat and barley.

Welsh Plant Breeding Station, Aberystwyth, for brown rust of wheat and
barley, mildew and crown rust of oats and Rhynchosporium of barley.

At these centres, virulence tests are carried out using spores multiplied from the disease samples. In the mildews, virulence is measured by inoculating detached seedling leaf segments. In the rusts, both seedling leaves (attached) and adult plants are usually inoculated as previous work has shown that a number of the resistances involved are ineffective at the seedling stage. Seedling tests are usually carried out under controlled environment conditions. Adult plant tests are carried out in the field in the following season.

RESULTS

The United Kingdom Cereal Pathogen Virulence Survey Committee meets annually to discuss the scientific and agricultural significance of the results of virulence tests carried out during the year. The results of wheat yellow rust and barley mildew tests are used to place winter wheat and spring barley varieties in diversification groups on the basis of their specific resistances. The results of the virulence tests and the diversification schemes are published shortly afterwards in the Annual Report.

The information provided by the Survey is used in a number of ways. Isolates possessing new virulences are used by the National Institute of Agricultural Botany to evaluate the resistance of cereal cultivars in trial in England & Wales. These isolates are also used by plant breeders to select lines with effective forms of resistance. Isolates are also supplied to Universities and Colleges for research projects and to illustrate to students the principles of resistance in host-pathogen systems. Versions of one or both varietal diversification schemes, modified to meet regional requirements, are published in the National Institute of Agricultural Botany Farmers Leaflet No. 8 'Recommended varieties of cereals', the Scottish Agricultural Colleges leaflet 'Recommended varieties of cereals', and the Agricultural Development & Advisory Service booklet 'The use of fungicides on cereals'.

FUTURE DEVELOPMENTS

In order to realise its objectives the Survey is actively engaged in research projects through those Committee members working at the three testing centres. The projects are aimed at improving our knowledge of the interaction between host and pathogen populations and at present include the use of mobile nurseries, the improved detection of adult plant virulence, the effect of variety mixtures on the pathogen population, the recognition of durable forms of resistance and the development of improved numerical techniques for analysing host-pathogen data matrices.

MILDEW OF WHEAT

Fiona G. A. Bennett

Plant Breeding Institute, Cambridge

Powdery mildew on wheat was more severe than it had been since 1974. The high number of samples from Flanders and Maris Huntsman reflected increased infection levels on these cultivars.

Anvil was assigned to wheat mildew resistance (WMR) group 4 and TW275 to WMR 2+4+6. No new resistance factors or combinations were discovered. One pathogen isolate was found to have a new pathogenicity combination and was assigned to wheat mildew virulence (WMV) group 2+6+8. It was isolated from a breeder's line thought to have the corresponding resistance combination.

Assessment of population pathogenicity levels showed that WMV 4 and 9 are decreasing whilst the combination WMV 2+6 is increasing. WMV 1, 2, 5 and 8 are all relatively common.

The behaviour of WMV 8 and WMV 2+6 in populations from different sources was examined. It was concluded that selection does operate against non-corresponding WMV types. Therefore diversification in this instance would be expected to be beneficial.

INTRODUCTION

Mildew levels nationally were higher than they had been since 1974, although the ADAS Foliar Disease Survey recorded an overall mean value of only 2.38 per cent mildew on leaf 2 of commercial wheat cultivars (J.E. King; pers. comm.). However, Rowe (1979) has suggested that the apparently low values obtained in this annual survey are due to the time and method of assessment and indeed in NIAB trials levels of mildew on winter wheat were as high as those on spring barley (Rowe, 1979). Of cultivars for which figures are available for 1978 and 1979, only Hobbit did not show any increase in infection whereas Maris Huntsman and Flanders both showed large increases. This was surprising in view of

Hobbit's lack of identified resistance characters and the similar number of crops sampled (and therefore presumably of acreages) of each cultivar in the two years. Analysis of wheat mildew samples has been carried out with these considerations in mind. Discussion of the results assesses the evidence that diversification would indeed be useful (Bennett, 1979) in controlling powdery mildew on widely grown cultivars of wheat.

METHOD OF SAMPLE TESTING

Samples received

Samples were requested from commercial crops of 20 cultivars. Table 1 gives details of samples received, their subsequent fate and the wheat mildew resistances (WMR) and the corresponding groups to which the source cultivars belong. The importance of minimising the delay between sampling and isolate reception is shown by Table 2. The condition of two-thirds of the isolates failing to establish was classified as "poor" on arrival; only about 13 per cent of those which were successfully cultured were similarly classified and half of these subsequently died in culture. The majority of the isolates which were in good condition on arrival and yet failed to establish had only been one day in transit; they probably represent samples of poor pathogenicity which anyway would have been lost. Records show that samples from infected leaves folded in on themselves and placed in plastic bags together with damp blotting paper have the best chance of survival. If these are posted by first-class letter post early in the week, delay in transit is minimised.

Differential tests

Bulk isolates from each sample were inoculated on to detached seedling leaves of 15 differential cultivars, listed in Table 3. Differentials were the same as those used in 1978 except for the substitution of Bounty for Sportsman as the WMR 2 differential and the addition of Brigand as an extra WMR 2+6 differential. These changes are in accordance with the practice of making differential cultivars relevant to agriculture. The identified resistance genes upon which WMR groups are based are also given in Table 3.

Colony numbers were counted with an automatic colony counter. Mean pathogenicity (sensu Scott et al., 1979) of a bulk isolate on a host cultivar was assessed as the number of colonies which was produced on detached seedling leaf segments of that cultivar expressed as a percentage of the number of colonies produced on Hobbit (the susceptible control).

Table 1. Details of samples received in 1979

WMR Group	Source cultivar	Number of samples		
		Received	Failed to establish	Died in culture
0	Atou	1	0	0
	Hobbit	3	1	1
	Minister	7	5	1
	Prince	10	3	4
2	Avalon	10	5	0
	Bilbo	1	0	0
	Bounty	13	7	1
	Sentry	11	5	1
	Sportsman	3	1	0
	Wizard	10	4	1
4	Armada	1	0	0
	Anvil	9	3	1
8	Aquila	15	8	0
	Flanders	21	8	0
	Granta	14	8	2
2+6	Brigand	15	9	0
	Hustler	16	9	1
	Kinsman	14	7	2
	Mardler	15	9	3
	Maris Huntsman	16	8	1
	Marksman	1	0	0
	Virtue	13	9	1
5+8+?	Sicco	2	0	0
2+4+6	Timmo	2	0	1
	TW275	1	0	0
?	Broom	1	0	0
	Highbury	1	0	0
	Kador	2	0	1
	RPB numbered varieties	3	0	0
-	Mixture	2	0	2
	Total	233	109	24

Table 5b. Mean pathogenicity level of WMV carriers on matching WMR differentials*

Year	Matching WMV/WMR Group											
	1	2	4	5	6	7	8	9	2+4	2+6†	5+8+?	2+4+6
1976	80	88	-	-	-	116	70	115	106	59	-	-
1977	-	152	99	-	-	143	79	-	123	55	-	-
1978	-	56	101	-	40	103	71	-	-	100	-	-
1979	-	72	77	-	55 ^δ	-	91	-	-	80	-	58

* a dash indicates that no samples were obtained from that WMR group

^δ estimated from samples from WMR 2+6 sources

† Maris Huntsman

In Table 5a, figures for 1976-78 supercede those given previously (Bennett, 1979; Table 5), as they include isolate results which were not available earlier. WMV 1, 2, 5 and 8 were the most pathogenic types, as in previous years, but all were lower than 50 per cent. The higher values for WMV 1 and 2 in 1976 and 1977 may be misleading because the differential cultivars (Axminster and Ulka respectively) used in those years are more susceptible than those used currently.

The decrease in WMV 4 pathogenicity is probably temporary since WMR 4 cultivars (e.g. Armada, Anvil) are likely to become more common. The steady decline of WMV 9 can be related to the disappearance of Maris Dove from commerce. Conversely, the rapid increase of WMV 2+6 is related to the increasing popularity of WMR 2+6 cultivars. Inspection of the mean pathogenicity levels of WMV 2+6 isolates from WMR 2+6 cultivars on the matching differential (Table 5b) over the same period suggests that the increase of WMV 2+6 in the general population, probably due to greater numbers of individuals carrying the character, may have been accompanied by an increase in actual fitness on WMR 2+6 cultivars as a consequence of selection. The declining NIAB mildew resistance ratings of some WMR 2+6 cultivars on NIAB recommended lists in recent years support this view. Therefore new WMR 2+6 cultivars will be at greater risk than previously.

Behaviour of WMV 8 and WMV 2+6 in populations

The influence of host cultivar on pathogen variation is crucial to the

question of diversification. If the mean pathogenicity of non-matching WMV types is independent of host WMR group, cultivar diversification is unlikely to be of value. If, on the other hand, certain WMV types are selected against on non-matching hosts, some disease control could be expected from diversification. To study this question, the behaviour of WMV 8 and WMV 2+6, the two most relevant types, has been examined in detail.

Table 6. Effect of source cultivar on mean pathogenicity of WMV 8 and WMV 2+6 in survey samples

Source	Year	Mean pathogenicity			Number of samples
		WMV 8*	WMV 2+6*	Other WMVs†	
WMR 8	78	71	13	22	15
Cultivars	79	91	30	21	17
WMR 2+6	78	14	100	8	10
Cultivars	79	30	80	8	23
Other ^δ	78	35	14	14	42
Cultivars	79	51	29	21	19

* Differentials: WMV 8, Flanders; WMV 2+6, Huntsman

† Mean of WMV 1, 4, 5, 7, 9, 2+4, 5+8+?, 2+4+6, but omitting samples in which specific interaction occurs.

^δ 1978 - Armada, Arminda, Cerco, Hobbit, Holdfast, Iona, Kador, Little Joss, Minister, Ranger, Sentry, Stuart, Timgalen, TW256, UWWMN cv., Wizard; 1979 - Anvil, Avalon, Bounty, Hobbit, Sentry, Wizard, TW275.

Table 6 shows the effect of source cultivar on WMV 8 and WMV 2+6 pathogenicity in survey samples as measured by colony numbers. Despite its high pathogenicity in populations from all other non-matching cultivars, WMV 8 is relatively less pathogenic in populations from WMR 2+6 cultivars. This result implies that there is greater selection against WMV 8 on WMR 2+6 cultivars than on others. WMV 2+6, however, is poorly pathogenic in populations both from Flanders (WMR 8) and from other non-matching cultivars, suggesting strong selection against WMV 2+6 in the absence of WMR 2+6 cultivars. The relatively low pathogenicity of both WMV 2+6 and

of other WMV types in populations from non-matching cultivars also suggests that combinations of virulence are uncommon. It is particularly noticeable that selection against other specific WMV types is stronger in populations from WMR 2+6 cultivars than in those from all other cultivars.

Table 7. Colony numbers occurring on seedlings of Flanders, Huntsman and other differentials when placed in crops or trial plots of Flanders, WMR 2+6 cultivars and other unrelated cultivars, expressed as percentage of colony number occurring on seedlings of Minister

Source Cultivar	Exposure site	No. of sites	WMV 8	WMV 2+6	Other ^δ WMVs
Flanders (WMR 8)	Fields*	4	170	21	24
	Plots	1	138	62	-
WMR 2+6 cultivars	Fields	4	61	41	11
	Plots [†]	2	55	48	-
Other cultivars	Fields	5	73	26	33
	Plots	2	65	49	-

* Flanders fields - Ely, Longstanton, Trumpington, Waterbeach;
WMR 2+6 cultivar fields - Maris Huntsman and Mardler at Ely, Hustler at Lolworth, Kinsman at Longstanton; Other fields - Kador, Maris Dove and Sicco at Waterbeach, Sportsman at Longstanton, spring wheat plots at PBI.

[†] WMR 2+6 plots - Maris Huntsman and Kinsman; other plots - Hobbit and Iona.

^δ Same as Table 6.

Mobile nursery results obtained in 1979 lead to similar conclusions (Table 7). The pattern is essentially the same whether the exposure sites are field crops or trial plots. Selection has again operated against WMV 8 or WMR 2+6 cultivars more than on other cultivars. WMV 2+6 on the other hand appears to be relatively uncompetitive in populations from all cultivars including those from WMR 2+6 cultivars. The high value of 62 for WMV 2+6 in the Flanders plot population is artificially inflated because of an adjacent upwind plot of Huntsman. Again, other WMV types are strongly selected against on WMR 2+6 cultivars. The reason for the unusually high and low values of WMV 8 and 2+6 respectively in WMR 2+6 populations is not known; it may be due to some form of interaction with the

control cultivar, Minister instead of Hobbit in this case, or because whole seedlings instead of detached leaves were used to analyse the populations directly.

Further evidence that WMV types are selected against on non-matching hosts comes from secondary analysis of mildew samples. Isolates were taken from seedlings of Flanders, Huntsman and Hobbit exposed in PBI trial plots (including these three cultivars), and maintained on their selective cultivars. They were inoculated onto detached leaves of Flanders, Huntsman and Hobbit and colony numbers visually counted. The results are shown in Table 8 and are expressed as relative percentages, taking the colony number on Hobbit as 100 per cent. Maximum pathogenicity was always obtained on the selective cultivar. WMV 8 was less pathogenic in the samples from Huntsman than in those from Hobbit confirming the results in Tables 6 and 7.

Table 8. Colony numbers occurring on detached leaves of Hobbit, Huntsman and Flanders in a secondary analysis of samples taken from infected seedlings of Hobbit, Huntsman and Flanders. Values are expressed as percentages of colony number occurring on Hobbit

Selective Cultivar	WMR Group	Test cultivar and WMV group			Number of samples
		Flanders 8	Huntsman 2+6	Hobbit 0	
Flanders	8	110	2	100	12
Huntsman	2+6	30	117	100	8
Hobbit	0	76	50	100	14

The samples from Flanders, however, compared with those from Hobbit had lower pathogenicity levels of WMV 2+6 than expected from other results, probably because several cycles of selection had occurred on Flanders between sampling and testing. Other results have shown that WMV 2+6 is uncompetitive (Bennett, 1979 & unpublished) and under these circumstances might be expected to decline in the population. Although Hobbit has no identified resistance, it was less susceptible to isolates selected on Flanders and Huntsman than to those selected on itself suggesting that it too may be of use in a diversification scheme, especially with WMR 2+6 cultivars.

These results suggest that selection does occur against non-matching

WMV types and this effect is greatest with WMV 2+6. Other WMV types seem to behave in a similar fashion.

A preliminary analysis, similar to that done for barley mildew (Wolfe & Slater, this report) with the limited data available, shows that WMV 4, WMV 8 and WMV 2+6 are all heavily selected against on non-matching hosts and indicates that the corresponding WMR groups would all be of value in diversification schemes.

In conclusion, the spread of disease is expected to be reduced if diversification is practised with cultivars from different WMR groups, especially WMR 8 and 2+6.

REFERENCES

- BENNETT, F.G.A. (1979). Mildew of Wheat. UK Cereal Pathogen Virulence Survey 1978 Annual Report, pp. 1-13.
- ROWE, J. (1979). The use of cultivar trials records in assessing the incidence of cereal disease in England and Wales. II. Erysiphe graminis in barley, wheat and oats. Annals of Applied Biology **93**, 257-265.
- SCOTT, P.R., JOHNSON, R., WOLFE, M.S., LOWE, H.J.B. & BENNETT, F.G.A. (1979). Host-specificity in cereal parasites in relation to their control. Plant Breeding Institute Annual Report, 1978, pp. 27-62.

YELLOW RUST OF WHEAT

R H Priestley & P Byford

National Institute of Agricultural Botany, Cambridge

The results of seedling tests indicate that the United Kingdom population of Puccinia striiformis is still in a relatively stable position relative to the overall resistances R 1 - 10. The results of adult plant tests have confirmed that isolates virulent on the cultivar Hobbit are also virulent on cultivars Brigand and Kador. Brigand and Kador have therefore been removed from diversification group DG 1 and placed in DG 6. Results from Polythene tunnel tests carried out during the period 1975-79 are presented as evidence for the effectiveness of cultivar diversification in reducing the spread of P. striiformis.

INTRODUCTION

The principal aim of the wheat yellow rust survey is the early detection of increased virulence in Puccinia striiformis compatible with resistances being exploited in commercial cultivars and breeding lines. Methods of detecting increased virulence and the current UK detection system have been described by Priestley (1978). Table 1 shows the specific resistances (WYR factors) identified in wheat cultivars to date, the resistance genes where known, a test cultivar possessing each resistance and the year of first detection of virulence (WYV) in the UK population of P. striiformis. If increased virulence is not found after a few years, it is an indication that the resistance may be of a durable nature.

VIRULENCE TEST METHODS

Seedling tests with 1979 isolates

A total of 52 samples was received by post. This is fewer than 1978 (168 samples) but greater than 1977 (39 samples). Samples were collected in a non-random way from Hobbit (7 samples), Kinsman (4), Kador (4), Maris Huntsman (3) and 36 other cultivars. Isolates were made from 32 samples; the remainder failed to sporulate after inoculation on to seedlings of the universally susceptible cultivar Sappo. Tests were carried out on all 32 isolates to identify virulences compatible with the overall resistances R 1 - R 10. Two isolates were found to be

Table 1. Resistance factors to *P. striiformis*

WYR factor	Gene	Type*	Test cultivar	WYV detected
WYR 1	Yr 1	overall	Chinese 166	1957
WYR 2	Yr 2	overall	Heine VII	1955
WYR 3	-	overall	Vilmorin 23	1932
WYR 4	Yr 3b + 4b	overall	Hybrid 46	1965
WYR 5	Yr 5	overall	<u>T. spelta album</u>	.
WYR 6	-	overall	Heine Kolben	1958
WYR 7	Yr 7	overall	Lee	1971
WYR 8	Yr 8	overall	Compair	1976
WYR 9	-	overall	Riebesel 47/51	1974
WYR 10	-	overall	Moro	.
WYR 11	-	adult plant	Joss Cambier	1971
WYR 12	-	adult plant	Mega	1969
WYR 13	-	adult plant	Maris Huntsman	1974
WYR 14	-	adult plant	Hobbit	1972

* sensu Zadoks (1961); overall resistances are effective at all growth stages, adult plant resistances are ineffective at seedling growth stages.

. = virulence not yet detected.

mixtures and have been excluded from the virulence frequency calculations.

Adult plant tests with 1978 and control isolates

Virulences compatible with both overall and adult plant resistances were identified in 24 isolates and an isolate mixture using the Polythene tunnel technique described by Priestley & Byford (1978). Tussocks of 36 cultivars were sown on 23-24 November 1978, inoculated on 20 March and 11 April 1979, and assessed for percentage leaf area infection using the International Scale (Doling, 1967) on 10 May (GS 32-37), 24 May (GS 45-58), 7 June (GS 64-68) and 21 June (GS 75-85).

The isolates comprised seven controls of known virulence, fifteen collected in 1978 from plants with a greater than expected disease level, two from various inoculated plots and a mixture of sixtytwo other 1978 isolates (Table 2). Plants in two tunnels were inoculated with isolate 76/71 to measure between-tunnel variation. These are referred to as 76/71A and 76/71B in later Tables.

Table 2. Isolates used in adult plant tests

Code	Cultivar	Region/	Site	WYV factors*
<u>Control isolates</u>				
71/493	Capta	Sc	Duns	WYV 1, 2, 3, 7
72/852	Maris Ranger	EM	Market Harborough	WYV (2), 3, 4, 6
75/109	Kinsman	WM	Harper Adams	WYV 2, 3, 4, 6
76/15	Clement	EM	Boston	WYV 2, 3, 4, 8, 9
76/71	Grenade	Sc	Mains of Ravensby	WYV 1, 2, 3
77/20	Maris Ranger	E	PBI Trial Ground	WYV 1, 2, 3, (4), 6
77/26	Hobbit	EM	Tydd St Mary	WYV 1, 2, 3
<u>1978 Isolates</u>				
78/11	Waggoner	EM	Holbeach	WYV 1, 2, 3
78/14	Armada	EM	Alford	WYV 1, 2, 3, (4), (6)
78/77	Hobbit	SE	Cholsey	WYV 1, 2, 3, (6), (7)
78/79	Kador	EM	Boston	WYV 1, 2, 3
78/89	Kinsman	E	Terrington	WYV 2, 3, 4, 6
78/100	Mardler	E	Boxworth	WYV 1, 2, 3
78/112	Hobbit	YL	Othringham	WYV 1, 2, 3
78/118	Kinsman	WM	Kireton	WYV (2), 3, 4, 6
78/129	Kinsman	E	Friskney	WYV 2, 3, 4, 6
78/131	Venture	EM	Kelstern	WYV 1, 2, 3
78/133	Hobbit	SE	Finstock	WYV 1, 2, 3
78/135	Kador	EM	Gt Brington	WYV 1, 2, 3
78/138	Kador	WM	Pershore	WYV 1, 2, 3
78/141	Maris Huntsman	Sc	North Berwick	WYV 1, 2, 3
78/150	Mega	E	Morley	WYV 2, 3, 4, 6

Other Isolates

PBI/75/27	Hobbit inoculated with WYR 72/23	WYV 2, 3, 4
77/A5	Hustler inoculated with isolated mixture	WYV 1, 2, 3
mix	Mixture of 62 1978 isolates	

() partially virulent on corresponding resistance

* virulence compatible with overall resistances (WYR 1 - 10) shown only (see Table 1).

/ Sc, Scotland; EM, East Midlands; YL, Yorks & Lancs; WM, West Midlands; E, East; SE, South East.

Table 3. Virulence factor frequency (%)

V factor	Common name	1976	1977	1978	1979
V 1	Chinese 166 virulence	92	73	73	83
V 2	Heine VII virulence	100	100	97	100
V 3	Vilmorin 23 virulence	100	100	100	100
V 4	Hybrid 46 virulence	12	24	27	17
V 5	<u>T. spelta album</u> virulence	0	0	0	0
V 6	Heine Kolben virulence	4	16	26	17
V 7	Lee virulence	0	8	0	0
V 8	Compair virulence	2	4	0	0
V 9	Riebesel 47/51 virulence	6	0	0	0
V 10	Moro virulence	0	0	0	0
	number of isolates tested	52	26	66	30

VIRULENCE TEST RESULTS

Seedling tests with 1979 isolates

Sampling was not carried out on a random basis and thus the virulence frequencies shown for 1976-79 (Table 3) should be interpreted with caution. Comparable data for the period 1970-75 was given by Priestley & Byford (1976). The 1979 data confirms the view postulated earlier (Priestley & Byford 1979) that the U.K. population of P. striiformis has reached a relatively stable position relative to the overall resistances WYR 1 to WYR 10. It is appreciated that the situation could change rapidly should changes occur in the relative popularity of cultivars possessing these resistances.

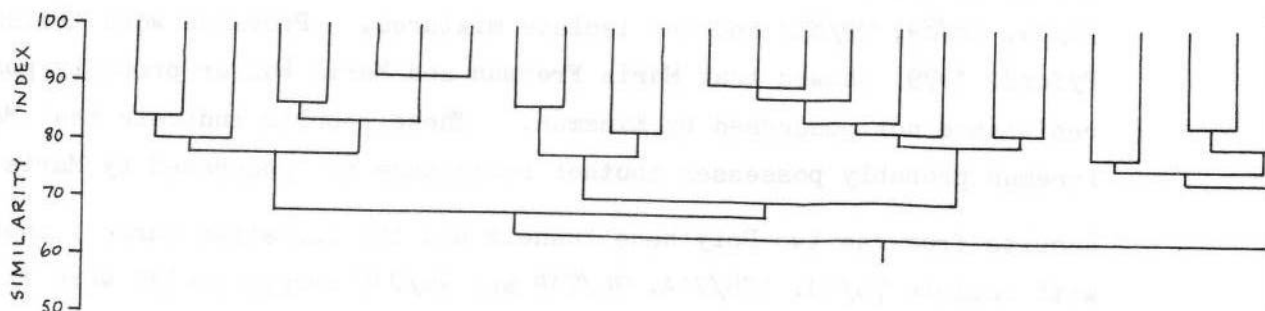
Adult plant tests with 1978 and control isolates

The results of the adult plant tests are given (Table 4). Values are mean percent leaf area infection calculated from two replicate tussocks assessed on four occasions. Underlined values show the position in the matrix of those two-factor residuals that are significantly ($P = 0.1$) greater than zero. These indicate a large positive interaction between the cultivar and isolate concerned. The order of cultivars and isolates in the table and the similarity dendrograms have been derived from average linkage cluster analysis of the two-factor residuals as described by Priestley & Byford (1979). Some rotation of stems of the dendrogram has been carried out as described by Sneath & Sokall (1973). The boxes are used solely to identify particular areas of the matrix; values within boxes have no particular statistical

Table 4. Results of adult plant tests 1979

Values are mean percent leaf area infection. For explanation of boxes, dendrograms and underlined values, see text.

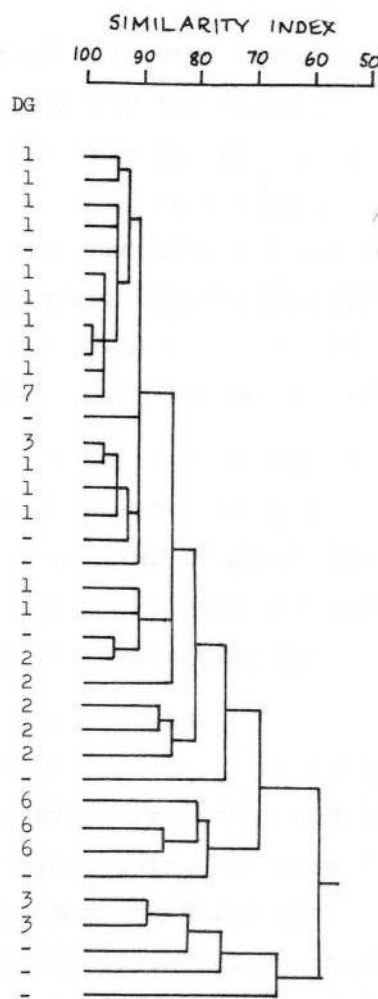
Isolate:		78/ 129	78/ 100	76/ 15	71/ 493	78/ 11	77/ A5	76/ 71A	78/ 131	76/ 71B	78/ 141	76/ 14	78/ 71C	78/ 79	76/ 112	78/ 77	75/ 133	78/ 27	77/ 135	78/ 26	75/ 138	78/ 150	75/ 109	78/ 118	78/ 89		
Cultivar	WYR factors																										
Bouquet	WYR var	0	0	0	1	0	1	0	0	1	1	0	0	2	1	1	3	1	4	6	6	1	1	1	0		
Anvil	WYR var	2	1	1	1	1	3	2	1	5	3	1	1	3	3	3	3	3	4	3	4	3	2	2	1		
Flanders	WYR 1, var	1	1	1	1	3	3	3	3	2	5	3	1	2	3	1	3	2	3	1	4	2	1	1	1		
Granta	WYR 1, var	0	0	0	3	1	1	1	1	0	1	2	1	1	1	1	1	0	4	0	3	1	0	0	0		
Authari	WYR 0	0	2	0	0	0	2	1	2	2	2	1	1	2	1	0	1	0	3	1	3	3	1	1	2		
Atou	WYR var	0	0	0	0	1	0	1	0	0	1	0	0	1	0	0	1	0	2	2	0	1	0	0	0		
Flinor	WYR 0	0	1	1	1	0	1	0	0	1	1	1	0	1	1	0	1	0	1	3	1	1	1	2	0		
Aquila	WYR 2, var	0	0	0	1	0	0	0	0	2	0	0	0	0	0	0	1	1	1	0	0	2	0	2	0		
Bounty	WYR 1	0	0	0	0	0	1	0	0	2	2	0	1	0	0	0	0	0	1	0	0	2	0	1	0		
Avalon	WYR 4	0	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1	1	1	0		
Stuart	WYR 9	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0		
Clement	WYR 9	0	0	<u>A 2</u>	0	1	1	0	0	0	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	0	
Maris Freeman	WYR 6	1	0	0	0	1	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	0	1	0	0	0	
Armada	WYR 3,12,var	1	0	3	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1	1	6	3	6	1		
Sentry	WYR 4,12?	0	0	2	0	1	0	0	0	0	0	0	1	0	0	0	0	0	1	0	2	2	4	0	5	2	
Wizard	WYR 4,12?	1	0	1	0	0	0	0	0	0	0	1	0	0	0	0	0	2	0	3	0	3	3	2	0		
Mega	WYR 12,var	0	0	0	0	1	0	0	1	3	1	1	1	0	0	1	1	0	2	0	2	6	0	2	1		
Tommy	WYR 7	0	0	0	<u>26</u>	0	1	1	1	0	1	1	0	0	0	0	0	0	1	1	0	1	0	1	1		
Mardler	WYR 1,2,3	0	4	0	1	1	5	3	2	8	9	6	9	1	1	1	2	0	4	1	4	7	0	0	0		
Prince	WYR 3	1	3	1	3	2	5	6	4	3	7	8	10	4	1	3	5	2	9	5	8	8	3	4	1		
Cappelle-Desprez	WYR 3	0	0	2	2	2	4	5	3	5	8	1	4	2	2	1	3	1	3	3	5	2	1	1			
Sportsman	WYR 1	0	3	0	1	0	4	3	1	<u>B</u>	4	5	3	2	1	1	1	0	4	2	2	0	0	0	0		
Maris Huntsman	WYR 2,i3	2	3	2	2	1	4	2	8	<u>18</u>	10	8	4	4	5	3	6	0	8	6	3	6	8	4	5		
Hustler	WYR 1,2,13	0	7	0	1	1	<u>13</u>	8	<u>15</u>	<u>12</u>	<u>13</u>	10	<u>13</u>	2	2	1	3	0	3	1	2	8	0	0	0		
Copain	WYR 13	1	6	1	2	4	<u>8</u>	6	<u>13</u>	<u>11</u>	<u>12</u>	9	<u>15</u>	2	4	2	2	1	3	4	3	3	10	5	10		
Virtue	WYR 1,13	0	5	0	2	7	<u>10</u>	8	<u>10</u>	<u>10</u>	<u>11</u>	<u>18</u>	<u>18</u>	3	7	3	4	1	6	4	9	2	0	2	0		
Maris Templar	WYR 1,3	0	3	0	6	17	9	9	10	9	12	15	10	6	7	7	5	0	10	14	17	6	0	1	0		
Hobbit	WYR 14	0	0	0	1	1	0	3	0	3	5	1	2	8	<u>14</u>	<u>10</u>	<u>13</u>	<u>10</u>	<u>14</u>	<u>25</u>	<u>10</u>	2	2	0	1		
Kador	WYR 14	0	3	1	5	6	5	5	7	6	7	7	2	10	7	5	12	11	<u>12</u>	<u>14</u>	<u>17</u>	7	6	4	3		
Brigand	WYR 3,14	0	0	1	3	1	2	1	1	5	3	1	1	5	5	4	5	<u>C</u>	3	7	10	10	3	2	1		
Maris Bilbo	WYR 14	2	3	5	6	6	10	8	10	7	10	12	11	17	14	13	<u>20</u>	<u>18</u>	<u>15</u>	<u>13</u>	<u>23</u>	12	8	12	11		
Kinsman	WYR 6	2	1	0	1	0	2	0	0	1	0	0	0	0	2	1	1	0	1	0	2	10	14	12	9		
Maris Ranger	WYR 6	2	0	0	0	1	0	0	0	0	0	0	0	<u>E</u>	<u>11</u>	<u>5</u>	1	1	0	0	0	1	0	2			
Waggoner	WYR 3,12,var	2	3	5	3	3	4	3	4	7	6	3	3	3	1	5	3	7	8	8	6	12	12	9	3		
Maris Beacon	WYR 4	0	0	7	0	0	0	0	0	1	0	3	0	0	1	0	0	10	1	0	0	<u>24</u>	<u>23</u>	9	<u>13</u>		
Michigan Amber	WYR 0	8	5	6	10	9	12	8	7	18	14	23	21	9	10	8	11	<u>23</u>	10	19	<u>26</u>	<u>26</u>	<u>34</u>	21	<u>26</u>		



stance factors ascribed to each cultivar are not only on results presented here but also on sly. Diversification Group (DG) numbers are lvars.

1979 adult plant tests were lower than those carried despite this, clear evidence of cultivar-isolate

ix 77/ 72/
20 852



Granta, Stuart and Prince) were included in the

The resistance of Avalon and Granta was highly plates. Isolate 76/15 produced relatively high Stuart and cultivar Clement (WYR 9) indicating that resistance (Box A). Prince resembles cultivars and Sportsman in that isolates virulent on them virulent on cultivars Maris Huntsman, Hustler, R 13).

and the earlier postulation (Priestley & Byford, 1979) cultivar Hobbit (WYR 14) also tend to be virulent C). These isolates are also virulent on cultivar or have therefore been removed from DG 1 and placed

own that isolate 75/109 produced a higher level of Kinsman than on cultivar Maris Ranger or Maris (rd), 1978, 1979). The results shown in Table 4 reveal previously unknown differences between Maris

A number of isolates produce relatively high ris Ranger (Boxes D and E) but relatively low ris Freeman. Examples of this effect are isolates the isolate mixtures. Previous work (Priestley & Maris Freeman and Maris Ranger probably possess a by Kinsman. These results indicate that Maris s another resistance not possessed by Maris Ranger.

these tunnels and the isolation nursery inoculated 1A, 76/71B and 76/71C respectively) were fairly s revealed the increased virulence of this isolate eties possessing R 13 (Box B).

IVENESS OF CULTIVAR DIVERSIFICATION

civar diversification in reducing disease spread in

Table 5. Mean percent leaf area infection produced by isolates of *P. striiformis* collected from, and inoculated onto, wheat varieties possessing particular specific resistances.

Figures in parenthesis are number of isolates used.

		isolates collected from varieties possessing:		
	varieties inoculated	R 6	R 13	R 14
R 6	Kinsman	7.2 (12)	2.4 (18)	0.9 (10)
	Maris Freeman	4.7 (12)	0.9 (18)	0.2 (10)
	Maris Ranger	5.5 (12)	1.4 (18)	0.6 (10)
R 13	Maris Huntsman	3.2 (12)	7.3 (18)	4.0 (10)
	Sportsman	0.7 (11)	4.0 (15)	1.2 (10)
	Hustler	1.1 (8)	14.3 (9)	1.8 (10)
R 14	Hobbit	1.8 (12)	1.0 (18)	13.1 (10)

the field depends on the extent to which non-corresponding virulence can survive in the pathogen population. For example, inoculum generated by a cultivar possessing R x will tend to consist largely of the corresponding virulence V x. However, a certain proportion may also possess non-corresponding virulence such as V y. If the frequency of V y is small, then inoculum generated by cultivars possessing R x will be largely non-virulent on adjacent fields and plants of cultivars possessing R y. Thus, diversification between R x and R y will be effective. If, however, the non-corresponding virulence V y is frequently generated on R x, then diversification between R x and R y will be much less effective.

Table 5 shows infection levels produced by isolates collected from, and inoculated onto, varieties possessing resistances R 6, R 13 and R 14. The results are taken from adult plant polythene tunnel tests carried out during 1975 to 1979. It can be seen that the frequencies of non-corresponding virulences are low. For example, infection levels resulting from inoculation of varieties possessing R 6 are lower with isolates collected from varieties possessing R 13 or R 14 than with isolates from varieties possessing R 6. Thus the frequency of V 6 generated on R 13 and R 14 must be relatively small. Similarly, V 13 must be relatively uncommon on varieties possessing R 6 or R 14, V 14 on varieties possessing either R 6 or

R 13. Diversification between varieties possessing those resistances is therefore likely to be effective in reducing disease spread between adjacent fields or in variety seed mixtures. The use of cultivar diversification on this and other scales has recently been described (Priestley, 1979).

REFERENCES

- DOLING, D. A. (1967). Evaluation of the reaction to yellow rust, Puccinia striiformis, of wheat varieties, 1957 - 66. Journal of the National Institute of Agricultural Botany, 11, 80-90.
- PRIESTLEY, R. H. (1978). Detection of increased virulence in populations of wheat yellow rust. In Plant Disease Epidemiology. Ed P R Scott & A Bainbridge, pp 63-70. Blackwell Scientific Publications, Oxford.
- PRIESTLEY, R. H. (1979). The management of resistant varieties. Proceedings 1979 British Crop Protection Conference - Pests and Diseases, 3, 753-760.
- PRIESTLEY, R. H. & BYFORD, P. (1976). Yellow rust of wheat. Physiologic Race Survey (Cereal Pathogens) 1975 Annual Report, 6-13.
- PRIESTLEY, R. H. & BYFORD, P. (1978). Yellow rust of wheat. United Kingdom Cereal Pathogen Virulence Survey 1977 Annual Report, 3-11.
- PRIESTLEY, R. H. & BYFORD, P. (1979). Yellow rust of wheat. United Kingdom Cereal Pathogen Virulence Survey 1978 Annual Report, 14-24.
- SNEATH, P. H. A. & SOKAL, R. R. (1973). Numerical taxonomy. W H Freeman & Co., San Francisco.

APPENDIX

Identification of isolates from the UK and Eire section of the International Survey of Factors of Virulence of Puccinia striiformis

The following virulences have been identified:

Isolate number	Cultivar	Virulence factors	World and European race number
79/29	Tadorna	V1,2,3	41 E 136
79/30	Lutin	V1,2,3	41 E 136
79/32	<u>Triticum Spelta Saharensense</u>	V1,2,3	41 E 136
79/33	Cama	V1,2,3	41 E 136
79/34	Nugaines	V1,2,3	41 E 136
79/35	Persian	V1,2,3	41 E 136
79/36	Lucas	V1,2,3	41 E 136
79/37	Heines Vll	V1,2,3	41 E 136
79/38	Kormora	V1,2,3	41 E 136
79/39	Bon Fermer	V1,2,3	41 E 136
79/40	Harvest Queen	V1,2,3	41 E 136
79/41	Nuranowskja 808	V1,2,3	41 E 136
79/45	Libellula	mixture*	-
79/48	Saturn	V1,2,3	41 E 136

*V1,2,3 and V1,3,4,6

Six other samples were received but they failed to sporulate after inoculation onto the susceptible cultivar Sappo.

BROWN RUST OF WHEAT

R.H. Priestley

National Institute of Agricultural Botany, Cambridge

E.R.L. Jones & B.C. Clifford

Welsh Plant Breeding Station, Aberystwyth

Only twelve disease samples were received in 1979. Results from adult plant nurseries grown and inoculated in the open at the Welsh Plant Breeding Station in 1979 and in Polythene tunnels at the National Institute of Agricultural Botany from 1976-1979 were complementary, despite a few anomalies. Clear evidence of three specific resistance factors (WBR 1, WBR 2 and WBR 5) was found. Corresponding virulence for all three was found amongst the isolates sent in to the Survey.

SEEDLING TESTS WITH 1979 ISOLATES

Only 12 disease samples were received during 1979. Isolates of Puccinia recondita were made from five of the samples collected from the cultivars Maris Huntsman (2), Mardler (2) and Brigand (1). Four of these were virulent on Mega, Aquila, Sentry, Galahad and Mithras but non-virulent on Clement, Sterna, Sappo, Maris Halberd, Hobbit, Wizard, Norman, Baron and Abele. The fifth (79/4) had additional virulence on Sterna, Wizard, Norman, Baron and Abele but was non-virulent on Galahad. Isolates could not be made from the remaining seven disease samples.

ADULT PLANT TESTS

Adult plant tests in which plants were inoculated with specific isolates of P. recondita (Table 1) were carried out in field isolation nurseries either in Polythene tunnels (National Institute of Agricultural Botany, Cambridge) or in the open (Welsh Plant Breeding Station, Aberystwyth). Results from the 1979 WPBS nurseries are given in Table 2. Results from the 1979 NIAB nurseries are given in Table 3 together with results from earlier tests in 1976, 1977 and 1978. The boxes in Table 3 delimit mean percent leaf area infection values produced by certain isolates that are relatively large in comparison with those produced on the same cultivars by other isolates. This is taken to indicate that the cultivars concerned share one or more specific resistances to P. recondita and that the isolates causing relatively high levels of infection possess the corresponding specific virulences. Specific

Table 1. Isolates used in adult plant tests

Isolate code	Cultivar	Location
61/37	?	Plant Breeding Institute, Cambridge
73/7	Maris Nimrod	Somerset
74/2	Maris Huntsman	Morley, Norfolk
74/11	Maris Fundin	Seale Hayne, Devon
77/9	Maris Ranger	WPBS nursery inoc with 76/1
77/22	Aquila	North Coates, Humberside
78/5	Aquila	Romney Marsh, Kent
78/A2	Maris Ranger	NIAB Poly tunnel inoc with 77/9
77/15	Sportsman	WPBS nursery

resistances (R) are numbered sequentially with the prefixes W (Wheat) and B (Brown rust), hence WBR 1. The genetic origin of these resistances is unknown. Specific resistances are further classified in the text as being of the 'overall' or 'adult plant' type (sensu Zadoks, 1961).

In general, the results from the nurseries grown under the two sets of conditions are complementary. Some anomalies occur, for example on Maris Fundin, Maris Bilbo and Maris Templar. Previous work (Clifford, Jones & Priestley, 1977) has shown that the expression of resistance in Maris Fundin is temperature sensitive. As all three cultivars are closely related, some of the anomalies may be due to a different expression of resistance under the warmer Polythene tunnel conditions. Some anomalous results from nurseries grown in the open may be due to cross-contamination of isolates. This is much less of a problem in adjacent Polythene tunnels because the walls mechanically prevent the dispersal of spores between tunnels. Evidently, neither type of test is entirely satisfactory.

The results confirm that Clement and Stuart have the same overall resistance (WBR 1). The resistance of Aquila shows a similar breadth of effectiveness but it is expressed only at the adult plant stage. This, and its parentage, suggest that the resistance is genetically different from that of Clement and Stuart which derives from the wheat-rye translocation line Mildress.

Resistance WBR 2, which is temperature sensitive, is present in Maris Fundin and also appears to be present in the related cultivars Maris Bilbo and Maris Templar. Maris Huntsman, Brigand and Mardler all appear to possess the adult plant resistance WBR 5. The picture is less clear with other largely

Table 2 Results of adult plant tests at WPBS, 1979

Values are mean percent leaf area infection from 4 replicate plots assessed on two dates. Letters are reaction type (S = susceptible; MS = moderately susceptible; MR = moderately resistant; R = resistant; R-S = variable due to temperature sensitivity).

Cultivar	isolate				Mean
	77/9	77/15	77/22	74/2*	
Aquila	6MS	11S	20S	17S	-
Clement	13S	22S	29S	23S	-
Bounty	1R	2MR	5MS	6MR	-
Sentry	6MS	8MS	10MS	7RS	-
Wizard	7MS	8MS	14S	4MS	-
Kinsman	7MR	8R	13MS	7R	-
Sportsman	1R	1R	8MS	4MR	-
Hobbit	3R	5R	6MS	6MR	-
Avalon	1R	2R	6MS	6MS	-
Norman	3R	2R	7MS	6MS	-
Mithras	6MR	5MR	9MS	8MS	-
Virtue	0	1R	1R	1R	-
Sterna	0	0	1R	0	-
Hustler	0	0	1R	1R	-
Maris Huntsman	11MS	9MS	13S	13S	-
Brigand	10MS	6MS	10MS	11MS	-
Galahad	8MS	9MS	9MS	6MS	-
Mardler	11MS	12MS	12MS	10MS	-
Maris Fundin	12R-S	16R-S	16R-S	16R-S	-
Maris Ranger	8S	15S	16S	-	-
Mega	9MS	14S	18S	11S	13.0
Bouquet	7S	14S	20S	13S	13.5
Atou	7MS	14MS	19S	14S	13.5
Anvil	6S	13S	25S	16S	15.0
Flinor	11S	18S	20S	11S	15.0
Maris Freeman	11S	14S	22S	12S	17.2
Waggoner	6MS	18S	28S	19S	17.7
Cappelle Desprez	13S	14S	24S	21S	18.0
Granta	11S	21S	24S	16S	18.0
Champléin	14S	19S	25S	21S	19.7
Prince	19S	17S	26S	25S	21.7
Armada	22S	26S	28S	25S	25.2
Copain	12S	22S	32S	29S	26.2

*contaminated; results atypical

Table 3 Results of adult plant tests at NIAB, 1976-1979

Values are mean percent leaf area infection usually from 2 replicate plots assessed on at least four dates. Boxes delimit infection values that are relatively large in comparison with those produced by other isolates on that cultivar. Results for isolate 77/22 in 1979 excluded.

Resistance factor	Cultivar	Isolate 73/3		74/2			61/37		74/11		77/9	78/A2	77/22	78/5
		year of test	76	76	77	78	79	76	76	79	78	79	78	79
WBR 1	Clement	1	0	0	1	3	0	0	1	18	16	39	26	
	Aquila*	.	.	1	1	0	.	.	1	12	10	25	9	
	Stuart	3	.	.	0	.	17	.	17	
WBR 2	M Fundin	1	2	9	.	.	28	40
	M Bilbo	.	.	3	2	2	.	.	18	26	4	13	.	0
	M Templar	.	.	4	3	.	.	.	4	17	12	19	.	4
WBR 5	M Huntsman	17	27	25	24	16	9	10	2	19	4	7	4	
	Mardler	.	.	19	28	14	.	.	2	20	7	4	3	
	Brigand	.	.	.	34	13	.	.	2	13	0	8	0	
WBR ?	M Ranger	0	1	3	1	0	0	1	1	12	12	12	8	
	Kinsman	0	1	2	3	0	0	0	0	5	7	5	6	
WBR ?	Sportsman	0	1	0	1	0	0	1	0	14	0	3	0	
	Bounty	.	.	.	1	0	.	.	0	6	0	3	0	
	Avalon	0	.	.	0	.	0	.	0	
	Hustler	.	.	0	1	0	.	.	0	0	0	0	0	
	Virtue	.	.	.	2	0	.	.	1	0	0	1	0	
	Hobbit	0	1	1	2	0	0	1	1	3	6	1	0	
	Wizard	.	.	.	8	0	.	.	3	5	3	5	1	
	Bouquet	1	2	13	14	3	5	3	3	4	1	7	1	
	Sentry	.	.	.	12	5	.	.	2	5	3	5	1	
	Flinor	2	4	7	15	3	0	5	2	7	3	10	2	
	Prince	4	.	.	2	.	7	.	4	
	Granta	2	.	.	4	.	7	.	6	
	M Freeman	3	6	12	15	2	5	7	1	11	6	12	2	
	Atou	3	4	8	18	2	5	5	7	10	6	15	3	
	Flanders	6	15	16	17	8	6	13	3	11	13	16	5	
	Kador	5	12	14	22	9	1	6	4	12	15	21	9	
	Anvil	.	.	.	25	5	.	.	5	18	5	18	3	
	Armada	.	.	22	32	15	.	.	11	34	13	29	9	
	Copain	.	.	.	33	23	.	.	25	31	20	30	13	

. = not tested

* = resistance not identical with other cultivars possessing WBR 1 (see text)

resistant varieties. Sportsman, Kinsman and Bounty responded similarly in the isolation nurseries grown in the open in 1979, being susceptible only to isolate 77/22 (Table 2), whereas in the nurseries grown in Polythene tunnels in 1976-79 Sportsman appeared similar to Bounty and Kinsman, and Maris Ranger appeared similar to Clement, Stuart and Aquila. Hustler and Virtue were very resistant to all isolates in nurseries grown under either condition. Some variation in pathogenicity for Avalon, Hobbit and Wizard was found. This is being investigated further.

REFERENCES

- CLIFFORD B.C. JONES E.R.L. & PRIESTLEY R.H. (1977). Brown Rust of Wheat. Physiologic Race Survey (Cereal Pathogens) 1976 Annual Report, 21-24
- ZADOKS J.C. (1961). Yellow rust studies in epidemiology and physiologic specialization. Tijdschrift over Plantenziekten 67, 69-256.

MILDEW OF BARLEY

M S Wolfe and Susan E Slater

Plant Breeding Institute, Cambridge

There were few additions to the known resistance groups or pathogenicity factors. The occurrence has yet to be confirmed of mlo in a commercial cultivar, Atem. Pathogenicity corresponding with BMR 6 + Ab 12 in Triumph was recorded for the second year.

A review of the pathogen population structure on the major resistance groups of barley cultivars revealed a relatively consistent pattern for the past five years. Analysis of the survival of pathogenicity factors on non-corresponding hosts illustrated the greater value of some groups for use in cultivar diversification and mixing. The analysis also revealed further evidence of specific interactions between certain BMR groups and non-corresponding BMV groups.

Provision of a large amount of material from the north of England facilitated analysis of geographical variation in pathogen populations dependent on cultivar distribution over the areas involved. BMV 3 and BMV 6 varied reciprocally, BMV 3 being more common and BMV 6 less so, in the north than in the south.

A comparison for the past three years of conventional sampling versus the use of mobile nurseries for the analysis of pathogen populations showed similar outcomes from both methods. The mobile nurseries also provided more evidence for background or unidentified resistance.

A total of 221 bulk isolates was received in 1979, of which 60 failed to establish. Table 1 shows the number of samples received from each cultivar and the BMR group to which they belong.

New identifications

No new resistance genes or pathogenicity factors were identified in the survey.

Table 1. Number of samples received in 1979, and BMR group definitions

BMR group	Gene	Cultivar and number of samples
0	-	Golden Promise (20) Maris Otter (1)
1	'Mlh' (2 genes)	a) Igri (3) b) Gerbel (2), Mammut* (-)
2	Mlg (2 genes)	Julia (15) Zephyr (1)
3	Mla6	Midas (15)
4	Mlv (2 genes)	Lofa (16) Aurea (5)
5	Mlas	Hassan (16)
6	Mla4/7 (2 genes)	Wing (14) Ark Royal (2) Keg (2)
7	Mla	Tyra (14)
8	Mla4/9 (2 genes)	Simon (1)
2+3		Impala (1)
2+4		Sundance (6) Koru (6) Georgie (5) Flare (1) Cerise* (-)
2+5		Porthos (7) Athos (5) Tintern* (5) Aramir (2) Piccolo (1)
2+5+?		Maris Mink (14)
2+7		MG 109/74* (-)
2+8		RPB475/74* (-)
3+4		Goldmarker (6) Jupiter (2)
4+5		Egmont* (1)
4+6		Dram* (4) Claret (2) Belfor (1)
4+?		Magnum (15)
6+Ab		Triumph (3)

*new and confirmed identifications in 1979

However, MG 109/74 and RPB 475/4 represent new BMR groups in that they combine Mlg respectively with Mla and Mla9. The cultivars Atem and Rendo are thought to possess Mlv1, 2 (Vada resistance) combined with mlo (Ir L Groenewegen, pers. comm.). In tests they were resistant to all isolates used but occasional isolates obtained from them were pathogenic on BMR4 (Mlv1/2).

Table 2 provides the complete data for the samples sent in during 1979. The number of test differentials used was increased to include more cultivars

with combined resistance factors for testing isolates obtained from those cultivars grown in the field. Isolates obtained from recently introduced cultivars with combined resistance factors (Jupiter, Goldmarker, Triumph and Dram) produced relatively fewer colonies on their corresponding seedling test cultivars than did isolates from older established cultivars on their 'own' test cultivars.

Isolates pathogenic on Triumph were again obtained from this new cultivar, and the same pathogenicity was detected at low levels on other cultivars, particularly those in BMR 6.

Mean pathogenicity of bulk isolates

Since 1975, pathogen isolates have been treated as bulk samples, except where information on the performance of particular genotypes has been required. This has considerably reduced the amount of work involved in assessing each sample, although some loss of information is involved. It has, however, facilitated more extensive sampling of the pathogen population which is a primary objective of the survey.

The major loss of information with this technique relates to the accuracy with which the frequency of genotypes with combinations of pathogenicity factors can be assessed. However, the data in Table 8 indicate that this presumed loss of accuracy may not be serious since the pathogenicity of bulk isolates on cultivars with combined resistance factors relates closely to expected performance derived from their pathogenicity on cultivars that carry the resistance factors separately.

The method of measuring the mean pathogenicity of a bulk isolate on a host cultivar is by counting the colony number produced on detached seedling leaf segments of that cultivar and of the susceptible control cultivar, Golden Promise. The colony number on the test cultivar is then expressed as a percentage of that on Golden Promise. This estimate does not distinguish between the components of mean pathogenicity: the frequency of individuals with host-specific pathogenicity matching the test cultivar, the proportion of those individuals whose pathogenicity is sufficient to cause recognisable colonies, and the size distribution of those colonies.

Pathogen population dynamics

At the beginning of an epidemic, when fields are being infected by spores

Table 2. Mean pathogenicity[†] of bulk isolates on test seedlings of BMR group cultivars, omitting isolates which were non-pathogenic on the cultivar from which they were collected

BMR group	Source of isolate	Number of pathogenic isolates	BMV group represented by test seedlings*															
			1	2	3	4	5	6	7	8	2+3	2+4	2+5	2+5+?	2+6	3+4	4+6	6+Ab
0	Maris Otter Golden Promise	1 13	44 46	46 65	31 46	19 27	37 32	24 28	0 6	0 1	24 24	7 5	52 27	3 20	44 32	2 2	1 5	8 3
1	Igri Gerbel	3 2	<u>41</u> 52	* 54	36 8	29 2	20 16	48 39	0 0	0 0	24 2	25 6	11 19	13 14	28 34	16 0	2 2	4 2
2	Zephyr Julia	1 13	29 43	<u>57</u> <u>73</u>	6 23	0 18	33 35	24 21	0 0	0 2	7 23	0 16	46 40	27 33	25 33	0 3	0 1	11 2
3	Midas	12	45	59	<u>68</u>	18	29	28	<1	0	72	11	27	21	24	6	1	1
4	Aurea Lofa	1 13	32 28	58 64	66 27	<u>69</u> <u>71</u>	8 27	1 6	0 0	0 0	63 26	58 44	4 32	3 20	0 10	42 21	<1 4	0 1
5	Hassan	13	43	66	18	7	<u>57</u>	8	<1	<1	16	7	57	48	7	3	1	1
6	Ark Royal Keg Wing	2 2 11	44 57 49	67 59 68	0 17 24	2 7 4	37 23 16	<u>91</u> <u>75</u> <u>73</u>	0 0 0	0 0 7	0 17 22	<1 1 1	20 42 16	19 6 13	87 71 67	0 4 2	5 4 2	3 21 5
7	Tyra	11	51	66	22	3	47	15	<u>75</u>	<1	20	2	49	37	12	<1	<1	2
8	Simon	1	70	44	0	0	15	82	0	<u>124</u>	0	0	23	9	57	0	2	3
2+3	Impala	1	68	<u>73</u>	<u>83</u>	12	65	3	0	0	<u>80</u>	4	49	76	5	11	0	0

Table 2 (contd.)

2+4	Georgie	1	48	63	27	53	11	0	0	0	29	56	13	1	0	9	0	0
	Koru	1	60	79	0	60	62	0	0	0	0	5	30	5	0	0	0	0
	Flare	1	28	51	41	61	0	0	0	0	54	39	0	0	0	39	0	0
	Sundance	4	22	62	53	54	27	11	0	0	58	65	44	9	8	31	<1	2
2+5	Aramir	2	47	73	1	5	40	22	2	0	1	8	60	44	8	0	<1	1
	Athos	2	56	66	46	8	36	17	0	0	57	1	42	35	13	<1	0	0
	Porthos	3	42	72	21	13	57	5	0	0	8	10	75	65	12	4	<1	1
	Tintern	3	55	64	49	3	57	24	0	0	52	4	60	46	19	4	<1	4
2+5+?	Mink	8	30	77	21	4	66	12	2	0	24	2	52	58	7	<1	<1	1
3+4	Jupiter	2	44	89	87	56	21	4	0	0	79	48	17	12	12	22	1	0
	Goldmarker	6	32	66	63	48	19	5	0	0	64	42	18	12	4	36	2	1
4+6	Dram	1	25	60	10	41	0	58	0	0	12	4	0	0	87	1	16	0
6+Ab	Triumph	2	51	50	57	25	34	82	0	0	82	1	24	32	35	0	7	43
4+5	Egmont	1	40	60	24	30	58	0	0	0	27	51	87	11	0	2	0	7

BMV group	test cultivars	BMV group	test cultivars
1	37/136, 41/145, Astrix	2+3	Inis
2	Goldfoil, Zephyr, Julia, Union	2+4	Georgie
3	Maris Concord, Midas	2+5	Aramir
4	Vada, Lofa Abed	2+5+?	Maris Mink
5	Hassan, Sultan	2+6	Mazurka
6	H.1063, Wing, Tern	3+4	Jupiter
7	Tyra	4+6	Dram, Claret
8	Akka	6+Ab	Triumph

† see text for definition of mean pathogenicity

* see text for explanation of boxes

coming from a general spore pool outside the crop, selection favours pathogen genotypes than can be established on a wide range of different cultivars. As the season progresses, however, selection then favours those pathogen genotypes in each field that are best adapted to the cultivar which they have infected (Wolfe & Schwarzbach, 1978). Consequently, the pathogen population structure on each cultivar is characterised by high pathogenicity for that cultivar (i.e. the diagonal line of boxes in Table 2), and lower values for the other cultivars depending on the frequency of genotypes which are also pathogenic on other cultivars (i.e. the remaining data in the body of Table 2). From Table 2, the mean value of each BMV column, excluding the boxed figures, represents the mean pathogenicity of that BMV factor on all hosts that do not possess the corresponding BMR factor; it is a measure of the fitness of that BMV factor on all non-corresponding hosts.

If the net effect of the two directions of selection, at the beginning and during the season, is to favour pathogen genotypes able to attack more than one cultivar, then the pathogenicity on each BMR group for other BMR groups should increase with time. However, their low initial frequency indicates that fungal genotypes with combined pathogenicity will have lower rates of reproduction on the cultivars that they can attack compared with simpler genotypes growing on their 'own' hosts. Consequently, an increase in combined pathogenicity may be accompanied by a decrease in 'own' pathogenicity.

The mean pathogenicity of isolates from each BMR group tested on its 'own' group is given in Table 3a for each year of the bulk sampling procedure. For BMV groups 1, 2, 3, 5 and 6, the best fit to linear regression with time is negative, consistent with a decline in pathogenicity, but none are significant, or significantly different from a slope of 0. For BMV 4 and 7, the best fit is positive, but not significant; for BMV 8 the regression is positive and significant ($P < 0.05$). However, BMR 8 is a relatively new and uncommon host group so that selection for increased pathogenicity within the pathogen population growing on this group may still be in progress.

Table 3b shows the mean pathogenicity for each BMR group on all other BMR groups. The values are similar and consistent between years, although for BMV 4 and BMV 5 there is a low rate of linear increase with time, which is

Table 3a. Mean pathogenicity of field isolates for the cultivar groups from which they were collected

Year	BMV group							
	1	2	3	4	5	6	7	8
1975	43	98	159	53	145	101	-	-
1976	83	54	40	60	63	70	72	-
1977	34	58	-	33	41	65	57	53
1978	43	78	70	62	68	89	66	68
1979	46	65	68	70	57	80	75	124
mean	50	71	84	56	75	81	68	82

Table 3b. Mean pathogenicity for each BMV factor on all hosts with non-corresponding resistance during the period 1975-1979

Year	BMV group							
	1	2	3	4	5	6	7	8
1975	40	74	21	3	15	17	-	-
1976	58	35	22	4	13	11	0	-
1977	40	46	9	4	14	9	1	<1
1978	44	72	22	7	25	18	<1	1
1979	44	61	21	8	26	16	<1	<1
mean	45	58	19	5	19	14	<1	<1

Table 3c. Mean pathogenicity for each BMV factor on all hosts with non-corresponding resistance during the period 1975-1979, corrected by the appropriate value for pathogenicity on 'own' host group (i.e. values in 3b divided by those in 3a, and multiplied by 100)

Year	BMV group							
	1	2	3	4	5	6	7	8
1975	92	75	13	5	10	17	-	-
1976	70	64	56	7	21	15	0	-
1977	118	80	15	13	34	14	1	<1
1978	103	92	31	12	37	20	<1	1
1979	96	94	31	11	45	20	<1	<1
mean	98	86	29	9	30	21	<1	<1

significant (for BMV 4, $P < 0.01$; for BMV 5, $P < 0.05$).

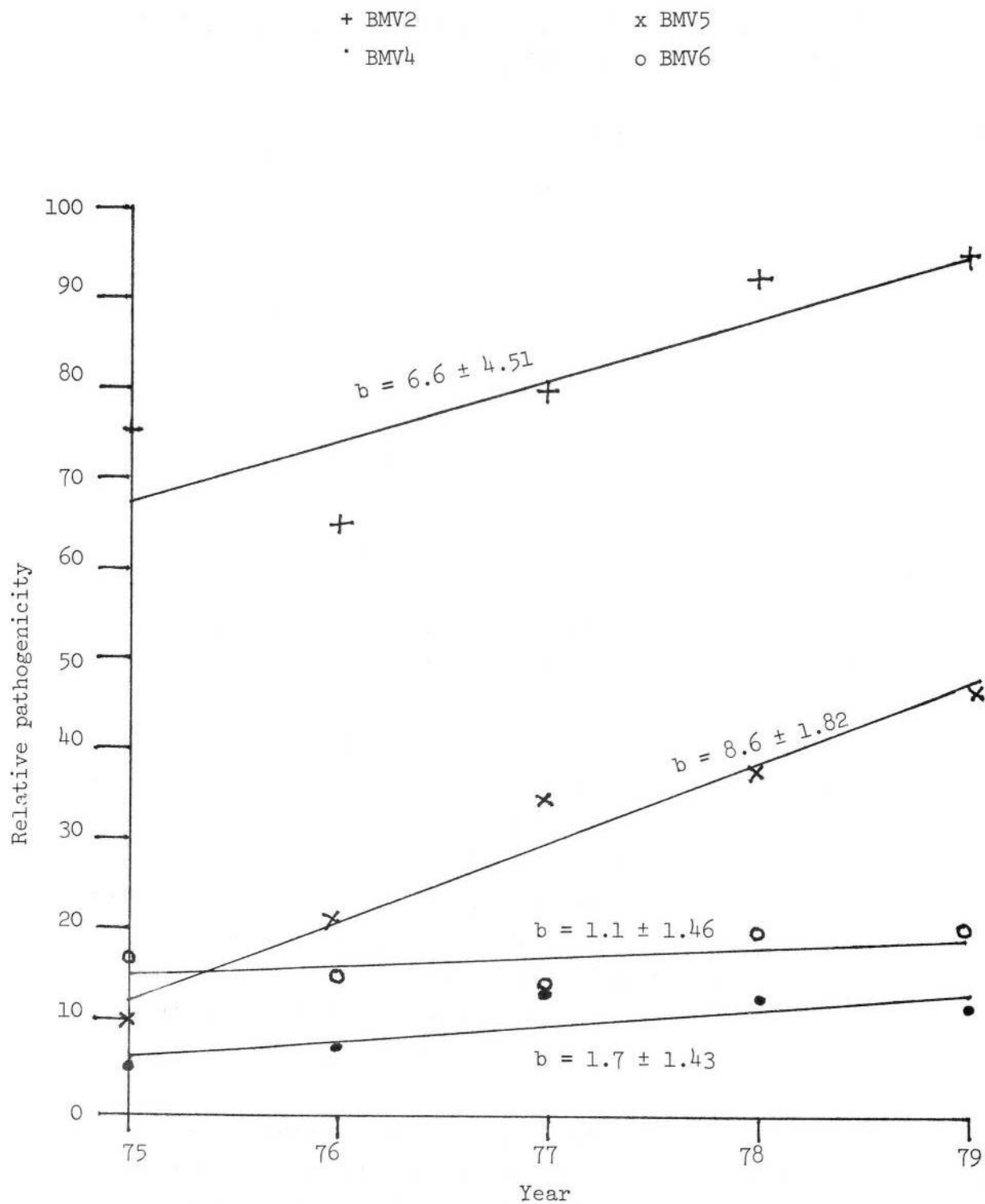
In Table 3c, the values for pathogenicity on non-corresponding BMR groups in each year (Table 3b) are expressed relative to the appropriate pathogenicity value for the corresponding BMR group in each year (Table 3a). This correction allows for any underlying trend in pathogenicity on 'own' host, and for other sources of variation in tests between years. However, the values in Table 3a are based on a smaller number of isolates than those in 3b and will therefore have larger variances; this reduces the accuracy of the values in Table 3c compared with those in 3b.

In Table 3c, there is a tendency for all groups towards an increase in pathogenicity with time. The linear regressions on time, using the data from Table 3c (Fig. 1) are all positive, although for BMV 1 and 3, they are not significant. Most of the remainder are significant, but with different probabilities (for BMV 2 and 4, $P < 0.05$; for BMV 5, $P < 0.001$, and for BMV 6, $P = 0.1 - 0.05$). If the value for 1976 on BMV 3 is omitted, the regression is significant ($P < 0.05$) and similar to that for BMV2 and 5.

From Fig. 1, pathogenicity for BMV 4 and 6 has increased rather slowly on non-corresponding cultivars, whereas that for BMV 2 and 5 has increased more rapidly. It is not known to what extent these differences are due to differences in the amounts of inoculum coming from cultivars with corresponding resistances, or to differences in the reproductive ability on non-corresponding hosts of pathogen genotypes carrying BMV 4 and 6 compared with those carrying BMV 2 and 5. On the basis of the data presented in Fig. 1, it would seem that BMR 3, 4, 6, 7 and 8, and to a lesser extent BMR 5 will continue to provide useful diversification for several more years. This situation contrasts strongly with that observed in the early 1960's when, with only two BMR groups in general use (BMR2 and 3), together with the combination BMR 2+3, there was a rapid increase in BMV 2+3. It seems that the current diversity of resistance factors limits the rapidity of selection for all possible combinations of pathogenicity factors.

The mean data for each BMV group in Table 3c and Fig. 1 conceal differences in the effects of different BMR groups on particular non-corresponding BMV factors. The differences may be positive, indicating less selection

Fig. 1. Linear regression of corrected mean pathogenicity values for each of the BMV groups on all BMR groups except that with corresponding resistance. BMV 1 and 3 are omitted because the regressions were not significant; BMV 7 and 8 are omitted because the pathogenicity levels were too low to be indicated on the graph



than average against particular BMV factors, or negative, indicating greater than average selection; the major differences are summarised in Table 4 for the period 1975-79 and approximate to P values less than 0.05.

Table 4. Deviations from the average pathogenicity of each BMV group on each non-corresponding BMR group

BMR group	1	2	BMR group 3	4	5	6
1		+
2
3
4	-	-	.		.	-
5		-
6	+	.	-	.	-	
7	+	.
8	+	.	-	.	-	.

. no deviation

+ positive deviation, i.e. more pathogenic than expected

- negative deviation, i.e. less pathogenic than expected

Greater than average selection against BMV 5 on BMR 6, and against BMV 6 on BMR 5, has been noted previously and has been of considerable value in relation to cultivar diversification and mixing. There is some indication, however, that the effect is declining on BMR 6, though not on BMR 5. The apparently small degree of selection against BMV 5 on BMR 7 suggests that in future there may be an increased risk from growing BMR 5 and 7 in proximity or together, than from growing members of either group with cultivars from BMR groups 3, 4, 6 or 8. The degree of risk depends on the susceptibility in the field of the cultivars involved.

Also apparent in Table 4, is the similarity in the patterns of deviations from average selection against non-corresponding pathogenicity factors caused by BMR 6 and 8. Since the only known similarity between all cultivars in these two groups is common possession of the resistance gene *Mla4*, it seems that possession of the corresponding pathogenicity, *Va4*,

may be responsible for the correlated effects on survival of the pathogen on BMR 1, 3, 4 and 5.

Value for diversification and variety mixing

To assess the current value for diversification of different pairwise combinations of BMR groups, corrected pathogenicity values for 1978 and 1979 combined are presented in Table 5. For example, to observe the potential influence of populations from each of BMR 2 and BMR 4 on the other, Table 5 indicates that the combination BMV 2+4 on BMR 2 has a pathogenicity value of 81, whilst BMV 2+4 on BMR 4 has a value of 15. Given equal areas of equally susceptible cultivars from BMR 2 and BMR 4, then the average of the two values for BMV 2+4, 48, provides an index of the pathogenicity of the population towards both cultivars at the beginning of the epidemic (Table 6), compared with the average pathogenicity for BMV 2 on BMR 2, and BMV 4 on BMR 4 (= 100).

Table 5. Pathogenicity of population samples from the major BMR groups on test seedlings of the same groups, corrected for pathogenicity on the groups from which they were collected.
Mean values for 1978 and 1979

Isolate source BMR	Corrected pathogenicity (colony numbers) on seedling leaves of BMR test cultivars							
	1	2	3	4	5	6	7	8
1	100	94	36	19	38	50	0	1
2	75	100	50	15	43	29	0	2
3	117	90	100	25	46	36	1	0
4	65	81	39	100	33	3	0	0
5	100	100	18	13	100	10	0	0
6	112	98	18	8	33	100	0	3
7	106	99	32	6	68	25	100	1
8	150	87	5	1	27	(100)*	0	100
mean of non-selective hosts	104	93	28	12	41	26	<1	1

* all isolates from BMR 8 are pathogenic on BMR 6 since they probably evolved from pathogen populations on BMR 6.

All pairs of data in Table 5 were averaged in this way to give Table 6 which is laid out in the form of a Diversification Group Table.

Table 6. Pathogenicity indices for all possible pairs of BMR groups, derived from Table 5. Positive (+) deviations from expectation indicate that the number is greater than expected, negative (-) deviations indicate less than expected (see Table 4)

Chosen BMR group	Mean pathogenicity for chosen BMR group and companion group							
	1	2	3	4	5	6	7	8
1	100	85	77	43	69	81	53	76
deviation				-		+		+
2	85	100	70	48	72	64	50	45
deviation			+					
3	77	70	100	32	32	27	17	3
deviation		+			-	-		-
4	43	48	32	100	23	6	3	1
deviation						-	-	-
5	69	72	32	23	100	22	34	14
deviation			-			-		-
6	81	64	27	6	22	100	13	(52)
deviation	+		-	-	-			+
7	53	50	17	3	34	13	100	1
deviation				-				
8	76	45	3	1	14	(52)	1	100
deviation	+		-	-	-	+		

By selecting any one BMR group in the left-hand column (Table 6), the appropriate pathogenicity index can be read off for any other BMR group with the chosen one. The deviations in Table 4 have been incorporated in Table 6. As an example, the combination BMR 4 with BMR 6 is particularly suitable for diversification, because the pathogenicity index is not only low, but significantly lower than the average for BMV 4 or BMV 6 in combination with any other BMV factor.

Combinations of BMR 1 and 2 together or with any other group (with the possible exception of BMR 1-4) have limited value (pathogenicity range 43-85), and are probably of little practical significance. Combinations of all other groups have a mean pathogenicity index of 34 or less (with the exception of BMR 6-8 in which the cultivars involved have Mla4 in common) and are thus potentially useful in a diversification scheme. The Table can only provide a guide; the real value of each combination in the

field depends on the susceptibility of the cultivars in question, their total area and their relative disposition as pure cultivars.

Cultivar mixtures are commonly made up from three or more components. Table 7 illustrates all possible three-way combinations of BMR groups (excluding those with BMR 1 and 2, and BMR 6 with 8). Each pathogenicity index in the Table is calculated as the mean of the appropriate two-way values in Table 6. For example, for BMR 3-5-6 (27), the values used were BMR 3-5 (32); BMR 3-6 (27); and BMR 5-6 (22). Again, the real value of each combination in the field will depend to some extent on the susceptibility of the particular cultivars used.

Cultivars with combined resistances

Pathogenicity data from each BMR group can provide estimates for the expected pathogenicity of all combination of BMV factors on that group. The estimates can be tested either by analysing individual pathogen genotypes extracted from the populations, or by assessing the performance of the populations on test cultivars that carry combinations of BMR factors. Assessments of the latter type can also be compared with the performance of pathogen populations obtained from cultivars with the same combination of BMR factors, using test cultivars with single BMR factors or with combinations. A reciprocal analysis using currently important combinations of BMR factors is given in Table 8. Estimates were obtained as products of the appropriate corrected mean pathogenicity values. The observed values were corrected against the mean of the pathogenicity values of each component factor on its 'own' host. Examples of the calculations are given in Table 8.

From Table 8, pathogenicity for BMR 2+5 is close to expectation on all hosts including BMR 2+5; this is also reflected in Table 4 in that the pathogenicity of BMV 2 on BMR 5, and of BMV 5 on BMR 2 does not deviate from the average. On the other hand, pathogenicity for BMR 2+4, and 3+4, is less than expected in almost all cases, including the samples obtained respectively from BMR 2+4 and BMR 3+4. Because BMV 4 has a value of less than 100 in the isolates obtained from BMR 2+4 and 3+4, the expected values for corresponding pathogenicity are also low. The observed values are, nevertheless, still lower than expected, further indicating the limited pathogenicity of these combinations. It is also possible that

Table 7. Pathogenicity indices for 16 3-way combinations of BMR groups.
Values given are means of the three appropriate values from
Table 6, e.g. for BMR 3-5-6, the values used are from BMR 3-5,
3-6 and 5-6. BMR 1 and 2 are excluded because of the high
values for corresponding pathogenicity on all other groups.
Combinations involving BMR 6 and 8 are omitted because of the
close relationship of the corresponding pathogen populations

<u>BMR groups</u>	<u>Mean pathog.</u>	<u>BMR groups</u>	<u>Mean pathog.</u>
3-4-5	29	4-3-5	29
3-4-6	22	4-3-6	22
3-4-7	14	4-3-7	14
3-4-8	12	4-3-8	12
3-5-6	27	4-5-6	17
3-5-7	28	4-5-7	20
3-5-8	16	4-5-8	13
3-6-7	19	4-6-7	7
3-7-8	7	4-7-8	2

<u>BMR groups</u>	<u>Mean pathog.</u>	<u>BMR groups</u>	<u>Mean pathog.</u>
5-3-4	29	6-3-4	22
5-3-6	27	6-3-5	27
5-3-7	28	6-3-7	19
5-3-8	16	6-4-5	17
5-4-6	17	6-4-7	7
5-4-7	20	6-5-7	23
5-4-8	13		
5-6-7	23		
5-7-8	16		

<u>BMR groups</u>	<u>Mean pathog.</u>	<u>BMR groups</u>	<u>Mean pathog.</u>
7-3-4	14	8-3-4	12
7-3-5	28	8-3-5	16
7-3-6	19	8-3-7	7
7-3-8	7	8-4-5	13
7-4-5	20	8-4-7	2
7-4-6	7	8-5-7	16
7-4-8	2		
7-5-6	23		
7-5-8	16		

Table 8. Pathogenicity for BMR groups with separate and combined resistances; data combined from 1978 and 1979. Derivation of the observed and expected values is given in the footnote

Field source BMR	2	3	4	5	2+4	2+5	3+4
2 obs	100	50	15	43	7*	45	4
exp					15†	43	8
3 obs	90	100	25	46	16	49	16
exp					23	41	25
4 obs	81	39	100	33	60	33	25
exp					81	27	39
5 obs	100	18	13	100	6	85	3
exp					13	100	2
2+4 obs	100	46	88	49	64	43	31
exp					88	49	40
2+5 obs	108	39	8	94	7	107	3
exp					9	102	3
3+4 obs	96	109	82	33	55	37	56
exp					79	32	89
mean obs					31	57	20
exp					44	56	29

$$* \text{ obs value} = \text{obs.} \div \left\{ \frac{\text{max. var 1} + \text{max. var 2}}{2} \right\} \times 100$$

$$\text{e.g. obs for BMR 2+5 on BMR 2+5} = \left\{ 72 \div \left(\frac{71 + 63}{2} \right) \right\} \times 100 = 107$$

$$† \text{ exp. value} = \{\text{obs. var 1}\} \times \{\text{obs. var 2}\} \times \frac{1}{100}$$

$$\text{e.g. exp. for BMR 2+5 on BMR 2+5} = \{108\} \times \{94\} \times \frac{1}{100} = 102$$

Uncorrected observed values derived from Table 2, and the corresponding Table for 1978 (CPVS, 1978)

the test cultivars with combinations of BMR factors possess unidentified resistance factors of small effect, not matched by all of the isolates obtained. Earlier evidence indicated that BMV 2+5 occurred initially at lower than expected values, but this effect seems now to have disappeared.

Geographical distribution of pathogenicity

Pathogen population structure on a cultivar depends on the nature of the initial population, which in turn is determined by the distribution and susceptibility of cultivars in the locality, and the susceptibility of the cultivar itself. A large survey of mildew populations at different sites in northern England carried out by Dr J T Fletcher and Dr M J Hims facilitated an analysis of geographical effects on population structures (Table 9).

Table 9. Geographical distribution of major pathogenicity factors.
Values are calculated as the mean pathogenicity for each BMR
group observed on all non-corresponding BMR's. 1979 data
unless otherwise stated

Location	BMR					
	1	2	3	4	5	6
Scotland (1978)	59	57	63	6	15	18
Scotland	46	59	51	26	22	9
Border	43	60	36	16	37	11
Northumbria	39	63	23	9	27	18
York Vale	37	74	10	6	27	23
Midlands [†]	53	65	13	7	29	2
E. Anglia	41	68	18	8	25	20

[†]Set of samples from Harper Adams only.

Samples from the northern survey were divided into three source areas (Border, Northumbria and York Vale) and compared with samples from three other areas, Scotland, Midlands and East Anglia. The comparison between sites was made on the mean values for pathogenicity on non-corresponding hosts, as in Table 3b. Pathogenicity for BMR 1, 2, 4 and 5 was similar in all localities (and indeed for all years: Table 3b). However, BMV 3 declined in value from Scotland southwards, which probably reflects the high density of crops of Midas in Scotland and the north compared with

the remainder of England. This difference is correlated with the relatively poor figure for the mildew resistance of Midas on the Scottish Recommended List compared with the higher figure on the NIAB Recommended List.

Disregarding the low value of the single sample from the Midlands, there is a suggestion of a converse relationship with BMV 6, related presumably to the low frequency of BMR 6 cultivars in Scotland and the north, compared with the rest of England. The apparent small size of this difference may reflect interplot interference in that many of the Scottish samples were obtained from plot trials which had a higher frequency of BMR 6 plants than on farms generally.

The major implication of these observations is that the value of BMR 3 cultivars for diversification and mixing must be less in Scotland and northern England than further south because of the over-exposure of this source of resistance in the north.

The lack of universal susceptibility

In the course of analysing the geographical effects, it was found that isolates obtained from Golden Promise gave different mean values for pathogenicity than did those from a range of other cultivars (Table 10).

Table 10. Comparison of non-corresponding pathogenicity values obtained in isolates from Golden Promise with the means of those obtained from a range of cultivars

		BMV					
		1	2	3	4	5	6
Scotland 1978	Golden Promise	52	63	36	18	18	22
	Other cultivars	59	57	63	6	15	18
North England 1979	Golden Promise	44	65	13	12	33	34
	Other cultivars	40	66	23	10	30	17

In both surveys in Table 10, mean pathogenicity values for BMR 1, 2, 4 and 5 are similar, but those for BMV 3 are lower, and for BMV 6 higher, than the corresponding values obtained from other cultivars. This indicates a differential interaction between Golden Promise and BMV 3 and 6, which, because of the high frequency of Golden Promise in Scotland,

would tend to reduce survival of BMV 3 and enhance survival of BMV 6, relative to other cultivars which also lack BMR 3 and 6.

Because of an earlier consideration that such effects might occur, a programme was started in 1979 to select other 'universal susceptibles' from the collection to substitute for, or to supplement, Golden Promise in the survey.

The use of mobile nurseries

In Table 11, the conventional survey method is compared with the use of mobile nurseries over three years to assess pathogen populations. The data for this comparison were obtained by correcting the mean pathogenicity values on non-corresponding hosts by the values for pathogenicity on the corresponding host; the data in Table 11 are thus directly comparable with the bottom line in Table 3c.

Table 11. Comparison of the conventional survey and mobile nursery methods for assessing barley mildew populations. The values given are mean pathogenicity levels for each BMV group averaged over all non-corresponding cultivars

		BMV										
Source		1	2	3	4	5	6	7	8	2+4	2+5	3+4
1977	Survey	118	80	15	13	34	14	1	<1	15	38	4
	Nursery	105	82	18	80	20	37	1	2	45	63	21
1978	Survey	103	92	31	12	37	20	<1	1	18	42	9
	Nursery	104	94	23	43	41	28	4	1	51	39	5
1979	Survey	96	94	31	11	45	20	<1	<1	29	42	16
	Nursery	93	87	11	37	48	22	7	1	74	39	7

The values for BMV 1, 2, 5, 7, 8 and 2+5 are similar between methods and between years. The suggestion of an increase in BMV 5 may be due to higher values of BMV 5 on BMR 7 in the last two years. The values for BMV 4 differ widely between techniques, which appears to be due to the temperature sensitivity of BMR 4 resistance in test seedlings (CPVS Report, 1978). The same effect is evident for BMV 2+4, but not for BMV 3+4, which suggests that the action of BMR 4 may be epistatic to that of BMR 2, but hypostatic to that of BMR 3.

Lower values for BMV 3 were produced in the nursery than in the survey, whereas the reverse was true for BMV 6. These trends are similar to the effects noted above in the geographical analysis and probably have the same cause. The nurseries have been restricted to a relatively small area in the vicinity of Cambridge, in which the frequency of BMR 3 fields has been low, and of BMR 6 fields high. The survey isolates have a much wider geographical distribution, although the majority for 1979 came from the north of England.

Mobile nurseries thus seem to prove a reasonable alternative to conventional leaf sampling. They are less efficient for detecting new BMV factors, but more so for detailed assessment of the population structure on individual cultivars (see below).

Unidentified or background resistance

The use of mobile nurseries allowed detailed observation of some interactions of individual cultivars with the pathogen (CPVS Report, 1978). For example, the two BMR 4 cultivars, Vada and Lofa Abed, were exposed in fields of different cultivars (Table 12).

Table 12. Mean pathogenicity on seedlings of Vada and Lofa Abed exposed in fields of different cultivars

Fields (no)	Test seedlings	
	Vada	Lofa Abed
Non-BMR ⁴ (7)	64	32
Goldmarker (1)	180	88
Magnum (1)	168	91
Lofa Abed (1)	110	79

In fields of non-BMR 4 cultivars, or of Goldmarker or Magnum, seedlings of Lofa Abed supported approximately half as many colonies as did those of Vada. After exposure in a field of Lofa Abed, however, there were almost threequarters as many colonies on seedlings of that cultivar as there were on those of Vada, indicating greater adaptation of the field population towards the host cultivar.

Similarly, among the BMR 5 cultivars, Sultan, Hassan and Maris Trojan, seedlings of Hassan were relatively more infected than those of the other two cultivars when exposed in a field of Hassan than when they were exposed in fields of other cultivars (Table 13):

Table 13. Mean pathogenicity on seedlings of BMR 5 cultivars exposed in fields of a range of non-BMR5 cultivars, BMR 6 cultivars and Hassan

Fields (no.)	Test seedlings		
	Sultan	Hassan	Maris Trojan
Non-BMR 5 (7)	51	53	45
BMR 6 (2)	14	26	25
Hassan (1)	109	159	31

Selection for different BMR 2+5 cultivars was evident from exposure of seedlings of Maris Mink, Athos, Porthos and Aramir in fields of non-BMR 2+5 cultivars, and in fields of Aramir and Porthos (BMR 2+5; Table 14).

Table 14. Mean pathogenicity on seedlings of BMR 2+5 cultivars exposed in fields of non-BMR 2+5 cultivars, and Aramir and Porthos (BMR 2+5)

Fields (no.)	Test seedlings			
	Maris Mink	Athos	Porthos	Aramir
Non-BMR 2+5 (7)	13	18	20	30
Aramir (1)	18	64	59	70
Porthos (1)	50	18	55	50

The relative infection of the Aramir and Porthos seedlings was similar in the non-BMR 2+5 fields and in the field of Aramir. In the field of Porthos, however, there was relatively more infection on the Porthos seedlings. It was also noted that Maris Mink and Athos showed a differential interaction with reversed ranking, Athos being more susceptible to the Aramir population and Maris Mink being more susceptible to the Porthos population.

Reference

Wolfe, M.S. & Schwarzbach, E. (1978). Patterns of race changes in powdery mildews. Annual Review of Phytopathology, 16, 159-180.

YELLOW RUST OF BARLEY

R H Priestley & P Byford

National Institute of Agricultural Botany, Cambridge

The number of samples of Puccinia striiformis received in 1979 was the lowest since 1971. The effect of BYR 2 in reducing infection levels was less well marked in winter and spring sown adult plant cultivar tests than in previous years. Overall, there was reasonable agreement between the relative resistance of cultivars inoculated in a Polythene tunnel and in a comparable isolation nursery.

INTRODUCTION

The specific resistances (BYR factors) identified in barley cultivars to date, a test cultivar possessing each resistance and the year of first detection of virulence (V) in the UK population of P. striiformis are given in Table 1.

VIRULENCE TEST METHODS

Methods are similar to those described for wheat yellow rust by Priestley (1978).

Seedling tests with 1979 isolates

A total of 18 disease samples was received by post during 1979. This is the lowest since 1971 and reflects the relatively low incidence of barley yellow rust in 1979. The samples were collected in a non-random way from Sonja (6 samples), Gerbal (2) and Igri (2). Only a few samples were received from spring barley cultivars.

Adult plant tests with 1978 and control isolates

Tests to measure the virulence of P. striiformis isolates on adult barley plants were continued in 1979 using the Polythene tunnel method developed for wheat yellow rust (Priestley & Byford, 1978). Details of the isolates used are given in Table 2.

Tests involving winter barley cultivars were carried out for the first time. Six replicate tussocks of 12 cultivars were sown on 23-24 November 1978, inoculated on 20 March and 11 April and assessed for percentage leaf area infection on 10 May (GS 45-50), 18 May (GS 50-58), 23 May (GS 54-68) and 31 May (GS 71). Spring barley tests consisted of 2 replicate tussocks of

Table 1. Resistance factors to *P. striiformis*

BYR factor	Test cultivar	Type*	Year virulence detected
BYR 1	Astrix	overall	1960
BYR 2	Bigo, Varunda	overall	1972-5
	Mazurka	seedling	

* Sensu Zadoks (1961); overall resistance is effective at all growth stages, seedling resistance is ineffective at adult plant growth stages.

Table 2. Isolates used in adult plant tests

Code	Cultivar	Region	Site	BYV factors
74/33	Malta	N	Morpeth	BYV 1
75/101	Varunda	YL	Boroughbridge	BYV 1, 2
77/1	Athene	E	Morley	BYV 1, 2
78/11	Magnum	SW	Cannington	BYV (1)
78/45	Goldmarker	WM	Farndon	BYV 1
78/48	Igri	S	Kirkliston	BYV 1

() = partially virulent or corresponding resistance

N = North

YL = Yorks and Lancs

E = East

SW = South West

WM = West Midlands

S = Scotland

36 cultivars sown on 15 March 1979, inoculated on 26 April and 10 May and assessed on 18 May (GS 31-39), 31 May (GS 45-56), 14 June (GS 68-75) and 27 June (GS 71-76). In addition an isolated field nursery, of identical design and spring cultivar composition but not covered with a Polythene tunnel was sown on 15 March 1979, inoculated on 26 April, 10 May and 19 May and assessed on 5 June (GS 32-33), 20 June (GS 49-53), 5 July (GS 71-72) and 17 July (77-78).

VIRULENCE TEST RESULTS

Seedling tests with 1979 isolates

Isolation was made from only one sample; the remaining 17 samples failed to infect the susceptible cultivar Berac. The isolate which was successfully cultured was found to be virulent on Astrix (R1) but not Bigo (R2).

Table 3. Results of adult plant winter barley tests, 1979

Values are mean percent leaf area infection.

Boxes are used to identify particular parts of the table and have no statistical significance.

BYR factor	cultivar	BYV factor	BYV 1	BYV 1, 2	mean
		isolate	78/ 48	75/ 101	
BYR 1	Astrix		37	29	33.0
BYR 2	Bigo		7	10	-
	Varunda	A	9	18	-
	Mazurka		25	34	-
BYR 0	Maris Trojan		8	5	6.5
	Emir		10	6	8.0
	Igri		16	13	14.5
	Maris Otter		19	14	16.5
	Hoppel		23	25	24.0
	Gerbél		24	26	25.0
	Athene		27	23	25.0
	Sonja		41	36	38.5

Adult plant tests with 1978 and control isolates

The results of the winter and spring barley adult plant tests are shown in Tables 3 and 4 respectively. Infection levels in the spring cultivar tests were lower than in previous years.

In both the winter and spring sown tests, the difference in infection level on cultivars possessing BYR 2 (ie Bigo, Varunda and Mazurka) produced by isolates possessing BYV 2 and lacking this virulence (Boxes A and B) was less well marked than in previous years.

Of the winter barley cultivars evaluated (all of those in Table 3 except Bigo, Varunda, Mazurka and Emir), only Astrix has been shown to possess any specific resistance. Seedling tests with this cultivar over a number of years have shown that it possesses BYR 1. However, all the isolates used in the tests summarised in Table 3 possess the corresponding virulence and so the resistance was not expressed.

Table 4. Results of adult plant spring barley tests, 1979

Values are mean percent leaf area infection.

Boxes are used to identify particular parts of the table and have no statistical significance.

BYV facto.		BYV 1			BYV 1, 2			
	isolate	78/ 11	78/ 45	74/ 33	77/ 1	75/ 101A*	75/ 101B*	
BYR factor	cultivar	mean						
BYR 1	Atem	9	18	30	16	14	4	15.7
	Sundance	12	16	26	17	23	3	16.2
	UD 3101	15	20	25	13	24	1	16.7
	Zephyr	15	21	28	17	22	4	17.8
	Julia	24	28	33	24	30	23	27.0
BYR 2	Bigo	1	0	2	6	10	4	-
	Varunda	8	8	11	15	25	12	-
	Mazurka	14	12	26	23	35	19	-
BYR 0	HW 56/43	0	1	3	0	2	0	1.0
	Emir	1	3	2	3	3	0	2.0
	Athos	9	6	4	5	10	0	5.7
	Tintern	4	7	11	6	8	0	6.0
	Abed 2257	5	6	9	7	4	1	6.0
	14754 Co 50	5	6	9	4	9	6	6.5
	Aramir	10	7	13	5	13	1	8.2
	Hassan	5	9	16	9	15	2	9.3
	Goldmarker	12	8	12	6	14	6	9.7
	Porthos	6	12	13	14	16	1	10.3
	Triumph	10	7	16	15	17	2	11.2
	Lofa Abed	9	11	16	13	21	3	12.2
	Cebeco 7607	14	9	17	12	20	3	12.5
	Egmont	11	16	22	13	17	7	14.3
	Ark Royal	7	11	22	13	27	7	14.5
	Midas	11	14	20	16	20	16	16.2
	HJ 69/641/4	15	17	24	12	25	9	17.0
	Aurea	11	19	31	19	28	15	20.5
	Koru	19	25	29	18	26	9	21.0
	Claret	22	21	28	18	31	7	21.2
	Tyra	19	24	29	22	33	19	24.3
	Georgie	18	24	36	26	31	12	24.5
	Jupiter	25	30	29	22	31	19	26.0
	Simon	22	34	34	20	36	14	26.7
	Dram	16	26	27	28	38	28	27.2
	Keg	25	27	35	28	39	11	27.5
	RPB 393/73	16	32	40	28	35	15	27.7

* 75/101A under Polythene; 75/101B not under Polythene.

The winter barley cultivars lacking specific resistances (BYR 0) became infected to markedly different degrees, as did the spring barley cultivars lacking specific resistance (RO) shown in Table 4. Seedling tests carried out as part of the NIAB cultivar evaluation programme indicate that Atem and UD 3101 possess BYR 1 (personal communication, R A Bayles). This cannot be deduced from Table 4 because all the isolates in those tests possessed the corresponding virulence BYV 1.

Infection levels on cultivars grown in the isolation nursery (75/101B) were consistently lower than those in the comparable Polythene tunnel (75/101A). There was general agreement between the relative resistance of cultivars grown under the two regimes but a few cultivars were more resistant in the isolation nursery than in the comparable Polythene tunnel (eg Claret).

REFERENCES

- PRIESTLEY, R. H. (1978). Detection of increased virulence in populations of wheat yellow rust. In Plant Disease Epidemiology Ed P R Scott & A Bainbridge pp 63-70. Blackwell Scientific Publications, Oxford.
- PRIESTLEY, R. H. & BYFORD, P. (1978). Yellow rust of barley. United Kingdom Cereal Pathogen Virulence Survey 1977 Annual Report, 12-16.

BROWN RUST OF BARLEY

E.R.L. Jones & B.C.Clifford

Welsh Plant Breeding Station, Aberystwyth

Only the common race F of Puccinia hordei Otth. which carries virulence for Pa, Pa₂, Pa₄, Pa₅, Pa₆ and Pa₈ was identified in seedling tests in 1979. Tests in isolation nurseries in the field confirmed the specific resistance of Simon (Pa₃) and Triumph (Pa₉?). The field monitoring of partially-expressed resistance (slow rusting) by use of trap nurseries continued in 1979. No evidence was found of higher than expected levels of infection on such resistant cultivars. An octal/binary system of notation is proposed for the designation of virulence gene combinations carried by isolates (races) of P. hordei.

SEEDLING TESTS

Of the 29 samples received from the 1979 Survey, 12 were from winter barley varieties from a trial in south Wales and 17 were from the WPBS/ADAS spring barley trap nurseries for brown rust grown in Devon and Cornwall. Twenty six samples were successfully cultured and all isolates were identified as the common race F which carries virulence for genes Pa, Pa₂, Pa₄, Pa₅, Pa₆ and Pa₈. Virulences for Pa₃ and Pa₉, which are apparently carried by Simon and Triumph respectively, were not detected.

ADULT PLANT TESTS

1. Isolation Nurseries

Twenty spring barley cultivars were again grown in four isolation nurseries each of which was inoculated with one of four isolates of P. hordei with different virulence combinations selected from previous surveys (Table 1).

Isolate 1	ex cultivar Simon
Isolate 2	Standard isolate 76-12
Isolate 3	ex cultivar Mirena
Isolate 4	Standard isolate Race F

The results confirmed previously reported findings. The only currently grown cultivars with race-specific resistance are Simon (gene Pa₃) and Triumph (Pa₉?). Triumph was resistant in the field to all four cultures but Simon was susceptible to the isolate cultured from that cultivar. Virulence for Pa₃ has been detected in previous surveys but not in the current one. Isolate 76-12 appeared to have lost virulence for Pa₃ which

Table 1. Response* of spring barley cultivars to specific isolates of Puccinia hordei in field isolation nurseries.

Cultivar	Isolate				
	ex Simon	76-12	ex Mirena	F	Mean
Simon	16S	1MS	8MS	6MR	-
Triumph	7R	3MR	7 R	8MR	-
Sundance	10MS	9MS	9MS	9MR	9.2
Tyra	13S	8S	12S	8MS	10.2
Armelle	12MS	12MS	10MS	11MS	11.2
Tintern	17S	9MS	11MS	9MR	11.5
Mirena	15MS	11MS	12MS	9MR	11.7
Hassan	16MS	11MS	12MS	11MS	12.5
Athos	15S	15MS	12MS	10MR	13.0
Mala Abed	16MS	10MS	16S	10MS	13.0
Porthos	16MS	15MR	11MS	10MR	13.0
Magnum	16S	12S	14S	11MS	13.2
Lofa Abed	14MS	15S	14MS	11MS	13.5
Coracle	14MS	14MS	15S	15MS	14.5
Maris Mink	15MS	19MS	15MS	14MS	15.7
Jupiter	19S	19S	16S	17S	17.7
Goldmarker	20S	26S	21S	21S	19.5
Proctor	17S	25S	21S	20S	20.7
Midas	22S	29S	29S	21S	25.2

*Adult plant assessment GS 10,1) of infection percentage and reaction type (average of 4 replicates).

it carried previously as judged by the resistance of Simon. Glasshouse tests confirmed this and so the culture has been discarded in favour of a known carrier of virulence to Pa_3 for future use. Mirena is not resistant to isolates carrying Pa_3 virulence as reported previously (Clifford, Jones and Priestley, 1978) and is in fact seedling susceptible to a wide range of common races in glasshouse tests.

2. ADAS/WPBS Trap Nurseries

The programme to monitor virulence in relation to partially-expressed host resistance by the use of trap nurseries sown in commercial crops continued in 1979. Because of the difficulties of the 1979 season only two nurseries were sown, one in Devon and one in Cornwall, and neither gave evidence of higher than expected levels of infection on resistant cultivars. The programme will continue in the 1980 season.

DESIGNATION OF SPECIFIC VIRULENCE GENES

Proposals for standardising procedures for surveying virulence in P. hordei were agreed by a group of European rust workers at the European and Mediterranean Cereal Rusts Conference at Interlaken, Switzerland in 1976 (Clifford, 1977). They included a suggestion for a formal system of race (virulence) nomenclature that should be short, simple, logical, flexible and informative. It was suggested that the octal/binary system of notation proposed by Gilmour (1973) fulfilled these criteria. As stated in the proposal, the system is particularly useful to designate virulence genotypes i.e. carriers of specific combinations of virulence which correspond to combinations of host resistances deployed in mixtures, multi-gene or multiline cultivars.

Nine barley genotypes that carry specific factors for reaction to P. hordei are listed in Table 2. These are numbered from 1 to 9 in a manner that corresponds to their Pa gene number and are ranked in a fixed linear order with numbers in ascending order from right to left. The binary numbers generated by allocating 0 for resistant and 1 for susceptible and the corresponding octal numbers are given in Table 3 for all the races of P. hordei identified from the Surveys from 1968 to 1976.

Table 2. Barley genotypes used to identify virulence factors in Puccinia hordei and their ranking for octal notation.

<u>Genotype</u>	<u>C.I. No.</u>	<u>Gene</u>	<u>Ranking</u>
Sudan	6489	Pa	1
Peruvian	935	Pa ₂	2
Ribari	-	Pa ₃	3
Gold	1145	Pa ₄	4
Quinn	1024	Pa ₅	5
Bolivia	1257	Pa ₆	6
Cebada Capa	6193	Pa ₇	7
Egypt 4	6481	Pa ₈	8
CI 1243	1243	Pa ₉	9

Table 3. Octal/binary system for the designation of virulence gene combinations carried by specific races of Puccinia hordei Otth.

Race	Differential Binary Number									Octal Number	Year Identified
	9	8	7	6	5	4	3	2	1		
A	-	0*	0	0	0	1	0	0	1	11	1968
B	-	1	0	0	0	1	0	0	1	211	1968
C	-	1	0	0	0	1	0	1	0	212	1968
D	-	0	0	0	1	1	0	1	0	32	1968
E	-	1	0	1	1	1	0	0	1	271	1968
F	-	1	0	1	1	1	0	1	1	273	1969
G	-	0	0	0	0	1	0	0	0	10	1969
H	-	0	0	1	1	1	0	0	0	70	1969
J	-	1	0	0	1	1	0	1	0	232	1969
K	-	1	0	1	0	1	0	0	1	251	1973
L	-	1	0	0	0	1	0	0	0	210	1973
76/2	0	1	0	1	0	1	1	1	1	257	1976
76/3	1	1	0	1	1	1	0	1	1	673	1976
76/12	1	1	0	1	1	1	1	1	1	677	1976

*0 = Resistant

1 = Susceptible

REFERENCES

- CLIFFORD, B.C. (1977). Monitoring virulence in Puccinia hordei: A proposal for the choice of host genotypes and survey procedures. Cereal Rusts Bulletin 5, 34-37
- CLIFFORD, B.C., JONES, E.R.L. & PRIESTLEY, R.H. (1979). Brown rust of barley. Report UK Cereal Pathogen Virulence Survey for 1978, pp.49-53.
- GILMOUR, J. (1973). Octal notation for designating physiologic races of plant pathogens. Nature Lond. 242, 620.

RHYNCHOSPORIUM OF BARLEY

E R L. Jones & B.C. Clifford

Welsh Plant Breeding Station, Aberystwyth

Of the 65 samples received in the 1979 Survey, the majority were from south west England, Wales and Scotland. Forty five samples were successfully cultured, of which 36 were from winter cultivars. Race UK 2 of Rhynchosporium secalis was identified from 36 of the samples but no isolate of race UK 1 was detected. A race identified for the first time in 1978 and which differs from UK 2 by being avirulent on Armelle, Katy, Mirra and Astrix, was isolated from five samples: it has been designated race UK 3. In addition, a previously unidentified race, designated race UK 4, was isolated from four samples. This is similar to race UK 2 but is avirulent on Katy, Mirra and Astrix.

GLASSHOUSE SEEDLING TESTS

The majority of 65 samples received were from south west England, Wales and Scotland and of the 45 successfully cultured, 36 were from the winter cultivars Athene (13 samples), Igri (8), Sonja (4), Maris Otter, Maris Trojan and Hoppel (3 each), Astrix and Katy (2 each) Gerbel and HJ 57/2 (1 each).

The results of glasshouse seedling tests using standard procedures and differential cultivars (Jones and Clifford, 1979) are given in Table 1. In the column headed RT for the reaction type in Table 1, the predominant reaction type is given first. A range of reactions on a plant is indicated by a comma and a range between plants by a hyphen. Race UK 2 was identified from 36 samples but no isolate of race UK 1 was detected. Five samples were of a previously identified 'variant' which is distinguished from race UK 2 by being avirulent on Armelle, Katy, Mirra and Astrix (Jones and Clifford, 1979). This is tentatively designated race UK 3. The remaining four isolates represent a hitherto undetected spectrum of virulence which is similar to that of race UK 2 with the exception of avirulence on Astrix, Mirra and Katy. This has been designated race UK 4.

Further tests of selected isolates of UK 3 and UK 4 were carried out on differential sets of winter cultivars which had previously been vernalised together with the spring differentials. Vernalisation did not affect the pattern of responses and the results served to confirm the identity of races UK 3 and UK 4. The differential interactions are summarised in Table 2.

Table 1. Response of spring and winter differential cultivars to isolates of Rhyn

Isolate	Differential		Maris		Triumph		La Mesita		Magnum		Armelle		Otter		Astrix		M
	Rs-79		%	RT	%	RT	%	RT	%	RT	%	RT	%	RT	%	RT	
3. Athene	70	4	70	4	0	-	0	-	50	4,3	70	4	60	4	3		
5. Igri	50	4,3	50	4,3	0	-	0	-	40	4-2	60	4	50	4,3	4		
6. Otter	60	4-2	70	4-2	1	1	0	-	30	3-1	60	4-1	0	-			
8. Astrix	60	4,3	60	4-2	0	-	0	-	40	4-2	60	4,3	30	4-2	3		
9. Hoppel	60	4,3	70	4	0	-	0	-	60	4,3	60	4-2	25	4-2	3		
10. Athene	60	4	60	4,3	1	1	1	1	10	3-1	70	4	10	3-1	1		
12. Athene	50	4-2	50	4	0	-	0	-	40	4-3	50	4,3	40	4-2	2		
13. Athene	60	4	50	4	0	-	0	-	50	4	60	4	20	4	1		
15. Igri	70	4-2	60	4-2	0	-	0	-	50	4-2	70	4	15	3-1	2		
16. Athene	60	4,3	60	4,3	0	-	0	-	50	4-2	60	4-2	15	4-1	2		
20. Hoppel	50	4-1	50	4,2	0	-	0	-	20	3-1	40	4-1	30	3-1	3		
21. Otter	50	4,3	50	4,3	0	-	0	-	0	-	50	4-1	0	-			
22. Athene	50	4-2	60	4-2	0	-	0	-	40	4-2	60	4-2	25	3-1	2		
24. Katy	60	4	70	4	0	-	0	-	50	4,3	60	4	30	4-2	3		
25. Athene	50	4,2	50	4	0	-	0	-	40	4,3	50	4,3	30	4-2	2		
26. Athene	60	4,3	60	4	0	-	0	-	50	4	60	4	35	4,3	3		
27. Igri	70	4	70	4	0	-	0	-	50	4,3	60	4	20	3-1	2		
28. Igri	60	4,3	60	4	0	-	0	-	50	4	60	4	20	3,2	2		
29. Katy	60	4	60	4	0	-	0	-	40	4,3	60	4	25	4-2	3		
30. Athene	50	4-2	60	4,3	0	-	0	-	40	4-2	79	4	30	4-2	2		
31. Igri	70	4	70	4	0	-	0	-	50	4	60	4	20	3-1	2		
33. Trojan	70	4	70	4	0	-	0	-	50	4,3	60	4-2	15	4-1	2		
35. Gerbel	60	4	60	4	0	-	0	-	40	4-2	60	4	25	3-1	2		
36. Astrix	60	4	70	4	0	-	0	-	50	4	60	4	30	4,3	2		
37. HJ57/2	50	4,3	60	4	0	-	0	-	50	4,2	60	4-2	25	3-1	2		
39. Sonja	50	4-2	60	4-2	0	-	0	-	40	4-2	70	4	25	3-1	2		
40. Athene	60	4	70	4	0	-	0	-	50	4,3	70	4	40	4-2	3		
41. Sonja	50	4-2	60	4,2	0	-	0	-	40	4-2	60	4,2	25	3-1	2		
42. Hoppel	50	4-2	50	4-2	0	-	0	-	30	4-1	50	4-2	30	4-1	2		
43. Otter	60	4	70	4	0	-	0	-	25	4-1	60	4	0	-			
44. Trojan	60	4,3	60	4	0	-	0	-	40	4-2	60	4	20	4-1	2		
45. Athene	60	4	60	4,3	0	-	0	-	20	4-1	60	4	15	3-1	1		
46. Igri	70	4	60	4	0	-	0	-	50	4,2	70	4	20	3-1	2		
47. Igri	70	4	70	4	0	-	0	-	50	4,2	60	4	15	3-1	2		
48. Athene	70	4,3	60	4-2	0	-	0	-	0	-	70	4	0	-			
49. Sonja	60	4	60	4	0	-	0	-	40	4-2	60	4	20	3-1	2		
50. Athene	60	4,3	60	4	0	-	0	-	50	4,2	60	4,2	20	4-1	2		
53. Trojan	70	4	60	4	0	-	0	-	50	4,2	70	4	30	4-1	2		
54. Sonja	50	4,2	60	4	0	-	0	-	40	4-2	60	4	25	4-1	2		
55. Igri	70	4	60	4,2	0	-	0	-	50	4,2	60	4	20	3-1	2		
56. Igri	70	4	50	4-1	0	-	0	-	30	4-1	70	4	5	3-1			
62. Jupiter	60	4,3	60	4,3	0	-	0	-	20	4,3	50	4	0	-			
63. Sundance	70	4	50	4	0	-	0	-	0	-	60	4,3	0	-			
64. Hassan	50	4-2	50	4	0	-	0	-	0	-	60	4-2	0	-			
65. Tyra	50	4,3	50	4	0	-	0	-	0	-	40	4,3	0	-			

Table 2. Interactions between specific isolates of *Rhynchosporium secalis* and differential cultivars (R=resistant, S=susceptible)

Race	Differential cultivar		
	Armelle	Astrix	Athene
UK 1	R	R	R
UK 2	S	S	S
UK 3	R	R	S
UK 4	S	R	S

From these results it may be inferred that the winter cultivars and Armelle carry different resistance genes whereas it was previously assumed that they shared a common resistance. An interpretation of these results is that the cultivars have a gene in common which is present on it own in Athene but Armelle and Astrix have additional different resistance factors.

It must be emphasised that these results relate to glasshouse tests on seedling plants and their relevance to the field remains to be determined. For example, within the group of cultivars represented by Athene, quantitative differences in infection occur with virulent isolates. In seedling tests, Athene and Maris Trojan are similarly susceptible, Igri and Sonja are less so and Hoppel is relatively resistant. This general pattern is repeated in the field but Igri shows relatively greater resistance.

ADULT PLANT FIELD TESTS

Isolation nurseries of winter and spring cultivars were sown in 1978-1979 to assess field responses to the important virulences detected in the 1978 survey. These included a UK 3 isolate which also gave high levels of infection on Igri in the glasshouse (isolate 78-50) and another (isolate 78-51) which was specifically adapted to Athene and Hoppel. However, considerable contamination of the nurseries from local natural infection rendered the results difficult to interpret. In general, confirmation of the known relative resistance of the winter cultivars was obtained but differential interactions were obscured. Igri and Hoppel expressed their known levels of resistance in all nurseries.

The detection of virulence in relation to quantitatively expressed resistance which may also vary with host ontogeny remains a central problem, especially in the assessment of winter cultivars. Research is in progress to assess the expression of resistance in relation to host development as a necessary preliminary to the assessment of corresponding pathogen variation.

REFERENCE

- JONES, E.R.L. & CLIFFORD, B.C. (1979). Rhynchosporium of barley. Report UK Cereal Pathogen Virulence Survey for 1978, pp.54-58.

MILDEW OF OATS

I.T. Jones & E.R.L. Jones,

Welsh Plant Breeding Station, Aberystwyth

Very few viable samples were received, probably due to the general low incidence of oat mildew in the United Kingdom during 1979, consequently only limited conclusions can be drawn from the results.

There was a slight change in the virulence pattern from previous years in that the virulences OMV 1 (Race 2) and OMV 1 + 3 (Race 4) were not detected.

There was a marked increase in OMV 1 + 2 (Race 3) which attacks cultivars with Cc 4146 resistance e.g. the spring cultivars Maris Tabard and Trafalgar (OMR group 2), but lacks the virulence to colonise OMR group 3 cultivars with 9065 Cn resistance such as Mostyn. A corresponding decrease occurred in the more complex virulence combination OMV 1 + 2 + 3 (Race 5) which is able to attack both OMR groups 2 and 3.

Virulence to the Avena barbata resistance in the translocation line Cc 6490 was not detected in the survey samples.

The adult plant response of ten cultivars with known seedling factors, when grown in four different field isolation nurseries, generally reflected the seedling mildew resistance pattern, thus confirming that satisfactory isolation had been maintained.

Whether this level of isolation would prevail in a season with higher incidence of mildew remains to be seen. Interesting and important differences in level of adult plant resistance between cultivars of the same resistance group were apparent.

INTRODUCTION

In common with the other disease surveys, the aim of the oat mildew survey is to detect at the earliest possible stage any increased virulence to the resistances present in established cultivars and breeders' lines. Virulence to all the hypersensitive seedling and overall resistances (Zadoks, 1961) now being used commercially has been detected in oat mildew. The combination of virulences in Race 5 (OMV 1 + 2 + 3) renders all present commercial cultivars

susceptible to this particular race which, indeed, in 1978 was the most prevalent (Jones and Jones, 1979).

In addition to other sources of hypersensitive seedling resistance such as that from the tetraploid Avena barbata, which is being developed and incorporated in high yielding lines, resistances effective only in the adult plant stage are also being utilised. Since cultivars with this type of resistance will probably be increasingly grown in the near future early detection of any erosion of such resistance is important. A preliminary experiment was therefore conducted in 1979 to ascertain whether exposed isolation nurseries can be used in testing different isolates of mildew. The results are reported in this paper.

METHODS OF TESTING

Seedling Test

Leaf samples are placed on filter paper soaked in 5% sucrose solution to multiply the spores. Bulk inoculum and/or single pustules from the leaf segments are further increased on seedlings kept in 'xylonite' transparent isolation cylinders at 15-17°C and receiving 16h artificial light from fluorescent tubes. The inoculum thus multiplied is then brushed onto seedlings of the test varieties grown in a spore proof glasshouse. The seedlings are isolated after inoculation by placing 'xylonite' covers over the small trays in which they have been grown. These are then maintained at 15- 17°C and disease reactions recorded 8-14 days after inoculation.

Forty one samples of mildew were received in 1979, comprising 36 spring and five winter oat cultivars. Thirty one of the samples were from Eire, but due to postal delays none gave viable inoculum. Among the remainder, however, six from Harper Adams Agricultural College, Salop, and one each from central Dyfed and the winter oat breeding nursery at WPBS, Aberystwyth were successfully cultured.

Adult plant tests

Twenty genotypes including 10 of the 1979 recommended winter and spring oat cultivars (Table 3) and 10 WPBS breeders' lines were sown in four well separated isolation nurseries at WPBS. Four replicates of each of the 20 genotypes were sown as foot squares (2g seed) in a randomised block layout. Squares were spaced 2 feet (60 cm) apart, with a disease spreader drill of a susceptible cultivar sown alongside. When the spreader drill plants were at

the 4-5 leaf stage, small pots containing seedlings infected with one of the four main races of oat mildew namely, Races 2, 3, 4 or 5, were transplanted into the drills of spreader plants at 1 metre intervals in order to establish an epidemic level of mildew in each isolation nursery. Recordings of reaction type (0-4) and percent leaf area covered with mildew were made on three successive dates during the season, namely, 28 June - 2 July, 9-12 July and 16-19 July, 1979.

RESULTS AND DISCUSSION

Seedling tests

Few viable samples, from only three locations in the United Kingdom were received in 1979, probably due to the very low incidence of oat mildew during this season. The interpretation of the virulence frequencies presented in Table 1 should, therefore, be viewed with caution.

Table 1. Race and virulence group frequencies identified from samples received in 1979 compared with the results of the previous two years

Virulence group (Race)	No. of isolates in 1979	Frequency (% of total)		
		1979	1978	1977
OMV 1 (2)	0	0	3	5
OMV 1+2 (3)	5	62	42	25
OMV 1+3 (4)	0	0	3	35
OMV 1+2+3 (5)	3	38	52	35

Details of the host cultivars from which mildew cultures were satisfactorily established and the location from which the leaf samples were received are given in Table 2.

Table 2. Virulence identification giving location and cultivars from which mildew samples were received

Location	Cultivars (with virulence identified in parenthesis)
Harper Adams College, Newport, Salop	Pennal (OMV 1+2), Peniarth (OMV 1+2), Maris Osprey (OMV 1+2), Maris Quest (OMV 1+2), Panema (OMV 1+2+3), Trafalgar (OMV 1+2+3)
Lledrod, Dyfed	Condor (OMV 1+2)
WPBS, Aberystwyth, Dyfed	Dubois (OMV 1+2+3)

The trend shown in 1978 is again evident in 1979 in that OMV 1+3 or Race 4, (virulent on the differential cultivars Manod and 9065 Cn) has decreased still further probably due to the much reduced area sown to the spring cultivar Mostyn which has resistance to this virulence combination. Furthermore, there appears to be an increase in the OMV 1+2 (Race 3) virulence, (which produces a compatible reaction on Cc 4146 and cultivars with this resistance such as the spring cultivars Maris Tabard and Trafalgar), relative to the more complex combination OMV 1+2+3 present in Race 5. This may again be a reflection of the decreased area sown to cultivars of the OMR group 3 (9065 Cn resistance) such as Mostyn, that area now being sown mainly to cultivars of the OMR group 2 having Cc 4146 resistance. Thus the mildew population, in order to survive under the present situation, does not require to possess combined virulence (as in Race 5) to both resistance groups.

However, other cultivars with resistance belonging to the OMR group 3 are again being commercially grown, for example, Panema winter oat. As greater areas become occupied by these new cultivars it will be interesting to observe whether the frequency of the combined virulence OMV 1+2+3 will again become predominant. The sample of mildew from Panema received in 1979 although giving a Race 5 (OMV 1+2+3) reaction in the initial test, appeared in subsequent independent tests of the inoculum from the tester varieties Cc 4146 and 9065 Cn to lose virulence to 9065 Cn when the isolate had only been transferred through Cc 4146. This phenomenon could explain the rather rapid apparent decrease in the OMV 1+2+3 (Race 5) in 1979 and consequent increase in OMV 1+2 (Race 3). Single spore tests are now being undertaken, either to confirm that this happens, or to ascertain whether the original inoculum contained a mixture of virulence combinations.

Virulence to the A. barbata resistance in the translocation line Cc 6490 reported in 1978 (Jones and Jones, 1979) was not detected in any of the survey samples received in 1979.

Adult plant tests

A summary of the adult plant reactions on the ten commercial cultivars is given in Table 3, the observations on the breeders' lines being omitted. The mean percentage leaf area infected and reaction type over the four replicates and three scoring dates are given for each of the four nurseries.

Table 3. Percentage leaf area covered with mildew (%) and reaction type 0-4 scale (R.T.) of 10 oat cultivars grown in four isolation nurseries in the field and inoculated with four different mildew isolates

Cultivar	OMR group	OMV 1 (Race 2) % R.T.	OMV 1+2 (Race 3) % R.T.	OMV 1+3 (Race 4) % R.T.	OMV 1+2+3 (Race 5) % R.T.
Leanda	0	26 4,2	18 3,2,1	23 4,2	15 3,1
Saladin	0	22 3,2	15 4,2	26 4,2	12 4,2
Peniarth	1	22 4	4 2,3	24 4	13 4
Pennal	1	17 3,2	12 3,2	12 3,2	11 2,3
Maris Osprey	1	19 4	1 2,3	20 4	12 4
Maris Oberon	2	4 1,2,3	23 3,2	18 3,2	9 1,3
Maris Tabard	2	4 On, 2	18 4	18 4,2	6 On, 1,2
Trafalgar	2	12 3	19 4	28 4	21 4
Panema	3	7 3,2	2 On, 2,3	20 4,2	15 4
Mostyn	3	12 3,2	1 On, 2	31 4	15 3,2

R.T. = Standard reaction types, most frequent in the range given in order of predominance

% = Percentage leaf area covered with mildew

It appears that, at least under the conditions prevailing in 1979, acceptably reliable results can be obtained from mildew isolation nurseries. For instance, in the nursery inoculated with OMV 1 (Race 2) the cultivars with known specific seedling resistance to this virulence, e.g. Panema and Mostyn (OMR group 3), and Maris Oberon, Maris Tabard and Trafalgar (OMR group 2) showed significantly lower percentage mildew infection than those with no known resistance factors, e.g. Leanda, Saladin (OMR group 0) and Peniarth, Fennal and Maris Osprey (OMR group 1).

On the other hand, in the nursery inoculated with OMV 1+2 (Race 3) cultivars of the OMR group 2, with resistance derived from Cc 4146 showed a high reaction type and a percentage area infected of 18-23% as would be expected. In contrast, Panema and Mostyn (OMR group 3) with specific resistance to this virulence had a highly resistant reaction type and only 1-2% leaf area infected.

However, in the OMV 1+3 (Race 4) nursery, Panema and Mostyn appeared very susceptible, as expected since they lack seedling resistance to this isolate. In this nursery all other cultivars showed a considerable amount of mildew, including unexpectedly OMR group 2 cultivars. This could have been due to cross contamination but it is more likely due to known slight contamination in the original inoculum released in this particular nursery.

In the nursery inoculated with OMV 1+2+3 (Race 5) isolate, which has seedling virulence to all entries, there were significant differences in percentage mildew between cultivars at the adult plant stage. For example, within the OMR group 2 cultivars Maris Oberon and Maris Tabard showed a considerable level of resistance with highly resistant reaction type, while Trafalgar, with seedling resistance derived from the same source (Cc 4146) had 21% leaf area infected and a susceptible type 4 reaction. A rather similar result was observed when these cultivars were grown in the nurseries inoculated with OMV 1+3 (Race 4) and OMV 1 (Race 2) and confirms adult plant tests carried out in the laboratory and glasshouse.

Since the results obtained in the four nurseries reflect, in general fairly closely, the seedling resistance patterns of the 10 cultivars selected for this experiment, it appears that there has been almost no cross-contamination between the four nurseries. However, during 1979 the level of oat mildew in the Aberystwyth area and throughout the country was extremely low, and whether this level of isolation would be maintained in a more normal disease year situation remains to be seen.

REFERENCES

- JONES, I.T. & JONES, E.R.L. (1979). Mildew of oats. Report UK Cereal Pathogen Virulence Survey for 1978, pp. 59-63.
- ZADOKS, J.C. (1961). Yellow rust on wheat: studies in epidemiology and physiologic specialization. Tijdschrift over Plantenziekten 67, 69-256.

EVIDENCE FOR THE EFFECTIVENESS OF CULTIVAR DIVERSIFICATION IN REDUCING THE SPREAD OF YELLOW RUST AND MILDEW IN CEREALS

R H Priestley

National Institute of Agricultural Botany, Cambridge

and M S Wolfe

Plant Breeding Institute, Cambridge

The effectiveness of cultivar diversification in reducing the spread of yellow rust and mildew depends firstly on the proposition that cultivars with specific resistance generate populations of pathogen spores that are more adapted to themselves than to other cultivars, and secondly that spores from such populations produced on one field pass to neighbouring fields. Evidence is briefly described to show that these propositions generally hold true. Diversification is also valuable as an insurance against sudden outbreaks of disease on previously resistant or partially resistant cultivars affecting a farmers entire cereal acreage. Growing a range of cultivars lessens the chance that all will be susceptible to any new pathogenic race. This is regardless of any reduction in disease spread.

INTRODUCTION

Cereal growing in the United Kingdom is characterised by the widespread cultivation of few cultivars. The main advantage to the farmer is that it allows him to concentrate his resources into one or a few cultivars that are likely to give him the greatest return. The main disadvantage is that such a high degree of genetic uniformity creates ideal conditions for the rapid spread of new forms of plant pathogens, should they occur.

The deliberate re-introduction of genetic diversity has been proposed by Marshall (1977) and many workers as a method of reducing this vulnerability. In the United Kingdom, this has taken the form of cultivar diversification schemes. These encourage farmers to grow a number of cultivars possessing different specific resistance factors either in adjacent fields or in the same field in cultivar mixtures. Schemes for diversification against wheat yellow rust and barley mildew were proposed in 1975 (Priestley and Wolfe, 1977) and have been revised annually to take account of new cultivars.

Recently the wheat scheme has been extended to include resistance to mildew (this Report, p.78). The development of these schemes and examples of the potential benefits of cultivar diversification in reducing disease spread has recently been described by Priestley & Bayles (1980). The role of cultivar diversification in an integrated disease management system has also been discussed (Priestley, 1979).

It can be postulated that, because of natural selection, most of the pathogen spores generated on cultivars with a particular specific resistance will tend to possess virulence for these cultivars. However, these spores will tend not to possess virulence for cultivars with other specific resistances because there is no selection for those characters. Consequently, disease will tend to spread less rapidly between fields of cultivars with different specific resistances than between those with identical resistances, assuming that spores pass between neighbouring fields. These postulates can be tested using the results of virulence surveys and observations of field interactions and cultivar mixtures.

EVIDENCE FROM VIRULENCE SURVEYS

In these surveys, disease samples are collected from field crops of particular cultivars and subsequently inoculated onto a set of other cultivars under standard conditions. The results of surveys of barley mildew (Wolfe & Slater, 1980), wheat yellow rust (Priestley & Byford, 1980) and wheat mildew (Bennett, 1980) have all shown that isolates collected from cultivars possessing specific resistances are less virulent on cultivars possessing other resistances than on cultivars possessing that same resistance.

EVIDENCE FROM FIELD INTERACTIONS

Yarham, Bacon & Haywood (1971), Jenkyn & Bainbridge (1974) and Wolfe (unpublished) observed gradients of mildew infection from one crop into neighbouring areas. Yarham et al (1971) showed that the average incidence of mildew in fields of spring barley increased with their proximity to winter barley, indicating shallow disease gradients over large distances. Jenkyn et al (1974) discussed the difficulty of arranging field trials on barley mildew to avoid the problem of interference between small plots, illustrating that on this scale spore transfer readily occurs. Wolfe (unpublished) has shown that the population structure of the pathogen in one field could be influenced for a considerable distance by spores arriving from a neighbouring field. Clearly, if the neighbouring fields are of cultivars with different specific resistances, more spores will be non-infective following such

transfers than if the cultivars possess identical specific resistances.

EVIDENCE FROM CULTIVAR MIXTURES

Cultivar mixtures, in effect, reduce the field size to that of single plants and thus maximise the interactions between populations of spores generated on cultivars with different specific resistances (Wolfe, 1978; Wolfe and Barrett, 1980). In such systems, reductions in disease intensity of more than 50% are usual, and may be as high as 80%, principally due to a proportion of the spores generated being non-infective on the plants on which they are deposited. The reductions will not be so great between conventional fields, but in the early part of the season when the diseases are at their most damaging, epidemic development is likely to be retarded until the numbers of spores produced within the crop greatly exceed those being blown in from neighbouring crops, (Wolfe and Schwarzbach, 1978).

DIVERSIFICATION AS INSURANCE

Diversification between fields and between seasons reduces the risk of overall disease loss on the farm regardless of the extent to which diversification may reduce disease spread between neighbouring fields. It is not possible at present to predict accurately on a local basis a sudden increase of disease on a previously resistant or partially resistant cultivar. When such events do occur, it is usually extremely difficult and expensive to attempt to contain the loss by the use of fungicides. The use of cultivars with different resistances therefore provides insurance against such a flare-up; the larger the number of different cultivars used, the greater the degree of insurance.

REFERENCES

- BENNETT, F G A (1980). Mildew of wheat. United Kingdom Cereal Pathogen Virulence Survey 1979 Annual Report, (this volume)
- JENKYN J F & BAINBRIDGE A (1974). Disease gradients and small plot experiments on barley mildew. Annals of Applied Biology 76, 269-279
- MARSHALL, D R (1977). The advantages and hazards of genetic homogeneity. Annals of New York Academy of Sciences 287, 1-20.
- PRIESTLEY, R H (1979). The management of resistant varieties. Proceedings 1979 British Crop Protection Conference - Pests and Diseases 3, 753-760.

- PRIESTLEY, R H & BAYLES, R A (1980). Varietal diversification as a means of reducing the spread of cereal diseases in the United Kingdom. Journal of the National Institute of Agricultural Botany 15, (in press).
- PRIESTLEY, R H & BYFORD, P (1980). Yellow rust of wheat. United Kingdom Cereal Pathogen Virulence Survey 1979 Annual Report, (this volume).
- PRIESTLEY, R H & WOLFE, M S (1977). Crop protection by cultivar diversification. Proceedings 1977 British Crop Protection Conference - Pests and Diseases 1, 135-140.
- WOLFE, M S (1978). Some practical implications of the use of cereal variety mixtures. In Plant Disease Epidemiology (Ed. by P R Scott & A Bainbridge) pp. 201-207. Blackwell Scientific Publications, Oxford.
- WOLFE, M S & BARRETT, J A (1980). Can we lead the pathogen astray? Plant Disease 64, 148-155.
- WOLFE, M S & SCHWARZBACH, E (1978). Patterns of race changes in powdery mildews. Annual Review of Phytopathology 16, 159-180.
- WOLFE, M S & SLATER, S E (1980). Mildew of barley. United Kingdom Cereal Pathogen Virulence Survey 1979 Annual Report, (this volume).
- YARHAM D J, BACON, E T G & HAYWARD, C F (1971). The effect on mildew development of the widespread use of fungicide on winter barley. Proceedings of the 6th British Insecticide and Fungicide Conference 1, 15-25.

CULTIVAR DIVERSIFICATION SCHEMES FOR WINTER WHEAT AND SPRING BARLEY, 1980

Cultivar diversification schemes to reduce the spread of yellow rust in winter wheat and mildew in spring barley have been produced by the UKCPVS Committee since 1975. The first two schemes (following) are 1980 versions which update those in the 1978 Annual Report. The third scheme is an extension of the winter wheat scheme to include resistance to mildew as well as yellow rust. Diversification Group (DG) numbers refer to yellow rust resistances, as previously. DG letters refer to mildew resistances.

All three schemes were sent to the authorities responsible for evaluating new cereal cultivars in England & Wales, Scotland and Northern Ireland, and to the Agricultural Development & Advisory Service.

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE RISK OF YELLOW RUST IN WINTER WHEAT 1980

Where winter wheat crops are grown adjacent to each other, or in the same field in successive years, the risk of severe yellow rust infections can be reduced by using cultivars that possess different resistance factors. Each diversification group (DG) below consists of cultivars possessing similar resistance factors. Thus the risk of disease in an area can be reduced by growing only one cultivar from within each diversification group. The only exception to this is that cultivars in DG 1 may be sown adjacent to one another because they are resistant to yellow rust spreading from all other varieties.

DG 1	Anvil	Copain	no varieties	DG 5
Aquila	Hustler			
Armada	Maris Huntsman			DG 6
Atou	Sportsman			Brigand
Avalon	Virtue			Hobbit
Bouquet				Kador
Bounty	DG 3			
Flanders	Kinsman			DG 7
Flinor	Maris Freeman			Stuart
Mardler				
Prince	DG 4			
Sentry	no varieties			

Choosing cultivars to grow adjacent to one another

In the table below, + signs indicate those combinations of DGs that can reduce the risk of yellow rust when single cultivars from different groups are grown adjacent to one another. It should be noted that DG 1 is exceptional in that individual cultivars from this group may be grown adjacent to each other.

DGs that can reduce risk of yellow rust when grown with chosen DG

Chosen DG	DG 1	DG 2	DG 3	DG 6	DG 7
DG 1	+	+	+	+	+
DG 2	+	•	+	+	+
DG 3	+	+	•	+	+
DG 6	+	+	+	•	+
DG 7	+	+	+	+	•

Spring wheat cultivars

Spring wheat crops should not be grown adjacent to susceptible winter wheat cultivars as these may act as a source of infection for the spring crop. If this is unavoidable, choose spring cultivars with a high level of resistance.

Where spring barley crops are grown adjacent to each other, or in the same field in successive years, the spread of mildew between crops can be reduced by using cultivars that possess different resistance factors. Each diversification group (DG) below consists of cultivars possessing similar resistance factors. Thus, disease levels in an area can be reduced by growing only one cultivar from within each diversification group. The only exceptions to this are a) cultivars in DG 0 which do not contribute because they are susceptible to mildew spreading from all other cultivars, and b) cultivars in DG 1 which may be sown adjacent to one another because they are resistant to mildew spreading from all other cultivars.

DG 0	Armelle	DG 4	Goldmarker	Tyra	DG 7
DG 1	Atem	DG 5	Aramir	Simon	DG 8
DG 2	Midas	Athos	Hassan	Claret	DG 9
DG 3	Abacus	Piccolo	Portos	Dram	DG 10
DG 5	Aurea	Tintern	Maris Mink	Egmont	
Flare	Georgeie	Ark Royal	Keg		
Koru	Lofa Abed	Mazurka	Triumph		
Sundance	Wing				

Choosing cultivars to grow adjacent to one another

In the table below, + signs indicate those combinations of DGs that can reduce mildew spread when single cultivars from different groups are grown adjacent to one another. It should be noted that DG 1 is exceptional in that individual cultivars from this group may be grown adjacent to each other.

DGs that can reduce mildew spread when grown with chosen DG

Chosen

DG	DG 1	DG 2	DG 3	DG 4	DG 5	DG 6	DG 7	DG 8	DG 9	DG 10
DG 1	+	+	+	+	+	+	+	+	+	+
DG 2	+	+	+	+	+	+	+	+	+	+
DG 3	+	+	+	+	+	+	+	+	+	+
DG 4	+	+	+	+	+	+	+	+	+	+
DG 5	+	+	+	+	+	+	+	+	+	+
DG 6	+	+	+	+	+	+	+	+	+	+
DG 7	+	+	+	+	+	+	+	+	+	+
DG 8	+	+	+	+	+	+	+	+	+	+
DG 9	+	+	+	+	+	+	+	+	+	+
DG 10	+	+	+	+	+	+	+	+	+	+

Winter barley cultivars

Susceptible winter barley cultivars may act as a source of infection for local spring barley crops. Thus, susceptible spring barley cultivars should not be grown close to these crops.

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

- CULTIVAR DIVERSIFICATION SCHEME TO REDUCE SPREAD OF YELLOW RUST AND MILDEW IN

