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CONTENTS

EDITORAL

I am now ready to receive contributions for Volume 8, Part 2 of the Cereal Rusts Bulletin which will be published towards the end of 1980. Research papers will be particularly welcomed but the results of surveys can also be accepted.

Please send papers and articles to me as soon as possible at the address below:

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

Gordon E. Russell
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A PRELIMINARY ANALYSIS OF GENES FOR RECEPTIVITY OF WHEAT TO *PUCCINIA RECONDITA*

BY ALBERTO PALYART DO CARMO E FREITAS

Estação Agronómica Nacional, Oeiras, Portugal

A difficulty that may arise in establishing and maintaining cultures of obligate parasites having gene-for-gene relationships with their hosts (Flor, 1955, 1971) is that material may be lost if the host variety carries a gene for low receptivity (low reaction, resistance) corresponding with a gene for low pathogenicity (avirulence) in the pathogen. When unexpected factors for low receptivity are detected in a variety used for establishment of pathogen cultures it will be necessary to select another variety without such genes or, in other words, possessing corresponding alleles for high receptivity (high reaction, susceptibility).

A variety with universally high receptivity is desirable for use in surveys but we can never be sure of high receptivity at all loci. This is also true for varieties used as recurrent parents in the production of isogenic lines with and without genes for low receptivity. It is therefore important to know as much as possible about genes for receptivity in such varieties.

Some well-known wheat varieties carrying different genes for high receptivity to *Puccinia recondita* were inoculated at the seedling stage in the greenhouse with Portuguese cultures of this pathogen (Table 1). The cultures referred to in this table were also tested on several wheat selections and backcrossed lines to identify their genes for high pathogenicity (Freitas, 1973; Freitas & Freitas, 1972, 1978). From these results the most profitable backcross lines and varieties for identifying different genes or groups of genes for receptivity were selected (Table 2). With these lines it was possible to identify, in the pathogen cultures, the corresponding genes for pathogenicity (Table 3).

If a culture carries an identified gene either for low or for high pathogenicity and gives a high infection type in association with a variety, it can be argued that this variety possesses a corresponding

Table 1. Infection types on wheat varieties

Wheat variety	*Puccinia recondita* culture															
	1A	1B	11A	11B	11D	15	20	57	58A	58B	58C	68	84	87	107	150
Little Club	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Morocco	4	4	4	4	4	4	4	4	0	4	4	4	4	4	4	4
Mocho de Espiga Branca	4	4	4	4	4−	4	2	4	4	4	2	4	4	4	4	4
Michigan Amber	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Novosadska	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Restauração	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Thatcher	0f1	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Prelude	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Red Bobs	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4

Table 2. Wheat selections carrying genes for low receptivity

Wheat selection	Gene	Wheat selection	Gene
Tremês Rijo 854	Prx:T	Aiv/6*Tc RL 6035	Pr3ka:T
Xerês 1165	Pry:T	Ech/6*Tc RL 6004	Pr10:T
Ardito 61534	Prz:T	ME/6*Tc RL 6006	Pr14b:T
Ctr/6*Tc RL 6033	Pr1:T	Ech/6*Tc RL 6037	Pr16:T
Wst/6*Tc RL 6016	Pr2a:T	RLco/6*Tc RL 6038	Pr17:T
Ls/6*Tc RL 6025	Pr2c:T	Af 43/7*Tc RL 6009	Pr18:T
Do/6*Tc RL 6002	Pr3:T		

x, y, z are provisional symbols

Table 3. Pathogenicity genes in *P. recondita* cultures

Culture	Pathogenicity gene												
	Tx:Pr	Ty:Pr	Tz:Pr	T1:Pr	T2a:Pr	T2c:Pr	T3:Pr	T3ka:Pr	T10:Pr	T14b:Pr	T16:Pr	T17:Pr	T18:Pr
1A	+	+	–	–	–	–	–	–	–	–	–	–	–
1B	–	–	–	–	–	–	–	–	+	+	–	–	+
11D	–	+	+	–	–	+	–	–	–	+	–	–	+
20	–	–	–	+	+	+	–	–	+	+	–	+	+
58A	–	+	+	–	–	+	+	–	+	+	–	–	+
58C	–	–	+	–	–	+	+	–	+	+	–	–	+

\+ : high pathogenicity
– : low pathogenicity

Table 4. Some receptivity genes carried by three varieties which produced low infection type with 13 cultures of *P. recondita*.

Variety	Receptivity gene												
	Prx	Pry	Prz	Pr1	Pr2a	Pr2c	Pr3	Pr3ka	Pr10	Pr14b	Pr16	Pr17	Pr18
Thatcher	+	+	+	+	+	+	+	+	+	?	+	+	?
Morocco	+	+	+	+	+	+	+	+	+	+	+	+	+
Mocho de Espiga Branca	+	+	+	+	+	+	+	+	+	+	+	+	+

\+ : high receptivity

gene for high receptivity. If a culture carries high pathogenicity genes corresponding to recognised genes for low receptivity but gives a low infection type towards a variety, it can be argued that the observed low infection type on the varieties is not due to those recognised genes for low receptivity. Furthermore, if other cultures have genes for low pathogenicity corresponding to those recognised genes but give high infection type towards the variety, then the variety carries high receptivity alleles at the loci of those corresponding genes.

For example, the variety Thatcher gives an infection type 'Ofl' with the culture 1A and an infection type "4" with all others (Table 1). So Thatcher carries a gene or genes for low receptivity. However, several cultures, which produce high infection types on Thatcher, possess genes for low pathogenicity corresponding to all recognised genes for low receptivity tested (Table 3). It can therefore be assumed that Thatcher carries all the corresponding alleles for high receptivity. In addition, the culture 1A carries high pathogenicity genes corresponding to low receptivity genes Prx and Pry whereas other cultures, which produce a high infection type on Thatcher, carry genes for low pathogenicity corresponding to those genes. From this it can be deduced that Thatcher carries high receptivity alleles at the Prx and Pry loci also. Culture 1A, however, carries genes for low pathogenicity corresponding to the recognised genes for low receptivity, Pr14b and Pr18, and produces a low infection type on Thatcher. All cultures which produce a high infection type on Thatcher carry high pathogenicity genes corresponding to Pr14b and Pr18. The possibility cannot therefore be excluded that the low infection type on Thatcher produced with culture 1A is due to any of these genes for low receptivity (Table 4).

Using similar lines of argument it is possible to deduce that the observed low infection type on Morocco and Mocho de Espiga Branca was not due to any of the recognised genes in Table 3 and that they carry corresponding alleles for high receptivity to all the tested genes (Table 4).

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PHYSIOLOGIC SPECIALIZATION OF STEM RUST AND LEAF RUST OF WHEAT IN CZECHOSLOVAKIA IN THE LAST 17 YEARS

BY P. BARTOŠ

Research Institute for Crop Production
Praha-Ruzyně, Czechoslovakia

In Czechoslovakia race surveys of stem and leaf rust were initiated in 1962. The results of the surveys have already been published in detail (Šebesta & Bartoš, 1967, 1968, 1969a, 1969b; Bartoš, 1972, 1975; Bartoš & Šebesta, 1971; Bartoš, Hladká & Vyvadilová, 1978; Bartoš & Hladká, 1978). This paper describes the developmental trend in the rust population in relation to the cultivars that have been grown.

STEM RUST (Puccinia graminis f.sp. tritici)

At the beginning of the surveys, races 21 and 14 predominated and this situation prevailed for almost a decade. At first no cultivars with specific resistance to stem rust were grown, but resistant cultivars from the USSR were introduced later. In this decade the proportion of isolates virulent on varieties carrying resistance gene Sr 5 increased, probably because the cultivar Bezostaya 1 and derived cultivars possessing Sr 5 was grown in the regions from which stem rust is supposed to invade Middle Europe. In 1972 races virulent on Sr 5 were more common in Czechoslovakia than races 14 and 21. Races 34 and 11 became the most important ones and formed almost 90% of the stem rust population in 1977. Therefore Sr 5, which is common in many East European cultivars, is of little value in Czechoslovakia now. However, some East European cultivars possess gene(s) in addition to Sr 5 which are partially effective against races 34 and 11. No race virulent to the resistance gene occurring in 1R/1B substitutions or translocations, which are common sources of stem rust resistance in breeding programmes, have been found in Czechoslovakia. Of the races determined in Czechoslovakia race 15 is the most virulent but its incidence is sporadic.

LEAF RUST (*Puccinia recondita*)

At the beginning of the rust surveys race UN 10-14 predominated and no cultivars with specific resistance to leaf rust were grown. In the year 1966 the cultivar Mironovskaya 808, which carries the resistance gene *Lr 3* and is therefore resistant to race UN 10-14, was registered in Czechoslovakia. The proportion of isolates of race UN 13-77, which up to that time was of secondary importance, became more prevalent. In 1968 race UN 13-77 was more common than race UN 10-14 and has remained so since that time. The cultivar Salzmünder Bartweizen (1R/1B substitution) was tested in Czechoslovakia in 1968 and a biotype of race UN 10-14 which was virulent on this cultivar, designated as UN 10-14 SaBa, was discovered. In 1972 cultivars Kavkaz and Aurora (also 1R/1B translocations) were introduced to Czechoslovakia. A biotype of race UN 13-77, designated UN 13-77 SaBa, virulent on these cultivars had been found in 1970 and this biotype has predominated in the leaf rust population since 1972.

Two cultivars, Sava and Zlatna Dolina, from Yugoslavia have been grown on a limited scale in Czechoslovakia since 1975. Initially they were resistant to leaf rust, but in 1977 virulent isolates of leaf rust were discovered on them. Most of the field isolates from the 1978 race survey were virulent on these cultivars.

CONCLUSIONS

A comparison of the stem and leaf rust variability in Czechoslovakia indicates that changes in the leaf rust population have been affected to a greater extent by the cultivars that were grown than those in the stem rust population. This is probably due to the fact that leaf rust overwinters in Czechoslovakia, stem rust epidemics usually being caused by airborne inoculum from abroad. The race composition of *P. graminis tritici* therefore seems not to have been greatly affected by the cultivars grown in Czechoslovakia.

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GENETICS OF FIELD RESISTANCE OF THE WINTER WHEATS BENNO, ZORBA, ORLANDO AND SALADIN TO RACE 232 E137 ("CLEMENT" RACE) OF *PUCCINIA STRIIFORMIS*

BY V. SLOVENČÍKOVÁ

Research Institute of Crop Production,
Praha-Ruzyně, Czechoslovakia

INTRODUCTION

Translocations or substitutions between wheat chromosome 1B and rye chromosome 1R are important sources of yellow rust resistance in wheat. However, the race of *Puccinia striiformis* identified as 232 E137 (the so-called "Clement" race) caused the breakdown of resistance in some cultivars derived from these sources. However, some of these cultivars that became susceptible as seedlings were still resistant as adult plants to this race and this was why we investigated the genetics of their field resistance to yellow rust.

MATERIALS AND METHODS

Tests of seedlings were carried out from February to the end of May in a partly air-conditioned greenhouse. The temperature and light during this period favoured the development of yellow rust.

Infection types on adult plants were evaluated in field nurseries at the end of flowering and in the milky ripe stage, when the infection level of yellow rust was at its highest. In some cases F_2 plants were evaluated both in 1977 and 1978 to eliminate the effects of environment. In 1977 the scale I-IV was used and this was converted to the scale of McNeal *et al* (1971) as follows: 0-3 = resistant; 4-6 = intermediately resistant; 7-9 = susceptible. This latter scale was used in our tests from 1978 onwards. The plants were inoculated with race 232 E137 ("Clement") which can overcome the "rye resistance"; uredospores of this race were obtained from Dr. Stubbs, IPO, Wageningen.

RESULTS

The results of the experiments are summarized in Tables 1, 2, 3.

Cv. Benno was susceptible at the seedling stage while a 1-2 infection type was observed on adult plants. The cross between cv. Salzmünder Bartweizen (susceptible) x cv. Benno suggests that one dominant and some recessive genes are active in adult plants. Similar conclusions were reached from the cross between cv. Clement (susceptible) x cv. Benno.

Cv. Orlando was susceptible at the seedling stage and resistant at the adult stage in field tests. Genes for field resistance were found to be identical in cv. Orlando and cv. Zorba and no segregation of susceptible plants occurred (Table 3).

Cv. Saladin was resistant at both the seedling and adult stages. Field resistance was found in progenies of the cross between cv. Winnetou and cv. Saladin. From the cross with susceptible Salzmünder Bartweizen, 2 or 3 dominant genes and some recessive genes were postulated at the adult stage; the F_3 progenies indicated the presence of more dominant genes, the effects of which can sometimes be reduced, resulting in reduced levels of resistance (infection types 2-4).

Cv. Winnetou was susceptible as seedlings but adult plants were resistant. There was no segregation of susceptible adult plants in the crosses Winnetou x Saladin and Zorba x Winnetou, indicating that these varieties may possess identical resistance genes.

Cv. Zorba was susceptible as seedlings and resistant as adult plants. Cv. Zorba may possess adult-plant resistance genes which are identical with those in cvs. Orlando and Winnetou.

DISCUSSION

Earlier work showed that at least one identical gene for resistance was found in the crosses Salzmünder Bartweizen x Orlando, Orlando x Zorba (Stubbs et al.,1977), Zorba x Winnetou (Slovenčíková,1973) when the race 40E 8, avirulent to the "rye resistance", was used. However, these varieties reacted differently in tests with the Clement race 232 E137 which overcomes the "rye resistance". For example, Salzmünder Bartweizen was susceptible at both seedling and adult stages, whereas Zorba and Winnetou were susceptible as seedlings in greenhouse tests but resistant as adults in field nurseries. Saladin was resistant in both greenhouse and field tests.

Table 1. Reaction of parental varieties to the "Clement" race of *Puccinia striiformis* in the greenhouse and in field nurseries

Variety	Infection type in greenhouse (seedlings)	Infection type in field nurseries (adult plants)
Benno	8	1-2
Clement	8	8
Orlando	8	1-3
Saladin	0	0
Salzmünder Bartweizen	8	7-8
Winnetou	8	0-1
Zorba	8	1

Table 2. Evaluation of some F_3 progenies from five crosses

Cross	Expected resistance genes	F_3 progenies			Total	P-value
		resistant	segregating	susceptible		
Salzmünder Bartweizen x Benno	1 dominant 2 recessive	10	16	0	26	0.80-0.90
Salzmünder Bartweizen x Saladin	3 dominant 1 recessive	24	2	0	26	0.70-0.50
Winnetou x Saladin	at least 1 gene in common	30 (type 0-1)			30	
Orlando x Zorba	at least 1 gene in common	30*			30	
Zorba x Winnetou	at least 1 gene in common	30 (immune reaction in all progenies)			30	

* In 6 progenies there were several plants of infection type 3; in 2 progenies several plants were of type 6. In the next year only some of these plants segregated in the F_4 with infection types 0-4.

Table 3. Adult-plant reactions of F_1 and F_2 progenies from seven crosses to the "Clement" race of *Puccinia striiformis* in field nurseries in 1977 (A) and 1978 (B)

Cross	F_1		F_2 Number of plants in infection types: resistant 0	1	2	3	4	5	6	susceptible 7	8	9	Total	Expected resistance genes	P-value
Salzmünder Bartweizen x Benno	1	A	90	111						3	27		231	1 dominant + 2 recessive	0.30-0.50
		B		65	29	77	78	47	19	24	20	2	361	1 dominant + 2 recessive	0.10-0.30
Clement x Benno	1	A	129				30				19		178	1 dominant + 2 recessive	0.90-0.95
		B	37	25	43		90	117	74	32	18		436	1 dominant + 3 recessive	0.80-0.90
Salzmünder Bartweizen x Saladin	0	A	112							4			116)	2 dominant +	0.95
		A	193			1	6	2	2	4			208)	2 recessive	0.30-0.10
		B	168		1	5	6		2	2			184	3 dominant + 2 recessive	0.90-0.95
Orlando x S. Bartweizen	0-1	B	76	3	13		15	1	2	2	27	3	142	1 dominant + 2 recessive	0.30-0.10
Winnetou x Saladin	0-1	A	232										232	at least 1 gene in common	
Orlando x Zorba	1	A	300			8			2				310	at least 1 gene in common	
Zorba x Winnetou	0	A	313										313	at least 1 gene in common	

Although P-values agreed with the expected ratio for the postulated genes in studies of the field resistance, more extensive genetic analyses will be necessary to confirm these results.

The presumption that major genes govern this adult-plant resistance is supported by the identity of at least one gene for resistance in the crosses Winnetou x Saladin, Orlando x Zorba, Zorba x Winnetou. The distribution of F_2 plants into infection types (Table 2) and a number of homozygous F_3 families (Table 3) indicate also the oligogenic character of the field resistance.

The expression of genes for resistance was sometimes modified. For example, in F_3 progenies of the crosses Salzmünder Bartweizen x Saladin, Salzmünder Bartweizen x Benno, Orlando x Zorba, derived from F_2 plants which had been classified as homozygous resistant with infection type 0, susceptibility ranged from 0-4. Some plants with infection type 0 as seedlings became less resistant in the later growth stages. In contrast, progenies of plants that were classified as homozygous susceptible (type 9), showed variability ranging from 7-9.

Further genetic analyses will be carried out to confirm the data presented in this paper and to find how the genetic background can influence the final infection type of plants.

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WHEAT RUST EPIDEMICS IN INDIA SINCE 1970

BY L.M. JOSHI, K.D. SRIVASTAVA, D.V. SINGH AND K. RAMANUJAM

Division of Mycology and Plant Pathology,
Indian Agricultural Research Institute, New Delhi-110012, India

Epidemics of wheat rusts have been recorded in India for a long time and it appears that the earliest severe epidemic in Central India occurred in 1783 (Sleeman, 1839). A chronological account of rust epidemics in India has been given by Nagarajan and Joshi (1975).

Since 1968, a wheat disease survey and surveillance programme has been in operation in India (Joshi, 1975) and as a result of this, the rust position has been very well documented during the last 13 years. During this period there have been two major epidemics of leaf rust (Puccinia recondita) and stripe rust (Puccinia striiformis) in the North-Western region and three localised epidemics of stem rust (Puccinia graminis) and leaf rust in Central and Peninsular India. A brief account of these epidemics is given below.

Epidemics of leaf and stripe rusts in 1971-72 and 1972-73 in North-Western India

In 1971-72 and 1972-73, leaf rust and stripe rust appeared in epidemic proportions in Punjab, Haryana and western Uttar Pradesh. In 1972-73 the leaf rust epidemic covered the whole of Uttar Pradesh, but stripe rust appeared very late on the then predominant variety, Kalyansona, and therefore did not do much damage. Stem rust was insignificant in both years and appeared only at the end of the season. The chief difference between the two successive epidemics was that, in the former, the rust epidemic suddenly flared up after 15th March due to a rise in temperature and to the presence of late-sown crops. However, in the second season, the epidemic started in February but developed only gradually.

Because of the seriousness of these epidemics, an attempt was made to calculate the losses which had occurred. Survey teams were sent out in epidemic areas at harvest time to collect samples of grain. A total number of 107 and 122 samples of different cultivars was collected from the threshing floors during 1971-72 and 1972-73 respectively for analysis of losses caused by rusts. The losses were calculated on the basis of 1000-grain weight in affected crops compared with the known 1000-grain weight of the same variety in the absence of disease. The losses were then used to calculate the yield loss of each variety in a particular State taking into consideration the area under cultivation and production (Table 1).

The total wheat production in 1971-72 from the areas which were affected by rust epidemics was probably about 11.5 million tons (tonnes) (Punjab 5.8 million tons, Northern Haryana 2.0 million tons and Western Uttar Pradesh approximately 3.5 million tons). Similarly, in 1972-73 production was approximately 13.0 million tons (Punjab 5.8 million tons, Haryana 2.0 million tons and Uttar Pradesh 6.0 million tons). Table 1 shows that during 1971-72 the average loss in 1000-grain weight due to rusts was 5.9 per cent in cv. Kalyansona, 2.0 per cent in cv. Sonalika, 24.1 per cent in cv. K 68 and 9.9 per cent in cv. Pb.C.306. Consequently, calculated losses in yield amounted to 0.22 million tons in Uttar Pradesh, 0.17 million tons in Haryana and 0.32 million tons in Punjab. The total loss in the 1971-72 crop was 0.71 million tons. In 1972-73, percentage losses in grain weight were 9.9, 0.82, 6.4 and 8.4 in cvs Kalyansona, Sonalika, Pb.C.306 and K 68 respectively and the total calculated loss in yield was 1.37 million tons (Joshi *et al.*, 1975).

Epidemics of stem and leaf rusts in Western India in 1973 and 1975

An isolated but severe epidemic of stem and leaf rusts was recorded on wheat in the Nad area of Sanchore in the Jalore district (Rajasthan) during November 1973 over an area of 20 000 hectares (Joshi *et al.*, 1974). In some fields even the soil was covered with uredospores after the farmers had removed the crop for use as cattle food, since no grain yield was expected from such heavily rusted crops. In this region a local cultivar, cv. Kharachia, was badly affected whereas some improved varieties like cvs. Kalyansona and S 227 had only traces of rust infection. It is estimated that nearly one-third of the entire crop (approximately 8000 hectares) was

Table 1. Estimated losses due to rust epidemics in 1971-72 and 1972-73 based on 1000-grain weights

Wheat Variety	No. of samples analysed	Average 1000-grain weight (g)			Approximate grain yields (million tons)			Estimated losses (million tons)			Total estimated losses
		Affected samples	Healthy samples	Calculated loss (%)	Uttar Pradesh	Haryana	Punjab	Uttar Pradesh	Haryana	Punjab	
1971-72											
Kalyansona	57	30.10	32.00	5.90	3.15	1.2	5.5	0.19	0.07	0.32	
Sonalika	33	45.08	46.00	2.00	0.28	0.2	0.05	0.01	0.04	0.001	
K 68	9	34.14	45.00	24.10	0.07	-	-	0.02	-	-	
Pb.C. 306	8	35.13	39.00	9.90	-	0.6	-	-	0.06	-	
Other varieties	-	-	-	-	-	-	0.25	-	-	-	
TOTAL	107	-	-	-	3.5	2.0	5.8	0.22	0.17	0.32	0.71
1972-73											
Kalyansona	54	28.82	32.00	9.90	0.9	1.6	5.5	0.09	0.16	0.54	
Sonalika	32	45.62	46.00	0.82	2.1	0.2	0.17	0.02	0.001	0.001	
K 68	4	41.20	45.00	8.40	1.2	-	-	0.10	-	-	
Pb.C. 306	13	36.50	39.00	6.40	1.8	0.2	-	-	0.01	-	
Local wheats	19	-	-	25.0	-	-	0.13	0.45	-	-	
TOTAL	122	-	-	-	6.0	2.0	5.8	0.66	0.17	0.54	1.37

completely destroyed and not even harvested. An epidemic occurred again in this region in 1975, but damage was much less in that season as most of the farmers had by then changed to cv. Kalyansona, which is resistant to stem rust. The chief reasons for early appearance of disease involve certain agronomic practices followed by farmers, including the early sowings of highly susceptible cultivars, the early spread of rust spores from the infection foci and the presence of temperatures and humidity that favour disease development.

Epidemics of stem and leaf rusts in Central India in 1978-79

In 1978-79 a large area of the Narmada valley of Madhya Pradesh, where two local wheat varieties Pissi and Malvi Local predominated, was once again hit badly by leaf and stem rust epidemics. A large number of grain samples was collected and these showed that the 1000-grain weight of infected crops was 6.4 - 10.5 g, whereas samples from rust-free crops weighed 35 - 40 g. Results of field tests suggested that rusts caused losses of between 60-75 per cent. However, improved varieties in the same area yielded well and there was little disease on them. This epidemic was caused by the early arrival of rust spores from South India (Joshi *et al.*, 1979) and this was followed by an extremely favourable temperature and abundant moisture.

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