CEREAL RUSTS BULLETIN

EDITED BY

G. E. RUSSELL

VOLUME 6 PART 1

PUBLISHED BY

THE EUROPEAN AND MEDITERRANEAN CEREAL RUSTS FOUNDATION

Cereal Rusts Bulletin Vol. 6, Part 1, 1978

CONTENTS

EDITORIAL

I would first like to pay tribute to my predecessor, Dr John Manners, for his excellent work in establishing the Cereal Rusts Bulletin so successfully. I am sure that we are all very grateful to him for his hard work. I will find it difficult to equal his achievements.

The late arrival of Volume 6, Part 1 has been due partly to unavoidable delays concerned with the setting up of new editorial and printing procedures and partly to an insufficiency of suitable papers for the Bulletin.

I hope that I will be inundated with good contributions within the next few weeks so that I can produce Part II for you in the Autumn! After that, future issues should be published on time provided that I am sent plenty of good papers and notes.

A special section in each issue will in future be devoted to Short Notes and Communications. Items of general and topical interest including descriptions of techniques and notices of forthcoming meetings, will be very welcome.

Contributors should send (by airmail if necessary) two copies of typescripts to me at the address below. Scientific papers will then be sent to one or more independent referees for comment.

Professor G.E. Russell
Department of Agricultural Biology
The University
Newcastle upon Tyne
NE1 7RU
England

SUBSCRIPTIONS

The Bulletin is published twice a year and is sent directly to subscribers on payment of an annual subscription. Alternatively, orders can be placed through booksellers at extra cost. Enquiries regarding subscriptions and orders should be sent to Dr J.E. Parlevliet, Treasurer, European and Mediterranean Cereal Rusts Foundation, Institute of Plant Breeding, 166 Lawickse Allee, Wageningen, The Netherlands.

Cereal Rusts Bulletin Vol. 6, Part 1, 1978

INDUCED MUTATIONS FOR VIRULENCE TO BARLEY IN BROWN RUST, PUCCINIA HORDEI OTTH.

BY J.E. PARLEVLIET AND G. VAN EIJK-BOS

Plant Breeding Department (I.v.P.), Agricultural University, Wageningen, The Netherlands

SUMMARY

Plants of the cultivars Rika x (Baladi 16 x Rika No. 7) and Cebada Capa, each carrying one resistance gene, Pa3 and Pa7 respectively, were inoculated with brown rust spores irradiated with 150 Krad γ-rays. The isolate used was avirulent to Pa3 and Pa7, but virulent to Pa, Pa2, Pa4, Pa5 and Pa8. A mutation frequency to virulence for Pa3 of up to 0.4% was observed. No mutations to virulence for Pa7 could be obtained.

INTRODUCTION

In barley, several major genes for resistance to brown rust, Puccinia hordei Otth. are known (Roane and Starling, 1967; Parlevliet, 1976; Clifford, 1977 and Tan, 1977). These genes are designated Pa, Pa2, Pa3 etc. and are assumed to operate on a gene-for-gene basis with corresponding virulence genes in the pathogen. Virulence to these resistance genes occurs widely in Europe (Bruckner, 1970; Clifford, 1974 and Rintelen, 1976). Only one gene, Pa7, is still effective everywhere in Europe, while virulence against Pa3 occurs rather infrequently (Parlevliet, 1976). Flor (1958) reported a parallel between the rate of spontaneous and induced mutations to virulence for various loci in the flax rust. With this in mind a study was done to find out how easily mutations towards virulence to Pa3 and Pa7 could be induced.

MATERIALS AND METHODS

A monospore culture was made from isolate 1-2, virulent on Pa, Pa2,

Pa4, Pa5 and Pa8, but avirulent on Pa3 and Pa7. This culture was used throughout and was multiplied on cultivar L98 which is very susceptible to brown rust. The cultivars Rika x (Baladi 16 x Rika No. 7) (Ribari) and Cebada Capa (CC), carrying Pa3 and Pa7 respectively were used to identify possible mutants for virulence to Pa3 and Pa7 as they carry no other effective resistance genes for this culture (Parlevliet, 1976).

To determine the irradiation dose to be used in the actual experiment, spores were irradiated within 24 h after collection with 0, 50, 100, 150 and 200 Krad γ-rays from a Co^{60} source with a dose rate of 850 Krad/h. The treated spores were, after mixing with Lycopodium spores to obtain a more equal distribution, dusted over L98 seedlings and the number of resulting uredosori per cm^2 leaf determined. This was repeated and the second series was used also to measure the latent period as described by Parlevliet (1975).

In the actual experiment the spores, diluted with Lycopodium spores, received either 0 or 150 Krad of irradiation. Non-irradiated (control) spores, mixed with Lycopodium, were dusted over 50 plants each of the cultivars Ribari, CC and Julia (Ju). For the irradiated treatment, 150 plants of Ribari and CC were used and 50 of Julia. The plants, in the tillering stage with five to six leaves, were grown in square plastic pots, five plants to a pot which were placed at random for inoculation. Per 50 plants, 4.3 mg of control spores or 13.0 mg of irradiated spores were applied. The cultivar Ju served to estimate the number of infections (compatible or incompatible) on Ribari and CC. Ju was chosen as it has an infection frequency representing a large group of cultivars with an intermediate level of infection frequency. On the Ju plants the infection frequency in uredosori per plant was determined, and this was used as a measure for the infection frequency per plant of Ribari and CC. Such an estimate is needed to derive estimates for mutations frequencies.

Mutants were obtained for virulence to Pa3, and a culture which was obtained by mixing several mutant cultures was compared with the mother culture 1-2 and an isolate from Israel, known to be virulent on Pa3 (culture 202). Spores of the three cultures, after multiplication on L98 plants, were used to inoculate seedlings of ten differential barley cultivars and the infection types were recorded four to five days after the uredosori or incompatible lesions became visible.

In all experiments, inoculated plants were incubated at 100% RH for at least 16 h in the dark.

RESULTS

The infection frequency decreased sharply and the latent period increased with higher doses of irradiation (Table 1). The LD 50, when measured by the frequency of sporulating infections, was in both series about 120 to 130 Krad. Schwinghamer (1959), studying flax rust, observed the highest mutation rate at 50 to 65% lethality. For the actual experiments, therefore, a dose of 150 Krad was chosen.

No sporulating infections could be found on the plants of Ribari and CC inoculated with control spores and on CC inoculated with irradiated spores. The irradiated spores produced a fairly large number of small sporulating infections on Ribari. These uredosori appeared very slowly, the first one 17 days after incubation, whereas most of the uredosori on Ju became visible between 8 and 16 days after incubation. The number of uredosori on Ribari increased to 85 after 21 days, to 129 after 27 days and to 248 after 34 days.

The average numbers of uredosori per plant on Julia were 298.2 for the control spores and 285.8 for the irradiated spores.

Clean seedlings of Ribari were inoculated with spores of 19 uredosori visible on Ribari 21 days after incubation. Of the sori visible on Ribari 34 days after incubation, 16 sori were multiplied separately on seedlings of L98 and then tested on Ribari seedlings in the same way as the 19 sori of the first group. Table 2 shows the results. Sixteen out of 35 isolated cultures showed no reaction at all when brought onto seedlings of Ribari or L98. Of the remaining 19 cultures, six gave an incompatible and 13 a compatible reaction with Ribari. Several of these were multiplied on Ribari seedlings and invariably showed a susceptible infection type. However they were not easy to multiply, probably due to a slower growth and a lower rate of spore production. After mixing these mutant cultures, a culture was obtained which was hardly discernible from the original culture 1-2. Its multiplication was easy and sporulation was abundant. This culture was compared with the cultures 1-2 and 202 on a series of differential barley cultivars (Table 3).

Table 1. Infection frequency in uredosori per cm^2 and latent period in days on seedlings of cultivar L98 for uredospores of Puccinia hordei irradiated with various doses of γ-rays.

Dose in Krad	Number of sori/cm^2		Latent period
	series I	series II	series II
0	10.6	14.6	10.3
50	10.0	12.0	10.6
100	6.8	9.2	10.8
150	3.2	5.4	11.0
200	2.2	4.2	12.0

Table 2. Classification of 35 Puccinia hordei isolates, derived from 35 sporulating pustules on Ribari, according to their interaction with Ribari; 19 were tested directly on Ribari, the others following culture on L98.

Reaction type of isolate	Number of isolates		
	Ribari	ex L98	ex Ribari*
None	8	8	-
0;	4	0	2
4	7	8	6

* via L98

Table 3. Infection types of Puccinia hordei culture 1-2, a mutant of it, M 1-2, and a culture from Israel, 202, on a differential barley cultivar series.

Cultivar	Resistance Gene	Culture		
		1-2	M 1-2	202
Sultan	-	4	4	4
Sudan	Pa	4	4	4
Peruvian	Pa2	4⁻	4⁻	4
Ribari	Pa3	0;	4	4
Estate	Pa3	0;	4⁻	4
Gold	Pa4	4	4	4
Quinn	Pa2, Pa5	4⁻	4⁻	4
Cebada Capa	Pa7	0;	0;	0;
Egypt 4	Pa8	4	4⁻	4
EP75	Paz [2)]	2	2	4⁻

1) Standard conventions

2) This gene is not yet designated (Parlevliet, 1976), but it appears different from the other eight already designated.

The mutant culture differed from 1-2 only in its virulence to Pa3 and from the culture from Israel in being avirulent on EP75. It apparently carries virulence to at least seven resistance genes.

DISCUSSION

The sporulating lesions on Ribari were nearly all small, retarded uredosori, not representative of a fully compatible reaction. Of the 35 sori tested, 16 gave no reaction at all, probably because of failure to infect. This is not surprising as the number of spores collected from the small lesions was often minute. Of the 19 sori giving a reaction on Ribari, six were incompatible indicating that not all sporulating lesions on Ribari represented mutations. Thirteen sori, however, appeared to be mutants.

If 13 out of 19 sori on Ribari were mutants, 13/19 x 248 = 170 mutants approximately were present on the 150 Ribari plants. The total number of infections, compatible and incompatible could be estimated through Ju, which had a mean of 285.8 uredosori per plant. This would give a calculated number of 42870 sori on 150 plants. Assuming a similar infection frequency on Ribari, 170 out of 42870 infections or approximately 0.4% produced a mutant virulent to Pa3.

This of course is a rough estimate. The ratio, mutant to non-mutant, of 13 : 19 among sporulating lesions on Ribari could be too high. The 35 sporulating lesions taken for testing might have been the larger ones, although there was no conscious selection for larger pustules. Among the larger pustules the mutants may occur with a higher frequency more than two- to three-fold. Another inaccuracy in the estimate is the assumption that Ju and Ribari have similar infection frequencies. Although the necrotic infection courts were not counted the impression was that the number of 0; type infections on Ribari was not considerably larger or smaller than the number of 4 type infections on Julia. Also the size of the leaf area did not differ much and the plants of the three cultivars included were not discernibly different in size. Taken together the error in the estimate for the mutation frequency may be considerable, but even with a four-fold over-estimation a relative high mutation frequency of 0.1% remains.

Contrasting to that is the total absence of mutations on CC. A possible explanation could be that isolate 1-2 is heterozygous for avirulence to Pa3

and homozygous for avirulence to Pa7. In the former, a single mutation could result in a homozygous virulent condition (from Va3 va3 to va3 va3) assuming that virulence is inherited in a recessive way. In the latter situation a double mutation would be required (from Va7Va7 to va7va7). This hypothesis is not unlikely as virulence to Pa3 occurs in Europe, but virulence to Pa7 had not been recorded yet (Parlevliet, 1976).

Mixing several fairly weak mutant cultures gave a more vigorous culture and it is tempting to consider the possibility of somatic recombination between the cultures, each of which was fairly weak (probably due to irradiation damage e.g. very small chromosomal aberrations). Recombination between such differently damaged chromosome sets could restore the vigour.

REFERENCES

BRÜCKNER, F. (1970). Varietal resistance of summer barley to leaf rust (Puccinia hordei Otth.) in Czechoslovakia. Genetica a Slechteni 6, 143-151

CLIFFORD, B.C. (1974). The choice of barley genotypes to differentiate races of Puccinia hordei Otth. Cereal Rusts Bull. 2, 5-6

CLIFFORD, B.C. (1977). Monitoring virulence in Puccinia hordei: A proposal for the choice of host genotypes and survey procedures. Cereal Rusts Bull. 5, 34-38

FLOR, H.H. (1958). Mutation to wider virulence in Melampsora lini. Phytopathology 48, 297-301

PARLEVLIET, J.E. (1975). Partial resistance of barley to leaf rust, Puccinia hordei. I. Effect of cultivar and development stage on latent period. Euphytica 24, 21-27

PARLEVLIET, J.E. (1976). The genetics of seedling resistance to leaf rust, Puccinia hordei Otth. in some spring barley cultivars. Euphytica 25, 249-254

RINTELEN, J. (1976). Physiologic specialisation of Puccinia hordei Otth. and reactions of varieties and cultivars to physiological races. Barley Genetics III, Proc. of 3rd Int.Barley Gen.Symp. Garching, 1975, 464-467

ROANE, C.W. & STARLING, T.M. (1967). Inheritance of reaction to Puccinia hordei in barley. II. Gene symbols for loci in differential cultivars. Phytopathology 57, 66-78

SCHWINGHAMER, E.A. (1959). The relation between radiation dose and the frequency of mutations for pathogenicity in Melampsora lini. Phytopathology 49, 260-269

TAN, B.H. (1977). A new gene for resistance to Puccinia hordei in certain Ethiopian barleys. Cereal Rusts Bull. 5, 39-43

Cereal Rusts Bulletin Vol. 6, Part 1, 1978

OCCURRENCE OF VIRULENCE IN PUCCINIA STRIIFORMIS FOR COMPAIR WHEAT IN ENGLAND

BY R. JOHNSON
Plant Breeding Institute, Trumpington, Cambridge
R.H. PRIESTLEY
National Institute of Agricultural Botany, Huntingdon Road, Cambridge
AND E.C. TAYLOR
Southampton University, Department of Biology, Southampton

INTRODUCTION

The hexaploid wheat cultivar Compair has resistance to yellow rust derived from Aegilops comosa carried as a translocation of a segment of chromosome 2M of Aegilops comosa to chromosome 20 of wheat (Riley, Chapman and Johnson, 1968a, 1968b). Until 1976 Compair was resistant to all isolates of Puccinia striiformis Westend. in Britain although it was susceptible in Mediterranean and Asiatic regions (Stubbs, Fuchs, Vecht and Basset, 1974). Since the beginning of 1976 races with virulence for Compair have been detected at three centres in England, in January 1976 at the University of Southampton, in June 1976 at the National Institute of Agricultural Botany (NIAB) Cambridge and in July 1976 at the Plant Breeding Institute (PBI) Cambridge. This paper describes the different circumstances under which Compair virulence was detected and discusses evidence for resistance to yellow rust in A. comosa in addition to that transferred to Compair.

METHODS AND RESULTS

Physiologic race numbers (eg 37 E132) are given according to the World and European nomenclature system described by Johnson, Stubbs, Fuchs and Chamberlain (1972). The individual phenotypic virulence factors present in each race (eg V 1, 2, 6) are numbered according to the system described by Priestley and Byford (1977) in which the numbers refer to virulence for the corresponding numbered differential cultivars in a set.

At Southampton, an isolate of race 36 E132 (= V 2, 6) was derived by screening the progeny of a mixed population of uredospores of races 37 E132 (= V 1, 2, 6) and an albino isolate of race 104 E137 (= V 2, 3, 4) (Taylor, 1976). Spores of race 36 E132 (= V 2, 6) were irradiated with ultra violet for 25 minutes at 20cm from a germicidal lamp which reduced their germination to about 1%. Some cultivars from the World and European sets of differential cultivars were inoculated with uredospores that were the progeny of the irradiated spores and pustules developed on Compair which had hitherto been resistant in Britain. Spores from these pustules proved to belong to race 36 E148 (= V 2, 6, 8) thus differing from the parent race only in virulence for Compair (V 8).

At the NIAB, a sample of yellow rust collected from a field crop of the winter wheat cultivar Clement at Boston, Lincolnshire was found to possess V 2, 3, 4, 8, 9. The virulence for Compair (V 8) was confirmed by a test on a further stock of Compair from PBI. Although it has not been tested on the World and European differential cultivars it is probable that this isolate would be classified as race 232 E153.

At the PBI, a virulent colony was observed on Compair in a differential test of race 108 E9 (= V 3, 4, 6). A culture from this colony was identified as race 108 E25 (= V, 3, 4, 6, 8) which differs from the parent colony only in virulence for Compair (V 8). Race 108 E25 (= V 3, 4, 6, 8) was virulent on lines developed by backcrossing resistance from Compair into Maris Widgeon and also on chromosome substitution lines in which chromosome 2D of Maris Widgeon was replaced by the whole chromosome 2M of A. comosa. In contrast, A. comosa itself was highly resistant.

DISCUSSION

It was surprising that, after almost ten years in which Compair was resistant to all isolates of P. striiformis available in Britain, cultures virulent on it were obtained in three different places within seven months. There seems, however, to be no relationship between the three events because the circumstances of occurrence were different and, in each case, virulence for Compair was combined with a different range of other virulence factors.

The virulence on Compair evidently originated by mutation. At Southampton a mutagen was used and both there and at PBI only the single change, to virulence for Compair, was detected to distinguish the races

from the parental cultures in which they originated. If virulence is recessive a double mutation would have been required in each case indicating that the genetic locus concerned is readily mutable. The gene for resistance to yellow rust in Compair may, therefore, be of little use alone, in protecting wheat crops from this disease in Europe.

Evidently A. comosa contains resistance additional to that which was transferred to Compair and this must be carried on a chromosome other than 2M. This may provide a useful marker in further investigations of the genome of A. comosa.

REFERENCES

JOHNSON, R., STUBBS, R.W., FUCHS, E. & CHAMBERLAIN, N.H. (1972). Nomenclature for physiologic races of Puccinia striiformis infecting wheat. Trans.Br.Mycol.Soc. 58: 475-480

PRIESTLEY, R.H. & BYFORD, P. (1977). Yellow rust of wheat. Ann. Rep. Physiologic race survey (Cereal Pathogens) 1976 Cambridge, England: 6-14

RILEY, R., CHAPMAN, V. & JOHNSON, R. (1968a). Introduction of yellow rust resistance of Aegilops comosa into wheat by genetically induced homoeologous recombination. Nature Lond. 217: 383-384

RILEY, R., CHAPMAN, V. & JOHNSON, R. (1968b). The incorporation of alien disease resistance in wheat by genetic interference with the regulation of meiotic chromosome synapsis. Genet.Res.Camb. 12: 199-219

STUBBS, R.W., FUCHS, E., VECHT, H. & BASSET, E.J.W. (1974). The international survey of factors of virulence of Puccinia striiformis Westend. in 1969, 1970 and 1971. Stichting Netherlands Graan-Centrum, Technisch Bericht 21: 1-88

TAYLOR, E.C. (1976). The production and behaviour of somatic recombinants in Puccinia striiformis. Proc. 4th European and Mediterranean Cereal Rusts Conference 1976 Interlaken, Switzerland: 36-38

Cereal Rusts Bulletin Vol. 6, Part 1, 1978

CHANGES IN THE RACE COMPOSITION OF PUCCINIA GRAMINIS f.sp. TRITICI IN FRANCE, IN 1977

BY M. MASSENOT

Laboratoire de Pathologie végétale, Institut National Agronomique, 78850 Thiverval-Grignon, France

In the years 1971-76, there were few epidemics of wheat black stem rust (Puccinia graminis f.sp. tritici) in France. Race 21, as in previous years, was the most prevalent race, although others, including races 14, 133 and 186 were also present. Race 1 was found in France for the first time in 1972 but was not found again until 1977. Several foci of black rust were detected in the South of France in early June 1977, and wheat crops later became badly affected in some localities, particularly in the Rhone Valley. In the vicinity of Grignon (35 km to the West of Paris), the disease was found in the second fortnight of June and in Normandy by the end of June. However, damage was much more severe in the South than in the North.

On the basis of 70 samples collected from different parts of France (42 from the South, 28 from the North West), the following conclusions can be drawn:

1) Race 21, previously prevalent and widespread, was not detected in 1977

2) Race 14 was common in 1977, being identified in 32 per cent of the samples studied

3) Two virulent races, race 11 and race 34, were identified in 8 per cent and 60 per cent of the samples, respectively

These changes in the race composition of P. graminis f.sp. tritici and the unexpected prevalence of virulent races may have important future implications for many countries of Western Europe.

Cereal Rusts Bulletin Vol. 6, Part 1, 1978

EPIDEMIC OF STRIPE RUST IN CZECHOSLOVAKIA IN 1977

BY V. SLOVENČÍKOVÁ AND I. BAREŠ

Research Institute of Crop Production, Praha-Ryzyně, Czechoslovakia

Since the introduction of the winter wheat cultivar Mironovskaja 808 in Czechoslovakia in 1966, epidemics of stripe rust (Puccinia striiformis Westend.) have been greatly reduced. Rust nurseries have shown that Mironovskaja 808 possesses a moderate level of adult plant resistance to those races that have been widespread in Czechoslovakia.

The recent epidemic of stripe rust in Czechoslovakia was caused by the cultivation of the Yugoslavian varieties Sava and Zlatna Dolina (released in Czechoslovakia in 1976). These varieties had produced high yields in Moravia and in West and South Slovakia and their known susceptibility to stripe rust was not considered important because of the absence of stripe rust epidemics for about 10 years.

The first outbreaks of stripe rust were observed in South Moravia and Slovakia in the autumn of 1976. The winter was relatively mild and so the rust spread very early and many foci were observed at the end of April. During the epidemic Zlatna Dolina was infected more than Sava and yield losses of these varieties were estimated as 30% in comparison with the yields of the same varieties when treated with Bayleton WP 25.

The spring wheat Janus also became severely infected with stripe rust in 1977.

Four races were identified from samples of P. striiformis spores isolated from field infections (Table 1). All isolates were virulent on Strub. Dickopf and Heines VII (R2). The race possessing virulence for Heines Kolben (R6) and Heines Peko was isolated once only. Two races were frequently identified: the first possessing virulence for the varieties Chinese 166 (R1) and Ibis, the second possessing virulence for Vilmorin 23 (R3), Carsten V and Nord Desprez.

Table 1. Infection types (after McNeal et al., 1971) of seedlings of differential varieties inoculated in the glasshouse with field isolates of Puccinia striiformis.

(0-3 = resistant, 4-6 = moderately resistant or susceptible, 7-9 = susceptible)

	Differentials																	
	Clement (R9)	Suwon 92 x Omar	Strub. Dickopf	Moro (R10)	Vilmorin 23 (R2)	Heines Kolben (R6)	Lee (R7)	Chinese 166 (R1)	Heines VII (R2)	Spal. Prolific	Carsten	Compair (R8)	Nord Desprez	Heines Peko	Reichersberg 42	Hybrid 46 (R4)	Flevina	Ibis
Isolated from:																		
Kormoran, Ibis, Sava, Zlatna Dolina	0	0	9	0	1	1	1	8	9	1	1	0	1	1	1	1	1	8
Zlatna Dolina, Sava	0	0	8	0	8	0	4	0	9	4	5	0	8	1	4	8	8	0
Zlatna Dolina (only 1 isolate)	0	0	8	0	0	9	0	0	8	8	0	0	0	8	0	0	0	0
Sava, Zlatna Dolina, Vuka, Biserka	0	0	8	0	8	1	1	0	8	7	8	0	8	0	0	0	0	0

Table 2. Infection type (after McNeal et al., 1971) of adult plants of 83 varieties in plots naturally infected with Puccinia striiformis

Variety	Infection type	% flag leaf area attacked	Variety	Infection type	% flag leaf area attacked	Variety	Infection type	% flag leaf area attacked
Alcedo	0,5*	0,10	Kaliakra	5	10	Poleskaja 70	7	80
Airus	1,6*	0,10	Kavkaz	1	0	Przevalskaja	4	10
Arminda	1	0	Kragujevčka	7	20	Rannaja 47	5	50
Aurora	1	0	Krasnodarskaja 46	5	20	Ricardo	1	0
Benno	1	0	Kormoran	7	25-70	Rothwell Perdix	4	20
Biserka	8	90	Lena	5-6	50	Rotonde	4,7*	20,80
Bocquiau	3	1	Lovrin 26	8	70	Sadovo 1	8	80
Bussard	1,8*	0,79	Ludogorka	6	10	Saladin	0	0
Cama	1,7*	0,20	Mamut	1	0	Salami	4	10
Capitole Vilmorin	5	30	Maris Beacon	6	40	Sanja	4	1
Cappelle Desprez	8	70	Maris Bilbo	6	20	Saturn	6	20
Danubius	8,5*	80,20	Maris Freeman	7	30	Sava	7	50-70
Disponent	1	0	Maris Huntsman	5	20	Silvana	4	5
Dněprovskaja 521	6	30	Maris Ranger	6	20	Slavia	5	10
Drina	8	90	Maris Widgeon	1	0	Slavjanka	8	70
Essor	5	20	Marijana	7	80	Sojuz 50	5	20
Feldman	0	0	Markus	3	5	Solaris	3	5
Flevina	7	50	Mephisto	6	30	Storm	3	5
Grana	5-6	20	Merkur	1	0	Talent	1	0
Hadmer. Qualitas	3	50	Mildress	0	0	Tumult	1	0
Hybrid 46	1	0	Mironovskaja 10	1,4*	0,0	Tundra	4-5,8*	10,70
Ibis	8	50	Mironovskaja 25	7	40	Vega	9	80
Ilijčovka	4	5	Mironovskaja 808	5	5	Vuka	7	70
Jana	7	80	Mironovskaja ulučšenaja	5	10	Winnetou	1	0
Janus	7	80	Moisson	6	20	Zlatna Dolina	8	70
Jara	8	50	Nadežnaja 45	7	40	Zora	4	20
Jubilar	4	1	Nimbus	0	0	Zorba	0	0
Jubilejnaja 50	5	10-25	Oděskaja 66	7	50			

* Some plants showed different infection types

Plots of wheat varieties sown in three sites, where the natural epidemic was very severe, were assessed for infection type and infection level (Table 2). In these localities three races were isolated, the virulence of which is described in Table 1. Races attacking Heines Kolben and Heines Peko were absent.

At these sites the following varieties remained resistant: Alcedo, Almus, Arminda, Aurora, Benno, Bocquiau, Bussard, Cama, Disponent, Felfman, Hybrid 46, Kavkaz, Mamut, Maris Widgeon, Merkur, Muldress, Mironovskaja 10, Nimbus, Ricardo, Saladin, Solaris, Storm, Talent, Tumult, Winnetou, Zorba.

Many varieties were moderately resistant, including the economically important new Czechoslovak variety Slavia (licenced in 1976). Inoculated tests have shown that this variety is moderately resistant as an adult plant to races 40 E 8 (3/55) and 232 E 137 (Clement race).

ACKNOWLEDGEMENT

The authors are indebted to Dr. R.W. Stubbs, I.P.O. Wageningen, for his kind help in the race identification.

Cereal Rusts Bulletin Vol. 6, Part 1, 1978

THE ATTEMPTED PRODUCTION OF NEW PHYSIOLOGIC RACES BY SOMATIC RECOMBINATION IN PUCCINIA HORDEI

BY MAHROKH FALAHATI-RASTEGAR, J.G. MAN[illegible]'S AND J. SMARTT

University of Southampton

The alternate hosts of P. hordei, Ornithogalum spp. are confined to Europe, North Africa and western Asia, and the aecia of P. hordei are everywhere rare (Gaumann, 1959). The rust is, however, common on barley virtually wherever that crop is grown, and in many countries numbers of physiologic races have been reported. In Britain, several races are known (Clifford, 1974), but the aecial stage has only been reported once (Dennis and Sandwith, 1948). It seems likely that hybridization, as well as mutation is often involved in the production of new rust races, and the demonstration of the existence of somatic recombination in other cereal rusts (see Day, 1974), suggested to us that it might occur in P. hordei.

Material of races A, D, F, J and K of P. hordei was kindly supplied by Dr. B.C. Clifford, and an orange mutant of race A (designated A_1) was obtained by treating urediospores of race A with ethyl methane sulphonate. Single spore cultures of each race and of the mutant were established, and the following mixtures of urediospores derived from them made: A_1 + D, A_1 + F, A_1 + J, A_1 + K, A_1 + D + F + J. Each mixture was inoculated on to seedlings of Proctor, susceptible to all P. hordei races known to occur in Britain.

Recombinants were sought by screening spores derived from mixed inoculations on appropriate hosts chosen from those listed by Clifford (1974). Orange pustules occurring on Quinn, Reka 1, Egypt 4, Cebada Capa, Estate or Ricardo (resistant to A_1) or brown pustules occurring on hosts resistant to the brown race used, namely Sudan (mixtures A_1 + D, A_1 + J only), Ricardo (mixture A_1 + D only), Cebada Capa or Estate, were isolated. All 366 pustules screened proved to belong to the relevant parent race. In addition, spores from 113 brown pustules and 83 two coloured pustules on susceptible hosts were cultured and tested: again, no recombinant was found.

The failure to detect recombination may have been due to the absence or rarity of this process in P. hordei (some 16,000 seedlings were used in screening experiments). The short incubation period and limited rate of mycelial growth in this species (Falahati-Rastegar, 1978) imply that the chances of mycelia of different infections mixing within the leaf are less than with other cereal rusts. It may be that recombination occurs, but that the particular isolates used are incompatible. Recent work on P. striiformis (Taylor, 1976) suggests that in that species, recombinants involving virulence on Chinese 166 are easier to obtain than others: it may be that the races and hosts used in our work on P. hordei were unsuitable for recombination studies. Some workers have succeeded in producing new races of P. recondita from mixed inoculations on wheat, but others have failed (Bartos, Fleischmann, Samborski and Shipton, 1969: Sharma and Prasada, 1970).

REFERENCES

BARTOS, P., FLEISCHMANN, G., SAMBORSKI, D.J. & SHIPTON, W.A. (1969). Studies on asexual variation in the virulence of oat crown rust, Puccinia coronata f. sp. avenae, and wheat leaf rust Puccinia recondita. Can.J. Bot. 47: 1383-1387

CLIFFORD, B.C. (1974). The choice of barley genotypes to differentiate races of Puccinia hordei Otth. Cereal Rusts Bull. 2: 5-6, 1974.

DAY, P.R. (1974). Genetics of host-parasite interaction. San Francisco: Freeman.

FALAHATI-RASTEGAR, M. (1978). Studies on variation within races, and competition and genetical r combination between races in Puccinia hordei. PhD Thesis, University of Southampton.

GAUMANN, E. (1959). Die Rostpilze Mitteleuropas mit besonderer Berucksichtigung der Schweiz. Bern: Buchler.

SHARMA, Somatic recombination in leaf rust of wheat caused by Puccinia recondita Rob. ex Desm. Phytopath.Z. 67: 240-244.

Cereal Rusts Bulletin Vol. 6, Part 1, 1978

CEREAL RUSTS IN CYPRUS IN 1976/77

BY ATHENA DELLA

Agricultural Research Institute, Nicosia, Cyprus

In Cyprus cereals are grown mainly under rain fed conditions with 200-400 mm annual rainfall and a hot dry climate from March to May from heading to maturity.

There have been no epidemics of cereal rusts in Cyprus in recent years. Important factors contributing to this have been a series of hot dry seasons and the tendency for the modern varieties to mature earlier than older varieties.

The main barley is the local variety Athenais. It is moderately susceptible to cereal rusts but is almost mature by the time infection can be observed on later varieties. Some later varieties were observed to reach 90% severity of leaf rust in 1976 at the Agricultural Research Institute at Athalassa.

Occasional slight infections of leaf and stem rust have been observed in some wheat fields but in irrigated fields at Athalassa a range of severities of leaf rust from 0-90% was observed, with most varieties at about 5%. Some varieties also had stem rust infections up to 30%, but very little yellow rust was observed.

EDITOR'S NOTE

I have been asked to draw the attention of readers to a short paper on urediniospore collectors by P.S. Teng and R.C. Close in the New Zealand Journal of Experimental Agriculture 5, 197-199 (February 1977). The paper describes the construction of two types of spore collectors and compares their efficiency. A membrane filter impaction collector was efficient for spore quantities of 0.05-2.00 mg and a miniature cyclone collector for quantities of 1-100 mg.

Cereal Rusts Bulletin Vol. 6, Part 1, 1978

OBITUARY

DR JOSÉ VALLEGA (1909-1978)

José Vallega was born in Vado Ligure on 29 March, 1909 and died in Rome on 10 April, 1978.

His scientific career started in 1931 at the University of Buenos Aires where he eventually became Assistant Professor of Plant Pathology in the Agricultural and Veterinary Faculty. During the period 1934-1943 he carried out research in the Phytotechnical Institute of Santa Catalina and in the Agricultural Faculty of the University of La Plata, Argentina, where he was Associate Professor of Plant Pathology. He initiated research there on physiological specialization of fungal pathogens, particularly cereal and flax rust fungi; he later became interested in the genetics of resistance to diseases in wheat, oats and flax. In 1939 he was awarded a Fellowship to visit the USA to study plant pathology at the University of Minnesota.

From 1944 to 1960 he successively held the following posts in Argentina: Assistant Director General for Agricultural Research; Director of the Experimental Stations of the Agriculture and Animal Husbandry Ministry; Chief of the Division of Plant Immunology, Introduction and Plant Exploration and Plant Genetics; Director of the National Centre of Agricultural Research; Founder of the National Institute of "Agropecuaria" Technology, and Professor of Plant Pathology. Experimental activity carried out during this time included: the genetics of parasites and of resistance in plants; epidemiology of the main parasites; the genetic improvement of cereals and of fruit and forest trees in Argentina.

Dr Vallega was Director of the FAO Division for Plant Production and Plant Protection in Rome from 1960 to 1969. In 1969 he was appointed Agricultural Minister in the Argentine Embassy in Rome, but he still found time to carry out studies on wheat and animal husbandry. On a voluntary basis, in his spare time, he collaborated with the Italian Institute of Genetics for Cereal Research "N. Strampelli", especially with Dr G. Zitelli, in the study of rusts and other parasites, in breeding high yielding durum wheats with resistance to diseases and lodging.

In 1974 he retired from his post at the Embassy in order to concentrate on his favourite subject, interactions between parasites and host plants. This work was carried out for the first two years with the Plant Pathology Institute of Bari University and for the last few years with the Experimental Institute for Cereal Research in Rome. He also kept in touch with the Genetic Source Unit of FAO in Rome.

Dr Vallega published about 200 technical and scientific papers, attended numerous professional meetings and travelled to more than 60 countries in the course of his work.

Among the many awards he received are the following:

- a Fellowship of the Argentine Association for Advancement of Sciences to enable him to study phytopathology and plant breeding in the United States (1938)
- Fellowship of Minnesota University (USA, 1939)
- Membership of Minnesota Society of Sigma XI (1939)
- Award for Regional Scientific Production of the National Commission of Culture (1942)
- Membership of the Society of National Sciences of Venezuela (1945)
- Rockefeller Foundation Fellowship to encourage contact with European scientists in the field of phytopathology and plant breeding (1953)
- Medal of the Colombian Association of Agronomists (1955)
- Membership of the Brasilian Society of Genetics (1960)
- Awarded the Stakman Medal and Diploma for achievements and human attitudes (1960)
- Membership of the National Academy of Agriculture of Bologna (1963)
- Membership of the Mondial Academy of Sciences and Humanities of USA (1963)
- Medal from Foggia - International Meeting on durum wheat in Italy and in the MEC countries (1969)
- Foreign Membership of the Agricultural Sciences Academy of URSS (1970)
- Golden Ear and Diploma awarded by Tres Arroyos Argentina (1973)

Dr Giuseppina Zitelli,
Instituto Sperimentale per
la Cerealicoltura,
Rome.

25 May 1978