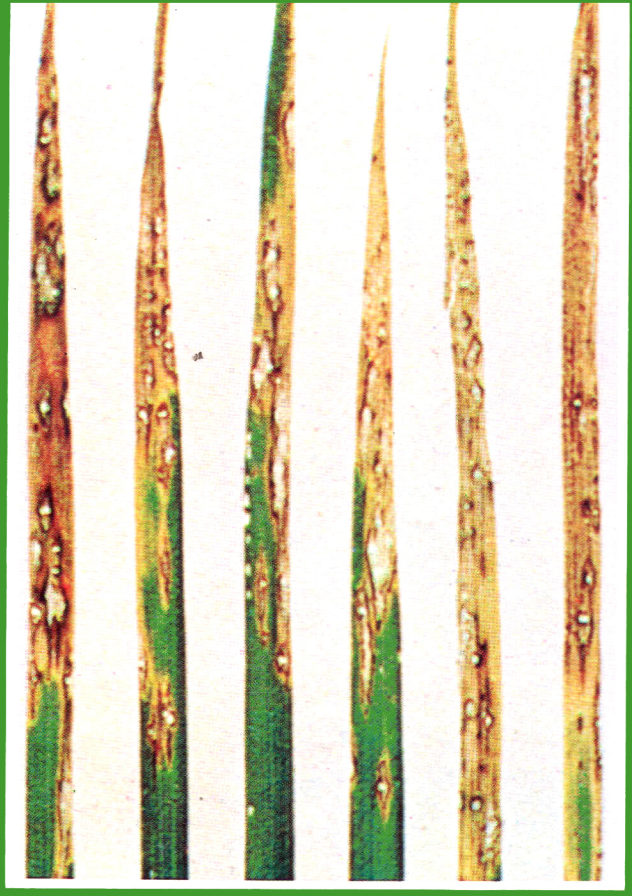

PROCEEDINGS OF THE RICE BLAST WORKSHOP

INTERNATIONAL RICE RESEARCH INSTITUTE

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Contents

Foreword

DISEASES AND INSECT PESTS seriously constrain rice yields, particularly in the tropics. Among the most widespread of the diseases is rice blast, caused by the fungus *Pyricularia oryzae,* which is found in most commercial rice-growing areas of the world. This disease is common in areas using wetland and bunded rice culture, but it is more serious where dryland or upbunded upland rice is grown. Such areas characterize most of the rice lands of South America and Africa and are found in many rice-growing regions of Asia.

Because of the "instability" of the rice blast fungus and the marked variability in pathogenicity, which results in different strains of this organism, its control and management are difficult. Isolates have been shown to rapidly lose their pathogenicity or to become more virulent, depending upon a variety of conditions and circumstances. The use of fungicides has been limited largely to the more affluent countries and the development of host resistance has met with only mixed success.

There is incomplete agreement among plant pathologists and plant breeders from different rice-growing countries as to the host-blast pathogen relationships and, consequently, as to the appropriate research methods to be used in developing blast-resistant varieties. Some have argued for approaches aimed at developing "horizontal" resistance; others maintain that varieties with strong "vertical" resistance should be developed.

The primary purposes of this symposium were to summarize what was known about rice blast and its control, and to develop research strategies to improve that control. Participants in the symposium included rice scientists with markedly different opinions as to the research strategies to be used. They were joined by plant breeders and plant pathologists working on fungus diseases of other crops. The interaction among these scientists was most useful in developing the recommendations for future research on blast.

IRRI is indebted to the participants of this conference for their recommendations for future research strategies. Special recognition should be given to the work of the Organizing Committee chaired by Dr. S. H. Ou, head of IRRI's Plant Pathology Department, and for the technical editing which this group performed. Thanks are also given to the professional editors Dr. J. C. Torio and Ms. Corazon V. Mendoza.

OPENING REMARKS

N. C. BRADY

I AM PLEASED TO WELCOME you this morning to the RICE BLAST WORKSHOP, to the International Rice Research Institute (IRRI). and to Los Baños. I wish to express IRRI's appreciation to you, first, for taking time to prepare the written papers that you made available to Dr. S. H. Ou and to the organizers of the workshop, and second, for your attendance at the workshop and your willingness to participate as speakers and discussants. This workshop focuses on a long-standing phytopathological problem of international economic significance.

Workshops and conferences held at IRRI must be on a topic of international significance and have specific objectives not only for IRRI, but for all participants, many of whom come long distances to attend. Scientists and researchers are invited to such meetings because of their expertise and experience and not because of organizational affiliation or nationality.

The two primary purposes of workshops and conferences sponsored and organized by IRRI are:

- the exchange of information among scientists from different locations on the subject of the conference, in this case, the rice blast disease,
- the development of future research strategies which may accelerate the acquisition of new knowledge. Throughout the IRRI system of conferences, this has become increasingly important,

The International Rice Research Institute has broad responsibilities and its scientists are somewhat isolated (even though they travel considerably) because they do not have the opportunity to come into contact with as many scientists as they would like, to help them identify priorities and to develop strategies for research. These strategies relate both to IRRI scientists and to scientists in national programs who cooperate and collaborate with the Institute. I believe that not only in the area of rice blast, but also in other areas for which IRRI has responsibilities, significant progress has been achieved as a result of collaborative efforts among scientists from different disciplines.

We at this workshop, share the mutual concern about the continuing economic significance of rice blast disease throughout the rice-growing world and know that it is of greater importance in some areas and under certain conditions than others. While progress has been made and is being made to manage and control this disease, satisfactory control is a goal yet to be achieved. Some of the methods that have been tried and others that are under development, i.e., breeding varieties that are resistant in various degrees to the rice blast, have given less than the desired control or have failed. As we discuss these disappointing results, we should endeavor to suggest strategies that might increase our successes in the future and reduce the failures.

You know better than I of the success, and lack of success, of different research techniques to better understand the causal organism, the genetics of host resistance, and the host-pathogen relations and interactions. We are all concerned with the need to improve the present procedures and to develop innovative approaches so significant progress can be made to better manage and control rice blast.

This workshop offers an opportunity and a challenge to critically evaluate what has been done in the past and to develop future research strategies. At this 3-day workshop, scientists from different disciplines (plant breeding, plant pathology, entomology, and others) and researchers from different bioclimatic zones of the rice-growing world (zones that differ in the strains of the pathogen and in the varieties of the rice plants) can bring to the discussions their collective experience in using different techniques to study rice blast and to seek methods of its control. Additionally, the scientists who are involved in research on the control and management of diseases of crops other than rice (i.e., wheat, corn, and potatoes) may suggest research techniques that might be applicable to research for the control and management of rice blast.

I wish you success in this workshop and once again thank you for your attendance and participation. We look forward to this opportunity to exchange information with each other and to develop notions and strategies for future efforts and research to manage and control this critical cereal crop disease—rice blast.

THE NATURE OF THE BLAST FUNGUS AND VARIETAL RESISTANCE IN JAPAN

TAKUJI KOZAKA

WITHIN THE LIMITS of our experience with Japanese isolates of the blast fungus *Pyricularia oryzae* Cav., pathogenic variability of cultures seems to be quite different with different isolates. Some isolates are fairly stable: others are highly variable.

PATHOGENIC VARIABILITY OF RICE BLAST FUNGUS CULTURES

By the term "stable," I mean that an original reaction to the rice differentials has remained constant throughout successive transferring, not that all spores have uniform virulence. For instance, at the National Institute of Agricultural Sciences (NIAS) in Tokyo in 1968, check tests were made for virulence to the differentials of cultures that had been preserved for years by transferring the cultures to potato sucrose agar in slants every 6 months. The tests showed that of 199 isolates, 69 had maintained their original virulence, 60 had lost it, 20 had increased it, and 50 had decreased it (Table 1). Some of the spontaneous changes in pathogenicity occurred soon after isolation; some appeared suddenly many years later. Segregation of virulence often was observed among monoconidial subcultures (Table 2). In extreme cases, repeated monoconidial reisolations resulted in continued change into several different races in each generation (Goto et al 1961, 1964; Goto and Yamanaka 1968). During successive transfers, sectors differing in mycelial type and in color sometimes occurred, and occasionally they lacked the original virulence (Goto et al 1961, 1964).

In the early stages of *P. oryzae* race study in Japan, we could not hope to maintain cultures of the identified races because of their considerable pathogenic variability. With advancing knowledge, however, we have been able to select many representative isolates that are fairly stable and have clear-cut reactions to the differentials. For example, ever since they were isolated in 1953 and 1955, the isolates Naga 87 and Ina 72 have maintained their original virulence to cultivars with resistance genes *Pi-k* and *Pi-m* although no particular precautions

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Table 1. Change in virulence of the preserved cultures of *Pyricularia oryzae* transferred every 6 months on potato sucrose agar. NIAS. 1968.

Yearspreserved (no.)	Isolates (no.) showing				Isolates (total no.)
	Original virulence	Increased virulence	Reduced virulence	Lost virulence	
15	3	0	0	0	3
14	1	0	1	0	2
13	0	0	2	0	2
12	1	0	0	0	1
11	2	0	2	0	4
10	1	0	0	0	1
9	3	1	3	1	8
8	3	0	5	2	10
7	1	1	2	4	8
6	4	0	1	14	19
5	6	2	7	8	23
4	17	9	20	18	64
3	6	1	1	3	11
2	15	3	2	9	29
1	6	3	4	1	14
Total	69	20	50	60	199

had been taken to preserve it. Most of the monoconidial subcultures of the two isolates show uniform virulence.

Researchers today have no great problem in storing their own cultures for race studies. By 1970 they had essentially finished classifying almost all cultivars then being used in Japan, by determining cultivar reactions to the representative Japanese races, or by screening horizontally resistant cultivars in each reaction type (Iwata 1973). Extensive genetic analyses have also been made, and more than 10 race-specific major resistance genes, all dominant. have been identified.

Table 2. Segregation of virulence in each 10 monoconidial subcultures originating from newly isolated cultures of *Pyricularia oryrae* NIAS, 1962.

Name of original Isolate that showed	
Uniform virulence	Segregated virulence
Ken 62-04	Ken 62-01
" 62-05	" 62-02
" 62-26	" 62-03
" 62-28	" 62-07
" 62-35	" 62-30
" 62-39	" 62-49
" 62-43	" 62-51
" 62-46	" 62-52
" 62-60	
" 62-78	
" 62-80	
" 62-83	

Table 3. Reaction of some isolates on the new differential varieties (Yamada et al 1976).

Differential variety	Resistance gene[a]	Code no.	Reaction[b] of Isolate[c]							
			Ken 53-33 (137)	Hiro 63-20 (303)	Chu 65-673 (103)	Ken 64-52 (107)	Ken 60-19 (037)	Oita 65-114 (017)	Naga 87 (131)	Ina 72 (031)
Shin 2	*Pi-k*^{s}	1	S	S	S	S	S	S	S	S
Aichi-Asahi	*Pi-a*	2	S	S	S	S	S	S	R	R
Ishikari-Shiroke	*Pi-i*	4	S	R	R	S	S	S	R	R
Kanto 51	*Pi-k*	10	S	R	R	R	S	S	S	S
Tsuyuake	*Pi-m*	20	S	R	R	R	S	R	S	S
Fukunishiki	*Pi-z*	40	R	R	R	R	R	R	R	R
Yashiro Mochi	*Pi-ta*	100	S	S	S	S	R	R	S	R
Pi No. 4	*Pi-ta*^{2}	200	R	S	R	R	R	R	R	R
Toride 1	*Pi-zt*	400	R	R	R	R	R	R	R	R

Differential variety	Resistance gene[a]	Code no.	Reaction[b] of Isolate[c]								
			Ken 61-14 (017)	Naga 64-8 (033)	Naga 65-386 (035)	Hoku 1 (007)	Ken 64-38 (003)	Ken 54-04 (003)	Ina 168 (101)	Naga 66-16 (001)	Naga 61-14 (005)
Shin 2	*Pi-k*^{s}	1	S	S	S	S	S	S	S	S	S
Aichi-Asahi	*Pi-a*	2	S	S	R	S	S	S	R	R	R
Ishikari-Shiroke	*Pi-i*	4	S	R	S	S	R	R	R	R	S
Kanto 51	*Pi-k*	10	S	S	S	R	R	R	R	R	R
Tsuyuake	*Pi-m*	20	R	S	S	R	R	R	R	R	R
Fukunishiki	*Pi-z*	40	R	R	R	R	R	R	R	R	R
Yashiro Mochi	*Pi-ta*	100	R	R	R	R	R	R	S	R	R
Pi No. 4	*Pi-ta*^{2}	200	R	R	R	R	R	R	R	R	R
Toride 1	*Pi-zt*	400	R	R	R	R	R	R	R	R	R

[a] *Pi-k*^{s} is not active against Japanese races. [b] S = susceptible; R = resistant. [c] Numbers in parentheses are race numbers.

The accumulated knowledge about race-cultivar relationships has allowed workers to propose a new set of differentials with single, identified resistance genes that are found either in commercial cultivars or in cultivars used for rice breeding (Yamada et al 1976; Table 3).

The facts I have mentioned indicate that not all isolates are extremely variable, at least in Japan, and that, as in many other diseases, races can be studied with selected cultures.

From 1962 to 1965, Japanese workers used many isolates collected from many countries other than Japan. The reactions of the foreign isolates to the Japanese differentials were less consistent than those of the Japanese isolates. Sectors also occurred more frequently. However, when representative isolates of races from the USA and from Japan were selected by US and Japanese workers and were exchanged, 96.5% of the reactions obtained in 1963 were in agreement, as were 83.9% of those obtained in 1964 (Goto et al 1967).

Those results suggest that foreign isolates are likely to be more variable than native isolates but selected isolates are by no means extremely variable.

I believe that it is highly possible to obtain stable isolates by selection in any country.

The frequency of pathogenic variability was found to differ with different cultural media. Potato sucrose agar seems to promote variability (Matsuyama and Kozaka 1970). At NIAS the type cultures are preserved in slants with liquid paraffin layer or with gum rubber cap.

There are few reports of pathogenic variation of the fungus on the rice plants when contamination is carefully avoided. Kato and Sasaki (1974) showed that from 82 inoculations on the glumes of four cultivars, with 24 isolates representing 11 races, only 2 reisolates of 62 inoculations with succeeding reisolations differed in virulence from the original races (Table 4).

In an area where a single cultivar is grown on a large scale and a single race of the blast fungus is highly predominant, the race is considered to have minimum contamination by other races. In such a field, 50 monoconidial isolates from each of 2 lesions (Nakamura 1972) and 20 to 37 isolates from each of 4 lesions (Fujikawa et al 1972) showed uniform virulence. In fields contaminated by several races, the races were originally isolated from a single lesion, but

Table 4. Results of reisolation from infected spikelets that were inoculated with avirulent races (Kato and Sasaki 1974).

Race	Isolate inoculated	Reisolation[a] from cultivar			
		Te-tep	Tadukan	Usen	Kanto 51
N-1	Hoku 373	S	S	S	S
	Ken 62-03		S		
	TH 65-105-5		S	S	S
N-2	Ken 59-49	S	S	S	
	Ken 62-42	S	S	S	S
	Chu 66-45-3-1	S	S		S
N-3	Naga 61-34	S	S		(S)*
N-4	Ine 168				S
	Ken 60-11		S	S	(S)
N-5	Hoku 540	S	S	S	S
	Naga 61-14	S	S	S	S
C-1	Ken 60-19	S	S	S	—
	TH 63-454	S	S	S	—
	Chu 66-64		S		—
C-3	Naga 87	S	S	S	—
	Ken 66-13		S		—
C-5	Ken 61-14		S	S	—
	Ou 65-114	S	S	S	—
C-6	Ken 64-109		S	S	—
	TH 67-27		S	S	—
C-8	Hiro 63-104		S		—
	Ken 66-14	S	S		—
C-9	Ken 66-111	S	S	S	—
	Ken 64-144	S	S	S	—

[a]S = reisolation successful; (S) = reisolates differed in virulence from original race; blank = reisolation unsuccessful; — = no inoculation.

Table 5. Races in a leaf lesion (Nakamura et al 1972; Fujikawa et al 1972).

District	Cultivar	Lesions (no.)	Isolations (no.)	Isolates (no.) identified as race			
				C-8	N-2	N-3	Others
Usa, Oita	Kuju	1	20	20			
		2	20	20			
	Reiho	1	6		6		
		2	7		7		
Minogo, Hiroshima	Chokoto	1	50	48			2
	Nakate-senbon	1	50		44		6
		2	50		36	1	14
Hachihonmatsu, Hiroshima	Hirosake	1	50		50		
		2	50		48		2
		3	50		50		

most isolates showed a single race reaction (Nakamura 1972; Fujikawa et al 1971; Table 5).

If a large number of spores of a virulent race were introduced to a large number of plants, a few virulent mutants could sometimes be isolated from the lesions that were produced on rare occasions on the resistant cultivars. Using that technique, Niijeki et al (1973) succeeded in selecting several mutants. The mutation frequency varied with the isolates and the rice genotypes (Table 6).

Table 6. Mutation frequencies of acquiring virulence from avirulent isolates inoculated to resistant cultivars. based on the ratio of the number of lesions on resistant to the number of lesions on susceptible cultivars (Niijeki et al 1973).

Resistant cultivar inoculated	Isolates used	Mutation frequency
Kanto 51	P 2b	3×10^{-4}
	Hoku 1	$<2.4 \times 10^{-5}$
	Ken 54-20	1.4×10^{-4}
	Ine 168	1.1×10^{3}
Fukunishiki	P 2b	1.1×10^{-4}
	Ken 53-33	9.1×10^{-5}
	Hoku 1	1.5×10^{-4}
	Hoku 1	$<3.0 \times 10^{-5}$
Pi No. 1	Ine 72-a	1.2×10^{-3}
	Ine 72-a	7.0×10^{-4}
	Ken 54-20	1.6×10^{-3}
	Ken 54-04	1.7×10^{-5}
Pi No. 4	Ken 53-33	$<1.3 \times 10^{-5}$
	Ine 72-a	$<1.6 \times 10^{-5}$
	Hoku 1	$<2.9 \times 10^{-5}$
	Ken 54-20	$<1.6 \times 10^{-5}$
6bJ-69	P 2b	3.4×10^{-5}
	Ken 53-33	3.0×10^{-5}
	Hoku 1	$<4.7 \times 10^{-5}$
	Ken 54-20	$<1.8 \times 10^{-5}$

Table 7. Mutation frequency of fungicide-resistant strains in *Pyricularia oryzae* (Katagiri and Uesugi 1976).

Strain	Concn of fungicide[a] for selection	Frequency (× 10^7)	Reaction[b] to fungicide[a]			
			KSM	Benomyl	IBP	HPA
Wild	0	0	S	S	S	R
Wild	200 ppm KSM	100	R	S	S	R
PTL-R	"	110	R	S	R	S
Wild	2.0 mM IBP	34	S	S	R	S
KSM-R	"	22	R	S	R	S
Wild	3.4 μM Benomyl	1.1	S	R	S	R
PTL-R	0.1 mM HPA	4.0	S	S		R

[a]KSM = Kasugamycin = Kasumin; Benomyl = Methyl-1 (buthylcarbamoyl) -2-benzimidazole = Benlate, IBP = Isopropyl benethiophosphate 0.0 -Diisopropyl -S-benzylthiophosphate = Kitazin; HPA = Dihexyl -N-methyl -N-phenylphosphoramide. [b]S = susceptible; R = resistant

GENETICS OF THE FUNGUS

Mutants with altered virulence, nutritional requirements, and resistance to fungicides have been obtained by several methods (Yamasaki et al 1964). The mutants are quite stable genetically and are used for many kinds of research, including the identification of resistance genes of rice plants. Recently, strains resistant to kasugamycin, an antibiotic fungicide commercially used in Japan, increased on farms in Yamagata prefecture where the fungicide had been applied several times to each crop for more than 4 years (Miura et al 1976). Experimentally, kasugamycin-resistant strains could be easily selected at a mutation frequency rate of 10^{-5} from ordinary sensitive isolates in a medium containing the antibiotic at 200 ppm (Katagiri and Uesugi 1976; Table 7).

With few exceptions, the cells in conidiophores, conidiospores, germ tubes, and mycelia are uninuclear. About 1 to 4 % have two or more nuclei, but seem to be transitional (Table 8, 9). Yamasaki and Niijeki (1965) observed that in the course of anastomosis, the nucleus of a strain migrated into the cell of another strain to produce a heterodicaryotic cell and occasionally to become diploid

Table 8. Number of nuclei in spores of *Pyricularia oryzae* (Toriyama et al unpubl.).

Isolate	Nuclei (no./cell)			Cells observed (no.)
	1	2	3	
11	434	6	0	440
104	179	0	0	179
Hoku 1	142	2	0	144
188	421	1	0	422
15	693	7	0	700
A 168	212	2	0	214
Total	2081	18	0	2099

Table 9. Number of nuclei in mycelia of *Pyricularia oryzae* (Toriyama et al unpubl.).

Isolate	Nuclei (no./cell)			Cells observed (no.)
	1	2	3	
183	207	2	0	209
105	299	2	0	301
A 168	128	4	0	132
5420	244	4	0	248
Hoku 1	253	5	0	258
15	252	3	2	257
5404	275	10	1	286
104	197	2	0	199
Ai 72	40	0	0	40
P 2	296	11	2	309
Ken 60-19	227	4	0	231
1-2	140	4	0	144
188	165	1	0	166
11	173	2	1	176
Total	2896	54	4	2956

after fusing. They reported that many single-conidium isolates of heterodiploids showed recombinant characteristics for auxotrophy, hydrogen sulfide production, and copper sulfate resistance (Table 10). They suggested that parasexual recombination was a possible cause of variability of the cultures. Goto and Yamanaka (1968) reported that one monoconidial reisolate from a mixed culture containing two races differed in virulence from the parental isolates. I, however, have not yet succeeded in obtaining and variation of virulence with mixed culturing (Table 11). In my study, the cultures failed to anastomose.

The possibility of extra-chromosomal or cytoplasmic factors has also been suggested by Frederiksen (1975).

Following up the findings of Hebert (1971), studies of the perfect stage of the blast fungus are in progress in Japan. Isolates from rice, when mixed with isolates from finger millet *Eleusione coracana* and from goosegrass *Eleusione indica,* easily produce matured perithecia. In a cross of rice isolates with finger-millet isolates, ascospore progenies segregated 1:1 for pathogenicity to the original hosts (Kato et al 1976; Yaegashi and Nishihara 1976; Table 12).

REACTIONS OF RICE PLANTS

General reactions

With spray inoculation, even when fresh spores of a stable, single race are used, lesions of several different reaction-types are produced on a leaf (Goto et al 1961). The proportion of susceptible-type lesions decreases with aging of leaves (Fig. 1). When most plants produce susceptible-type lesions, they must be recognized as susceptible. On the other hand, cultivars should be recognized

Table 10. Characters of single-spore cultures isolated from heterodiploids in *Pyricularia oryzae* (Yamasaki and Niijeki 1965).

Heterodiplold or parent	Nutritional requirement				Hydrogen sulfide generation	Copper sulfate resistance color		Cultures (no.)	
	Adenosine	Methionine	Inositol	Lysine					
Parent 1	−	+	+	+	−	S	black		
Parent 2	+	+	−	+	−	R	black		
Heterodiploid I	+	+	+	+	−	R	black		
Single spore culture	+	+	−	+	−	R	pale black	31	
	+	−	+	+	−	S	black	110	
	+	+	+	+	−	R	brown	15	(Heterodiploid I)
	+	+	−	+	−	S	black brown	2	
	+	+	−	+	−	R	grey	1	
	−	+	+	+	−	R	grey	1	
Parent 3	−	+	+	+	−	S	black		
Parent 4	+	−	−	+	+	S	pale black		
Heterodiploid II	+	+	+	+	−	S	black		
Single spore culture	−	−	−	?	−	S	black	22	
	+	−	+	+	−	S	black	16	
	+	+	+	+	−	S	black	2	(Heterodiploid II)
	+	−	+	+	−	S	brown	72	
	+	−	+	+	−	S	white	8	
Parent 5	−	+	+	+	−	S	black		
Parent 6	+	−	+	+	+	R	black		
Heterodiploid III	+	+	+	+	−	R	black		
Single spore culture	+	−	+	+	+	R	black	35	(Parent 6)
	+	+	+	+	+	R	black	4	
	+	−	+	+	−	–	brown	65	
	+	−	+	+	−	–	white	5	
	+	−	+	+	+	–	white	1	
	+	−	+	+	+	R	black	9	

\+ =
– =
S = susceptible
R = resistant

Table 11. Races of monoconidial subcultures originating from the mixed culture of 2 races (National Institute of Agricultural Sciences 1967, unpubl.).

Mixed races (Isolates used)	Race identified	Subcultures (no.)
C-1, N-1 (Ken 60-19, Hoku 373)	C-1	30
	N-1	17
	T-1?	5
C-3, N-1 (Naga 87, Hoku 373)	C-3	181
	N-1	13
	N-2	1
C-3, N-1 (Hiro 65-182, Hoku 373)	C-3	41
	N-1	5
N-5, 0 (Naga 61-14, Ken 54-04 N)	N-5	21
	0	13

as resistant if they produce no lesion, of the susceptible type under conditions favoring blast disease. Reactions of plants differ greatly with differing environmental conditions before and after inoculation, and with aging (Goto et al 1961). The uppermost leaf of seedlings at the fourth- to seventh-leaf stage usually shows a very clear reaction (Table 13). Plants are so sensitive to environmental conditions that even persons with long experience fail sometimes to produce good infections by spray inoculation; low air humidity, or air temperature above 28°C (Table 14), or a restricted supply of nitrogen fertilizer makes the plants very resistant. Punch inoculation of the uppermost leaves, however, results in one of two very clear reactions: susceptible or resistsnt.

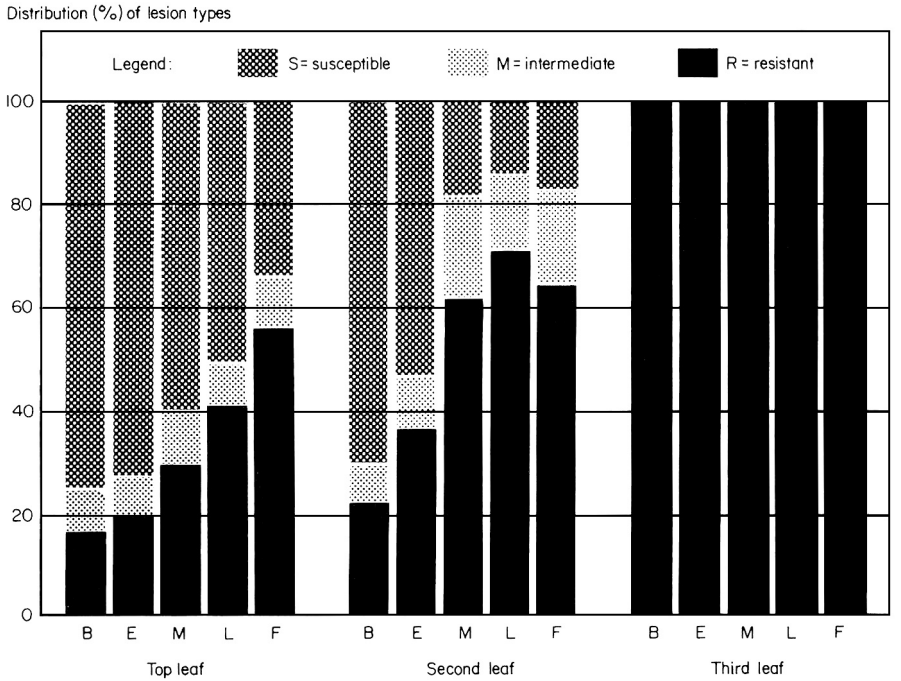
Plant reactions differ intrinsically in their different parts, e.g. glumes, panicle branches, and developing panicle necks are likely to be rather less complete in expression of resistance than are leaf blades. Inoculation tests showed that isolates that were avirulent to the leaf blades often produced a considerable number of susceptible lesions on the glumes and panicle branches of the same cultivars without any pathogenic variation (Kato and Sasaki 1974; Yamanaka et al 1970; Suzuki and Fujita 1976; Table 15). Avirulent races were obtained from glumes with high frequency (Yamanaka et al 1970; Koide 1976: Suzuki and Fujita 1976; Table 16).

The reason a leaf blade always shows an X-type reaction has been unclear. Goto and Yamanaka (1968) showed that the reisolates from lesions of different reaction-types on a leaf produced by an isolate were the same in their virulence except when most of the lesions were of the resistant type. Of the spores that exhibited difrerent lesion-types when used in spray inoculation, more than 80% could invade cells with uniformly developed mycelium when sheath inoculation was used. Those facts indicate that the spores used were less different in virulence than in their reactions on leaves. I believe the motor

Table 12. Mating reactions of isolates of *Pyricularia* from 20 gramineous species (Yaegashi and Nishihara 1976).

Host source	Country of origin	Isolates used (no.)	Host source and mating reaction[a]: *Stenotaphrum secundatum*	*Setaria viridis*	*Setaria italica*	*Phalaris arundinacea*	*Pennisetum typhoides*	*Pennisetum clandestinum*	*Panicum colaratum*	*Panicum bisulcatum*	*Oryza sativa*	*Lolium multiflorum*	*Leersia oryzoides*	*Festuca elatior*	*Eriochloa villosa*	*Eragrostis lehmaniana*	*Eragrostis curvula*	*Eleusine indica*	*Eleusine coracana*	*Digitaria sanguinalis*	*Cenchrus ciliaris*	*Anthoxanthum odoratum*
Anthoxanthum odoratum	Japan	6	–	–	+	–	–	–	+	–	–	–	–	–	–	–	–	+	+	–	–	–
Cenchrus ciliaris	Japan	3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	
Digitaria sanguinalis	Japan, US	20,9		–		–	–	–	–	–	–	–	–	–	–	–		–+	+	+		
Neusine coracana	Japan	9	+	+	+	+	–	+	+	–	+	–	–	–	+	+	+	+	+			
Eleusine indica	Japan	4	–	+	+	+	–	+	+	–	+	–	–	–	+	+	+	+				
Eragrostis curvula	Japan	5	–	–	+	–	–	–	+	–	–	–	–	–	–	–	–					
Eragrostis lehmaniana	Japan	3	–	–	+	–	–	–	+	–	–	–	–	–	–	–						
Eriochloa villosa	Japan	1	–	–	–	–	–	–	–	–	–	–	–	–	*							
Festuca elatior	Japan	1	–	–	–	–	–	–	–	–	–	–	–	*								
Leersia oryzoides	Japan	1	–	–	–	–	–	–	–	–	–	–	*									
Lolium multiflorum	US	1	–	–	–	–	–	–	–	–	–	*										
Oryza sativa	Japan	20	–	–	–	+	–	–	–	–	–											
Panicum bisulcatum	Japan	4	–	–	+	–	–	–	–	–												
Panicum colaratum	Japan	3	+	+	–	+	–	–	–													
Pennisetum clandestinum	Japan	2	–	–	–	–	–	–														
Pennisetum typhoides	US	2	–	–	–	–	–															
Phalaris arundinacea	Japan	2	–	–	+	–																
Setaria Italica	US	3	+	–	–																	
Setaria viridis	Japan	1	–	*																		
Stenotaphrum secundatum	US, Peru	2,1	–																			

[a] + = perithecia with ascospores produced; ± = perithecia without ascospores produced occasionally; – = no perithecia produced; * = no mating was conducted because of single isolate.



1. Variation in lesion type of the top to the third leaf in relation to the grade of emergence of the top leaf at the time of inoculation (seedling) (Goto et al 1961). B = beginning, 1-2%, E = early middle, 21-30%, M = middle. 41-60%, L = late middle, 61-80%, F = final, 81-100%, S. M. and R indicate susceptible, intermediate, and resistant reaction type of lesions, respectively.

cells where the infection occurs are not uniform in susceptibility.

Host-phenotypes in relation to resistance genes. The race-specific major genes in Japan are being identified by their different reactions to a representative number of fungus races, including mutants. Kinds or numbers of resistance genes, however, have not been distinguished by differences in behavior toward the same race, although there are a few genes of incomplete dominance to environmental conditions or of weak resistance (Yamasaki and Kiyosawa 1966). Experience with many isolates suggests that the cultivars that are likely to show fluctuating reactions to isolates or to environmental conditions seem to have several minor genes cooperating. For example, Homarenishiki and Ginga, members of an old set of Japanese differentials, which often showed inconsistent reactions, have one unidentified race-specific major gene and two minor genes (Kiyosawa 1970). Tatsumimochi and Fukei-69 have been recognized so far to be the same *Pi-k* genotype, but the former is less infected by race C-3 (*k*) than by race C-1 (*a, i, k*), while the latter is infected equally by both races (Yaegashi and Kobayashi 1976). Race C-3 attacks cultivars with *pi-k* gene as represented in (*k*). The dominance of *Pi-k* in Tatsumimochi may

Table 13. Change of varietal resistance of rice with aging in leaf blast (Central Agricultural Experiment Station 1967).

Cultivar	Leaf stage at inoculation	Disease incidence	Cultivar	Leaf stage at inoculation	Disease incidence
Kongo	4	●●●●●	Estunan 30	4	●●●●● ●●
	6	●●●●● ●		6	●●●●● ●●
	8	●●		8	●●●●
	10	●●		10	●●●●
	12			12	●
Kanto 59	4	●●●●● ●●●●●	Kusabue	4	●●●●● ●●●●●
	6	●●●●● ●●●●		6	●●●●● ●●●●
	8	●●●●● ●		8	●●●●● ●
	10	●●●●●		10	●●●●● ●
	12	●●		12	●
Shinanohikari	4	●●●●● ●●●●	Takanenishiki	4	●●●●● ●●●●
	6	●●●●● ●●●		6	●●●●● ●●●
	8	●●●		8	●●●●●
	10	●		10	●●●●
	12	●		12	●
Homarenishiki	4	●●●●● ●●	Aichiasahi	4	●●●●● ●●●●●
	6	●●●●● ●●		6	●●●●● ●●●●
	8	●●		8	●●●●● ●●
	10	●		10	●●●●● ●
	12			12	●●
Norin 22	4	●●●●● ●●●●	Norin 29	4	●●●●● ●●●●●
	6	●●●●● ●●●		6	●●●●● ●●●
	8	●●●●●		8	●●●●●
	10	●●		10	●●●●
	12	●		12	●

Table 14. Effects of air temperature after inoculation on varietal resistance to leaf blast (Central Agricultural Experiment Station 1968).

Cultivar	Temperature (°C)	Lesions (no./leaf)	Cultivar	Temperature (°C)	Lesions (no./leaf)
Sanin 68	20	●●●●●●●●●● ●●●●	Kinmaze	20	●●●●●●●●●●
	25	■■■■■■■■■■ ■		25	■■■■■■■■
	30	▲▲▲▲▲▲▲▲▲		30	▲▲
Senshuraku	20	●●●●●	Yamabiko	20	●●●●●●
	25	■■■■■■		25	■■■■■
	30	▲▲▲		30	▲▲▲
Kanto 51	20	●●●●●●●●●● ●●●●●	Chiyohikari	20	●●●●●
	25	■■■■■■■■■■ ■		25	■■■■■
	30	▲▲▲▲▲▲▲▲▲▲ ▲		30	▲▲▲
Kusabue	20	●●●●●●●●●●	Koganenishiki	20	●●●
	25	■■■■■■■■■■ ■		25	■■
	30	▲▲▲▲▲▲▲▲▲▲		30	▲
Ohu 248	20	●●●●●	Norin 29	20	●●●●●●●●●● ●●●
	25	■■■■		25	■■■■■■■■■■ ■■■■■■
	30	▲▲▲		30	▲▲▲▲▲▲▲▲▲
Senbonasahi	20	●●●●●	Tozan 38	20	●●●●●●●●●
	25	■■■■■■		25	■■■■■■■■■■
	30	▲		30	▲▲

Table 15. Infection of spikelets with races of *Pyricuiaria oryrae* avirulent to leaves (Suzuki and Fujita 1976).

Cultivar inoculated	Genotype	Race inoculated	Reaction[a] on leaves	Infected spikelets (%)
Fujiminori	*Pi-a*	N-1	S	73
		C-3	R	19
		N-4	R	7
Sasashigure	*Pi-a*	N-1	S	82
		C-3	R	13
		N-4	R	8
Ugonishiki	*Pi-k*	C-8	S	48
		N-1	R	35
		N-2	R	53
Fukei 69	*Pi-k*	C-8	S	29
		N-1	R	35
		N-2	R	23
Yoneshiro	*Pi-i*	N-1	S	65
		C-8	R	38
		N-2	R	36
Tohoku 111	*Pi-i*	N-1	S	70
		C-8	R	23
		N-2	R	20

[a] s = susceptible; R = resistant

be influenced by unknown genes. Goto (1970) reported that Shensho was controlled by three resistance genes, which contributed different degrees of resistance cumulatively to the isolates Ken 53-33, race T-1 (*a, i, k, ta*). In general, gene analysis of exotic cultivars with Japanese domestic races, including their mutants, seems to be rather more difficult than that of Japanese domestic cultivars. That probably is due to the rather complex gene constitution of the exotic cultivars. Most of them showed a continuous distribution of intensity of resistance in F_2 and F_3 progeny of crosses with susceptible Japanese cultivars. Kiyosawa (1971) mentions that, in general, gene analysis for resistance is simplest when the varieties to be analyzed are crossed with

Table 16. Isolation frequency of avirulent races from naturally infected spikelets (Suzuki and Fujita 1976).

Cultivar	Isolations (no.)	Avirulent races (%)
Himenomochi	52	58
Tatsumimochi	43	46
Dewanomochi	29	31
Sasakimochi	33	34
Fukunohana	58	24
Shimokita	12	100
Kairyo shinko	30	10

other varieties native to the same country and the hybrid progeny are inoculated with a native strain of fungus.

Pathogenic variability observed in the Philippines might be due partly to the complex gene constitution of the differentials.

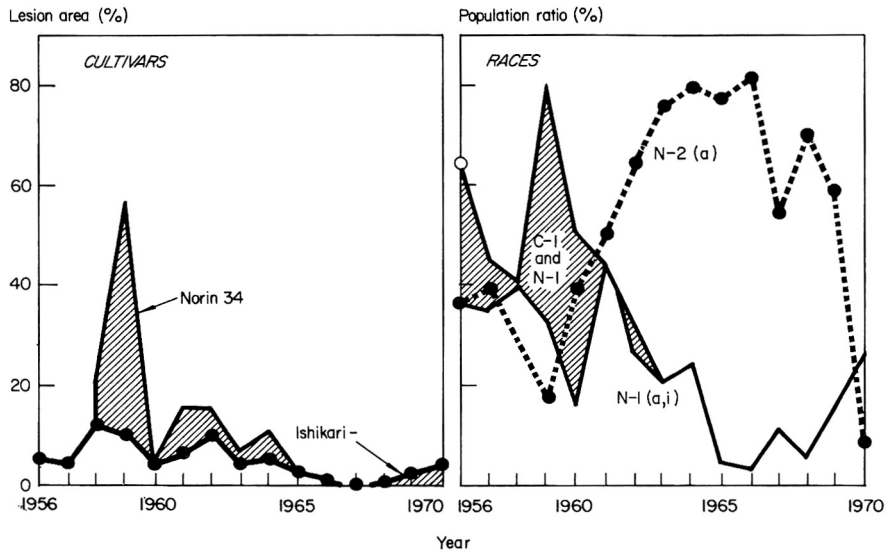
CHANGES IN PREVALENCE OF RACES

Race studies in Japan began in 1955. In the first 10 years of research, the identification and distribution of races had been made extensively on a national scale. In the next 10 years, research had focused on the changes of the prevailing races in some selected regions. The results (Goto et al 1961, 1964; Kozaka et al 1972) can be summarized as follows. The race flora in Japan is rather simple, probably because of the simple genotypes of the currently used cultivars (Table 17). The predominant races up to 1960 were race N-2 (*a*) in Southern Japan and race N-1 (*a,i*) in Northern Japan, followed by race C-1 (*a*, *i*, *k*) in Northern Japan, Race N-4 (+) and race N-5 (*i*) Mere detected locally. The prevalence of race N-1 (*a*, *i*) in Northern Japan was ascribed to the wide distribution of the cool-resistant cultivars carrying the resistance gene *Pi-i.* The gene *Pi-k* was found in Chinese cultivars and some other foreign cultivars, but not in the Japanese domestic cultivars; at the time, no cultivars with *Pi-k* were in commercial use. Accordingly, the reason for the predominance of race C-1 (a, i, k) in Northern Japan was not clear. In very ancient times when so-called Toboshi, which is assumed to be of Chinese origin, was cultivated in Japan, races virulent to it might have become naturalized. Since 1960 several cultivars newly developed from crosses with Chinese varieties were released for the control of blast. With the rapid increase in planting of the new cultivars, populations of the races C-1, C-5, C-6 (*a*, *i*, *k*), C-8 (*a*, *k*), C-9 (*i*, *k*), and C-3 (*k*), which had been minor, increased rapidly, and 2 to 4 years later a breakdown of resistance occurred even where the new cultivars had been widely introduced. Damage was more severe than that suffered by the old cultivars. The new

Table 17. Grouping Japanese rice cultivars collected by National Institute of Agricultural Sciences on the basis of the reaction to the selected races of *Pyricularia oryzae* in Japan (Yamada et al 1969).

Cultivar group	Reaction[a]							Possible genotype	Cultivars (no.)
	C-3	C-6	N-1	N-2	N-3[a]	N-4	N-5		
A	S	S	S	S	S	S	S	+	910 (67%)
B	R	S	S	S	S	R	R	*Pi-a*	332 (25%)
C	R	S	S	R	R	R	S	*Pi-i*	47 (3%)
D[b]	R	R	R	R	R	R	R		11
E	R	S	S	R	R	R	R	*Pi-a, Pi-i*	7
F	R	R	S	S	R	R	R	*Pi-a, ?*	4
G	S	R	S	S	R	S	S		3
H[b]	S	R	R	R	R	R	R		2

[a]S = susceptible, R = resistant [b]Includes the cultivars from the cross with foreign cultivars.

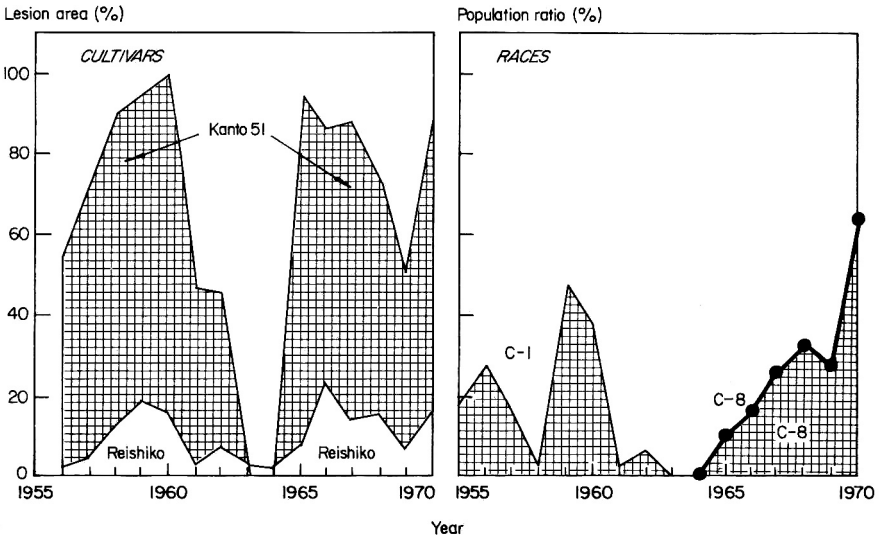


2. Annual variation in disease incidence of the cultivars in relation to the prevalence of races (Nakanishi and Nishioka 1972). Races C-1 and N-1 are virulent to both culttivars, but race N-2 is avirulent.

cultivars were replaced by the old. The planting of cultivars with *Pi-i* has also decreased gradually because of poor eating quality. Consequently, the races C-1, C-5, C-6 (*a, i, k*), C-3 (*k*). and N-1 (*a, i*) have decreased. In the 1970's the races N-2 (a) and C-8 (*a, k*) have predominated, except in Hokkaido and some few prefectures, where cultivars of the new type have still been widely grown. Quite recently, in Kyushu district in the southernmost part of Japan, the race T-2 (*a, ta*) increased because of the wide distribution of a new cultivar. Reiho, carrying the *Pi-ta* gene (Matsumoto 1974).

Such facts suggest that change in the prevalence of races depends mainly on the genotypes of the cultivars grown; evolution would normally take place in steps, starting from the basic races N-2 (*a*) and C-8 (*a, k*), and the unnecessary genes seem to have less fitness for survival. Disease incidence of cultivars in the field is determined by the population of virulent races found there, but not by the number of virulent races (Fig. 2, 3).

In attempting to breed a new cultivar resistant to all known races, a problem is to predict the occurrence of new virulent races as early as possible. There are a few reliable examples in Japan. Pi No. 1 is the first resistant cultivar developed from a cross with an indica variety, Tadukan, by repeated back-crossing. Extensive screening in nursery beds was conducted at Chugoku National Experiment Station during some 10 years following 1945. Susceptible cultivars were planted around each nursery as spreaders and inoculated with a local isolate at the third- or fourth-leaf stage. Infections were very severe



3. Annual variation in disease incidence of the cultivars in relation to the prevalence of races (Nakanishi and Nishioka 1972). Races C-1 and C-8 are virulent to both cultivars.

in all tests, but no new virulent races could be detected during the course of breeding. The races virulent to Pi No. 1 later were found in isolates collected from other districts. The circumstances were very similar in the case of Fukunishiki, whose gene was introduced from Zenith at Tohoku National Experiment Station in 1962. An unknown race was not found until 1966 when the new cultivar had been distributed widely in Fukushima prefecture (Hirano et al 1967).

While the breeding program for Pi No. 1 was under way, the existence of pathogenic specialization of the rice blast fungus was still unclear; breeders could not conceive of a strain that might be virulent to new cultivars. When Fukunishiki was being developed, breeders were well aware of the existence of races. But the new virulent race was not detected during its breeding.

Recently, a test to discover whether careful observations could find a new race was conducted for a cultivar crossed with Toride No. 1, which has a resistance gene from an indica variety, TKM. The test cultivar was grown in small fields of about 0.01 to 0.03 ha by ordinary cultural methods at 5 district experiment stations. In three stations where the disease was very severe in control plants, 0.1 to 1.3 % of the hills were slightly infected with 1 or 2 susceptible-type lesions, from which a new race was isolated (Asaga et al 1975).

Special attention could sometimes, but not always, detect unknown races.

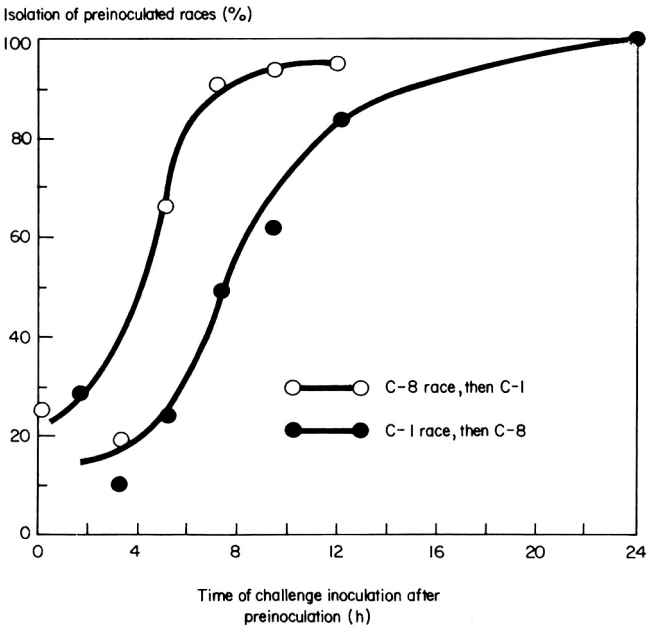
INTERACTION AMONG RACES

Interaction among virulent races

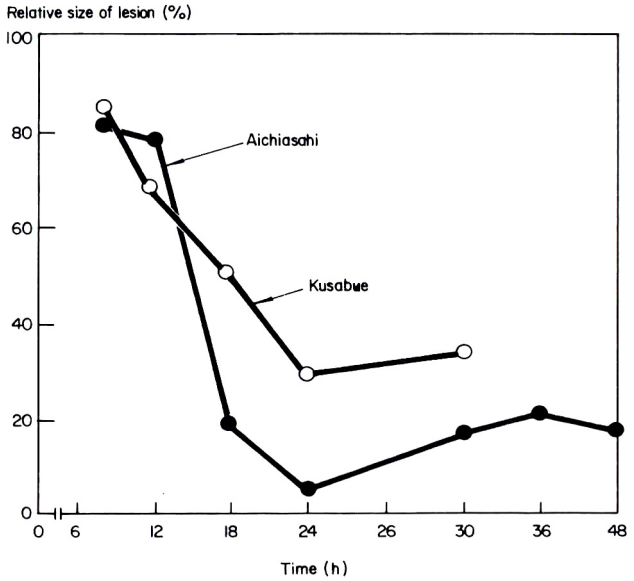
From lesions produced by either punch or spray inoculation with the spores of two different virulent races mixed in equal portions, the original two races could he reisolated, but one often predominated, showing competition between races or isolates (Yamada and Iwano 1971: Yamanaka 1974). That was true in a nursery-bed test in which several races were used in a mixture for inoculations (Shimoyama and Endo 1972). In double inoculations at several intervals, a race inoculated more than one-half day in advance of another was generally reisolated at a higher rate (Iwano 1976; Fig. 4).

Interaction between a virulent and an avirulent race

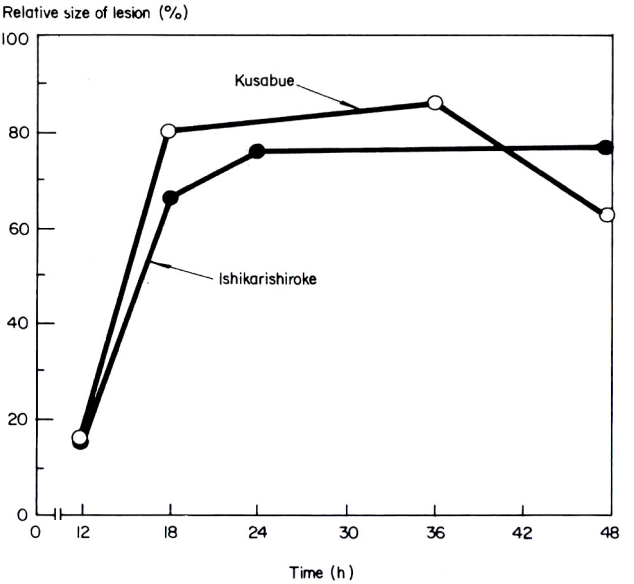
Induced resistance or cross protection is observed when an avirulent race is used to inoculate, and inoculation with a virulent race follows in not less than 18 hours (Takahashi 1956; Takayama and Kozaka 1976; Fig. 5). Similar preinoculation with a virulent race induces susceptibility, and infection then can occur upon inoculation with an avirulent race (Koraka unpubl.) (Fig. 6).



4. Reisolation ratio of preinoculated race in relation to the time of challenge inoculation in double inoculation with two virulent races (Iwano 1976)



5. Lesion sizes in double inoculation with avirulent and virulent races (Takayama and Kozaka 1975). The cultivars Aichiasahi and Kusabue were used independently.



6. Infection lesions by avirulent race in double inoculation with virulent and avirulent races (Kozaka unpubl.). The cultivars Ishikarishiroke and Kusabue were used independently.

In inoculation with two races at one time, protection is induced in proportion to the concentration of the avirulent race (Ohata and Kozaka 1967). The induced protection is local, not systemic.

Consequently, the complex interaction among races can be observed under such conditions as nursery-bed screening for resistant cultivars.

NATURE OF RACE-SPECIFIC RESISTANCE

The fungus penetrates motor cells of the leaf blade. Although the rate of penetration by appressoria differs with the concentration of inoculum, it is usually about 15 or 20% with spray inoculation of younger susceptible cultivars. In resistant cultivars, penetration ranges from 0 to about 15% (Yoshino 1972). With sheath inoculation, it is usually above 80% in both types of cultivar (Sakamoto 1968).

In compatible reactions in inner epidermal cells of the sheath, the invaded host cells maintain the ability for plasmolysis and neutral red staining for about 38 hours after penetration, without marked visible change. That indicates that like obligate parasites, the fungus does not kill susceptible host cells at the very early stage of infection. In incompatible reactions, the invaded host cells clearly show a hypersensitive reaction within 24 hours. The invaded mycelia die after that (Sakamoto 1968: Ohata et al 1963). Nonhost plants of *Monocotyledones* often produce marked papilla beneath the appressoria before penetration. The other responses of host cells are similar to those in resistant rice cultivars. The invading mycelia cannot develop beyond the primary infected cells, and they soon die (Hashioka and Kusadome 1975).

The mechanism of race-specific resistance is not yet understood exactly. But, as already indicated, infection by an avirulent race protects from challenging infections by virulent races. Also, a peculiar fluorescent substance, which is toxic to the fungus, is always produced in diseased plants, particularly in resistant tissues immediately after infection, but not in healthy plants. It seems reasonable to believe that resistance to blast, like resistance to many other diseases is caused by the induction of an unidentified phytoalexin-like substance (Ohata and Kozaka 1967).

NATURE OF NONSPECIFIC RESISTANCE

Varietal differences in nonspecific resistance can be measured by the number and size of susceptible lesions by virulent races. Horizontally resistant cultivars generally show fewer and smaller lesions. With sheath inoculation, mycelial development is less, and the invaded host cell shows cell-wall thickening or browning of cytoplasms.

On horizontally resistant cultivars, leaf lesions usually show earlier, and are surrounded by a darker brown color. Accumulation of phenols, melanin-like substances, lignins, and some other subtances is probably involved in making

chemical and physical barriers around infection areas.

The stunting of plants by infection is one of the most noticeable symptoms of leaf blast. Remarkable ethylene production occurs immediately after infection in susceptible cultivars; less ethylene is produced in resistant cultivars. Inoculation by punching leaves either with methionine, a precursor of ethylene, or with ethler, a commercial chemical that produces ethylene in plants, causes the same symptoms as does blast infection (Kozaka 1977). Such facts suggest that a major factor in stunting may be ethylene production. The remarkable increase of peroxidase in infected tissues might be related to ethylene production. The relationships between tolerance of ethylene injury and tolerance of leaf blast are under investigation.

CONCLUSIONS

The results obtained in Japan indicate that although the rice blast fungus is likely to be rather variable in pathogenicity during culture, selected isolates are quite suitable for use in genetic analysis and in many other studies. The genetic and biochemical aspects of the host-parasite interaction that are observed when using these isolates are essentially the same as those seen in many other diseases such as wheat rust and potato late blight. Much information accumulated from the study of many other diseases can also be used in breeding cultivars resistant to blast.

My conclusions on the pathogenic variability of cultures may differ somewhat from those of some foreign workers. Further research on the genetics of the blast fungus would resolve the disagreement. I suggest the use of the protoplast-fusion technique for research on the somatic inheritance of the fungus.

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BREEDING FOR AND GENETICS OF BLAST RESISTANCE IN JAPAN

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BLAST RESISTANCE HAS CONSISTENTLY been one of the most important objectives of rice breeders in Japan. The history of rice breeding in that country may be roughly divided into three stages: 1) breeding for field resistance, 2) breeding for true resistance, and 3) breakdown of true resistance and reassessment of field resistance. The terms "true resistance" and "field resistance" are more commonly used in Japan than are the terms "vertical resistance" and "horizontal resistance."

This paper discusses the genetic studies on, evaluation of, and strategy for future breeding for, blast resistance in Japan.

HISTORICAL REVIEW OF BREEDING FOR BLAST RESISTASCE

Breeding for field resistance

In the first stage of Japan's rice breeding history, the breeders' main occupation was to accumulate minor genes governing a moderate level of resistance in Japanese domestic varieties. Such resistance is considered field resistance or horizontal resistance in the light of present knowledge (Ezuka 1972).

Rice breeding work in Japan started in 1904 with the support of national funds (Matsuo 1967). Pure-line selection was the main breeding method. Some varieties such as Kameji and Aikoku were found to be resistant to blast disease in those days. Their resistance, however, was not sufficient to control the disease after Japanese farmers began to use nitrogenous fertilizers more intensively (Ito 1965). The effect of pure-line selection was too limited for developing more resistant varieties.

Hybridization was introduced into rice breeding around 1910 (Kiyosawa et al 1975). Rice breeding was systematically organized throughout the country in 1927 (Matsuo 1967). Since that time, a large number of crosses among domestic japonica varieties have been made at every agricultural experiment station. Many blast-resistant varieties have been developed and released for commercial

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cultivation (Ito 1965; Toriyama 1972: Kiyosawa et al 1975).

The variety Norin 6 was developed from the cross Joshu × Sen-ichi in 1936, and Norin 8 was developed from the cross Ginbozu × Asahi in 1937. Norin 6 was found to be resistant to neck blast, while Norin 8 was resistant to leaf blast. Norin 8 and Norin 6 were then crossed to develop varieties resistant to both leaf and neck blast. Norin 22 and Norin 23 were thus developed in 1943. Those two excellent varieties were used for further crossing with other leading varieties. A number of useful resistant varieties such as Honenwase, Chiyohikari, and Yamabiko were released to farmers by the year 1958.

Another group of resistant varieties was developed by Iwatsuki (1942) and Ujihara (1960). Iwatsuki noticed an upland variety, Sensho, to be more resistant to blast than paddy varieties. He crossed Sensho with a paddy variety, Kinai-ban 33, in 1923, and made further multiple crosses with paddy varieties to eliminate undesirable characters of upland rice. After he crossed it three or four times with paddy varieties, Shinju was developed in 1936 and Futaba in 1938. Iwatsuki and Ujihara further developed Shuho from the cross Futaba x Norin 6 in 1944. Shuho was then crossed with Norin 22 and Rikuu 132, both of which were also resistant varieties. From the cross Shuho × Norin 22, five excellent varieties, Wakaba, Wasewakaba, Koganenishiki, Ukonnishiki, and Homarenishiki were developed, and from the cross Shuho × Rikuu 132, Ginga was developed. Fujiminori and Reimei were also good varieties derived from the cross including Futaba.

The resistant varieties thus developed were cultivated successfully in blast-prevalent areas, and also were used as sources of genes for blast resistance. Distinct erosion of field resistance has rarely been observed in those varieties.

Breeding for true resistance

In 1942, a new breeding program was started to introduce into Japanese paddy rice a major gene or genes governing a high level of resistance in foreign varieties (Matsuo 1967). That type of resistance is what is today called true resistance or vertical resistance.

Matsuo (1952) found that some Chinese japonica varieties such as To-to and Reishiko were highly resistant to blast disease. It was convenient for the breeders that these varieties did not show the hybrid sterility that was often seen in japonica-indica crosses. The hybridization was first made in 1943. Koyama (1952) developed Kanto 51 and Kanto 52 from the cross Ginbozu × To-to, and Kanto 53, Kanto 54, and Kanto 55 from the cross Norin 10 × Reishiko in 1950. Many breeders used those varieties as gene sources for further crossing, and a number of excellent resistant varieties were developed. Among those resistant varieties, Kusabue (Ito et al 1961), Yuukara (Samoto and Ouchi 1968), and Senshuraku (Kunitake et al 1962) were outstanding. They were cultivated over a wide area because of their high yielding ability and good grain quality.

Ujihara and Nakanishi (1960) also used a Chinese variety, Hokushi Tami, as gene source in 1944 to incorporate its high resistance with the moderate

resistance of Shin-ju and Shuho. BR 1 (Kongo) and Minehikari were thus developed in 1957 and 1965, respectively. The resistance in BR 1 is known to be very stable with no sign of breakdown up to this point.

The hybrid sterility that occurs often in indica-japonica crosses makes it difficult to introduce the resistance gene or genes from indica to japonica. Kitamura (1962) made extensive studies on that hybrid sterility, and developed a method for eliminating it. He proposed the use of an indica variety as a male parent in the first cross, or of a japonica variety as a female parent in the backcross. An indica variety, Tadukan, was used by Shigemura and Kitamura (1954) as a gene source to cross with Norin 8 and Senbon Asahi in 1943. After three to four successive backcrosses with japonica varieties. Pi 1, Pi 2, Pi 3, Pi 4, and Pi 5 were developed during the 1950's. Those varieties and their relatives were used for further crossing to develop resistant varieties. Shimokita, Tosa Senbon, Asahikari, Satominori, and Akiji are the commercial varieties derived from those crosses. Some other indica varieties were also used as donor parents to provide true resistance. They were Oka-ine, Zenith, TKM 1, and CO 25. The varieties developed from them were Yashiro-mochi, Fukunishiki, Toride 1, and Toride 2, respectively (Toriyama 1972).

The gene or genes of true resistance introduced into Japanese varieties by those breeding programs are listed in Table 1. Among them, *Pi-z'* is noticeable because it has a broad spectrum against the races of blast fungus in Japan (Yokoo and Kiyosawa 1970).

Breakdown of true resistance and reassessment of field resistance

The breakdown of true resistance was really unexpected and shocking for Japanese breeders and plant pathologists. It began to occur only several years after the release of the varieties. Pi 5 was first broken down in Hiroshima in 1962. Kusabue was broken down in Fukushima, Tochigi, Toyama, and Hiroshima; and Senshuraku was broken down in Niigata in 1963. The breakdowns of other varieties such as Yuukara, Ugonishiki, Hatsuiwai-mochi, Koshihibiki, Senshuraku, Tatsumi-mochi, Sanpuku, and Fukunishiki were then reported one after another from various parts of Japan within a few years (Yamada 1965; Ezuka 1972).

The causal fungus was immediately isolated from the broken-down plants. The isolates were tested for pathogenicity to the differential varieties. Results showed that the unexpected outbreak of blast was due to the propagation of new or minor races that matched the true resistance involved in those varieties (Yamada 1965; Ezuka 1972).

The most striking scene was observed where a broken-down variety was found in a field adjoining one with a domestic susceptible variety. The broken-down variety was often damaged more severely than the domestic susceptible variety. That was later proved by many investigators to be due to the abnormally low level of the field resistance. The varieties were developed in the absence of the matching fungus races, so that they had never had the opportunity of

Table 1. Main varieties bred for high blast resistance from crosses between Japanese and foreign varieties (Ezuka 1972).

Foreign varieties used as donor parent	True resistance gene introduced [a]	Resistant varieties developed
Indica		
Zenith	*Pi-z*	Fukunishiki, Ou 243, Ou 244, Fukei 67, Fukei 73
TKM 1	*Pi-z*^{t}	Toride 1
CO. 25	*Pi-z*^{t}	Toride 2
Okaine	*Pi-ta*	Yashiro-mochi
Tadukan	*Pi-ta*	Shimokita, Tosa Senbon, Pi 1, Pi 2
"	*Pi-ta*^{2}	Satominori, Akiji, Asahikari, Pi 3, Pi 4, Pi 5
Modan	*Pi-k*	Chugoku 31
Te-tep	*Pi-k*	Mutsunishiki
Chinese japonica		
Reishiko	*Pi-k*	Kusabue, Teine, Yuukara, Oyodo, Matsumae, Ishikari, Kanto 53, Tachihonami, Senshuraku, Koshihibiki, Koshiminori, Imochishirazu, Yachiho, Tatsumi-mochi. Sasaki-mochi, Dewano-mochi, Hatsuiwai-mochi, Hida-mochi
To-to	*Pi-k*	Ugonishiki, Kanto 51, Mangetsu-mochi, Kagura-mochi, Tsukimi-mochi
Yakeiko	*Pi-k*	Chugoku 23
Hokushi Tami	*Pi-k, Pi-m*	Sanpuku, Tsuyuake, BR 1, Takara Senbon, Minehihari, Suzukaze, Himeno-mochi
Taiwan japonica		
Taichung Glu. Yu 26	*Pi-i*	Akishino-mochi, Takasago-mochi

[a]Among the resistance genes shown in this table, only *Pi-i* is found in Japanese domestic varieties.

being selected for field resistance before their release. They offer a typical example of "Vertifolia effect," described by Van der Plank (1963, 1968).

After such bitter experiences, the importance of field resistance is again attracting the attention of Japanese breeders and plant pathologists. Many studies of the nature of field resistance as well as of methods for evaluating it have been made. Since about 1967, it has become common to check the field resistance of newly developed varieties or strains before they are released to farmers. Severe breakdown has not yet been observed on the varieties passed through such a check.

Thus, field resistance is undoubtedly one of the effective countermeasures to be used against the breakdown of true resistance. However, field resistance itself is usually lower than true resistance. It fluctuates more because of environmental conditions than does true resistance. Not a few breeders fear that the field resistance alone may not always be sufficient to control the disease. They now look eagerly for the new strategy for the third stage of breeding for blast resistance.

GENETIC STUDIES OF BLAST RESISTANCE

Genetic studies of blast resistance in Japan can be traced back to Sasaki (1922). Not a few investigators reported on the subject before 1960. Their studies were reviewed by Takahashi (1965) and Yamasaki and Kiyosawa (1966). However, those studies were conducted without sufficient knowledge of the pathogenic specialization of the causal fungus. According to Yamasaki and Kiyosawa (1966), it is quite difficult to obtain a universal understanding from those early studies. Systematic studies that were first undertaken after Goto et al (1961, 1964) established the differential system for blast fungus races in Japan.

Inheritance of true resistance

The first step of the modern genetic studies was to classify rice varieties by their resistance to the different races of blast fungus. Yamasaki and Kiyosawa (1966) classified Japanese rice varieties into five groups using injection inoculation of seven representative fungus strains. Kiyosawa (1972a) later reported that 16 groups can be distinguished by the method.

Some representative varieties were chosen from each varietal group to analyze their blast resistance genetically. Yamasaki and Kiyosawa (1966), Kiyosawa (1972a, 1974), and his coworkers made a series of extensive studies of the genetic constitution of those varieties. Besides the ordinary method of gene analysis, cumulative distribution curves were sometimes used for analyzing resistance that was governed by several major genes. Pathogenic mutants of some fungus strains were also used with those methods (Kiyosawa 1976). The result was identification of 13 major genes governing blast resistance of rice, including some foreign varieties (Table 2). In addition to those genes, *Pi-k*^{c} was found in Chugoku 31 by Toriyama et al (1968) and Yunoki et al (1968), *Pi-ta*^{n} in Nakei 212 by Shinoda et al (1971a), and *Pi-s* in 65A-15 by Shinoda et al (1971b). Later, *Pi-k*^{c} was described as *Pi-k* by Shinoda et al (1971a), and *Pi-s* was found to be identical to *Pi-b* (Kiyosawa, personal communication).

Among those resistance genes. *Pi-k*^{s}, *Pi-a,* and *Pi-i* were found commonly in Japanese domestic varieties. *Pi-k*^{s} does not operate against any of the blast fungus races in Japan, so that it is usually neglected in breeding work. Similarly, *Pi-a* is not particularly effective in controlling the disease because the fungus races matching *Pi-a* are widely distributed throughout Japan. On the other hand, *Pi-i* is effective to some extent because the distribution of the races matching it is rather limited. Most of the other genes were found in foreign varieties or in the Japanese varieties derived from foreign varieties. Those genes, although they suffered from breakdown in some areas, are still effective in other areas where the matching races are not prevalent. The origin of *Pi-s* and *Pi-ta*^{n} is unknown.

It is noticeable that some of the genes have multiple alleles. *Pi-k, Pi-k*^{s}, *Pi-k*^{p}, *Pi-k*^{h}, and *Pi-k*^{c} are allelic and located at the *Pi-k* locus; *Pi-ta, Pi-ta*^{2},

Table 2. List of blast resistance genes identified in Japanese and foreign rice varieties (based on Kiyosawa 1974).

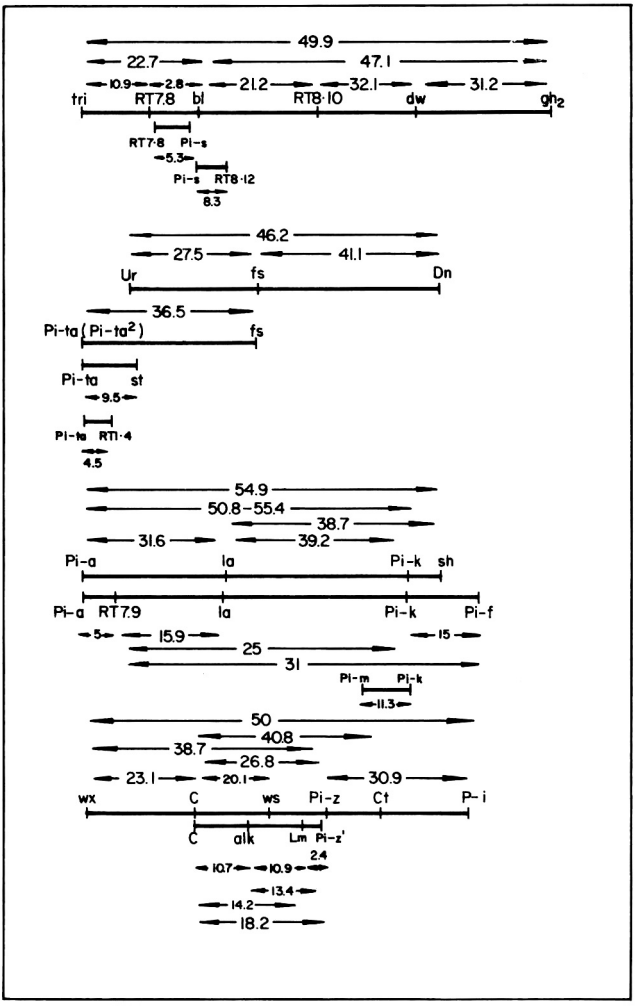
Resistance gene	Varieties in which the resistance gene was identified
Pi-a	Aichi Asahi type varieties in Japan (Japan), Jae Keum, Pal tal, etc. (Korea), Usen, To-to, Choko-to, Pe Bi Hun, Hokushi Tami, Kannonsen (China), CO. 25 (India), Pusur (Pakistan), Blue Bonnet, Zenith (USA)
Pi-i	Ishikari Shiroke, Fujisaka 5, Sekiyama 2, Hokuriku 12, Takasago-mochi, Akishino-mochi (Japan), Doazi chall (Korea), Dawn (USA)
Pi-k	Kanto 51, Kusabue, Minehikari (Japan, Introduced from Chinese varieties), Hokushi Tami, To-to, Choko-to, Reishiko, Yakeiko (China)
$Pi\text{-}k^s$	Shin 2, Norin 6, Shirogane, Kamenoo, Shinriki, Kinmaze, Sasashigure, Norin 17, Norin 18, Zuiho, Ishikari Shiroke, Fujisaka 5, Sekiyama 2, Hokuriku 12, Takanenishiki (Japan), Taichung 65, To-to (long grain) (China), Caloro, Lacrosse (USA)
$Pi\text{-}k^p$	Pusur (Pakistan)
$Pi\text{-}k^h$	HR-22 (India)
Pi-k?	Te-tep (Vietnam), Charnack (India), Tadukan (Philippines), Russia 33 (USSR), Dawn (USA)
Pi-m	Minehikari, Tsuyuake, Sanpuku (Japan, introduced from Chinese variety, Hokushi Tami)
Pi-ta	Yashiro-mochi, Pi 1, K 1 (Japan, introduced from foreign varieties), Okaine, Pai-kan-tao, Kannonsen (China), Tadukan (Philippines)
$Pi\text{-}ta^2$	Pi 4 (Japan, introduced from Philippine variety, Tadukan), Tadukan (Philippines)
Pi-z	Ou 244, Fukei 67 (Japan, introduced from USA variety, Zenith), Zenith (USA)
$Pi\text{-}z^t$	TKM 1, CO. 25, CO. 4 (India), Leuang Tawng 77-12-5, Chao Leuang 11 (Thailand), Morak Sepilai, Kontor (Malaysia)
Pi-b	Bengawan, Tjina, Tjahaja (Indonesia), Milek Kuning (Malaysia)
Pi-t	Tjahaja (Indonesia)

and $Pi\text{-}ta^n$ are at the *Pi-ta* locus; and *Pi-z* and $Pi\text{-}z^t$ are at the *Pi-z* locus. The allelic genes at the same locus differ in their reactions to fungus races.

Linkage relationships among the genes for blast resistance and other traits were studied by several investigators. The chromosome reciprocal-translocation method was used successfully in some studies. Toriyama and Kiyosawa compiled the studies and illustrated them in a chromosome map (Fig. 1; Kiyosawa 1972a, 1974). According to that map, all the genes for blast resistance tested so far belong to the four linkage groups: *bl, fs, la,* and *wx.*

In the course of those studies, Yamasaki and Kiyosawa (1966) and Kiyosawa (1967, 1974) suggested that the gene-for-gene theory can be applied to the host-pathogen relationship. But the direct demonstration was impossible for lack of the perfect state of the blast fungus.

The progress in genetic studies of blast resistance has greatly influenced the field of plant pathology. The differential system for identifying blast fungus races established by Goto et al (1961, 1964) has been widely used in Japan, but it has become clear that the differential varieties adopted there are not always the best choices in the light of genetic studies. Ezuka (1970) and Sakurai and Toriyama (1973) pointed out the defects of these differentials, and presented



1. Linkage relationship among genes for blast resistance and other traits in four group (Kiyozawa 1972, 1974).

the criteria to be fulfilled by ideal differential varieties. They especially stressed that each differential variety should have only one major true resistance gene which operates on Japanese races and is involved in commercial varieties in Japan. Yamada et al (1976) proposed a new system of differentiating races of blast fungus according to criteria presented by Sakurai and Toriyama (1973). In the new system, nine varieties, which have different single genes for true

resistance, were adopted as the differential varieties. They are Shin 2 (*Pi-k*^{s}), Aichi Asahi (*Pi-a*), Ishikari Shiroke (*Pi-i*), Kanto 51 (*Pi-k*). Tsuyuake (*Pi-m*), Fukunishiki (*Pi-z*), Yashiro-mochi (*Pi-ta*), Pi 4 (*Pi-ta*^{2}). and Toride 1 (*Pi-z*^{t}). The new system is expected to be much more useful than the older one, because the races identified by it directly indicate their virulence or avirulence to the commercial varieties cultivated in Japan.

Comparative studies of the resistance genes found in Japan and those found in other countries have not yet started. Blast fungus races vary from country to country as do rice varieties. It is difficult to compare genetic studies of the different varieties and races that have been conducted in different countries. A different gene or genes may be found in one and the same variety when different races are used for inoculation. Kiyosawa (1971) proposed comparing the genes found independently in different countries. The first step is to obtain in each country differential varieties that have different single genes. Then the differential varieties are exchanged and tested for their resistance to the differential races in the new country. If an exchanged variety showed the same reaction as any one of the domestic differential varieties, the resistance genes of both varieties may be identical (although, sometimes they may not be). The conclusion should be drawn from gene analysis using hybridization.

Inheritance of field resistance

Field resistance is considered commonly to be of polygenic nature. That is based on the fact that offspring of a cross between the varieties with different levels of field resistance show a continuous variation in field resistance. Asaga and Yoshimura (1969, 1970a, 1971) made crosses between a variety with a moderate level of field resistance, Yamabiko, and susceptible varieties, Kusabue and Norin 29. The F_3 and F_4 lines were tested for field resistance in a blast nursery. Continuous variation of field resistance was observed among those lines from the level of Yamabiko to the level of Kusabue or Norin 29. Ito and Takakuwa (1965) also stated that offspring from the crosses of Norin 22 with susceptible varieties showed continuous variation from resistant to susceptible.

In some varieties, however, a major gene or genes were reported to govern the field resistance. The high level of field resistance found in some Japanese upland varieties appears to fall into such a category. Shinoda et al (1971a) reported that the high field resistance of an upland variety, Kuroka, was governed by more than three major genes, including complementary genes. Abe et al (1974, 1976) investigated the high field resistance of two upland varieties. They found that the resistance of Upland Rice Norin Mochi 4 was expressed by the additive effect of more than three minor genes or polygenes, and that of upland rice Norin Mochi 26 was expressed by the additive or supplementary effects of several minor genes, including two or three with relatively high function. Goto (1970) reported that the field resistance of Sensho was governed by three additive genes.

The most conspicuous instance of major-gene-dependent field resistance

was observed in Chugoku 31. That variety was found to produce only a few susceptible-type lesions under such conditions that susceptible varieties were killed (Ezuka et al 1969b). Toriyama et al (1968) demonstrated that the field resistance of Chugoku 31 was governed by a single dominant gene, which they named *Pi-f.* Chugoku 31 was later found to be attacked severely by certain strains of the blast fungus (Yunoki et al 1970b). That is the same phenomenon as the breakdonm of true resistance. It would be a most exceptional case of field resistance, which is highly specific to fungus strains.

In spite of those exceptions, Japanese investigators still believe that field resistance in most cases is governed by polygenes and is nonspecific to races or fungus strains; that is, it is horizontal. Specific field resistance, just because it was exceptional, was emphasized too much in the literature. That may have caused some misunderstanding among the investigators in other countries. However, the greater part of field resistance is horizontal—as the terms field or horizontal resistance are commonly understood in Japan.

EVALUATION OF BLAST RESISTANCE

For efficient breeding of resistant varieties, methods of screening for resistance are important. The evaluation of blast resistance in Japan consists of two procedures: evaluation of true resistance and evaluation of field resistance. To prevent breakdown, the two types of resistance should be evaluated separately.

True resistance

To determine the genotype for true resistance of a given variety, laborious genetic study, including hybridization, is necessary. Such study is difficult in routine breeding work.

Yamasaki and Kiyosawa (1966) developed a rapid method for estimating the probable genotype for true resistance. Rice seedlings at the 3.2- to 4.3-leaf stage are inoculated by injection with seven representative fungus strains. The reaction to each fungus strain is evaluated as R^h, R, MR, M, MS, or S, according to the size and number of developed lesions. Then the pattern of reaction to seven fungus strains is compared with that of known genotypes. According to Kiyosawa (1972a), 16 groups can be distinguished by this method (Table 3). He (1973) classified 397 rice varieties into 12 groups. The method is now most commonly used by Japanese breeders to estimate the probable genotype for true resistance.

Ezuka et al (1969a) also tested 373 rice varieties for true resistance, and pointed out that a few genotypes cannot be distinguished by the Yamasaki and Kiyosawa (1966) method alone. Ezuka and his associates employed some other fungus strains and spray inoculation for some varieties. As a result, the Kanto 51 type was divided into three genotypes, *Pi-k, Pi-i, Pi-k,* and *Pi-k Pi-m;* To-to type into three, *Pi-a Pi-k, Pi-a Pi-i Pi-k,* and *Pi-a Pi-k Pi-m;* and Pi 4 type into *Pi-ta*^{2} and *Pi-a Pi-ta*^{2}.

Table 3. Reaction patterns of rice varieties to seven differential fungus strains inoculated by the injection method (Kiyosawa 1972).

Varietal group	Genotype of representative variety	Reaction[a] to fungus strain						
		P-2b	Ken 53-33	Ina 72	Hoku 1	Ken 54-20	Ken 54-04	Ina 168
Shin 2	*Pi-k*^{s}	S	S	S	S	S	MS	S
Aichi Asahi	*Pi-a*	S	S	R	S	S	MS	R
Kanto 51	*Pi-k*	MR	S	S	R^{h}	R^{h}	R^{h}	R^{h}
Ishikari Shiroke	*Pi-i*	M	S	M	S	MS	MR	M
Yashiro-mochi	*Pi-ta*	S	S	M	MR	M	MR	S
Pi 4	*Pi-ta*^{2}	S	M	R^{h}	R	R	R	MR
Fukunishiki	*Pi-z*	M	M	M	MR	M	MR	M
Toride1	*Piz*^{t}	R^{h}	R^{h}	R^{h}	R^{h}	R^{h}	R^{h}	R^{h}
To-to	*Pi-k Pi-a*	MR	S	R	R^{h}	R^{h}	R^{h}	R^{h}
Shinsetsu	*Pi-i Pi-a*	M	S	R	S	MS	MR	R
Shimokita	*Pi-ta Pi-a*	S	S	R	MR	M	MR	R
Zenith	*Pi-z Pi-a*	M	M	R	MR	M	MR	R
K 2	*Pi-k*^{p} *Pi-a*	S	S	R	R	R	R	R
K 3	*Pi-k*^{h}	MR	S	S	R	R	R	R
BL 8	*Pi-b*	MR	M	MR	MR	M	MR	MR
K 59	*Pi-t*	M	MR	M	M	MS	M	MS

[a]S = susceptible, R = resistant, MR = moderately resistant. R^{h} = highly resistant, MS = moderately susceptible.

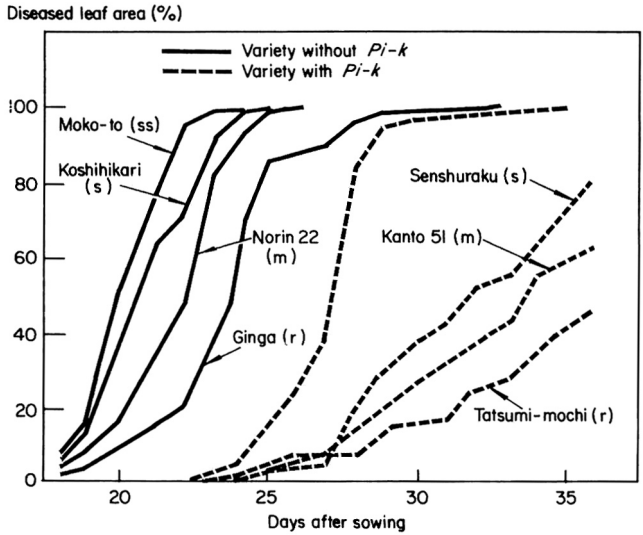
Yamada (1973) attempted to determine the genotypes by spray inoculation of the seven differential fungus strains of Kiyosawa and four other fungus strains. He classified a number of rice varieties into eight genotypes; he divided To-to type into three genotypes, *Pi-a Pi-k, Pi-i Pi-k,* and *Pi-a Pi-i Pi-k.* Several other classification systems of rice varieties using spray inoculation were presented by other investigators. The systems as reviewed by Kiyosawa (1974), were somewhat different from each other in the way of dividing varietal groups, but not essentially.

Through those investigations, most of the commercial varieties and important domestic varieties in Japan have been tested for their true-resistance genotypes.

Field resistance

To evaluate field resistance, it is necessary to exclude the effect of true resistance. There are many races in nature. Some may attack the test variety and others may not. Moreover, some varieties may be attacked by a race that does not attack others. Under such complicated conditions, field resistance cannot be evaluated, because both true resistance and field resistance may be included in the resistance observed in the field (Ezuka 1972).

Figure 2 shows an example of varietal differences in disease progress in a blast nursery. Four varieties (solid lines) do not have true-resistance genes, while the other four (broken lines) have the *Pi-k* gene. Disease occurred about a week later on the varieties with the *Pi-k* gene than on those without *Pi-k.* That is due to the prevalence of fungus races avirulent to *Pi-k* at the initial



2. The progress of leaf blast on some rice varieties with and without *Pi-k* gene in the blast nursery (Ezuka 1972). ss = highly susceptible, s = susceptible, m = intermediate, r = resistant—in the sense of field resistance.

stage of the disease. The difference between the two groups is ascribed to a difference in true resistance. On the other hand, differences between varieties in the same group are considered to be differences in field resistance. It is quite clear that the varietal groups should not be treated together in evaluating field resistance.

Ezuka et al (1969a, b) proposed two methods for excluding the effect of true resistance. One is to confine the comparison of resistance within the varietal groups of the same true-resistance genotype. The other is to make an artificial inoculation with a certain fungus strain that is virulent to all the varieties under the test. The former method is useful for tests in the open field or in the blast nursery, while the latter is adequate for tests in the glasshouse free from natural infection. In either case, it is essential to know the true-resistance genotype of the test varieties before evaluating field resistance.

The blast nursery is now most commonly used in Japan for evaluating field resistance. It has been used in breeding work for a long time, but true resistance and field resistance were not distinguished consciously by the early breeders. Narita and Iwata (1964a, b) were probably the first to distinguish field resistance from true resistance in the blast nursery. They tested 142, rice varieties for the true-resistance reaction to several fungus races, and classified them into seven groups. Of those varieties, 63 were tested repeatedly for resistance in the blast nursery, subjected to natural infection. Remarkable differences were observed in disease severity among the varieties within every group. Narita and Iwata

(1964b) supposed the difference to be due to field resistance.

Ezuka et al (1969b) tested some representative varieties of each true-resistance genotype for resistance in the blast nursery. The tests were repeated six times. Disease severity was found to differ widely even within varieties of the same true-resistance genotype. The differences in field resistance were more distinct when the disease developed slowly and became prevalent after the seventh-leaf stage than in the case of early occurrence. The same varieties were also tested for field resistance in paddy fields in the tillering-to-booting stage. The results were very similar to those obtained in the blast nurseries. Ezuka et al (1969b) then tested 412 rice varieties for field resistance in the blast nursery. The test varieties were grown in groups according to their true-resistance genotypes, and a susceptible check variety for each group of the same genotype was inserted at every fifth row. Disease severity was compared with that of the check variety in each group, and classified into five grades, rr (highly resistant), r (resistant), m (intermediate), s (susceptible). and ss (highly susceptible). Part of the results are presented in Table 4. Similar tests, in the blast nursery, were also conducted for field resistance of many varieties by Iwata et al (1971) and several other investigators (Kozaka et al 1972 : Agriculture, Forestry, and Fisheries Research Council 1973).

As a result of those experiments, Japanese breeders and plant pathologists accepted generally that field resistance could be evaluated by the blast nursery method.

Niizeki (1967) tried to evaluate field resistance in the glasshouse by seedling inoculation. He used a fungus strain virulent to all the test varieties and graded the field resistance on the basis of the ratio of the number of susceptible-type lesions to the total number of lesions. Yunoki et al (1970a) examined various factors affecting the evaluation of field resistance in the glasshouse. They recommended the following procedure as adequate for routine work: 1) Select a fungus strain virulent to all test varieties : 2) Grow seedlings with an excessive nitrogen supply; 3) Adjust the concentration of the inoculum to $10\text{-}25 \times 10^4$ spores/ml; 4) Inoculate the seedlings at the seventh-leaf-stage by spraying, and 5) Finally, evaluate the field resistance by the number of susceptible-type lesions that result. The result obtained was in fairly good agreement with that obtained by Ezuka et al (1969b) under field conditions.

Kiyosawa (1966b) used injection instead of spraying to evaluate field resistance in the glasshouse. He used a weakly aggressive fungus strain. Ken 54-04, for the inoculation and examined the ratio of the number of susceptible-type lesions to the total number of lesions. He considered the resistance thus evaluated to be field resistance, because it showed fairly high correlation with the field resistance evaluated under field conditions.

Those studies made it possible, to some extent, to evaluate field resistance in the glasshouse. However, it should be noted that the resistance evaluated by only one artificial inoculation can not represent the entire field resistance seen under field conditions (Kiyosawa 1970; Ezuka et al 1969b; Yunoki et al 1970a).

Table 4. Results of tests for field resistance of rice varieties to leaf blast in upland blast nursery (Ezuka 1972).

Genotype for true resistance	Grade of field resistance[a]	Variety
+	rr	Chiyohikari, Ou 247, San-in 63 Sensho, Fukuton, St 1
	r	Noun 22, Hatsunishiki, Honen Wase, Shin 2, Koshiji Wase, Ginga
	m	Norin 1, Koganenami, Yaeho Shin Yamabuki, Iwai-mochi
	s	Norin 6, Norin 8, Manryo, Tosan 38, Kinki 33
	ss	Norin 29, Norin 48, Koshihikari Kaho, Moko-to
Pi-a	rr	Heiroku-mochi, Upland Rice Norin 1, Shin Hakaburi, Kuroka
	r	Gin-masari, Yamabiko, Fujiminori, Reimei, Mutsu-kogane, Futaba, Homare Nishiki
	m	Towada, Fukuminori, Hamayu, Shuho, Senbon-masari
	s	Hoyoku, Kokumasari, Kochikare, Senbon Asahi, Chusei Shin Senbon
	ss	Norin 17, Norin 18, Mihonishiki, Sasashigure, Aichi Asahi, Jukkoku
Pi-i	rr	Yoneshiro, Isao-mochi, Kuiku 9, Joiku 232
	r	Norin 34, Hamaminori, Ishikari Shiroke, Fujisaka 5, Shinanohikari
	m	Asashio
	ss	Fukuyuki, Gohyakumangoku
Pi-a Pi-i	r	Shinsetsu, Naruho
	m	Miyoshi, Takane Nishiki
	s	Yamahibiki
	ss	Sawaminori
Pi-k	rr	Chugoku 31
	r	San-in 68, Tatsumi-mochi
	m	Ugonishiki, Kanto 51, Mangetsu-mochi
	s	Senshuraku, Koshiminori, Dewano-mochi, Hatsuiwai-mochi
	ss	Kusabue, Kanto 53, Tachihonami
Pi-k Pi-m	m	Etsunan 70
	s	Tsuyuake, Etsunan 24, Etsunan 49
Pi-a Pi-k	rr	Hokkai 184
	m	Sakaki-mochi
	s	Teine, Ou 248, Koshihibiki, Kayura-mochi, Tsukimi-mochi
	ss	Yuukara, Oyodo, Hokkai 183
Pi-a Pi-k Pi-m	r	Hokushin 1, BR 1
	m	Sanpuku, Takara Senbon, Minehikari
	s	Etsunan 52, Suzukaze
	ss	Etsunan 57
Pi-ta	s	Yashiro-mochi
	ss	Tosa Senbon
Pi-a Pi-ta	m	Shimokita, Tohoku 92
	ss	Tohoku 96, Pi 1, Pi 2
Pi-ta^2	m	Akiji, Saikai 104, Pi 3
	s	Satominori, Nankai 39, Chugoku 43, Pi 4, Pi 5, Asahikari
Pi-a Pi-ta^2	m	Kansai 13
	s	Kansai 14, San-in 77, San-in 79
Pi-z	rr	Ou 244, 54BC-68
	m	Fukunishiki, Ou 243
Pi-a Pi-z	s	Fukei 67
	ss	Fukei 73

[a]rr = highly resistant, r = resistant, m = intermediate, s = susceptible, ss = highly susceptible

Field resistance to panicle blast remains difficult to evaluate. That is due mainly to differences in heading dates among varieties tested. Suzuki and Yamada (1969, 1970) developed a method for evaluating field resistance to panicle blast by the use of regrowing panicles of cut-back plants. The differences in full-heading dates of 40 tested varieties were decreased from 45 days in the untreated plants to 10 days in the regrown plants, so that artificial inoculation could be performed simultaneously. The correlation between field resistance to panicle blast, and that to leaf blast was not always distinct. Asaga and Yoshimura (1970b) also reported that field resistance to panicle blast did not always coincide with that to leaf blast in their experiment.

STRATEGY FOR FUTURE BREEDING FOR BLAST RESISTANCE

To overcome difficulties caused by the breakdown of true resistance, many investigators have proposed various countermeasures, which are summarized as follows (Kiyosawa et al 1975): 1) utilization of field resistance, 2) incorporation of field resistance with true resistance, 3) mixed cultivation of varieties with different true resistances (multiline), 4) rotation of varieties with different true resistances, and 5) accumulation of many true-resistance genes in a single variety.

Use of field resistance

The use of field resistance is the simplest but most reasonable method for avoiding the breakdown, because field resistance in most cases is considered nonspecific to fungus races; that is, it is horizontal.

There are, however, several reports suggesting that the field resistance in some rice varieties varies more or less with the fungus strains. Toriyama (1975) gave a review of those reports and stated that field resistance was not equivalent to horizontal resistance. The most conspicuous evidence was the breakdown of field resistance in Chugoku 31 (Yunoki et al 1970b). The resistance of that variety is governed by a single major gene, *Pi-f,* as mentioned previously. That fact suggests that major-gene resistance is liable to breakdown even though its expression is quantitative. The resistance that depends on only one resistance gene can be broken down by only one chance of mutation in the causal fungus. The high level of field resistance in some Japanese upland varieties also appeared to be governed by relatively few major or submajor genes. The breakdown of those upland varieties has not yet been reported under field conditions, but its possibility is not negligible. Sekiguchi (unpubl.) found that a few isolates severely attack some highly resistant upland varieties such as Kuroka and Fukuton.

The moderate level of field resistance is generally accepted as race nonspecific, that is, horizontal. That type of resistance is considered to be governed by a polygene system, and that is why the resistance is hardly influenced by the shift of races or mutation of the fungi. Some exceptional instances of

variable field resistance were reported even in those varieties by Yunoki et al (1970b) and the Central Agricultural Experiment Station (1970). However, the data are not sufficient to determine the degree of variability under such circumstances. Many other reports support the nonspecificity of field resistance. A number of commercial varieties with field resistance have been cultivated in farmers' fields for many years with good results. It can be said that the polygenic field resistance is far more stable against shifting fungus races than is true resistance.

Breeding for polygenic resistance, however, is more difficult than breeding for major-gene resistance. There are usually more conflicts with yield, quality, and other traits. Moreover, polygenic resistance is lower than major-gene resistance. It is desirable to develop varieties with polygenic resistance that is as high as possible, to serve as intermediate breeding materials. The polygenic systems involved in different varieties may not be the same. Multiple crossing among them may be able to produce higher polygenic resistance than any one of the parental varieties. Such fundamental breeding work would be worth testing (Ezuka 1977).

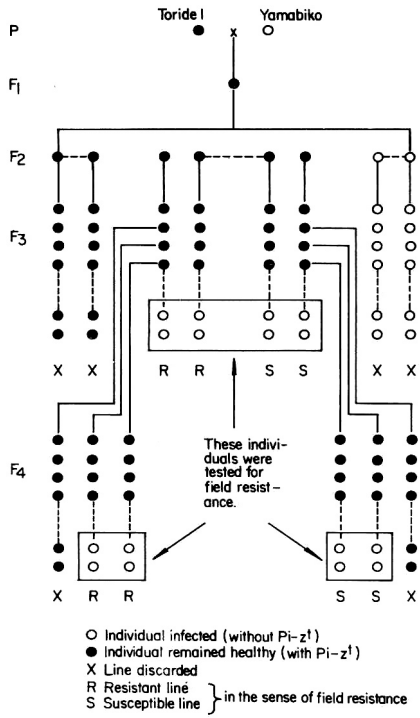
Incorporation of field resistance with true resistance

The severe breakdown of true resistance observed in the varieties Kusabue, Yuukara, Pi 5, and others is undoubtedly caused by the lack of field resistance that results from the 'Vertifolia effect.' True resistance itself appears to have no or little effect on field resistance (Asaga and Yoshimura 1969). If a useful level of field resistance is incorporated with true resistance into a variety, that variety will be more stable in its resistance than the varieties possessing true or field resistance separately.

A dificulty arises, however, in that the field resistance, when combined with true resistance, cannot be tested unless the race or races matching the true resistance are obtained. The more useful the true resistance is, the more difficult it is to obtain the matching races.

There are two ways to solve this difficulty (Kiyosawa et al 1975). One is to select a fungus mutant matching the true resistance, and use it for the inoculation in the glasshouse. In such a case, the mutant should be used very carefully to prevent its escape into the open field.

The other method is an indirect evaluation method developed by Asaga and Higashi (1973). They attempted to incorporate the field resistance of Yamabiko with the true resistance of Toride 1 by crossing both varieties. Yamabiko has *Pi-a* alone as a true-resistance gene and is infected by most of the fungus races in Japan, but has rather high field resistance. Toride 1 has the true resistance gene *Pi-z*^{t} introduced from an Indian variety, TKM 1, which is operative against all the fungus races in Japan. As shown in Figure 3, Asaga and Higashi picked out individuals without *Pi-z* ^{t} from the F_3 lines that segregated according to the resistance due to *Pi-z* ^{t}, and tested them for field resistance by the ordinary method in the glasshouse or in the field. From the results, they estimated the



3. Procedure for evaluating the field resistance of the progenies from the cross between Toride 1 and Yamabiko (Asaga and Higashi 1973).

field resistance of the remainder of the same lines, that is, individuals with *Pi-z*^{t}. They tested the F_4 plants again by the same method. They developed eight lines that combined the true resistance gene *Pi-z*^{t} from Toride 1 with field resistance as high as that of Yamabiko.

Mixed cultivation of varieties with different true resistances (multiline)

Mixed cultivation for the control of rusts of wheat, oats, and sunflower is already in practical use in the United States. Okabe (1967) reviewed those results and suggested the possibility of introducing the method into rice breeding. By mathematical analysis Kiyosawa and Shiyomi (1972) showed the effectiveness of multilines. Recently, Shindo (unpubl.) demonstrated that the mixed cultivation of three rice varieties with different true-resistance genotypes gave a remarkably lower blast occurrence than did monocultivation of each variety (Table 5).

However, it is not easy for rice breeders to develop a series of near-isogenic lines to serve as a multiline. Moreover, it is possible to produce a so-called super-race that matches all the true-resistance genes involved in the multiline system.

Table 5. Effect of mixed cultivation of varieties with different true resistances to blast (Shindo unpubl.).

Exp. no.	Variety	True resistance genotype	S-type lesions (no./hill)	Panicle infection index	Yield (kg/3.3 m^2)
I	Monocultivation				
	Fukei 69	*Pi-k*	12.25	8.68	1.58
	Tohoku 108	*Pi-a Pi-i*	25.75	23.78	1.58
	Toyonishiki	*Pi-a*	7.25	4.08	1.95
	Average		15.08	12.18	1.70
	Mixed cultivation of 3 varieties		2.25	1.68	1.98
II	Monocultivation				
	Bikei 91	*Pi-k*	0	0.03	1.83
	Miyoshi	*Pi-a Pi-i*	3.25	6.18	1.75
	Norin 41	*Pi-a*	15.50	23.10	1.75
	Average		6.25	9.97	1.80
	Mixed cultivation of 3 varieties		1.75	3.45	1.83

If all the available genes were exhausted by the appearance of such superraces, multilines could no longer be effective. Kiyosawa et al (1975) suggested that such a possibility may be avoided if the stabilizing selection described by Van der Plank (1963, 1968) exists in the rice varieties-blast fungus races relationship. Further studies will be required to determine that aspect.

Rotation of varieties with different true resistances

Rotation of varieties is characterized by the replacement of one variety with another before, or just after, the true resistance of the first variety is broken down. A series of varieties with different true resistances is necessary, but they do not always need to be near-isogenic. If a useful level of field resistance is combined with the true resistance in each variety, the longevity of each variety will be extended. Such a method seems likely to be more acceptable than the multiline system from the viewpoint of breeding technique, but creates difficulties in timely supply of seeds of the new varieties.

Moreover, the possibility of producing a superrace cannot be neglected. If no stabilizing pressure operates, virulent genes will be accumulated step by step in the fungus by replacing one variety with another. After one cycle of rotation, a superrace that is virulent to all the varieties of the series may occur (Ezuka 1977). The effectiveness of the method, therefore, depends on the stabilizing selection, as pointed out by Kiyosawa et al (1975).

Legislative control will be needed for effective utilization of the method.

Accumulation of many true-resistance genes in a single variety

Developing a supervariety that is resistant to all or most of the fungus races existent is accompanied by not a few technical difficulties. The hardest problem

is to identify the resistance genes accumulated in the hybrids. Ezuka (1977) discussed the matter and concluded that the more true-resistance genes that might be accumulated, the fewer fungus strains could be available for the identification of the accumulated genes. According to him, no fungus isolate is now available in Japan to distinguish the plant possessing four genes, *Pi-a, Pi-k, Pi-i,* and *Pi-z,* from the plants possessing three genes, *Pi-a, Pi-k,* and *Pi-z.* He also suggested the possibility that the superrace may be produced by the cultivation of a supervariety. Kiyosawa et al (1975) pointed out the importance of stabilizing selection also in this case.

Recently, Ou (1972, 1975) presented a new and noteworthy theory about the stable resistance in Tetep, Carreon, and some other varieties with a broad spectrum of resistance to races. His theory is based on the extraordinary high variability of the races of blast fungus. The selection pressure does not work in that case. The more vertical resistance genes that are accumulated, the higher is the stable resistance to be expected. The stable resistance is similar to the horizontal resistance in appearance. According to such a theory, breeders do not have to identify the accumulated major genes one by one. They only need to make as many crosses as possible among highly resistant varieties, and to select hybrids showing resistance of as high a level as possible. The higher the resistance in the fields, the greater is the number of major genes that are expected to be accumulated. Such plants must have broad spectrums of resistance to the races and produce only a few lesions even if matching superraces appear. That will be a labor-saving and convenient process for the breeders. However, it still is not known whether the variability of races is usually as high as reported by Ou (1972, 1975), whose view at present is not widely supported in Japan. The variability itself, however, has been reported (Goto et al 1964; Kiyosawa 1966a, 1976) or actually experienced by many Japanese investigators. Only the degree and frequency of the variation need to be determined.

Policy for future breeding

As mentioned above, various methods have been proposed for future breeding for blast resistance. Some are already in practical use. Others are under experimental or mathematical analysis (Kiyosawa and Shiyomi 1972: Kiyosawa 1972b). The utilization of field resistance and its incorporation with true resistance is probably the most practicable method for the urgent requirement (Ezuka 1972, 1977). However, it is not easy to establish a comprehensive strategy to solve all the difficulties we now face.

Robinson (1971) formulated 14 rules to assess the value of vertical resistance. In terms of such rules, rice blast is a compound-interest disease (rule 2) caused by a pathogen with a high vertical mutability (rule 3). The host population is genetically uniform and tends to be grown in large areas of a single cultivar (rule 4). Vertical resistance sometimes confers incomplete protection from neck and panicle blast (rule 10). Legislative control is not always easy (rule 12). Those factors may reduce the value of vertical resistance, but it can be more

valuable when reinforced with a useful level of horizontal resistance (rule 13). Whether stabilizing pressure can be exploited is not known (rule 5), nor is it known whether a high vertical resistance is correlated with a low horizontal pathogenicity (rule 14). Those aspects will be the most important problems to be solved in future studies.

Based on present knowledge of the host-parasite relationship in rice blast disease. vertical resistance alone cannot provide reliable control measures. Horizontal resistance alone, however, may not always be sufficient to control the disease in agriculture. Under such circumstances. Robinson (1973) suggested, a sound breeding policy would be to aim at horizontal resistance first. If adequate disease control is not achieved, horizontal resistance could then be reinforced by vertical resistance. That view is applicable to the present status of breeding for rice blast resistance in Japan.

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BLAST RESISTANCE IN INDIA

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BLAST DISEASE OF RICE occurs seasonally in the plains of India, but it is endemic in the hilly regions. It is the most important fungal disease of rice. Since the introduction of high yielding varieties of rice, attention has been directed toward bacterial blight, to which all the high yielding varieties are susceptible. Only a few varieties are susceptible to blast. Ratna and Pusa 2–21, two early maturing varieties that are widely accepted, are susceptible to blast. When grown in the kharif season, however, they are harvested at the beginning of October and therefore escape blast in most parts of India. In any event, the program of breeding for blast resistance is receiving due attention in India. Limited progress has been made in breeding varieties resistant to blast disease in some principal rice-growing states of India (Parthasarathy and Padmanabhan 1958: Padmanabhan et al 1960, 1964).

ISOLATION OF SOURCES OF RESISTANCE

As early as 1924, Co 4 was selected as resistant to blast at Madras; later TKM 1 was recognized as resistant. A procedure for raising seedlings for artificial infection tests, recording infection, and classifying varieties on the basis of infection scores was standardized in India in 1948 and 1949. It was used to screen the genetic stocks of rice varieties at the Central Rice Research Institute (CRRI). Cuttack (Padmanabhan and Ganguly 1959: Padmanabhan et al 1964; Veeraraghavan 1967).

The varieties found resistant at Cuttack were studied for their reaction at CRRI testing centers in different states ot India from 1954 to 1958 and 1960 to 1962 (Table 1).

S67 was resistant at all the centers: BJ 1 was resistant at all centers except at Kanke.

Yield trials with promising resistance selections were carried out in collaboration with rice specialists: appropriate recommendations then were made to the

Central Rice Research Institute, Cuttack, India.

Table 1. Reaction of 15 rice varieties selected as resistant to blast at Cuttack and its 14 testing centers during 1960–62.

Variety	Reaction[a] at seedling stage													
	Cuttack	Deras	Bhagalpur	Kalimpong	Lomavala	Kashmir	Malda	Jeypore	Kanpur	Coimbatore	Kanke	Mangalore	Ponampet	Majhra
AC 28	R	R	X	S	X	R	R	R	X	R	R	X	R	R
AC 75	R	R	R	R	X	X	R	R	R	R	S	S	R	R
AC 533	R	R	R	R	S	R	R	R	S	R	S	S	R	R
AC 1425	R	R	S	R	S	R	R	MR	R	R	R	R	R	MS
AC 1613	R	R	X	S	S	R	R	R	R	R	S	R	R	R
AC 1730	R	R	R	R	S	X	R	R	R	S	S	R	R	R
AC 1773	R	R	MR	R	S	X	R	S	R	S	S	S	S	R
AC 2250 (Co 29)	R	R	S	R	R	S	R	R	R	S	S	S	R	R
AC 2489 (Nep-vai)	R	R	R	R	R	R	R	R	R	S	R	R	R	R
AC 1443	R	R	R	R	R	X	R	R	R	S	S	R	R	R
S67	R	R	MR	R	R	X	R	R	R	R	R	R	R	X
Hyb. Cul. 1 (CR 906)	R	R	R	R	S	X	R	R	R	S	S	R	R	X
Hyb. Cul. 2 (CR 907)	R	R	R	R	R	X	R	R	R	R	S	R	S	X
BJ 1	R	R	R	R	R	X	R	R	R	R	S	R	R	R
Co 13	S	S	S	S	S	S	S	S	S	S	S	S	S	X

[a] R = resistant, MS = moderately susceptible, X = not tested, MR = moderately resistant, S = susceptible

states to use the best resistant varieties either as donors in hybridization programs or as direct introductions.

Meanwhile, screening trials for blast resistance were in progress at various state research stations (Table 2).

UNIFORM BLAST NURSERIES

The need for uniform worldwide testing procedures to select for resistance was stressed by Dr. N. Parthasarathy at a meeting of the International Rice Commission (IRC) in Japan in 1954. As suggested by the Food and Agriculture Organization of the United Nations (FAO), information on the procedures being followed throughout the world was collected and presented at an IRC meeting in Malaya in 1966 (Parthasarathy and Padmanabhan 1958). Resistant and susceptible varieties were obtained from various countries and tested at eight principal testing centers; the results were reported to the FAO in 1959 (Richharia et al 1961). In 1959, the working party meeting of the IRC in Ceylon considered the test reports and suggested that uniform blast nursery trials be

Table 2. Rice varieties reported resistant to *Pyricularia oryzae* in India.

Varieties	Reference
Co 25, (Co 4 × Adt 10), Co 29 (Co 13 × Co 4) and Co 30 (GFB 24 × Co 4)	Abdus Samad (1961)
Adt 25, Co 29 (T 6522 = Co 4 × Co 13), Co 30 (Co 4 × GFB 24)	Abdus Samad and Shanmugasundaram (1960)
T 900, T 1992, T 1978, T 1824, T 365, T 92, T 2289, T 545, T 544, T 540, T 537, T 536, T 530, T 1206, T 448, T 1210, T 945, T 60, T 555, T 82, T 175, T 289, T 1267, T 1222, T 864, T 651, T 421, T 397, T 195, T 336, T 130, T 531	Chandra Mohan and Palaniswamy (1968)
Rm 3, R 6, HL 12, HL 13, UM 1, HM 9, HM 10, SI 4, PL 15 and PI 16	Dutta and Chufran (1966)
Sm 8, T 100, N 12, T 22A, T 36, H 755, Bj 1, T 3, CP 9	Mathur and Misra (1961)
AC 2250, Co 29, AC 2489 (*Nep-vai*), S 67, CR 907, (Co 25 × Co 13)	Mathur et al (1963)
Ninnidhan	Murti et al (1963)
M 42	Narasinga Rao et al (1956)
Akp 8, Akp 9, Mtu 5, As 2, Mugad 249, CR 6, CP 9, Adt 12, Co 25, Co 26, Ptb 10, Bhogjira 1, S 67, Co 4, Ch 55, S 624, Bam 4, H 755, SM 6, SM 8, SM 9	Padmanabhan and Ganguly (1959)
Ch 27, Ch 28, Ch 71, Jap 7, T 1446, T 1715, T 2009, Seluz 44, T 6522 (Co 29), AC 2489, *(Nep-vai).* T 1026, T 1160	Padmanabhan et al (1964)
Fa Yiu Tsai, Malio 611, Modan Black, T 1453, Thekkan Cheera, Ptb 10, BC 17-1-3, Hbj Boro 8, BC 22-1-2, Hbj Boro 7, Khata Boro, Hbj Boro 4, Baahusali, Hbj Boro 6, Tepa Hbj, Boro 2, Cau 264, Ciong Chien 351, Tep Saigon 229, Tunsart, E 45, Karuthamodan, Ptb 29, T 137, Karivennal, T 33, Vary Lava 16, Vary Lava 90, Ca 902/b/2/2, Ca 902/b/3/3, Carolina 164, Cristal 161, Tunsart, Camoni, Carreon	Veeraraghavan (1967)
S 67, S 139, S 246, S 661, S 1053, Ch 45, Ch 47, H 55, H 320, C 113, B 1399	Venkata Krishnaiah and Delvi (1960a)

conducted. A committee consisting of Dr. L. H. Fernando (Ceylon), Dr. H. Okomoto (Japan), and Dr. S. Y. Padmanabhan (India) was appointed to prepare a draft recommendation. The draft was approved at the 1962 IRC meeting at Delhi. In the International Blast Symposium in Manila in 1963, the blast scoring system was simplified. Uniform blast nursery trials are now being conducted in 26 countries.

International entries

Since 1962 several sets of varieties assembled by the International Rice Research Institute (IRRI) have been tested in India at various sites. A number have emerged as resistant to moderately resistant to blast (Table 3).

Table 3. Rice varieties that emerged as resistant to moderately resistant in the International Rice Blast Nursery trials conducted from 1962 to 1975.

Year	Tests (no.)	Testing centers (no.)	Entries tested (no.)	Entries emerged as R-MR (no.)	R-MR entries (no.)	
					Indian	Other
1962–69	27	15	256	20	6 (Ptb 10, Cr 906, Cr 907, Co 25, S 67, Co 4)	14
1968–70	7	7	321	8	3 (Kasipichodi Basmati 370, Jampada)	5
1973	11	11	357	35	2 (Kasipichodi Basmati 370)	33
1974	6	6	362	18	3 (Kasipichodi, Np 130, Manoharsali)	15
1975	5	5	463	62	0	62

CRRI genetic stocks

The uniform blast nursery technique has been used in India since 1962 to screen the CRRI genetic stocks. The catalog of germplasm collection, published by CRRI (1971) covered 5,728 varieties, whose reactions to blast on the 0–9 scale, were listed. In addition, the results of tests of the entries against blast, helminthosporiose, stem rot, bacterial leaf blight, and bacterial streak have been presented in a CRRI bulletin, *Assessment of germplasm for resistance against major cereal diseases—rice.*

A total of 4,863 entries from the Assam Rice Collection were tested; only 62 were found resistant to blast.

Of the 1,465 varieties from the Jeypore Botanical Collection that were tested, only 5 entries (JBS 21, 41, 974, 1462, and 1637) were found resistant to blast.

The reactions of high yielding varieties were evaluated in multilocation tests in India (Table 4).

In breeding for resistance, India appears to be fortunate, as it has many good donors available. The stability of reaction to blast of Tetep, Tadukan, and other varieties is particularly helpful. Their resistance is being incorporated into the high yielding varieties.

PHYSIOLOGIC SPECIALIZATION IN *PYRICULARIA ORYZAE* CAV.

Padmanabhan (1954) tested five isolates of the fungus *Pyricularia oryzae* Cav.—two from Cuttack and one each from the Indian Agricultural Research Institute (New Delhi), Coimbatore, and Wynad (now Kera1a)—on nine rice varieties, but obtained no evidence of specialization in pathogenicity of the isolates.

Table 4. Overall reaction of high yielding rice cultivars to blast at various test locations in India during 1968–1971.[a]

Cultivar	Kalimpong	CRRI (Cuttack)	Raipur	Faizabad	Majhera	AICRIP (Hyderabad)	Anakapalli	Nellore	Hebbal	Ponnampet	Moncompu	Pattambi	Ratnagiri	Sirsi	Coimbatore	Aduthurai
IR8	3	2	3	–	–	3	1	4	–	6	–	–	–	–	–	–
Java	4	5	2	2	2	5	4	4	3	5	1	4	1	2	3	3
Padma	4	5	4	2	2	4	6	4	4	7	1	3	–	5	2	3
Cauvery	6	5	6	2	6	6	5	3	2	–	3	4	2	5	1	5
Sabarmati	6	1	5	–	–	3	5	5	–	6	–	–	–	–	2	3
Jamuna	6	3	2	–	–	3	5	4	–	6	–	–	–	–	–	3
TN1	–	4	–	–	–	6	4	2	–	7	–	(4)	–	–	1	4
Jagannath	–	5	7	–	2	7	7	7	–	7	–	(4)	1	–	1	6
Kanchi		4	2	–	6	6	5	4	–	6	–	(4)	1	3	3	2
Bala	–	4	4	–	–	5	7	6	–	7	–	(4)	1	–	2	2
Karuna	–	6	–	1	2	4	7	2	2	–	2	3	2	5	1	2
Krishna	–	6	4	–	5	6	3	4	–	7	–	3	–	–	2	6
Ratna	–	4 (6)	7	3	2	4	7	2	4	6	2	5	2	3	4	6
IR20	–	6	–	5	2	7	5	4	5	5	2	2 (4)	4	3	4	2
Vijaya	–	2	–	4	–	–	–	2	3	7	2	4	–	2	–	–
Jayanti	–	2	4	3	2	4	5	2	4	3	1	5	2	2	3	3
Pankaj	–	1	2	–	3	6	5	2	–	5	–	(4)	–	–	1	4
Sona	–	6	6	–	–	–	4	2	–	7	–	–	–	–	–	–
Kannagi	–	(5)	–	5	7	7	5	5	7	–	2	7	3	3	2	7
IR22	–	6	–	3	6	5	3	2	6	–	2	6	1	3	1	4
Mashuri	–	4	1	1	3	7	6	2	–	–	–	(3)	2	–	1	3

[a]Source: AICRIP (All–India Coordinated Rice Improvement Project) Reports. 1–2 = resistant; 3–4 = moderate; 5–7 = susceptible

Kulkarni et al (1959) reported marked differences in three isolates of *P. oryzae* that were cultured on different media : apricot-juice, potato-saccharose, and potato-dextrose agars. The isolates fell into three physiologic forms.

The occurrence of physiologic races in *P. oryzae* in India was first reported by Latterell et al (1960), who used the US blast differentials and adopted three categories of host-reaction: resistant, moderate, and susceptible. I reported the occurrence of physiologic races of the pathogen from India (Padmanabhan 1965b) after studying isolates of *P. oryzae* representing the different regions of the country. I used the US blast differentials and adopted three categories of host reaction : resistant, moderately resistant, and susceptible. Chakrabarti et al (1966), and my associates and I (Padmanabhan et al 1967) reported the occurrence of 12 and 13 races after analyzing 17 and 19 isolates of *P. oryzae,* respectively, using US blast differentials and adopting resistance and susceptibility as criteria for distinguishing physiologic races. Most of the isolates reported by those two communications were the same.

My associates and I (Padmanabhan et al 1970) screened 132 isolates of *P. oryzae* collected from different parts of India during the period 1962–68, and reported the occurrence of 31 pathogenic races of *P. oryzae,* adopting the nomenclature of Atkins (1967) for races of *P. oryzae,* based on their pathogenicity on the international blast differentials Raminad Str. 3, Zenith, NP. 125, Usen, Dular, Kanto 51, CI.8970 (S), and Caloro, adopting resistance and susceptibility as criteria for distinguishing physiologic races. Venkata Rao and Appa Rao (1976) reported the occurrence of 17 races in Andhra Pradesh during 1968; 15 had already been reported; IA-110 and IA-117 were new reports. Padhi and Chakrabarti (1972) reported three new races of *P. oryzae* on the basis of isolates obtained during 1971. Veeraraghavan and Premalatha Dath (1975) reported the identification of two physiolosic races IC-1 and IC-17 (according to the Ling and Ou, 1969 classification) during 1972 after analyzing 48 isolates of *P. oryzae* collected during 1971 and 1972. Veeraraghavan and Premalatha Dath (1976) again analyzed 110 isolates prevalent during 1973 and 1974 in 19 states in India, and found that only the race IC-17 was prevalent. It was the predominant race among the 31 races reported by my associates and me (Padmanabhan et al 1970). The exclusive occurrence of a single race over a period of 2 years, 1973 and 1974 in the country, according to these authors, might indicate a host-pathogen equilibrium marked by the absence of reports of breakdown of resistance in any rice variety during that period. Further tests conducted during 1975 also confirmed the exclusive prevalence of the race IC-17 in India. Thus during the period from 1972 to 1975, all the 246 isolates of *P. oryzae* screened for their racial identity confirmed the exclusive prevalence of the race IC-17.

Veeraraghavan (1975a) has further confirmed the stability under a different method of storage of isolates of *P. oryzae* belonging to the race IC-17.

Veeraraghavan and Premalatha Dath (1975) have suggested the need for classifying C-type spots—circular spots about 2 or 3 mm in diameter with a central ashy zone and a dark, purplish-brown margin—as a susceptible reaction, since both C and D spots possess grey centers and brown margins, and reveal sporulation. That approach is being adopted currently. Furthermore, spots which fall between type C and type D do occur on certain of the differentials. Veeraraghavan and Premalatha Dath have suggested that the international blast differential variety Usen gave an ambiguous reaction and should be replaced by a variety with clear-cut reaction. Goto (1964) who are among the authors of the international blast differentials, supported the suggestion. They described the ambiguous reaction of Usen. Caloro, whose reaction was ambiguous, has been described as a narrow-lesion variety (Veeraraghavan 1975a). Therefore, that differential also needs replacement. Another differential variety that needs replacement is Raminad Str. 3, since it does not set seed or flower at high latitudes, creating a problem for seed production.

Veeraraghavan (1975b) suggested a new method of classification and nomenclature of physiologic races of *P. oryzae* Cav. The 256 possible patho-

genicity-patterns on the eight international blast differentials are sorted into 16 groups with the English letters A to P indicating the race group, and numbers indicating the physiologic races. Under this system of classification, all isolates of *P. oryzae* producing a susceptible reaction on all the international blast differentials have been categorized into one race group. Similarly, those which produce a resistant reaction on all the differentials have been accommodated in a single race group. Susceptibility or resistance of an individual differential or sets of differentials, taken in a serial order from the list of differentials, have been the criterion for classification. The method is being adopted currently.

In studies of the identification of physiologic races, it is suggested that the set of differential varieties be thoroughly purified to ensure homogeneity of each host variety in its reaction to the pathogen or to a specific isolate of the pathogen. The existence of variation among the individual plants of a variety in their reactions to the pathogen should be taken cognizance of, lest results be distorted and fail to reflect the variation in the pathogenicity as it exists in the pathogen.

While discussing the extent of variation in pathogen *P. oryzae,* it may be relevant to point out that, although most of the literature has attributed the variation in pathogenicity to heterokaryosis, recent critical studies by Yaegashi and Hebert (1976) confirm the findings of Giatgong and Frederiksen (1969) and Yamasaki and Niizeki (1965) that the pathogen is uninucleate and that mutation remains as the only source of variation. The perfect stage of the pathogen has not yet been detected.

HYBRIDIZATION

In hybridization projects to evolve resistant varieties, field techniques with bombardment rows of a susceptible variety were used. A series of hybrids with Co 4 as the donor parent for resistance have been released in Tamil Nadu. They include Co 25 (Co 4/Adt 10), a long-duration (150 days) variety; Co 29 (Co 4/Co 13), an early maturing (120 days) variety; and Co 30 (GEB 24/ Co 4). a medium-duration (145 days) variety (Abdus Samad and Shanmugasundaram 1960).

Two hybridization projects were also initiated at CRRI in 1954. The first cross was that between Co 25, (Co 4/Adt 10) resistant to blast, and Co 13, susceptible to blast. Two blast-resistant cultivars, CR 906 and CR 907, were evolved. They were of medium duration (135 to 140 days) and gave a very high (76 to 80%) hull recovery. The second cross was between Co 25, resistant to blast but susceptible to helminthosporiose, and Bam 10, resistant to helminthosporiose but susceptible to blast. Three cultivars resistant to both blast and helminthosporiose were evolved.

Hybridization projects with Tetep, Tadukan, and Zenith as donor parents for resistance have been initiated at CRRI. Among the varieties being improted are Supriya, IR8, Jagannath, and IR22. Segregating populations of crosses

in the F_2 and resistant selections in F_3 to F_5 were subjected to disease pressure and artificial inoculation. Fixed cultivars were distributed for testing in blast-endemic areas. A few promising blast-resistant cultivars with high yield potential have been identified. One cultivar from each of the crosses IR8/Tetep, IR8/Tadukan, and Supriya/Tetep has shown promise (CRRI). Two cultivars from cross CRK 30 (IR8/Sigadis) possess resistance to blast as well as high yield potential in kharif season (Mathur and Mishra 1976).

IR22, a selection from the cross made between IR8 and Tadukan at IRRI, was susceptible to blast at many sites in India. Crosses were therefore made at CRRI between IR22 and the blast-resistant parent Zenith in 1971. A few blast-resistant cultivars possessing the high yield and fine grain quality of IR22 have evolved.

Jagannath, a susceptible mutant of T 141, was crossed with Tetep and Zenith in 1968 and with Jayanti (CR 10-4103) in 1971 to evolve cultivars with the duration of Jagannath and possessing resistance to blast. The first two crosses failed to meet the goal. However, blast-resistant selections from the cross Jagannath/Jayanti have shown promise, and a few are similar to Jagannath in duration and yield (Mathur and Choupatnaik unpubl.).

GENETICS OF RESISTANCE

Various workers in India have studied the genetics of resistance in rice to blast fungus with many crosses. Their findings are summarized below:

Leaf blast

The Genes

1. Monogenic dominant (+ modifiers): Bhapkar and D'Cruz (1960). Veeraraghavan and Padmanabhan (1969).
2. Monogenic recessive: Ramiah and Ramaswamy (1936).
3. One dominant gene for each race of the pathogen: Venkataswamy (1963), Padmanabhan et al (1967), Rath and Padmanabhan (1972c).
4. Two dominant independent genes : Padmanabhan (1965a).
5. Two dominant complementary genes: Padmanabhan et al (1973).
6. Two dominant duplicate genes: Rath and Padmanabhan (1972c), Padmanabhan et al (1973).
7. Two recessive duplicate genes : Rath and Padmanabhan (1972c).
8. Three dominant genes (any two of them conferring resistance): Padmanabhan et al (1973).
9. Inhibitory gene : Rath and Padmanabhan (1972c), Padmanabhan et al (1973)
10. Cytoplasmic factors: Padmanabhan et al (1967), Rath and Padmanabhan (1972b), Padmanabhan et al (1973).

Penetration phase and establishment phase

1. Genetics of penetration, as estimated by the number of penetration points,

was conditioned by polygenes (Rath unpubl. data of 1970: Rath and Padmanabhan 1973).
2. Genetics of establishment phase by major genes (Rath and Padmanabhan 1973).

The above findings have been further confirmed by sheath-inoculation, which permits a detailed and critical examination of the different phases of infection of host cells by the pathogen (Kaur et al 1974). On the basis of the findings, it was suggested that polygenes govern the resistance in the penetration phase, whereas major genes govern resistance to the spread of infection in the host (Kaur and Padmanabhan 1977).

Influence of modifers on major gene resistance

Using sheath inoculation, very precise values can be used to describe the extent of development of infection, and the resistance to development has been found to be highly variable. In certain varieties, such as BJ 1 and S67, the reaction was between the extreme resistance of Tetep and Zenith and the extreme susceptible reaction of Co 13; it was closer to resistance than to susceptibility. That might be due to modifiers or to the operation of polygenic systems of resistance along with major genes. The reaction of the variety S67 in many locations in India also suggested that a search of genetic stocks for its type of resistance may be rewarding.

Field tolerance to rice blast

Resistance to blast that is governed by major genes remains unaffected by environmental factors, but is liable to breakdown when a more virulent or specialized pathogen appears. In contrast, the resistance governed by a large number of genes or by major genes with large numbers of modifiers imparts "field resistance."

At CRRI, the varieties in its genetic stocks and in the Jeypore Botanical collections, the Assam Rice Collections, and the International Rice Blast Nurser) (IRBN) have been screened in the upland nurseries for over 15 years at the high nitrogen level of 100 kg/ha.

In recording observations of foliar infection, the type and the number of leaf lesions are taken into consideration, as suggested by Padmanabhan and Ganguly (1959). Infection is also scored on a 0–7 scale, as suggested by Ou (1965a,b). Furthermore, the extent of infection in a plot is scored on a 1–3 scale.

A numbcr of varieties consistently showing an infection of IC, ID, and IE or 3–1, 4–1, and 5–1 possibly possess field tolerance to rice blast.

The following varieties from the earlier IRBN sets are likely to have field resistance: Bir-me-fen, Cesariet, Chokoto, Dudkuning, Fa-vui-Tsai, Chianung Yu-242, Mayan Sugumpal, Mayan Ebos, Nato, Pusa 5-3-1-2, Suwon-215, Cr 10, R-167, Pusur, DNJ-146, PAU 470, ARC 14928, ARC 15603, IR305-4-20-3-3, A 36-3, and Seriraja.

From the recent IRBN sets, the five cultivars IR22 F.1 (IR930-147-5)

Col 1, Taichung-Shi, 183, DNJ 60, DL 9, and DD 89 appear to have "field resistance." They have been tested under heavy natural infection in upland and transplanted crops and under artificial infection. Based on the types and number of spots and the spread of the infection, those varieties stood out as hopeful candidates for "field resistance." Further tests are in progress to confirm the findings.

Neck blast

Resistance to neck blast is governed by two or more genes, which may be independent of those of leaf blast (Padmanabhan 1965a); three pairs of dominant genes were the same as those giving foliar blast (Padmanabhan et al 1975).

GENE SYMBOLIZATION

Rath and Padmanabhan (1972) suggested a method of naming *Pyricularia*-resistant genes in which the designation of *Pyricularia* with *Pi* is retained, followed by the source of resistance indicated as a subscript, and the international race against which the gene is operating is shown after a hyphen. For example, a dominant resistant gene of Zenith operating against race IA-11 may be designated as *Piz-All.*

INDUCED MUTATIONS FOR DISEASE RESISTANCE IN RICE

Work was initiated at CRRI during 1965 to investigate the possibilities of using induced mutations in a breeding program for disease resistance. Seeds of the variety Taichung 65 were treated with Gamma-ray (10 KR) and ethyl methane sulphonate (1/300, 1/400, and 1/500). Treated seeds were screened rigorously to eliminate unhealthy seedlings, and the second and succeeding generations were subjected to heavy disease pressure. A few selected lines bred true for resistance in third and fourth generations, and it was possible to establish lines with resistance to the International races IR-4, IE-3, and IA-121 of *P. oryzae* Cav. (Nayak and Padmanabhan 1970).

An attempt was made to induce blast resistance (1973–76) in the high yielding variety Ratna (IR8/TKm 6) through chemomutagenesis with EMS 0.1 and 0.2% concentrations (Kaur et al 1975, 1978). The mutagen treatment induced great variability; in different generators M_2 to M_5, higher resistance and higher susceptibility were found than in the parent.

The proportion of resistant plants isolated from selections for resistance gradually increased in M_4 generations over the proportions in M_3 and M_2 generations. In the M_5 generation, few of the lines were found to be breeding true for disease resistance.

Mutagen treatment (EMS) also induced variability in grain characteristics. Ratna has long, slender grains. Mutations with medium and slender, long and bold, and short and bold grains have also been obtained. Induction of

resistance through mutation and selection appears to be a hopeful approach in breeding for blast resistance in rice, as few true-breeding resistant lines retaining the characters of Ratna could be isolated in the M_5 generation.

Gangadharan and Mathur (1976) obtained Mtu 17 mutants possessing desirable agronomic characters through chemomutagenesis with dES; some of the mutants had blast resistance, whereas the parent Mtu 17 was susceptible.

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BREEDING FOR BLAST RESISTANCE AT CIAT

M. J. ROSERO

THE BLAST DISEASE caused by the fungus *Pyricularia oryzae* Cav. is present in all Latin American countries where rice is grown. Although in some countries the disease has been considered of minor economic importance, it is now recognized as a major problem.

The use of resistant varieties is widely recognized as the most practical and economic method by which the farmer can control blast disease.

However, chemical control has been practiced in various countries of Latin America, using compounds like Blasticidine S, Hinosan, Kasumin, and Benlate. Although those chemicals have been used extensively and have helped to stabilize rice production in the eastern area of Colombia as well as in other areas of Latin American countries, breeding for genetic resistance is considered, in the Cooperative International Center tor Tropical Agriculture (CIAT)—Colombian Institute of Agriculture (ICA) Rice Program, to be the most profitable way of controlling blast.

The main objective of the CIAT-ICA Rice Program is to select superior high yielding varieties with resistance to blast. All crosses made since 1969 have been focused on attaining that objective.

BREEDING RESULTS

To accomplish the selection of superior genotypes with resistance to blast, a total of 1,163 crosses were made from 1969 to 1976. In 1969, 48 crosses were made among several promising advanced lines, to combine the good characteristics of sister lines of CICA 4 and IR22 with those of lines from the International Rice Research Institute (IRRI) crosses IR822 and IR532, which have blast resistance from Pankhari and Tadukan. From these crosses, six fixed lines were selected in 1972 and tested in regional trials in the main rice areas of Colombia (Table 1).

The blast reaction and yields of the six lines were compared with those of four commercial varieties. From those results, line A was selected and released

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Table 1. Blast infection and yields of three lines and three commercial rice varieties, obtained in regional trials, Colombia, 1972–1974.[a]

Line	Cross and pedigree	Leaf blast[b]	Neck blast (%)	Grain yeld (t/ha)
1	IR930-2 x IR822-432 P723-6-3-1	1–3	6.0	6.0
8	IR930-53 x IR579-160 P738-137-4-1	3–4	25.5	5.8
16	IR5—Selection	1–2	2.5	5.6
	CICA 4	3–5	32.5	5.5
	IR22	3–4	28.5	4.5
	IRE	3–5	30.6	5.1

[a] Data averaged from 15 regional trials conducted in farmers' fields in areas favorable for blast disease incidence. [b] 1–2 = resistant; 2.1-3 = moderately resistant; 3.1–4 = moderately susceptible; 4.1-7 = susceptible

Table 2. Reaction of blast-resistant sources in blast beds, Colombia 1970–1976.[a]

Variety	Tests (no.)		
	Total	Susceptible	Resistant
Tetep	25	0	25
C46-15	11	0	11
Colombia I	100	1	99
Dissi Hatif	8	1	7
Mamoriaka	7	2	5
Carreon	8	1	7

[a] Tests made at experimental stations of the Colombian Institute of Agriculture in Palmira and Villavicencio.

to farmers as CICA 6. It was released as a resistant variety. However, after one cycle of planting, it became susceptible not only in Colombia but in other countries of Latin America.

During 1970–1971, 154 crosses were made with the primary purpose of combining blast resistance with other desired traits already present in several promising lines from IRRI. The varieties Colombia I, Tetep, Dissi Hatif, Mamoriaka, and C46-15 were used as sources of blast resistance. They have shown broad-spectrum resistance in several tests (Table 2). From those crosses, 14 promising lines were selected in early 1975 and tested in 33 regional trials in the major rice areas of Colombia. Six lines were selected from the 14, on the basis of yields, apparent blast resistance, and other desirable traits. Listed in Table 3 are the crosses, their pedigrees, and their sources of resistance to blast. Table 4 compares the reaction to blast and yields obtained in irrigated regional trials in Colombia under high disease pressure. The data represent an average of 11 plantings in 1975 and 2 in 1976. All the lines were tolerant of

Table 3. Six promising blast-resistant rice lines selected by the CIAT-ICA[a] Rice Program in 1976.

Line	Cross and pedigree	Source of blast resistance
4421	IR665-23-3-1//F_1 (IR841-63-5-104-1B/C46-15) P901-22-11-2-6-1-1B	C46-15
4422	IR665-23-3-1//F_1 (IR841-63-5-104-1B/C46-15) P901-22-11-5-3-2-1B	C46-15
4440	CICA 4//F_1 (IR665-23-3-1/Tetep) P918-25-1-4-2-3-1B	Tetep
4444	CICA 4//F_1 (IR665-23-3-1/Tetep) P918-25-15-2-3-2-1B	Tetep
4461	IR22//F_1 (IR930-147-8/Col 1) P881-19-24-12-1B-6-1B	Colombia 1
4462	IR22//F_1 (IR930-147-8/Col 1) P881-19-24-12-1B-7-1B	Colombia 1

[a]International Center for Tropical Agriculture-Colombian Institute of Agriculture

Table 4. Blast incidence and yields of six promisIng rice lines and four commercial varieties observed in CIAT-ICA[a] Rice Program regional trials in irrigated areas, Colombia.[b]

Line or variety	Leaf blast[c]		Neck blast (%)		Grain yield (t/ha)	
	1975	1976	1975	1976	1975	1976
4421	0.1	3.0	6.0	10.0	5.5	4.7
4422	0.3	2.0	13.0	30.0	5.7	3.6
4440	1.0	2.0	4.0	6.0	6.0	5.8
4444	0.4	0.0	5.5	6.5	5.7	5.5
4461	1.0	4.0	3.5	30.0	4.3	3.3
4462	1.0	2.0	5.0	30.0	4.8	3.7
CICA 4	4.4	5.5	61.5	38.5	4.3	4.1
CICA 6	3.6	5.0	49.5	47.5	3.7	3.5
IR8	3.9	4.0	52.2	50.0	3.5	3.5
IR22	4.1	5.5	45.0	40.0	3.9	3.8

[a]International Center for Tropical Agriculture-Colombian Institute of Agriculture [b]Data averaged from 11 regional trials in 1975 and 2 trials in 1976; all made in areas with high incidence of blast [c]At booting stage in all regional trials; 1-2 = resistant 2.1-3 = moderately resistant, 3.1-4 = moderately susceptible. 4.1-7 = susceptible.

blast in 1975 and the commercial varieties were susceptible. In 1976, lines 4421, 4440, and 4444 continued to be tolerant. But lines 4422, 4461, and 4462 were infected (30%) with neck rot. That probably indicates the failure of the three lines to inherit all the genetic components of their resistant source, or it may reveal the presence of a new biotype of the pathogen that affected the lines.

On the basis of the 1975 results and the results from 23 regional trials in the main rice areas of Colombia, lines 4421 and 4461 were selected to be named as varieties by CIAT and ICA rice technicians in May 1976. Line 4421 was named CICA 9 and line 4461, CICA 7.

The rice technicians released at the same time these two lines with a different source of resistance to blast because experience demonstrates that a variety with genes from a single source of resistance breaks down in one or two cycles of commercial planting. Two varieties with different sources of resistance, when grown simultaneously, would probably maintain their resistance through several seasons of commercial planting.

Table 5. Blast incidence and yields of the six promising rice lines in several Latin American countries, 1975.[a]

Line	Costa Rica		Honduras		Guatemala[b]		Nicaragua		Panama	
	Nb	Y	Nb	Y	Nb	Y	Nb	Y	Nb	Y
4421	0	7.9	0.4	6.4	13.0	5.5	5	8.3	5	5.2
4422	0	9.2	1.0	5.6	1.5	5.6	8	7.7	8	5.4
4440	0	6.8	0.3	6.7	22.5	5.9	1	7.1	5	4.4
4444	0	7.7	0.0	4.4	5.0	5.4	2	6.7	5	4.1
4461	0	8.5	0.2	4.8	2.0	4.6	15	5.0	3	5.6
4462	0	7.2	2.0	3.9	2.0	5.8	19	5.9	5	6.1
CICA 4	0	5.9	2.0	4.3	60.0	2.6	2	6.9	55	3.3
CICA 6	20	4.2	0.2	4.0	10.0	4.7	4	6.7	5	6.5

[a]Nb = neck blast (%), Y = yield (t/ha), Nicaragua and Panama tests were irrigated; others were upland tests with high rainfall. [b]Average of two tests

Seed of eight promising lines was provided in 1975 to national rice programs of all Central American countries, and to Ecuador, Venezuela, Argentina, Brazil, and Paraguay in South America. The results that had been reported (Table 5) indicated that all the lines were resistant in Costa Rica, Honduras, and Panama. Lines 4421 and 4440 were susceptible in Guatemala. Also, lines 4461 and 4462, both with resistance derived from Colombia 1, showed a moderate susceptibility in Nicaragua.

In October 1976, I personally observed in demonstration plots at Tocumen Experimental Station of the Universidad de Panama that both with and without irrigation, CICA 9 (line 4421) was severely affected by leaf and neck blast, while CICA 7 (line 4461) in adjacent plots was resistant.

There were indications, then, that the tolerance to blast of the new varieties would not be stable. The varieties would probably be susceptible within two or three cycles of commercial planting.

NEW APPROACHES TO BREEDING FOR BLAST RESISTANCE

Experience demonstrates that a single source of resistance to blast is broken down into two or three commercial plantings. There is an urgent need, therefore, for breeders to concentrate on selecting varieties with different sources to provide a broad spectrum of resistance.

In 1975 two new approaches to breeding for blast resistance were initiated in the CIAT-ICA Cooperative Rice Program.

The first approach attempts to combine multiple sources of resistance in new varieties. To accomplish this, 10 blast-resistant, advanced lines were selected from yield trials (Table 6). The sources of resistance in these lines were Tetep, C46-15, Dissi Hatif, and Colombia 1. The 10 lines were crossed in all possible combinations to give 45 single crosses, each with two sources of resistance, The F_1 plants from single crosses were intercrossed to combine three or four sources of resistance in one multiple cross. In these multiple crosses, the

Table 6. Advanced rice lines selected to combine multiple sources of resistance to blast in new varieties.

Line	Cross and pedigree
4418[a]	IR665-23-3-1//F_1 (IR841-63-5-104/1B/C46-15) P901-22-7-2-3-2-1B
4425[a]	F_1 (IR665-33-5-8/Dissi Hatif)/ (IR841-63-5-104-1B) P914-43-8-3-5-2-1B
4426	CICA 4//F_1 (IR665-33-5-8/Tetep) P917-30-3-3-3-2-1B
4436	CICA 4//F_1 (IR665-33-5-8/Terep) P917-57-45-6-1-1-1B
4438	CICA 4//F_1 (IR665-23-3-1/Tetep) P918-19-9-3-1-3-1B
4440[a]	CICA 4//F_1 (IR665-23-3-1/Tetep) P918-25-1-4-2-3-1B
4452	CICA 4//F_1 (IR665-23-3-1/C46-15) P920-7-4-3-3-1-1B
4462[a]	IR22//F_1 (IR930-147-8/Colombia 1) P881-19-2-12-1B-7-1B
4463	IR22//F_1 (IR930-147-13/Colombia 1) P882-12-6-1-5-3-1-1-1B
4467	(IR665-23-3-1/Tetep^2) P896-20-1-1-6-8-1B
1445[a]	(IR8^3/Carreon) IRRI 1445

[a] Selected as donor parents for the multiline variety project.

IRRI 1445 line with resistance from Carreon was added. A total of 587 multiple crosses were produced in August 1975. The F_1 seedlings of the multiple crosses were exposed, under seedbed conditions, to blast disease. Over 6,000 F_1 seedlings found to be resistant were transplanted in the field in October 1975. and 3,600 F_1 plants were harvested individually in February 1976. The F_2 population was grown in the ICA Experiment Station, La Libertad, in eastern Colombia. The station provides conditions favorable for blast development. From September to October 1976, 24,000 F_2 plants resistant to both leaf and neck blast were harvested. The evaluation of the material is being continued at the ICA Station in Palmira. That germplasm represents an enormous source of resistance for national programs of Latin America in the near future.

The second approach to blast-resistance breeding is focused on producing multiline varieties. Two high yielding lines, 4414 and 4421, well adapted in Latin America, were selected as recurrent parents.

The lines were crossed in March 1975 with five donor lines having good agronomic characteristics, and sources of resistance different from those of Tetep, Dissi Hatif, C46-15, or Colombia 1 (Table 6). F_1 resistant plants were backcrossed to recurrent parents in February 1976. One or two further backcrosses were to be made to reconstitute the characteristics of the recurrent parent in the resistant selections that will be carried through the pedigree system. In F_5 or F_6, several isogenic lines from each backcross will be available for international evaluation. National programs can bulk the seed of several isogenic lines carrying different sources of resistance to produce their own multiline varieties.

The two new approaches offer rice breeders better possibilities for selecting varieties with a broad spectrum of resistance to blast disease.

BREEDING FOR BLAST RESISTANCE AT IRRI

I. IKEHASHI and G. S. KHUSH

IN MOST OF THE IRRIGATED RICE FIELDS in the tropical plains, rice blast seems to be a rather insignificant disease. Many IRRI varieties with moderate resistance to blast have obtained wide acceptance in South and Southeast Asian countries. Nevertheless, blast is frequently a major constraint to stable rice production on many terraces in hilly areas, especially in cooler highlands or in upland fields. Increased use of fertilizers is likely to increase the incidence even further, not only in mountainous regions but also in the tropical plains during the wet season.

PAST EFFORTS IN BREEDING FOR BLAST RESISTANCE

Scientists at the International Rice Research Institute (IRRI) have had longer experience with blast than with any other disease or insect of rice. As soon as IRRI's research program was organized, plant pathologists developed the upland-nursery method of screening, and the available germplasm was screened to identify sources of blast resistance. Resistant parents were identified and used in the crossing program. Progenies of those crosses were regularly screened for several generations to identify resistant breeding lines with improved plant types.

Resistant donors in earlier stages

From the beginning, the complexity of race composition of the blast fungus was realized and numerous parents were used as sources of resistance (Beachell and Khush 1969; Khush and Beachell 1971). They included Dawn, Gam Pai 15, H-105, Leuang Yai 34, Sigadis, Kam Bau Ngan, Tadukan, TKM 6, Zenith, and several japonica varieties from Taiwan. Dawn was used rather extensively.

In the initial stages, the blast-resistant parents were crossed with Dee-geo-woo-gen and TN1, and improved-plant-type breeding lines with blast resistance were selected. During 1966, 1967, and 1968, IR8 and improved-plant-type

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Table 1. Blast-resistant lines developed from various donor parents.

Selection	Cross	Donor parent
IR4-93	H 105/Dee-geo-woo-gen	H 105
IR480-5-9	Nahng Mon S-4 ^{2}/TN1	Nahng Mon S-4
IR759-54-2-2	IR8/Peta ^{3}//Dawn	Dawn
IR1909-1-3-3	IR8 ^{4}/Dawn	Dawn
IR790-28-1-6	Peta ^{4}/TN1/4/IR8///H 105/Dgwg//B589A4 ^{2}/TN1	B589A4
IR1360-85-2-3	IR8/Kam Bau Ngan	Kam Bau Ngan
IR833-6-2-1	Peta ^{3}/TN1//Gam Pai 15	Gam Pai 15
IR1416-128-1-2	Peta ^{4}/TN1//Tetep	Tetep
IR1416-131-3-10	Peta ^{4}/TN1//Tetep	Tetep
IR1544-238-2-3	IR24/Tetep	Tetep
IR1544-340-6-1	IR24/Tetep	Tetep
IR1529-680-3	Sigadis ^{2}/TN1//IR24	Sigadis

lines from backcrosses of TN1 to Peta, such as IR262-43-8, were used extensively in crosses with blast-resistant parents. After 1968, IR23 was used as the improved-plant-type parent for about 2 years. Blast-resistant lines with high yield potential developed in the earlier years are listed in Table 1.

It was noticed in the earlier years that some resistant lines became susceptible after a few seasons' tests. For example, blast-resistant selections from the crosses of Dawn showed susceptible reaction in the 1972 wet season and later. Similarly, after showing resistance for several seasons, some lines from a particular cross showed susceptibility even though the resistant parent continued to show the resistant reaction. That indicated that more than one major gene governs resistance in the donor parent.

Identification of stable resistance through the International Rice Blast Nursery (IRBN)

To identify lines with broad spectrums of resistance, a set of breeding lines was added to the IRBN in 1968. In 1969 a set of 175 selections from several resistant sources was also tested in Thailand, Indonesia, Sri Lanka, Bangladesh, India, Colombia, and Guyana. Selections from crobses involving Sigadis were found resistant in Thailand, Sri Lanka, Guyana, and Colombia, but not in Indonesia. Selections from crosses involving H-105, T 172, and B589A4-18 proved resistant in the Philippines, Indonesia, Sri Lanka, Guyana, and Colombia but susceptible in Thailand. Selections from the crosses with Zenith and Dawn were resistant at all sites (IRRI 1970).

In 1969 two varieties, Carreon and Tetep, were identified from the IRBN tests. They showed broad spectrums of resistance in numerous tests conducted in 35 countries (IRRI 1970). Tetep was crossed that year with IR400-28-4 (Cross No. IR1416) and IR24 (Cross No. IR1544), while Carreon was crossed with IR24. Carreon proved to be a poor combiner and most of its progeny were discarded in the early stages. Excellent blast-resistant progeny were

obtained from the Tetep crosses. They were evaluated in the blast nursery in different months and several lines with high levels of resistance were identified (IRRI 1970). Several have continued to show resistance at Los Baños.

Progeny of IR1416 (IR400/Tetep) also have good grain quality and have proved to be good combiners. Similarly, progeny of IR1544 (IR24 Tetep) have excellent grain quality, good combining ability, and resistance to the green leafhopper. Those lines were used in a series of single and multiple crosses with other improved-plant-type lines, and many lines with blast resistance and multiple resistance to other diseases and insects were developed (Table 2).

Gam Pai 15 was identified to be resistant to blast in early years. It was crossed with a line from Peta^3/TN1, and a promising line. IR833-6-2, from this cross was used in a multiple cross, IR3061 (IR833-6-2/IR1561-149//IR24^4/*O. nivara*). From that cross, several blast-resistant lines with multiple resistance to other diseases and insects were selected: three of them were named IR28, IR29, and IR34.

Advanced-generation breeding lines from other crosses are regularly screened in the blast nursery, and new sources of resistance are sometimes discovered. As an example, when *Oryza nivara* was used in the breeding program as a source of resistance to grassy stunt disease, many lines from its third and fourth backcrosses to IR24 were found to be highly resistant to blast.

PRESENT STATUS OF BLAST RESISTANCE

Numerous breeding lines with high level of resistance to blast and multiple resistance to other major diseases and insects have been developed through the multidisciplinary approach at IRRI. They are being used in IRRI's hybridization program and are shared with national rice improvement programs through the International Rice Testing Program. Many of the lines, especially those from crosses involving Tetep, have shown broad spectrums of resistance at many sites.

Table 2. Breeding lines with multiple resistance to diseases and insects and with blast resistance inherited from Tetep.

Selection	Cross
IR1820-52-2-4	IR24//Mudgo/IR8///Peta^4/TN1//Tetep
IR2035-290-2-3	Peta^4/TN1//Tetep///Peta^3/TN1//HR21/4/IR24//Mudgo//R8///IR24^3/ *O. nivara*
IR2055-481-2-6	BPI121-407/4/Peta^4 /TN1//Tetep///IR22
IR2034-238-1-2-3	IR1539-60///Peta^3/TN1//HR21/4/IR24^4 / *O. nivara*
IR2058-78-1-3	Peta^4/TN1//Tetep///Peta^3/TN1//HR21/4/IR1366-120/1R1539-111
IR2588-48-3	IR24/Tetep///Sigadis^2/TN1//IR24
IR2823-103-5-1	CR94-13///Sigadis^2/TN1//IR24/4/IR24^3 / *O. nivara //IR400/Tetep*
IR2829-409-5-3	CR94-13///Sigadis^2/TN1//IR24/4/Tetep//IR1721-13
IR4427-28-3-2	BPI121-407/4/Peta^4/TN1//Tetep////IR22/5/IR2061-464
IR4442-207-2-3	IR2061-464/4/IR24//Mudgo/IR8///Peta^4/TN1//Tetep

Table 3. Blast-resistant breeding lines identified from 1975 IRBN, and their differential reactions at IRRI and Leyte, Philippines, in 1976.

Designation, cross	Reaction (%) in IRBN[a]			Locations (no.)	Reaction rating[a]	
	R	MR	S		At IRRI	At Leyte
IR29, IR833-6-2-1-1//1R1561-149-1/IR1737	74	22	4	23	1	7–8
IR1416-128-5-8, IR400-28-4-5/Tetep	80	16	4	25	1(3)	1(3)
IR1820-52-2-4-1, IR1539-60/1R1416-128-5	80	16	4	25	1	4
IR1905-81-3-1, IR8/Tetep	76	20	4		1	3
IR2035-255-2-3-2. IR1416-128/IR1364-37/// IR1539-269//IR24[3]/*O. nivara*	72	28	0	25	1	5-6
IR2053-521-1-1-2, IR1416-131-5/IR22//C4-63	60	32	8	25	1(3)	7
IR2058-435-3-2-2-2, IR1416-131-5/IR1364-37// IR1366-120-3/IR1539-111	80	16	4	25	1(3,4)	6–7
IR2061-464-2-4-4-6, IR833-6-2-1-1//IR1561-149-1/IR1737	72	24	4	25	1(3)	7
IR2588-2-3-3, IR1544-238-2-3/IR1529-680-3	68	24	8	25	1	7–8
IR2793-10-2, IR1416-131-5/IR1364-37// IR1514A-E666	76	20	4	25	6	8
IR2793-38-3, IR1416-131-5/IR1364-37// IR1514A-E666	72	20	8	25	1(3)	7
IR2793-80-1, IR1416-131-5/1R1364-37// IR1514A-E666	76	20	4	25	6	7
IR3259-P11-186-4. IR8[3]/Tetep					1	1(3)
IR3273-339-2-5, IR8[6]/PK 203					1(3,4)	1(3)
Dawn (check variety)	36	40	24	25	6	1(3)
Tetep (check variety)	79	21	0	24	1(3)	4

[a]IRBN = International Rice Blast Nursery, R (0-2) = resistant MR (3-4) = moderately resistant; S (5–9) = susceptible. Only a few plants in the population showed the reaction given in the parentheses

In the 1975 IRBN, 101 IRRI breeding lines were included. Several promising lines were identified to be as resistant as Tetep at many sites (Table 3). Several of their sibs, particularly the selections of IR2035, IR2053, IR2055, IR2058, IR2588, and IR2793, have continued to show resistance at IRRI for at least 4 years. With them a large number of crosses have been generated for further work. So far, most seem to show a fair level of resistance in IRRI's environments and at many sites of IRBN.

It was realized, however, that further progress would be limited if location of the blast nursery were confined to IRRI. An additional blast nursery was therefore initiated in 1976 to evaluate all entries in the replicated yield trials. The fungus races have been noted to be far different on Leyte island, Philippines, from that at Los Baños.

In the dry season, the blast nursery was located in Alan-alan, Leyte, under upland conditions; reactions of the breeding lines were similar to those obtained at Los Baños. However, in the wet-season blast nursery at San Isidro, Leyte, in a swampy field surrounded by hills, the reactions were distinctively different. Many entries that have shown moderate resistance at Los Baños were severely infected at San Isidro, while some entries that have never been very resistant at Los Baños were highly resistant in Leyte, suggesting a specific reaction

between the fungus race and some major genic resistance. Tracing the ancestry of these lines back through parents led us to assume that the resistance donors at San Isidro might be Tetep, Ptb 18, TKM 6, and Co 13.

The reaction found in the second blast nursery at San Isidro was more surprising; most of the breeding lines that were as resistant as Tetep in the IRBN were apparently susceptible (Table 3). The resistance of some lines. such as IR3259-PP11-184-4 and IR3273-339-2-5, which was derived from Tetep or Pankhari 203 through repeated backcrosses, suggested a major-gene nature. However, many other Tetep derivatives seem to have failed to inherit some of the major genes as they showed susceptibility at San Isidro in spite of their outstanding resistance in the IRBN (Table 3).

DISCUSSION OF PAST BREEDING PROCEDURES

Ou (1972) has pointed out the existence of wide-spectrum resistance and emphasized the importance of its genetic analysis but no extensive genetic research on it has been undertaken so far. On the basis of previous knowledge, we are only able to indicate some characteristic features that might throw light on the genetic aspects of past breeding work.

The fact that resistance from some sources had been transferred through backcrossing seems to suggest that the resistance may be conveyed by major genes. The resistance of Dawn and *O. nivara* was carried through three or four backcrosses. If that resistance had been due to the cumulative effect of a number of minor genes, it could not have been transferred so easily to the progeny through repeated backcrossing.

In a certain phase of breeding, it is obvious that even a single major gene can play a dominant role in conveying blast resistance. For instance, after an extensive backcrossing program with a number of exotic sources that had been resistant to all the native fungal races in Japan, only an identical gene was introduced from diverse indica donors originating in different geographical areas (Nagai et al 1973). Similarly, the resistance of many varieties derived from crosses between japonica and indica varieties of Chinese origin at some breeding centers in Japan had earlier been found to be governed by an identical gene, *Pi-k*, which for several years showed good resistance throughout Japan (Kiyosawa 1974).

Another feature of past breeding experience at IRRI was that some lines showed resistance only for one or two seasons while others kept it for a fairly long time in the blast nursery where dominant fungus races are supposed to be always shifting. Such differences in the life span of resistance are presumably caused by differences in the number of resistance genes in each line and the spectrums of resistancc conveyed by each gene. It should be noted that a few lines, selected from a large number of the progeny of a single cross with Tetep, have been found to be fairly resistant in various locations and seasons. That has been true of crosses like IR1416, IR1544, and IR1905. Those lines probably

have more than one resistance gene. If a few of several resistance genes that segregate in a given cross convey resistance to predominant races under a given condition, they may mask the action of the rest of the others and lead to random fixation of the latter.

If there are more than three or four resistance genes, the probability of combining the whole set in the same line would be quite small. Only when a large number of progenies are subjected to a large number of races at various locations during several generations would a number of resistance genes be accumulated. That appears to be the case in the selection of resistant lines from the single crosses involving Tetep, which seems to have at least three major genes (Kiyosawa 1967; Padmanabhan et al 1973).

Attention should be drawn to the genetic constitution of the so-called stable resistant sources such as Tetep and Carreon. Most likely those varieties have greater horizontal resistance, as some virulent races isolated from them could not make fast-spreading lesions when inoculated back to them (Ou 1972). Similar observations have been made of some Japanese varieties, particularly those originating from upland sources, in which the expansion of the lesions in the presence of virulent races was not as fast as it was in other groups of rices (Yunoki et al 1970). Many of the upland rices that have survived severe blast infection in the upland or rainfed rice fields for hundreds of years are noted for their resistance (Ito 1965; Yunoki et al 1970). They probably have some mechanism protecting them from extinction by blast. Such a mechanism could not be a single major gene, because a monogenic resistance could have been broken easily by continued exposure to severe epidemics. Instead, the resistance may be governed by a combination of many minor genes, and a few major genes that are often transferred through backcrossing. Stable resistance, may be due to a combination of vertical and horizontal resistance. The aim of the IRRI breeding program is to combine that type of resistance with improved agronomic backgrounds.

FUTURE STRATEGIES

In the light of recent advances on the genetics of the host-pathogen relationship, several strategies have been proposed for plant breeding. Even before some of the strategies have been tried, it may be possible to discuss in detail the possible advantages and drawbacks of certain strategies (Day 1974). In complementarity to considerations on the scope of manipulating major genic resistance, Van der Plank (1971) elaborated on the use of horizontal resistance specifically for rice breeding.

In such strategies, adequate attention should be paid to the considerable variability of blast races from region to region and from season to season. Systematic studies by Goto (1965), Padmanabhan (1965), Chiu et al (1965). Lee and Matsumoto (1966), Galvez and Lozano (1968), and Bandong and

Ou (1966) have led to the identification of distinct races of blast in several countries. Studies by Ou and Ayad (1968) and Giatgong and Frederiksen (1969) have shown the potential of variability in the fungus within one generation.

Identification of major genes

Most work on identification of genes for blast resistance has been carried out in Japan, and 10 loci for resistance to Japanese races have been identified (Kiyosawa 1974). Atkins and Johnston (1965) identified in Northrose and Nato a single dominant gene that conveyed resistance to USA race 1 of blast. Hsieh et al (1967) identified three dominant genes for resistance in japonica strains from Taiwan. However, except a single study on the inheritance of resistance by Padmanabhan et al (1973), no work has been done on the identification of genes for resistance to tropical races of blast.

The first step in genetic investigations probably should be an extensive collection of fungal races that could clearly discriminate varietal resistance, and the most careful manipulation to maintain the specific pathogenicity of those races with regular checks by a set of differentials. Although many studies have focused on the variability of the blast fungus, the possibility of maintaining specific races in the tropics should be explored. Veeraraghavan (1975) reported that isolates of *P. oryzae* Cav. revealed pathogenic stability in spite of repeated subculturing and storage at a temperature of 5°C in rice culms or oatmeal agar slants or in the latter at room temperatures of 30 ± 5°C. A fungus race from the Philippines (Matsumoto et al 1964) had been used for genetic work in Japan to identify a major gene, *Pi-k*^{s} (Kilosana et al 1967: Kiyosawa 1969). That is additional evidence that in spite of the variability of the fungus, genetic analysis can be feasible with certain races.

It is encouraging that repeated backcrosses to Tetep and other varieties were successful to some extent in maintaining resistance in the breeding materials at IRRI, indicating that major genes have a significant role. Lines derived from backcrosses could be good differentials for race identification and for genetic analysis to identify major genes. Genes identified in the temperate-zone differentials (Yamada et al 1976) also could be good tools for the identification of genes in the tropical varieties.

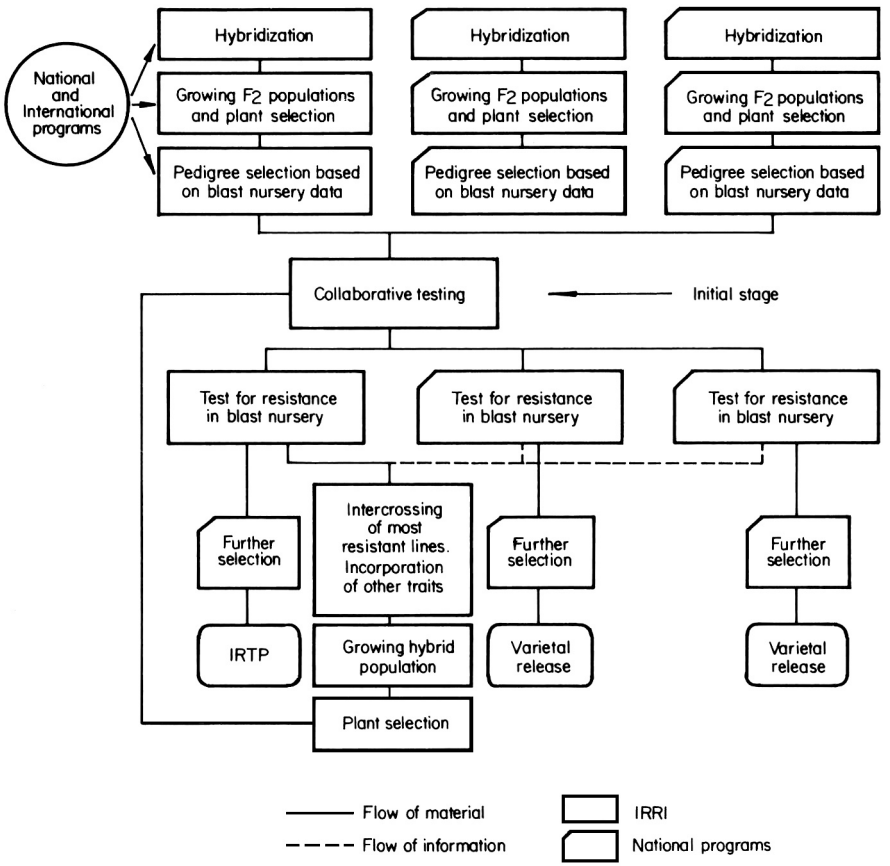
A coordinated international approach to develop differentials needs to be adopted at the present stage of the research. It should include identification of the most widely distributed blast races in the cooperating countries. identification of genes for blast resistance, and incorporation of distinct genes for resistance into isogenic lines. On the basis of such work, the programmed release of resistant lines, either sequentially or as multilineal lines, and the combining of several genes for resistance into the same variety should come within the reach of rice workers.

In the course of steady advance in the analysis of major-gene inheritance, the role of field resistance in a breeding program can be estimated properly.

Because one or two major genes mask thc effect of minor genes, the exploitation of minor gene resistance or field resistance is feasible only on the basis of the correct evaluation of such major genes.

Accumulation of diverse genes through multilocational tests

It is obvious that it will be many years before several genes for blast resistance are identified. Meanwhile, a practical approach can be adopted for accumulating diverse genes for resistance. Because of the diversification of fungus races over different locations, we can expect that different resistance genes will be included if the resistant breeding lines from diverse locations are intercrossed and hybrid lines are evaluated at many locations in cooperation with national breeding programs. A schematic diagram for the procedure is presented in Figure 1. It envisages the involvement of several national programs. Resistant



1. Collaborative breeding project on blast resistance for accumulation ofmajor genes for resistance.

Table 4. Differential reactions of sister lines at different locations in 1975 IRBN.

Breeding line	Reaction rating[a]		Breeding line	Reaction rating[a]	
	Indonesia	*Senegal 18*		*Indonesia*	*Senegal 18*
IR2071-176-1-3	0	0	IR2793-10-2	0	5
-636-5-5	0	6	-38-2	3	5
-176-2-3-4	9	1	-138-3	7	7
-344-3-4-5	7	3	-30-3	6	7
-621-2-3-6	7	6	-5-1	6	7
-774-4-5	7	8	-138-2	6	0
-588-5-4-3	3	9		*Malaysa 5*	*Senegal 18*
-2-3-3	0	0	IR1544-238-2-3	1	0
-5-1-2	0	8	-34-6-1	1	0
-29-3-3	0	0	-38-2-2	7	9
-19-1-1	6	0	-57-1-1	5	9
-132-1-2	6	0	-312-3-3	5	9
IR2793-12-1	5	0			
-15-2	9	0			
-18-5	4	0			
-38-3	6	0			
-53-2	6	0			
-80-1	0	0			
-83-2	6	0			
-123-1	6	1			

[a]0–2 = resistant, 3–4 = moderately resistant 5–9 = susceptible

selections from the different breeding programs would be tested in the IRBN. Most resistant selections would be intercrossed. The hybrid populations would be grown at one or more sites. Breeding lines originating from these intermatings would be tested collaboratively at many sites, and the lines showing resistance at the largest number of sites would be used for further hybridization. Repetition of the process, with rejection of segregating susceptible progenies, should lead to accumulation of resistance genes. Sister lines resistant at a single site may be shown to possess different genes by multilocation tests, as is evident in Table 4, which indicates that sister lines with resistant reaction at IRRI gave differential reactions at other locations. If lines showing resistance at many locations can be selected, they presumably will have more resistance genes than lines showing differential reaction at ditrerent locations.

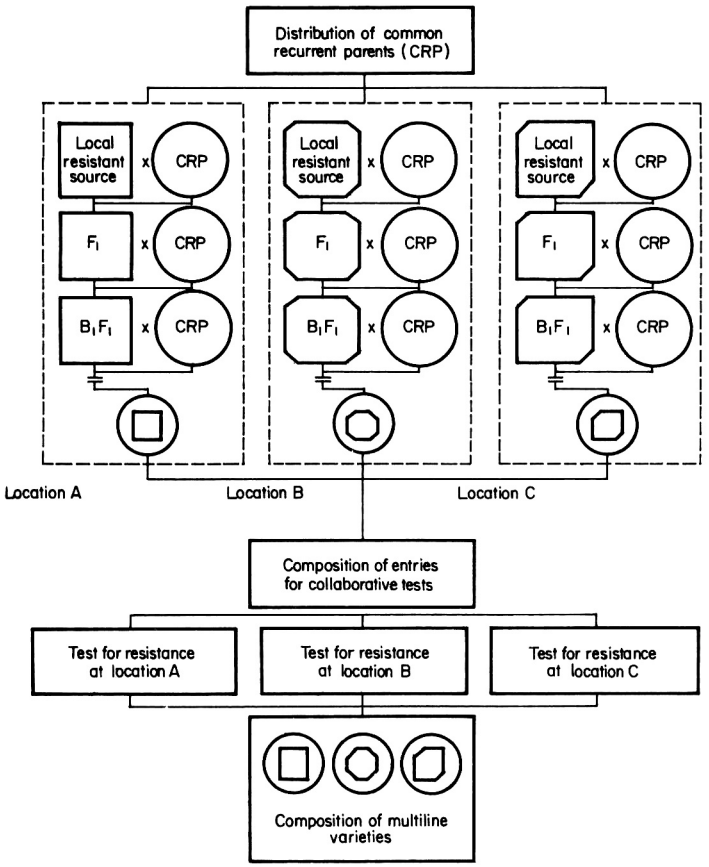
The proposed procedures should result not only in an accumulation of major genes but also in the incorporation of some minor genes. Horizontal resistance is manifested in fewer lesions and in slower developing lesions in the presence of virulent races. If the blast nursery is sensitive enough to identify moderate resistance and we intentionally choose the lines that are moderately resistant in the presence of different virulent races, instead of selecting only for vertical resistance, we should be able to accumulate minor genes for horizontal resistance.

In this proposed approach, if a number of resistance genes exist as multiple alleles at single locus, as has been demonstrated by Kiyosawa (1974), the

selections may inherit different alleles of the same major genes, but it should be possible to accumulate many of the minor genes.

A modified multiline variety

The development of a true multiline variety must await the identification of diverse genes for resistance. However, a modified approach can be made with international cooperation. Various steps for developing a modified multiline variety are presented in Figure 2. The scheme envisages the selection of resistant parents at several research centers and the adoption of a common recurrent parent or parents in a backcrossing program to develop isogenic lines. After five or six backcrosses, isogenic lines would become available, which would



2. Collaborative breeding project on blast resistance for developing a multiline variety.

be mixed to develop a multiline variety. Since the donor parents would be different and screening would be done at diverse locations with different races, different resistance genes presumably would be incorporated into the isogenic lines.

Need for diversified screening techniques

In view of the proposed genetic analysis of resistance, it is imperative that a facility be available that can handle artificial inoculation with several kinds of races. As a practical alternative to greenhouse screening, a multilocation blast nursery is to be developed so that breeding lines can routinely be exposed to diverse fungal races. The initial trial at Leyte has facilitated our selection procedures.

As has been recognized, the resistance of rice plants to blast increases during plant growth, and young seedlings are most sensitive to blast (Ezuka et al 1969). Also, the specific reaction of a variety to a fungus race seems very clear in young seedlings. Usually such a specific reaction becomes obscure in later stages. And in many cases the reaction to neck blast differs from that to leaf blast produced by the same fungus race. The present blast nursery seems to be showing more of such specific reaction. If the level of the field resistance or nonspecific resistance is to be increased, the blast nursery should also be sensitive enough to detect less specific infections. Delayed infection of the seedlings may be considered a means of doing so.

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BREEDING RICE FOR RESISTANCE TO BLAST- A CRITICAL REVIEW

S. H. OU

SACCARDO CREATED TWO GENERIC NAMES, *Pyricularia* and *Dactylaria* in 1880; both names have been used for the fungus that causes the rice blast disease. Also he spelled the first term *Pyricularia* and later changed it to *Piricularia.* Cavara in 1891 first described the fungus on rice and called it *P. oryzae.* An identical description appeared again in 1892 in Briosi and Caura. I Funghi parass. No. 188, as *P. oryzae* sp. nov., and again in the same year as *P. oryzae* sp. nov. under the authorship of Cavara. Many species of the genus on many gramineous and nongramineous hosts have been described. Some workers considered they should be lumped together into one or a few species. Others would split them, chiefly on the basis of cross-inoculation experiments. The controversy between "lumpers" and "splitters" has continued (see Asuyama 1965: Ou 1972a). Such confusions at the beginning were like portents of what would come in the work of the next 80 and more years on many aspects of the disease.

The work on breeding for blast resistance started more than a half century ago. Many resistant cultivars have been developed in several countries. But they have been unstable, have become susceptible within a few years after their release, or have not been useful in epidemic areas or under epidemic conditions. The basic problem seems to have been an insufficient knowledge of the fungus, and of the host resistance. Plant breeding is a technology, not a science; it produces not knowledge (the purpose of science) but material products (cultivars) (Riley 1976). Breeding for disease resistance requires knowledge from genetics and plant pathology. More knowledge in those areas may bring about more success in combating the blast disease.

The literature on subjects related to blast-resistance breeding is voluminous. This review attempts to cover the following areas:

1. variability of the causal fungus,
2. varietal reactions to infection,

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3. horizontal resistance, field resistance, broad-spectrum vertical resistance, and quantitative resistance,
4. genetics of resistance, and
5. breeding for blast resistance.

The critical discussions presented here aim to develop proper perspectives for future work and to suggest areas that require further investigation. The many tables offer detailed information and provide critical examination of results.[1]

PATHOGENIC VARIABILITY OF *PYRICULARIA ORYZAE*

A general review of the pathogenic variability of *P. oryzae* was given earlier (Ou 1972a). Briefly, physiologic races were noticed in Japan as early as 1922 by Sasaki. It was not until about 1950, when resistant cultivars developed by breeding, such as Futaba, became susceptible, that intensive study of races began in Japan. In about 1960 (Goto et al 1960; Goto 1965), 12 rice cultivars were selected as differentials. They included two tropical, four Chinese, and six Japanese selections. Thirteen races were identified and were classified into three groups: T (tropical), C (Chinese), and N (Nippon-Japan) according to the differentials they infected. The Japanese workers attached great importance to those race groups. F. M. Latterell reported races in the US in 1954. Race studies were begun in Taiwan in the mid-1950's. During the 1960's, races were studied in several other countries. In each country a few or many races were identified, depending upon the number of isolates collected and tested.

Because differing sets of differential cultivars were used in these national studies, the races identified in one country could not be compared with those of another. In an attempt to resolve this situation, a cooperative project between Japan and the US was initiated in 1963 to develop an international set of differentials. After 3 years of study, 8 cultivars were selected and 32 race groups were characterized (Atkins et al 1967), and a standardized system of numbering the international races was suggested to prevent confusion (Ling and Ou 1969). The eight international differential cultivars and other differentials used in various countries are listed in Table 1. There are drawbacks to using the international differentials in the tropics, but they will not be discussed in detail here.

Recently a committee in Japan (Yamada et al 1976) proposed a new set of nine differential cultivars for Japan on the basis of known resistance genes: Shan 2 (*Pi-k*^{s}), Aichi-asahi (*Pi-a*), Ishikarishiroke (*Pi-i*), Kanto 51 (*Pi-k*), Tsuyuake (*Pi-m*), Fukunishiki (*Pi-z*), Yashiromochi (*Pi-ta*), Pi No. 1 (*Pi-ta*^{2}), and Toride 1 (*Pi-z*^{t}). The committee also adopted Gilmour's octal system of numbering the races.

In the late 1960's *P. oryzae* was found to have a new dimension of variability.

[1]The wording and arrangement of some of the tables and figures have been modified from the original published form to provide easier reading and clarity.

Table 1. Differential varieties for *P. oryzae* used in different countries (Ou 1972a).

Internatlonal[a]	Japan[b]	U.S.A.[c]	Taiwan[d]
Raminad Str 3	Tetep	Zenith	Kung-shan-wu-shan-ken
Zenith	Tadukan	Lacrosse	Taichung 65
NP-125	Usen	Caloro	Pai-kan-tao
Usen	Chokoto	Sha-tiao-tsao (P)	Taichung 171
Dular	Yakeiko	(CI 8970-P)	Chianung 242
Kanto 51	Kanto51	Sha-tiao-tsao (S)	Kwangfu 1
Sha-tiao-tsao (s)	Ishikari-shiroke	CI 5309	Chianung 280
(CI 8970-S)	Hoare-nishiki	PI 180061	Taichung line 33
Caloro	Ginga	PI 201902	Kanto 51
	Norin 22	Wagwag	Norin 21
	Aichi-asahi	Raminad Str 3	Sensho
	Norin 20	(Rexoro)[i]	Cutsugulcul
		(Taichung 65)	Natala
			Kao-chio-liu-chou
			Kaohsiung-ta-li-chen-yu
			Taichung-ti-chio-wu-chien

Philippines[e]	India[f]	Korea[g]	Colombia[h]
Kataktara DA-2	AC. 1613	Zenith	Raminad Str 3
CI 5309	CR. 906	Ishikari-shiroke	Zenith
Chokoto	Bengawan	Pi 1	NP-125
Co 25	S.M. 6	Sensho	Usen
Wagwag	Mas	Kanto51	Dular
Pai-kan-tao	Intan	Ayanishiki	Kanto 51
Peta	CR. 907	Norin 17	Sha-tiao-tsao (s)
Raminad Str. 3	BJ-1	Norin 22	Caloro
Taichung t-c-w-c	S. 67	Norin 1	Aichi-asahi
Lacrosse		Tonewase	Ishikari-shiroke
Sha-tiao-tsao (s)			(Napal)[i]
Khao-tah-haeng 17			(Bluebonnet 50)[i]

[a]Atkins et al 1967. [b]Goto 1965. [c]Atkins 1968. [d]Chlu et al 1965 [e]Bandong and Ou 1966. [f]Padmanabhan 1965. [g]Ahn and Chung 1962. [h]alvez and Lozano 1968. [i]Used as a supplementary variety.

Conidial populations derived from single leaf-lesions and from monoconidial cultures differ in pathogenicity. Such populations consist of many races when the daughter conidia are separately cultured and used to inoculate differential cultivars (Ou and Ayad 1968; Ou et al 1970; Table 2, 3). Such changes in trace occurred in at least three consecutive conidial generations (Giatgong and Frederiksen 1969: Table 4). In other words, the conidia seem to lack homogene pathogenicity: they continue to change. Even hyphal tip cultures derived from single cells of a conidium may consist of many races (Ou 1972a; Fig. 1). This new dimension of variability necessitates a change in the conventional concept of races. The phenomenon is puzzling: it has nevertheless been demonstrated repeatedly. In home respects, it compares with the conidial variation in *Fusarium* shown by Snyder (1933).

No exact cytogenetic explanations for the variation are available. Yamasaki and Niizeki (1965) reported that most fungal cells were uninucleate, though in

Table 2. Pathogenic races of *Pyricularia oryzae* among monoconidial cultures from lesion no. 1, lesion no. 2, and monoconidial subcultures of culture L-1-43 and L-1-49 based upon reactions of the Philippine differential varieties (Ou and Ayad 1968).

Race designation	Reaction[a] to Philippine differential variety												Monoconidial cultures (no.)		Monoconidial subcultures (no.)	
	Katak-tara DA2	CI15309	Cho-koto	Co25	Wag-wag	Paikan tao	Peta	Raminad Str. 3	Taichung T-C-W-C-	La-crosse	Shatto-tsao(S)	Khao-tah-ha-eng 17	Lesion no.1	Lesion no. 2	Culture-L 1–43	Culture-L 1–49
P 6	R	R	R	S	R	S	S	S	S	S	S	S				
P 8	Rs	R	Rs	R	S	Rs	S	S	S	S	S	S		24	6	
P 12	R	R	R	R	S	S	S	S	S	S	S	S		11		
P 16	R	R	R	Rs	Rs	Rs	S	S	Rs	Rs	S	S	12		4	9
P 17	R	R	R	R	R	R	S	S	S	S	S	S			2	
P 26	R	R	R	Rs	R	R	Rs	Rs	R	R	R	S	4			
P 30	R	R	R	R	Rs	R	S	S	Rs	S	S	S	8		4	7
P 36	R	R	Rs	R	S	Rs	S	Rs	S	S	S	S		2		
P 38	R	R	R	R	R	R	S	Rs	R	R	R	S	1			
P 47	R	R	R	R	Rs	R	S	S	S	S	Rs	S			1	2
P 52	R	R	R	S	S	R	S	S	S	S	S	S	2			
P 54	R	R	R	S	R	R	S	S	S	R	S	S	1			
P 55	R	R	Rs	S	Rs	R	S	S	Rs	S	S	S	4			
P 56	Rs	R	R	S	R	R	S	S	R	Rs	S	S	4			
P 57	R	R	R	Rs	Rs	R	S	S	R	Rs	Rs	S	13		2	2
P 58	R	R	R	R	R	R	R	S	R	R	Rs	S	3			
P 59	R	R	R	R	S	S	R	S	S	Rs	R	S	1		1	
P 62	R	R	R	R	S	R	S	S	Rs	S	S	S		2		
P 63	R	R	S	S	Rs	R	S	Rs	R	Rs	R	S		1		
P 64	R	R	R	R	S	S	S	Rs	S	Rs	S	S		2		
P 69	R	R	R	S	R	R	S	S	R	S	R	S	1			
P 70	R	R	R	Rs	S	Rs	S	S	Rs	Rs	S	S	1		3	
P 71	R	R	R	Rs	S	S	S	Rs	S	S	S	S		1		
P 73	R	R	R	R	R	R	S	S	R	S	Rs	S	1			1
P 76	R	R	R	Rs	S	R	S	S	S	S	Rs	S	1			1
P 77	R	R	R	S	R	R	S	S	Rs	R	Rs	S				1
P 78	R	R	R	S	S	R	S	S	R	Rs	S	S				1
P 79	R	R	R	R	R	R	S	S	S	R	Rs	S				1
P 80	R	R	R	R	S	R	S	Rs	Rs	S	Rs	S			1	
P 81	R	R	R	Rs	S	R	S	S	S	Rs	S	S			1	

[a]R = resistant, S = susceptible. Where two or more cultures were inoculated, Rs may also include R reaction

Table 3. Pathogenic races of *P. oryzae* from monoconidial subcultures of eight parental isolates based on the reaction of Philippine differential varieties (Ou et al 1970).

Race	Parental isolate	Philippine races[a]	Races (total no.)	Monoconidial subcultures (total no.)
P15	FR-13	P 7 (4) P28 (4) P54 (2) P66 (2) P130 (1) P155 (1) P 8 (2) P30 (4) P55 (1) P88 (1) P132 (1) P158 (1) P15 (58) P35 (10) P56 (1) P98 (1) P133 (1) P164 (1) P16 (10) P36 (1) P57 (2) P102 (3) P134 (1) P17 (2) P38 (1) P60 (1) P125 (7) P141 (3) P18 (14) P45 (2) P61 (1) P126 (1) P147(1) P19 (8) P51 (1) P62 (1) P128 (5) P148 (8) P25 (1) P52 (4) P64 (1) P129 (4) P151 (1)	43	189
P8	FR-1	P8 (61) P18 (1) P32 (3) P64 (2) P118 (1) P149 (1) P12 (29) P19 (2) P36 (4) P70 (1) P125 (1) P150 (2) P15 (1) P21 (1) P50 (14) P80 (1) P141 (4) P153 (1) P16 (1) P25 (1) P52 (4) P81 (13) P142 (2) P17 (3) P30 (2) P62 (1) P87 (2) P143(1)	28	160
	FR-79	P8 (10) P35 (4) P102 (5) P168 (4) P12 (1) P81 (4) P120 (2) P169 (1) P16 (1) P90 (2) P165 (1) P170 (1) P17 (5) P92 (4) P166 (1) P177 (1) P18 (2) P100 (2) P167 (1)	19	52
P81	FR-1-138	P8 (19) P17 (6) P52 (1) P118 (1) P9 (1) P36 (2) P62 (1) P141 (1) P12 (10) P50 (4) P98 (1) P175 (1)	12	48
P36	FR-82	P23 (1) P92 (31) P120 (1) P177 (1) P87 (13) P114 (1) P150 (2) P181 (1)	8	51
	FR-78	P87 (2) P92 (33) P120 (1) P131 (2) P89 (1) P112 (4) P123 (1) P166 (1)	8	45
	FR-80	P8 (37) P17 (2) P52 (1) P117 (1) P12 (2) P50 (1) P62 (1)	7	45
	FR-81	P8 (28) P36 (2) P52 (1) P98 (1) P12 (4) P44 (1) P81 (1)	7	38

[a]Each figure enclosed within parentheses represents number of isolates.

certain strains, 13 to 20% of the cells were multinucleate, containing 2 to 6 nuclei. Anastomosis and migration of nuclei were observed. Nuclei had apparently fused to form heterodiploids. Haploidization could then bring about new combinations. Wu and Tsai (1974), crossing ultraviolet-induced mutants, also suggested that parasexuality might be the mechanism of variation. Another study at the International Rice Research Institute (IRRI) (unpubl.). using chemically induced mutants that differed in nutritional requirements or color, or both, also suggests that either heterocaryosis or parasexual recombination can occur. We also found (unpubl.) that in an artificial medium the conidia germinate and produce either appressoria or barrel-shapcd cells within 24 hours. The appressoria contain as many as 32 to 64 nuclei, and the barrel-shaped cells have 2 to 6 nuclei. From these two organs, normal uninucleate mycelia are produced. It seems that after spore germination there is always a multinucleate stage. Genetic changes could occur

Table 4. Disease reactions to 20 first-, second-, and third-generation monoconidial lines of *Pyricularia oryzae,* race 1, tested on 4 differential rice varieties (Giatgong and Frederiksen 1969).

Variety or C.I. number	Reaction class[a]							
	Parental isolate	Monoconidial isolates						
				First generation				
Zenith	S			S	S	S	R[b]	
C.I. 8970-P	R			R	S	R	S	
C.I. 8970-S	S			S	S	R	S	
P.I. 180061	S			S	S	S	R	
	Frequency			13	4	2	1	
				Second generation				
Zenith	R	R	R	R[b]	S	S	R	S
C.I. 8970-P	S	S	R	S	R	S	R	S
C.I. 8970-S	S	S	S	S	S	S	R	S
P.I. 180061	R	R	R	S	S	S	R	R
	Frequency	7	5	3	2	1	1	1
				Third generation				
Zenith	R			R	R	R	R	
C.I. 8970-P	S			S	S	R	S	
C.I. 8970-S	S			S	S	R	R	
P.I. 180061	S			S	R	R	R	
	Frequency			15	2	2	1	

[a] R = resistant, S = susceptible. [b] Isolate selected for testing in the next generation.

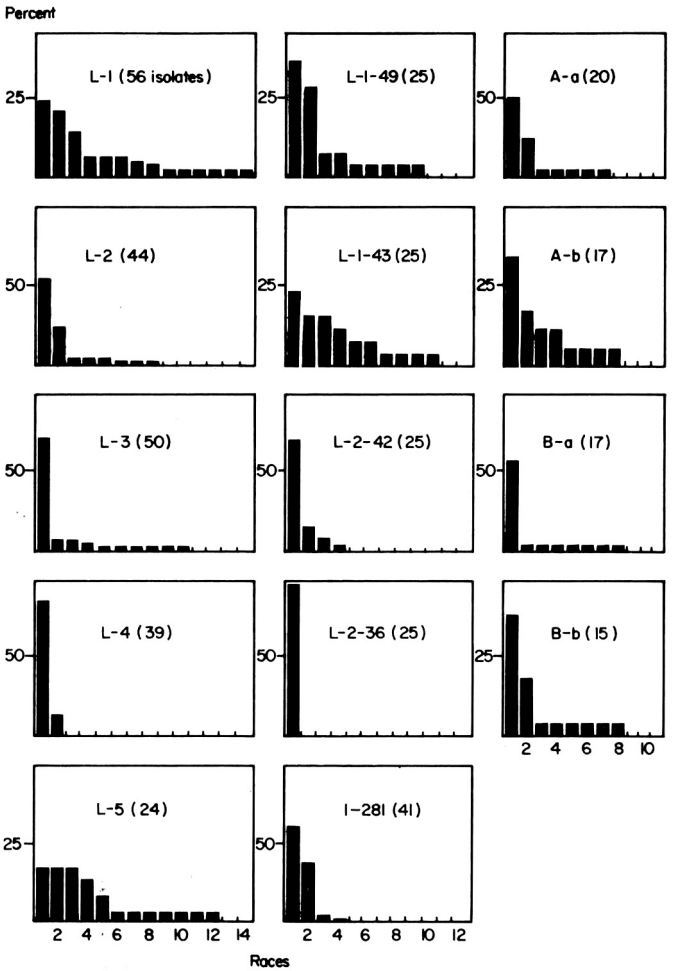
during that stage. Further cytogenetical studies are required to explain the variation.

Kato et al (1976) and Yaegashi and Nishihara (1976) were able to produce the perfect stage by mating *Pyricularia* sp. on other cereals and grass weeds with *P. oryzae.* Further success in the production of the sexual stage among isolates of *P. oryzae* may answer many of the pertinent questions.

Because of the unusual nature of the variability of *P. oryzae* and because no satisfactory genetical explanations are yet known, some think the reports of its existance to be "exaggerated" (Latterell 1972).

In fact, evidence and indications of such variability are frequently found in the literature, in more or less detail:

1. Goto et al (1964) in their extensive national cooperative work on the races of *P. oryzae* in Japan, in which 7 national agricultural experiment stations and 17 workers took part, also studied the variability among conidia from pure cultures. Among 10 monoconidial subcultures from isolate Ken 62-01, great variation in pathogenic pattern was observed. In the first inoculation, at least four new races—I, II, III, and IV—could be recognized (Table 5), and only one subculture had the same reaction pattern as the original. The second inoculation also yielded four new races; two, V and VI had not been found in the first inoculation and one culture had a pattern similar to that of the original. In the third inoculation,



1. Relative population of pathogenic races originating from single lesions (L-1 to L-5), single conidia (L-1–49 to 1–281), and single cells (A-a, apical to B-b, basal) of *P. oryzae* (Ou 1972a).

isolates were separated into five races, three of which were new (VII, VIII, and IX in Table 5). Thus in 3 inoculations the 10 monoconidial subcultures were separated into 10 races by their infection patterns.

It is particularly interesting to note that each of the 10 monoconidial subcultures differed in pathogenic pattern in each succeeding inoculation (Table 6). Each subculture differed in pathogenic pattern from the preceding culture.

Table 5. Pathogenicity of single-spore subcultures from isolate Ken 62-01 on differentials (from Table 59, p.75 Goto 1964, translated).

Original culture		Reaction[a] to differential variety												Race
		Tetep	Tadukan	Usen	Chokoto	Yakeiko	Kanto 51	Ishikari shiroke	Homare nishiki	Ginga	Norin 22	Aichi Asahi	Norin 20	
Ken 62-01		R[a]	R	R	R	R	R	S	S	S	S	S	S	0
1st inoculation (August)	Single spore subculture ①	R	R	R	R,M	R	R	M(S)	M,R	R,M,S	S	S	S	I
	②	R	R	R	R	R	R	S	R	R	M(S)	S	S	II
	③	R	R	R	M,R	R	R	S(M)	R	M(S)	S	S	S	I
	④	R	R	R	R(M)	R	R	R(S)	R(S)	S	S	S	S	0
	⑤	R	R	R	R	R	R	S,R	R	R(S)	M(S)	S	S	I
	⑥	R	R	R	R	R	R	M	R	M	S	S	S	IV
	⑦	R	R	R	S	R(M)	R(S)	S	R	S	S	S	S	III
	⑨	R	R	R	R	R(M)	R	R	R	*R*	S	S	S	IV
	⑩	R	R	R	R	R	R	R	R	R(M)	S	S	S	V
2nd inoculation (Nov.–Dec.)	Single spore subculture ①	R	R	R	M	R(M,S)	S	S	S	S	S	S	S	V
	②	R	R	R	R	S,R	S	S	S	S	S	S	S	V
	③	R	R	R	R	R	M	S	S	S	S	S	S	0
	④	R	R	R	R	R	M	S	R	R(S)	S	S	R(S)	I
	⑤	R	R	R	R	R	R	S	R	M	S	S	S	II
	⑥	R	R	R	R	R(S)	M	S	S	M	S	S	S	V
	⑦	R	R	R	R	S,R	S,R	S	S	S	S	S	S	V
	⑧	R	R	R	R	R	M	S	R	S	S	S	S	I
	⑨	R	R	R	R	R	R	S	R	S	S	S	S	I
	⑩	R	R	R	R	R(S)	S	S	S	S	S	S	S	V
3rd inoculation (Mar.–Apr.)	Single spore subculture ①	R	R	R	R	R	R	S	S	S,R	S	S	S	0
	②	R	R	R	R	R(M)	R(M)	S	S	S	S	S	S	0
	③	R	R	R	S	R	S	S	S	S,R	S	S	S	VII
	④	R	R	R	R(M)	R(M,S)	M	S	S	S	S	S	S	VIII
	⑤	R	R	R	R	R	M,S	S	S	S(M,R)	S	S	S	IX
	⑥	R	R	R	R	R	M	S	S	S	S	S	S	0
	⑦	R	R	R	R	R	R(M)	S	S	S	S	S	S	0
	⑧	R	R	R	R	R(S)	S(R)	S	S	S	S	S	S	V
	⑨	R	R	R	R	R	R	S	M(S)	S	S	S	S	0
	⑩	R	R	R	R	R	R	S	S	S	S	S	S	0

[a] R,M,S = resistant, moderately resistant, susceptible; R(S) = most seedlings resistant, a few susceptible; R,M,S = reaction types approximately equal in number
Underline indicates reaction which is considered significantly different from the original reaction by which races are identified by the present author.

Table 6. Reactions of 12 differential varieties to 10 single-spore subcultures from isolate Ken 62-01 in 3 consecutive inoculations (retabulated from Table 5; a, b, c = 1st, 2nd, and 3rd inoculation).

Subculture from Ken 62-01	Reaction[a] of												Race
	Tetep (R)	Tadukan (R)	Usen (R)	Chokoto (R)	Yakeiko (R)	Kanto 51 (R)	Ishikarishioke (S)	Honiarenishiki (S)	Ginga (S)	Norin 22 (S)	Aichi Asahi (S)	Norin 20 (S)	
1-a	R	R	R	R (M)	R	R	M (S)	M (R)	R, M, S	S	S	S	I
1-b	R	R	R	M	R (M,S)	S	S	S	S	S	S	S	V
1-c	R	R	R	R	R	R	S	S	S,R	S	S	S	0
2-a	R	R	R	R	R	R	S	R	R	M (S)	S	S	II
2-b	R	R	R	R	S,R	S	S	S	S	S	S	S	V
2-c	R	R	R	R	R	R (M)	S	S	S	S	S	S	0
3-a	R	R	R	M, R	R	R	S (M)	R	M (S)	S	S	S	I
3-b	R	R	R	R	R	M	S	S	S	S	S	S	0
3-c	R	R	R	S	R	S	S	S	S, R	S	S	S	VII
4-a	R	R	R	R (M)	R	R	R (S)	R (S)	S	S	S	S	0
4-b	R	R	R	R	R	M	S	R	R (S)	S	S	R (S)	I
4-c	R	R	R	R (M)	R, M, S	M	S	S	S	S	S	S	VIII
5-a	R	R	R	R	R	R	S R	R	R (S)	M (S)	S	S	I
5-b	R	R	R	R	R	R	S	R	M	S	S	S	II
5-c	R	R	R	R	R	M, S	S	S	S (M,R)	S	S	S	IX
6-a	R	R	R	R	R	R	M	R	M	S	S	S	IV
6-b	R	R	R	R	R (S)	M	S	S	M	S	S	S	VI
6-c	R	R	R	R	R	M	S	S	S	S	S	S	0
7-a	R	R	R	S	R (M)	R (S)	S	R	S	S	S	S	III
7-b	R	R	R	R	S,R	S,R	S	S	S	S	S	S	V
7-c	R	R	R	R	R	R (M)	S	S	S	S	S	S	0
8-a													
8-b	R	R	R	R	R	M	S	R	S	S	S	S	I
8-c	R	R	R	R	R (S)	S (R)	S	S	S	S	S	S	V
9-a	R	R	R	R	R	R	R	R	R	S	S	S	IV
9-b	R	R	R	R	R	R	S	R	S	S	S	S	I
9-c	R	R	R	R	R	R	S	M (S)	S	S	S	S	0
10-a	R	R	R	R	R	R	R	R	R (M)	S	S	S	IV
10-b	R	R	R	R	R (S)	S	S	S	S	S	S	S	V
10-c	R	R	R	R	R	R	S	S	S	S	S	S	0

[a]Letter in parentheses after each variety name indicates original reaction to isolate Ken 62-01, R = resistant, S = susceptible, M = moderately resistant

Goto et al (1964) also made similar studies of 20 isolates, 8 of which exhibited variations like those just noted (no details were given), while others retained their original patterns of pathogenicity.

Those results were identical with observations reported by Ou and Ayad (1968), and Giatgong and Frederiksen (1969). Furthermore, Goto et al (1964) showed that when the same isolates were tested at different cooperative stations in Japan, the pathogenic patterns differed greatly (Table 7). When tested in different years, each isolate also differed in pathogenic patterns; some isolates gained pathogenicity, i.e. some originally resistant cultivars became susceptible, while others lost their pathogenicity to varieties that originally had been susceptible (Table 8).

No emphasis was placed on, or attention paid to, this phenomenon of extreme variation. The above information (in Japanese) was "buried" in the voluminous literature and remained unknown to others for many years. The attitude of the workers toward the variability was reflected in their English summary on the topic (Goto et al 1964, p. 96):

> "Evidence is noted that in blast fungi the isolates vary frequently, but not always, during the course of successive culture. Stability of the isolates in pathogenicity is quite different with isolates. For instance, nearly more than five successive transfers during the three years from 1957 in the National Institute of Agricultural Sciences showed that 12 isolates out of 26 maintained well their original pathogenicity, while 5 isolates increased the range of pathogenicity, three decreased, and 6 lost entirely their pathogenicity to the differentials in the second or third year. . . .
>
> "Segregation of pathogenicity also occurs sometimes in single spore reisolates from a mother culture. For instance, all of the repeated single spore reisolates of certain isolates retained their original reaction, but many variants occurred from other isolates. . . . As a whole, it will be said that about 1/3 of the isolates caused variation within one year.
>
> "Variation of fungi is an obstruction to the identification of races, especially races in fields, although it is an interesting problem in relation to the origin of races. Therefore, it has been understood among the workers of the Joint Work that the identification of races must be done as early as possible after isolation. A period of at least three months is required for identification, as a month or more will be needed for preparation of spore materials, one or two months will be needed for the test with two or three repetitions, and another one or two months will be necessary for confirmation in the National Institute of Agricultural Sciences. *Therefore, only the isolate which have been stable throughout this period are considered to be legitimate races of Japan."*

It is unfortunate that the workers disregarded the variable cultures and considered them "illegitimate." The study of races should consider pathogenic variability of the organism as a whole: disregarding the variable cultures seems illogical. Stable isolates, if real, are useful in genetic and other studies, but in breeding for blast resistance, the variable isolates should also be taken into consideration. According to estimates of the Goto group, one-third of the fungus isolates were variable.

The statement that some isolates were stable and others were not may also be superficial. The "stable" designations were based upon reactions with the 12 differential cultivars that were being used in the testing. If

Table 7. Changes in reaction pattern of the same cultures tested at different cooperative stations in Japan—(Part of Table 65, Goto et al 1964, translated).

Race isolate	Test location	Reaction of differential variety												Date of inoculation
		Tetep	Tadukan	Usen	Chokoto	Yakeiko	Kanto 51	Ishikari-shiroke	Homare-nishiki	Ginga	Norin 22	Aichi Asahi	Norin 20	
TH-61-48 T-1	Hokkaido	R	R	R	M	M–S	M–S	S	S	S	S	S	S	
	Aichi (Anjo)	R	R	M	M	S	S	S	S	S	S	S	S	
	Oh-ita	R	R	R	M	S	S	S	S	S	S	S	S	
	Tohoku	M	M	M	S	S	S	S	S	S	S	S	S	
	NIAS	R	R	R	S	S	S	S	S	S	S	S	S	'61.10.16
	NIAS	R	R	R	M	R	S	S	S	S	S	S	S	'61.10.23
	NIAS	R	R	R	R	R	R	S	S	S	S	S	S	'61.11.24
Naga 60-36 C-4	Hokkaido	R	R	R	M	R	R	S	R	R	X	S	S	
	Nagano	R	R	R	M	R	S	S	V	V	S	S	S	
	Aichi (Anjo)	R	R	R	R(M)	R	R(S)	S	S	R	S	S	S	
	Gifu	R	R	R	M	R	S	S	S	S	S	S	S	
	Oh-ita	R	R	R	M	R	S	S	M–R?	S	S	S	S	
	Tohoku	R	R	M	M	R	S	S	R	R	S	S	S	
	NIAS	R	R	R	M	R	S	S	R	S	S	S	S	'61.10.16
	NIAS	R	R	R	R	R	M	S	M	M	S	S	S	'61.10.23
	NIAS	R	R	R	R	R	M	S	R	R	M	M	M	'61.12.10
	NIAS	R	R	R	R	R	R	M	R	R	M	S	S	'62.03.06
	NIAS	R	R	R	R	R	R	R	R	R	R	S	S	'62.04.02
Ai 35-3 N-2	Hokkaido	R	R	R	R	R	R	S	S	S	S	S	S	'61.10.16, 10.23
	Aichi (Anjo)	R	R	R	R	R	R	R–M	S	S	S	S	S	
	Oh-ita	R	R	R	R	R	R	R	R	S	S	R	S	
	Tohoku	R	R	R	R	R	R	R	R	R	S	S	S	

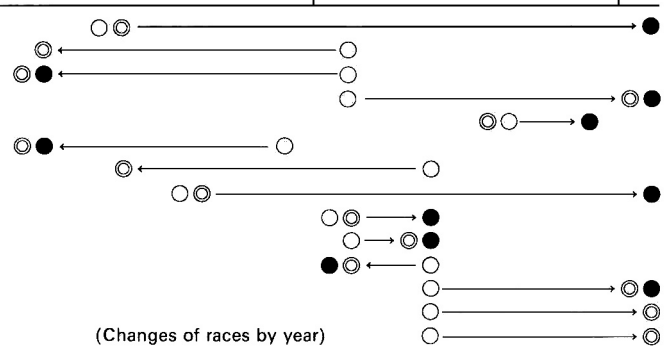
other cultivars had been used for differentiation, the stable ones might also have proved to be unstable.

2. Chien (1968) obtained 10 monoconidial isolates from a lesion of the susceptible type and 10 from a lesion of the resistant type: he made single conidial subcultures from the susceptible-type lesion for 5 generations and from the resistant-type lesions for 4 generations. The monoconidial isolates and their subcultures in each generation were tested on 10 cultivars for pathogenicity. All 20 isolates were found to change their pathogenicity in 1 to 4 generations on 2 to 6 of the cultivars. Each of the 10 monoconidial isolates from the susceptible-type lesion during the 5 single-spore generations (five spores altogether for each isolate) was separated into from 2 to 5 races (Table 9a). Each of the 10 monoconidial isolates from the resistant-type lesion during the four single-spore generations (four spores altogether for each isolate) was separated into 2 or 3 races (Table 9b).

It may be noted that the cultures always changed from S to R reaction,

Table 8. Changes in reaction pattern of isolates tested in different years (from part of Table 57; Goto et al 1964, translated).

	Differential	Race C				Race N				
		1	2	3	(d)	1	2	4		
Original reaction	Tetep	R	R	R	R	R	R	R	R	R
	Tadukan	R	R	R	R	R	R	R	R	R
	Chokoto	S	M	R	S	R	R	R	R	R
	Yakeiko	S	M	S	S	R	R	R	R	R
	Kanto 51	S	S	S	S	R	R	R	R	R
	Ishikarishiroke	S	S	R	R	S	R	R	R	R
	Homarenishiki	S	S	R	S	S	S	R	R	R
	Ginga	S	S	S	S	S	S	S	R	R
	Norin 22	S	S	S	S	S	S	S	R	R
	Aichi Asahi	S	S	R	S	S	S	R	R	R
	Norin 20	S	S	S	S	S	S	S	S	R
Isolate	57-04 (Hok)									
	57-05 (Hok)									
	57-06 (Hok)									
	57-15 (Hok)									
	57-57 (Tokyo)									
	57-58 (Tokyo)									
	57-61 (Tokyo)									
	57-77 (Naga)									
	57-87 (Naga)									
	57-94 (Nara)									
	57-98 (Tot)									
	57-114 (Ko)									
	57-115 (Ko)									
	57-132 (Kag)									



Year: ○ = 1957; ◎ = 1959; ● = 1960.

or lost pathogenicity. The reason is not known. Other data (Goto et al 1964, for example) showed changes in both directions, though many have been from S to R.

3. Nakanishi and Imamura (1960) briefly reported that isolates from the same lesion may differ in pathogenicity, and single-spore subcultures may also differ in pathogenicity from the original single-spore cultures. Goto and Yamanaka (1960) noted that when races A and B were mixed and inoculated, a third race appeared, which they thought might be the result of anastomosis between A and B. In view of recent findings on variability, the new race may have actually come from only one of the original races. Goto and Sakai (1963), in studying intracolonial variation of pathogenicity, found that in four of six colonial races, the variation among subclones within the same clone was statistically significant. They suggested that vegetative segregation might occur within isolates.
4. Katsuya and Kiyosawa (1969) and Kiyosawa (1976) reported the frequency of spontaneous mutation, in which a fungus strain previously

Table 9a. Reaction of 10 differential rice varieties to isolates of *P. oryzae* upon successive single spore transfers (Chien 1968, translated). Isolates from S-type lesion.[a]

Variety	S-1	S-2	S-3	S-4	S-5
	Gen. 1 2 3 4 5	Gen. 1 2 3 4 5	Gen. 1 2 3 4 5	Gen. 1 2 3 4 5	Gen. 1 2 3 4 5
Kungsan-wu-shang-ken	S S S S S	S S S R R	S S S S S	S S S S S	S R R R R
Taichung 65	S S S S S	S S S S R	S S S R R	S S S M M	S S S S S
Pai-kan-tao	S S R R R	S R R R R	S R R R R	S R R R R	S R R R R
Taichung 171	R R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Chia-nun-yu 280	R R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Kanto 51	M R R R R	S R R R R	S R R R R	R R R R R	R R R R R
Norin 21	R R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Cutsugulcul	R R R R R	R R R R R	S R R R R	S R R R R	R R R R R
Kaohsiung-ta-li-chen-yu	R R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Taichung-T-C-W-C	R R R R R	R R R R R	R R R R R	R R R R R	S R R R R

Variety	S-6	S-7	S-8	S-9	S-10
	Gen. 1 2 3 4 5	Gen. 1 2 3 4 5	Gen. 1 2 3 4 5	Gen. 1 2 3 4 5	Gen. 1 2 3 4 5
Kungsan-wu-shang-ken	S S S S S	S S S S R	S S S S S	S S S S S	S M M M M
Taichung 65	S R R R R	S R R R R	S S S S M	S S S S S	S M M M M
Pai-kan-tao	R R R R R	S R R R R	S S R R R	S M M M M	S R R R R
Taichung 171	R R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Chia-nun-yu 280	R R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Kanto 51	S R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Norin 21	R R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Cutsugulcul	S R R R R	S R R R R	R R R R R	S R R R R	R R R R R
Kaohsiung-ta-li-chen-yu	R R R R R	R R R R R	R R R R R	R R R R R	R R R R R
Taichung-T-C-W-C	M R R R R	S R R R R	R R R R R	R R R R R	S R R R R

[a]Gen. 1, 2, 3, 4, 5 = 1st, 2nd, 3rd, 4th, 5th single spore generation, respectively

avirulent to a cultivar becomes virulent to it. In some rice cultivar-fungus strain combinations, the rates of mutation are as high as 8 or 12% (Table 10, 11). A blast lesion produces 2,000 to 6,000 conidia each night for 2 weeks or more. Each milliliter of spore suspension generally used for inoculation contains 40,000 to 50,000 or more conidia. At such a high frequency of mutation, the change of pathogenicity among the conidial populations would be very common. In fact. one wonders if it should be called mutation.

In my thinking, the mutation rates depend upon the cultivars used; a cultivar with a broad spectrum of resistance gives a low frequency,

Table 9b. Reaction of 10 differential rice varieties to isolates of *P. oryzae* upon successive single-spore transfers (Chien 1968, translated). Isolates from R-type lesion.[a]

Variety	R-1				R-2				R-3				R-4				R-5			
	Gen. 1	2	3	4	Gen. 1	2	3	4	Gen. 1	2	3	4	Gen. 1	2	3	4	Gen. 1	2	3	4
Kungsan-wu-shang-ken	S	S	S	S	S	S	S	S	S	S	R	R	S	S	R	R	S	S	S	M
Taichung 65	S	S	S	S	S	S	S	S	S	S	R	R	S	S	R	R	S	S	M	M
Pai-kan-tao	S	S	R	R	S	S	S	S	S	R	R	R	R	R	R	R	M	M	R	R
Taichung 171	S	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
Chia-nun-yu 280	R	R	R	R	R	R	R	R	M	R	R	R	R	R	R	R	R	R	R	R
Kanto 51	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
Norin 21	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
Cutsugulcul	S	R	R	R	S	S	R	R	S	S	R	R	S	S	R	R	S	S	R	R
Kaohsiung-ta-li-chen-yu	S	S	R	R	M	R	R	R	S	S	R	R	R	R	R	R	R	R	R	R
Taichung-T-C-W-C	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	S	R	R

Variety	R-6				R-7				R-8				R-9				R-10			
	Gen. 1	2	3	4	Gen. 1	2	3	4	Gen. 1	2	3	4	Gen. 1	2	3	4	Gen. 1	2	3	4
Kungsan-wu-shang-ken	S	S	R	R	S	S	S	S	M	M	R	R	R	R	R	R	S	S	R	R
Taichung 65	R	R	R	R	S	S	S	S	R	R	R	R	S	S	S	S	S	S	S	S
Pai-kan-tao	M	R	R	R	R	R	R	R	S	S	S	R	R	R	R	R	M	R	R	R
Taichung 171	R	R	R	R	M	M	M	M	R	R	R	R	R	R	R	R	R	R	R	R
Chia-nun-yu 280	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
Kanto 51	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
Norin 21	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
Cutsugulcul	R	R	R	R	R	R	R	R	S	S	R	R	S	R	R	R	R	R	R	R
Kaohsiung-ta-li-chen-yu	R	R	R	R	S	R	R	R	R	R	R	R	S	S	R	R	R	R	R	R
Taichung-T-C-W-C	R	R	R	R	R	R	R	R	R	R	R	R	S	R	R	R	S	M	R	R

[a] Gen. 1, 2, 3, 4, = 1st, 2nd, 3rd, 4th single-spore generation, respectively.

while a cultivar with narrow spectrum gives a higher frequency. Furthermore, the mutations disclosed by the above studies are changes in pathogenicity to single cultivars. If each of the two groups of spores, avirulent and virulent, could be tested against several cultivars, further differences and many races might be found.

5. In more than 30 years of study of *P. oryzae,* Suzuki (1967) found great variation in physiology and pathogenicity and described the fungus as *"persistently heterocaryotic."* Although his interpretation of heterocaryosis as the cause of variation is questionable, the phenomenon of great variation existed. Here are a few paragraphs from his English summary:

> It has been firmly explained by a number of classical and modern extensive investigations that there is a considerable variation in the physiological characteristics as well as in the pathogenicity of *P. oryzae,* even though the isolates tested originate from a single spore. For instance, some of the workers who studied on the biologic species have pointed out that the capability of pathogenic variation in *P. oryzae* is so clearly great

Table 10. Frequencies of occurrence of virulent mutants from inoculated avirulent strains (Kiyosawa 1976).

Author	Gene for avirulence	Occurrence frequency ($\times 10^{-5}$) in fungus strain							
		P-2b	Ken 53-33	Ina 72	Hoku 1	Ken 54-20	Ken 54-04	Ina 168
Kiyosawa 1966	*Av-k*				3400	12000	400	11000
	Av-a			200				200
Niizeki 1967	*Av-k*	490			<200	1300	960	4200
	Av-a	41	24	18	<84	120	27	590
	Av-ta			190	2200	1600	9.7	
	Av-ta^2		<1.3	<6.8	<240	<130	22	<100
Kiyosawa and Katsuya 1969	*Av-k*				400	3100	500	4000
Niizeki *et al* 1973	*Av-k*	30			<2.4	14	29	110
	Av-k	27			340	34	58	46
	Av-z	11	9.1	15	<3.0	4.2	3.8	19
	Av-ta			120	70	160	1.7	
	Av-ta^2		<1.3	<1.6	<2.9	<160	<1.1	<1.3
	Av-zt	3.4	3.0		<4.7	<1.8	1.0	<1.7
	Av-zt	<1.0	<1.0		<4.7	<1.2	<1.8	<1.2
Kiyosawa 1976a	*Av-a*			40				0
	Av-k	290			1600	8400	0	100
	Av-z	0	0	0	0	0	0	0
	Av-ta^2		0	0	0	0	0	0
	Av-zt	0	0	0	0	0	0	0
	Av-b	0	0	0	0	0	0	0

Table 11. Frequencies of virulent mutants classified by original avirulence genes.

Author	Frequency ($\times 10^{-5}$) of mutant classified by										
	Av-a	*Av-i*	*Av-k*	*Av-kp*	*Av-kh*	*Av-z*	*Av-ta*	*Av-ta*^{2}	*Av-zt*	*Av-b*	*Av-t*
Kiyosawa 1966	200		6500								
Niizeki 1967			1400			120	1000	3.7			
Katsuya and Kiyosawa 1969			1100								
Niizeki et al 1973			37			4.4	88	0	1.2		
			81								
Kiyosawa 1976a	20		2100			0		0	0	0	
Frequency of failure[a]	2200	1900	15000	10000	1500	680	1700		0	380	0

[a]Of genetic experiment due to virulent mutation.

> that several of the biologic species originated as cultural sectors or as pathogenic variants, and that one grade difference in host reaction and the mesothetic reaction occur rather frequently or generally in the same biologic species although they originated from a single spore.
>
> The writer also had often obtained the contradictory results in the inoculation experiments with the monospore cultures of *P. oryzae* for the investigations on the nature of resistance of rice plant to the blast disease which were carried out for several years about thirty five years ago. It is said that such considerable variations *in vitro* and *in vivo* have caused much confusion and uncertainty about the identification of the fungus biologic species as well as the establishment of differential hosts during the four decades.
>
> It is beyond doubt that such remarkable variation is a great obstacle not only in all the basic studies of the blast disease and fungus but also in the establishment of differential hosts and in the identification of biologic species. No one has undertaken to explain the origin or mechanism of such variability in *P. oryzae.*

It seems to me that evidences of the high degree of variability in *P. oryzae* is overwhelming and not exaggerated.

Because of the wide variation in the fungus, a new concept of "race" must be adopted in the study of blast. A concept of a transitory race group consisting of conidia that differ genetically and are capable of changing in succeeding generations may be appropriate.

I wish to emphasize the significance of this type of variability for the following reasons:

1. It affects the study of races and of host genetics.
2. It is a crucial factor in discussing resistance to blast including the development of resistant cultivars, population shifts, stabilizing selection, and so forth.
3. It explains some of the common phenomena in varietal reaction that can hardly be explained otherwise, as will be discussed below.

I consider it to be the basic issue in blast work. Further studies of variability are most desirable to unify our thinking.

VARIETAL REACTIONS TO INFECTION

Type and number of lesions

The reaction of rice leaves to the invasion of the blast fungus is expressed by type and number of lesions. Many workers have given various designations to the different types of lesions (see Ou, 1972a, p. 135–137). Three types of designation are commonly used. One is numerical—1, 2, 3, 4, 5, and so on. Many Japanese workers use letters—b, bg, bG, pG, and so on. Indian workers use A, B, C, D, E. Relevant to our discussions, three general types of lesion are described below:

1 or 2 = b = A or B
: Minute (1 or A) to larger (2 or B) brown specks not more than 1 or 2 mm in diameter, indicating a resistant reaction (R).

3 = bg = C
: Roundish, restricted lesions with necrotic gray centers about 2 or 3 mm in

diameter, and brown margins, indicating a moderately resistant reaction (M).

4 or 5 = bG, pG = D or E

Large, elliptical lesions, 1 or 1 cm long with gray centers and brown (4, bG or D) or purple (5, pG, or E) margins, indicating a susceptible reaction (S).

The classification of lesions is arbitrary, with all gradations possible between the types.

All agree that lesion types are the result of a genetically controlled interaction between resistance of a rice cultivar and virulence of a pathogen.

More often than not, both in the field (blast nurseries) and in artificial inoculation experiments using single-spore pure cultures, the R, M, and S types appear in close proximity on the same leaf. Quamaruzzaman and Ou (1970), IRRI (l972), and others have shown that many races exist simultaneously in a field. The mixture of lesion types on the same leaves in the field are therefore easily explained as being produced by the presence of races of different pathogenicity.

The appearance of different lesion types on leaves artificially inoculated from single-spore pure culture is intriguing. It makes race identification, genetic analysis, and other studies difficult, if not impossible (Padmanabhan 1965; Yorinori and Thurston 1975; Cho and Kiyosawa 1973; Table 6, 12).

In the early stages of the nationwide cooperative studies on races in Japan, Goto et al (1960) reported that the reactions of 6 cultivars to 16 isolates varied in both type and number of lesions. A few paragraphs from the English translation of their discussion are quoted below:

> When the plants were susceptible, S type lessions were produced abundantly (5 to 10 lesions or more per plant), mixed with a few M and R type lesions. When the plants were moderately susceptible, M type lesions were usually produced less abundantly (leas than 5 lesions per plant), more or less mixed with a few R type lesions. When the plants were highly resistant, either very few to numerous R type lesions or no lesion, were produced-
>
> Some isolates were designated as X type, as in the case or the cereal rusts. As had been previously mentioned, they might be mixed with other races which are pathogenic to Chinese varieties, although they were derived from single spores.
>
> Some of the varieties used in these experiments seemed to be heterogenous with respect to their disease reaction, and they sometimes showed inconsistent lesion types.
>
> . . . some of the isolates such as 53-12, 55-71, 53-39, 54-18, and 54-24 completely lost their pathogenicity during the course of cultures on potato-sucrose agar.

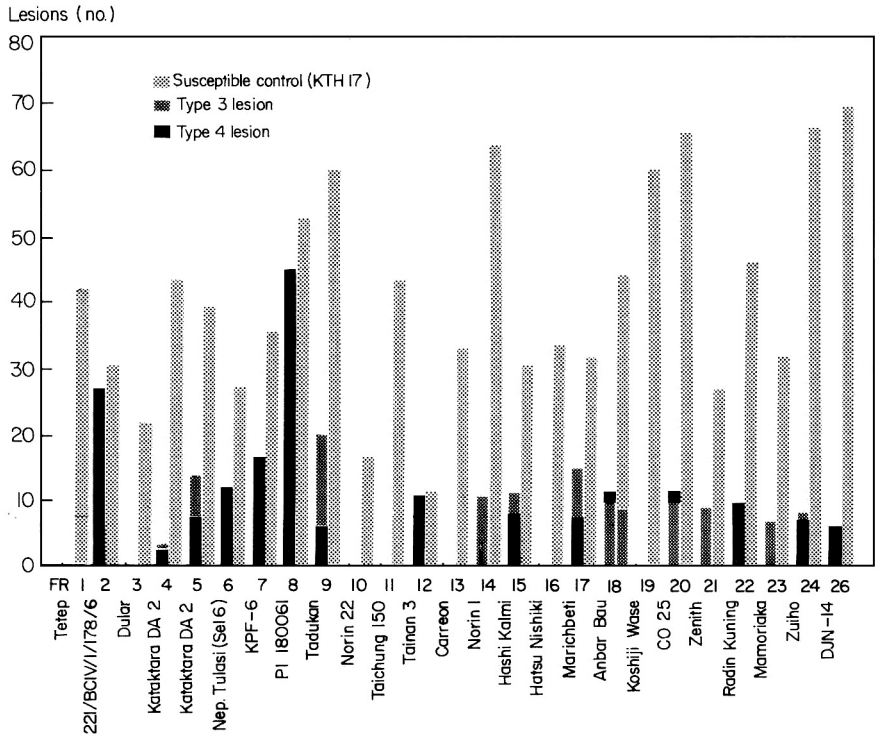
If the type of lesion is due to a genetically controlled interaction, mixtures of lesions of different types should be due to genetical differences among conidia in the inoculum, even though the conidia originated from single spores. That supports the concept mentioned above that the fungus is very variable and many different pathogenic races may develop from a single-culturc spore.

Observed as frequently as the mixture of lesions of different types is the wide variation in numbers of susceptible-type lesions on different rice cultivars in both the blast nurseries and artificial-inoculation experiments. Lesion numbers vary from very few to many. The differences are too large to be explained by

Table 12. Relative level of resistance to blast of some rice varieties, based upon the international blast nurseries (IBN) tests.

Variety group	Susceptible reading (no./total no. IBN tests)	Resistance (%)
IBN resistant varieties (partial listing)		
Tetep	6/302	98.0%
Nang chet cuc	12/282	95.8
C-46-15(II)	6/229	97.4
C-46-15(I)	19/307	93.8
Tadukan	17/310	94.0
Mamoriaka	5/227	97.8
Carreon	6/227	97.4
Huan-sen-go	8/215	97.2
Dissi Hatif	6/223	97.3
Ram Tulasi (sel)	5/194	97.4
Fanny (susceptible check)	203/252	19.5
Varieties used as donors in Japan—		
Sensho	121/267	54.7%
Norin 22	78/265	70.6
Pi No. 3 (from Tadukan)	11/174	93.7
Pi No. 4 (")	9/166	94.5
Pi No. 5 (")	12/150	92.0
Kanto 51 (from Toto)	73/280	73.9
Kanto 53 (from Reishiko)	48/212	77.4
Chugoku 31	13/82	84.2
St1	12/88	86.4
Varieties used as donors in India—		
Co 4	7/61	88.5%
Co 25 (from Co 4)	52/216	75.9
Co 30 (from Co 4)	65/123	47.2
Pusur	13/82	84.2
Varieties used as donor in Sri Lanka—		
H 4	31/247	87.5%
H 5	24/316	92.4
M 302	28/310	91.0
Varieties used as donors in Taiwan—		
Chia nung yu 181	34/180	78.9%
Chia nung yu 242	61/160	61.9
Chia nung yu 280 (2 entries)	113/386	70.7
Pai-kan-tao	12/98	87.8
Varieties used as donors in USA—		
CP 231 × HO 12 (Dawn)	18/206	91.3%

uneven distribution of the spores. Two cultivars with large differences in numbers of lesions are often in close proximity in blast nurseries and in inoculation chambers. When both cultivars are considered susceptible to the race with which they have been inoculated, the differences in numbers of lesions need to be accounted for. They may easily be explained if different pathogenic races are present in the inoculum. Experimental data to support this contention are discussed below.



2. Reaction (average number of lesions per seedling) of resistant host varieties when each variety is inoculated with a culture originally isolated from itself. Khao-tah-haeng 17 is a susceptible control (IRRI 1970).

Reaction of resistant cultivars to isolates from their own lesions

The International Rice Blast Nurseries have identified many highly resistant rice cultivars (discussed below). Occasionally, however, a few typical S-type lesion, are found on these cultivars. IRRI (1970) reported that when the fungus was isolated from these few lesions and used to inoculate the original cultivars, the isolates produced few or sometimes no lesions. The same isolates produced many lesions on the susceptible control variety inoculated at the same time (Fig. 2). When 37 isolates and reisolates from the resistant cultivar Tetep were used as inocula (IRRI 1970: Ou 1972b), an average of 2.2 type-4 lesions (and 1.7 type-3) were produced on Tetep while 32.7 type-4 (and 0.6 type-3) lesions were produced on Khao-tah-haeng (KTH), the susceptible control (Table 13) in the same inoculations. Yunoki et al (1970b) obtained several isolates from St 1 and Chugoku 31, two resistant cultivars, and used them to inoculate St 1, Chugoku 31, and some other cultivars. Only a few lesions appeared on St 1

Table 13. Lesion development on varieties Tetep, Carreon, and Khao-tah-haeng 17 inoculated at the same time with isolates and reisolates of *P. oryzae* from Tetep (Ou 1972b).

Isolate or reisolate from Tetep	Susceptible-type lesions[a] (no./seedling)		
	Carreon	Tetep	KTH
FR-1	0	0.0	63.4
FR-4A10	0	14.1	53.3
FR-13-141	0	0.1	67.3
FR-13-1a	0	0.3	42.5
FR-28	0	0.0	39.2
FR-30A2	0	0.4	20.3
-30A3	0	2.5	26.0
-30A5	0	5.8	44.5
-30A6	0	2.6	43.0
-30A7	0	2.1	61.4
-30A8	0	0.2	62.8
-30A42	0	0.4	15.2
-30A43	0	0.0	15.7
-30A44	0	0.9	17.0
-30A45	0	0.5	14.6
-30-1a	0	0.1	38.4
-30B1	0	0.8	14.1
-30B2	0	0.6	29.7
-30B3	0	0.0	14.6
FR-31	0	0.1	58.3
FR-35-1b	0	0.7	38.6
FR-50-1b	0	0.3	30.3
FR-52-1b	0	0.0	24.1
FR-54-1b	0	0.4	17.7
FR-56	0	4.8	55.6
-56A2	0	2.5	16.6
-56A9	0	5.0	16.3
FR-57	0	0.3	35.5
-57-1b	0	0.2	17.2
FR-59-A1	0	8.1	34.5
-59-1b	0	0.2	20.3
FR-78	0	0.8	22.4
-78A4(1)	0	3.7	44.0
-78A4(2)	0	3.8	19.9
-78-1a	0	3.1	21.5
-78-1b	0	1.3	9.7
-78-16	–	16.1	44.6
Av	0	2.2	32.7

[a]Counted from 20 plants

and Chugoku 31; many appeared on other cultivars (particularly on Mokoto, a well-known, very susceptible variety). For three consecutive conidial generations (reisolates) the results were the same (Table 14).

When an isolate is obtained from a type-4 lesion on a given cultivar, the isolate (race) is pathogenic to that cultivar. Why then does it produce few lesions or sometimes none when reinoculated? We (Ou et al 1971) obtained many single-spore cultures of six isolates from Tetep and one from Carreon, which we then used to inoculate Carreon, Tetep, KTH, and 12 Philippine

Table 14. Resistance of St No. 1 and Chugoku 31 to isolates from St No. 1 and Chugoku 31 (adapted from Yunoki et al 1970b, translated).

Isolate	Host variety	Race	Susceptible-type lesions (no./seedling)					
			Chugoku 31 *Pi–k^c*, *Pi–f*	St.1 *Pi–f*	Ginga +[a]	Norin 22 +	Koshi-hikari +	Mokoto +
C66-10	St No. 1	C-8	0	2.2	14.2	12.0	28.6	42.0
C66-11	"	C-8	0.5	0.9	8.5	12.9	25.4	55.0
C66-12	"	C-8	0.3	1.9	11.4	15.5	23.7	82.0
C66-13	"	?	1.8	2.9	7.9	16.3	32.1	50.6
C66-14	"	N-1	0	8.8	17.7	15.9	49.0	71.4
C66-15	"	N-1	0.3	0.2	9.2	23.0	35.6	134.9
C66-22	Chugoku 31	C-8	2.6	2.4	21.2	23.9	38.8	134.7
C66-23	"	?	1.6	0.8	10.3	18.8	26.1	89.7
C66-24	"	?	0.2	0.6	7.6	50.9	34.8	224.0
C66-25	"	?	0.2	1.4	6.6	21.4	33.3	81.4
C66-26	"	?	0.1	0.1	1.0	3.1	8.0	12.6
C66-27	"	?	0.2	1.3	5.6	19.7	33.6	112.6
Ken 60-19	Kanto 52	C-1	1.0	1.9	2.6	3.3	15.2	86.3
					1st reisolates			
C66-10	St No. 1		0	5.2	17.1	13.8	32.7	39.7
C66-11	"		0.6	0	1.0	2.1	2.2	12.2
C66-12	"		0.9	1.3	1.5	2.8	7.8	14.4
C66-13	"		0	0.2	0.5	0.3	1.2	1.4
C66-14	"		0	1.3	1.5	2.3	5.0	3.6
C66-15	"		8.9	3.5	17.1	6.5	27.6	48.8
C66-22	Chugoku 31		10.3	8.6	19.7	7.2	33.4	32.4
C66-23	"		0.2	1.2	3.6	2.2	2.4	8.8
C66-24	"		4.9	9.6	18.6	9.7	20.0	37.0
C66-25	"		0.5	1.1	1.9	0.4	4.0	6.2
C66-26	"		4.5	3.9	2.7	1.2	2.3	5.6
C66-27	"		1.2	2.3	6.3	2.0	11.3	17.6
Ken 60-19	—		2.4	4.3	18.5	7.2	12.2	22.7
					2nd reisolates			
C66-10	St No. 1		0	5.4	21.8	35.8	56.3	23.7
C66-11	"		0.8	1.0	5.8	8.1	11.9	70.6
C66-12	"		0.8	2.2	3.2	7.9	2.3	82.4
C66-13	"		3.3	2.7	9.8	33.5	50.3	201.9
C66-14	"		0	10.4	17.3	49.2	113.6	357.8
C66-15			0.3	3.7	10.3	28.5	36.0	97.9
C66-22	Chugoku 31		0.7	1.0	8.9	15.5	19.9	108.1
C66-23			0.4	1.6	5.9	21.2	10.6	179.5
C66-24	"		0.3	0.1	2.1	4.1	4.2	56.2
C66-25	"		0.1	0.2	6.3	24.0	39.2	213.3
C66-26	"		0.3	0.4	3.0	4.9	5.0	29.0
C66-27	"		0.1	0.1	4.1	9.9	16.1	103.3
Ken 60-19	—		1.2	1.6	1.6	6.2	20.4	115.0

[a]Resistance genes unknown

differentials. Below are some of the results:

1. There were many races among the single-spore cultures from each isolate.
2. Many of the races could not infect Tetep or Carreon, and those that were pathogenic (qualitatively) produced only a few lesions.

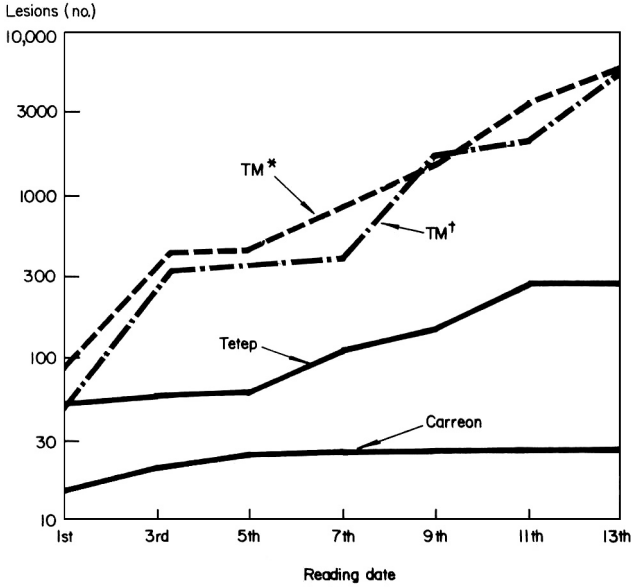
Table 15. Qualitative (pathogenic races) and quantitative (number of susceptible lesions), pathogenicity of monoconidial subcultures of isolates FR-1, FR-1-138, FR-78, FR-78-16, FR-79, and FR-80 of *Pyricularia oryzae* from Tetep and FR-13 from Carreon, artificially inoculated on Tetep, Carreon, and Khao-teh-haeng 17 (KTH) cultivars (Ou et al 1971).

Isolate	Cultivar	Races (no.) pathogenic to respective varieties[a]	Subcultures (no.) pathogenic to respective cultivars[b]	Lesions (av. no./plant) caused by	
				All subcultures	Pathogenic subculture
FR-1	Carreon	11 (28)	60 (160)	0.3	1.1
	Tetep	5 (28)	19 (160)	0.1	1.4
	KTH	28 (28)	160 (160)	33.9	33.9
FR-1-138	Carreon	6 (12)	15 (48)	0.8	2.8
	Tetep	1 (12)	3 (48)	0.1	6.2
	KTH	12 (12)	48 (48)	56.6	56.6
FR-78	Tetep	7 (8)	44 (45)	5.2	6.1
	KTH	8 (8)	45 (45)	22.6	22.6
FR-78-16	Carreon	1 (51)	1 (100)	0.1	8.7
	Tetep	17 (51)	43 (100)	3.6	8.9
	KTH	48 (51)	97 (100)	17.4	18.0
FR-79	Carreon	1 (19)	1 (52)	0.01	0.5
	Tetep	11 (19)	17 (52)	0.7	2.5
	KTH	19 (19)	52 (52)	46.3	46.3
FR-80	Carreon	3 (7)	7 (45)	0.6	4.1
	Tetep	1 (7)	1 (45)	0.2	7.9
	KTH	7 (7)	45 (45)	47.9	47.9
FR-13	Carreon	0 (43)	0 (189)	0	0
	Tetep	17 (43)	43 (189)	0.1	0.8
	KTH	43 (43)	189 (189)	50.3	50.3

[a] Figures in parentheses are total numbers of races. [b] Figures in paretheses are total numbers of subcultures.

Table 16. Pathogenic races of *Pyricularia oryzae* derived from isolates FR-1, FR-1-138, FR-13, FR-78, FR-78-16, FR-79, and FR-80 grouped by the number of the Philippine differential cultivars infected (Ou et al 1971).

Differential cultivars (no.)	Races (no.) derived from isolates: subcultures (no.) in each race group (in parentheses)						
	FR-1	FR-1-138	FR-78	FR-78-16	FR-79	FR-80	FR-13
1				1 (1)			
2	1 (1)			1 (1)			2 (11)
3	1 (1)			6 (6)			6 (68)
4	4 (4)			7 (9)	5 (12)		7 (45)
5	7 (15)	2 (2)		5 (7)	1 (1)		9 (31)
6	5 (22)	5 (11)		7 (10)	5 (17)	2 (3)	8 (12)
7	1 (61)	1 (19)		6 (12)	2 (11)	1 (37)	6 (14)
8	3 (34)	3 (12)	2 (3)	6 (13)	2 (2)	2 (3)	4 (7)
9	3 (17)	1 (4)	3 (6)	7 (11)	3 (5)	2 (2)	1 (1)
10			1 (33)	3 (17)	1 (4)		
11	2 (3)		2 (3)	2 (13)			
12	1 (2)						
Total no. races	28	12	8	51	19	7	43
Total no. subcultures	160	48	45	100	52	45	189



3. Number of lesions per 100 rice seedlings on resistant cultivars (Tetep, Carreon) and on susceptible cultivar Tjeremas adjacent to Tetep (TM*) and adjacent to Carreon (TM+) inoculated with isolate (FR-78-16) of *Pyricularia oryzae* from Tetep in blast nursery (Ou et al 1971).

3. There were always many lesions on KTH, the susceptible check.

The inoculation experiments are summarized in Table 15. The races that they differentiated are shown in Table 3. The range of variation in pathogenicity of the races to the differentials is shown in Table 16.

FR-78-16, one of the most pathogenic isolates from Tetep (Table 13), was used in a spore suspension to inoculate Tetep and Carreon in a field at the seedling stage. Again, few lesions were observed on Tetep or Carreon, but many were seen on the susceptible control Tjeremas in adjacent row 10 cm away (Fig. 3).

Several other cultivars that were similarly studied behaved like Tetep (IRRI, 1971; Table 17).

On the other hand, isolates from the susceptible cultivar KTH, when reinoculated, always produced large numbers of lesions (Table 18).

On the basis of the mentioned experiments I explain why few lesions appear on resistant cultivars and many on susceptible cultivars as follows. The isolates, when multiplied, produced many races or conidia of different genotypes. Only a few lesions appear on a cultivar with a broad spectrum of resistance because it can be infected by only a few of the races, or a few spores in the

Table 17. Qualitative (pathogenic races) and quantitative (no. of susceptible-type lesions) pathogenicity of monoconidial isolates of *Pyricularia oryzae* from six resistant varieties on their original host varieties, on other resistant varieties, and on the susceptible variety KTH 17 (IRRI 1971).

Fungus races isolated from	Races (no.)									
	Total	Pathogenic to								
		Carreon	Tetep	R 67	C46-15	Nang-chet-cuc	Dissi Hatif	Mamoriaka	Ca 435	KTH
Mamoriaka	6	0	0	0	—	—	6	4	—	6
Dissi Hatif	8	0	0	0	—	—	6	4	—	6
C46-15	3	0	0	—	0	1	1	0	—	3
Ca 435/b/5/1	3	0	0	—	1	—	1	0	0	3
R 67	2	1	0	0	1	2	—	—	—	2
Nang-chet-cuc	2	1	0	0	0	1	—	—	0	2
	Subcultures (no.)									
	Total	Pathogenic to								
		Carreon	Tetep	R 67	C46-15	Nang-chet-cuc	Dissi Hatif	Mamoriaka	Ca 435	KTH
Mamoriaka	49	0	0	1	—	—	45	43	—	49
Dissi Hatif	67	0	0	2	1	—	46	41	—	67
C46-15	28	0	0	—	0	2	2	0	—	28
Ca 435/b/5/1	20	0	0	—	2	—	1	0	0	20
R67	16	2	0	0	2	12	—	—	—	16
Nang-chet-cuc	5	1	0	0	0	2	—	—	—	5
	Lesions caused by all monoconidial subcultures (no./plant)									
	Carreon	Tetep	R 67	C46-15	Nang-chet-cuc	Dissi Hatif	Mamoriaka	Ca 435	KTH	
Mamoriaka	0.01	0.01	0.03	—	—	8.7	3.8	—	66	
Dissi Hatif	0.01	0	0.1	0.1	—	10.1	6.1	—	104	
C46-15	0.01	0	—	0	0.2	0.4	0.1	—	94	
Ca 435/b/5/1	0.02	0	—	0.1	—	0.1	0.1	0	47	
R 67	0.7	0.1	0	0.2	6.6	—	—	—	120	
Nang-chet-cuc	0.3	3	0	0	3.2	—	—	—	116	

Table 18. Number of lesions on Khao-teh-haeng 17(KTH) and other rice varieties when inoculated with isolates from KTH.

Variety	Susceptible-type lesions (no./seedling)								
	Isolate 1801			Isolate 1802			Isolate 1805		
	Rep 1[a]	Rep 2	Rep 3	Rep 1	Rep 2	Rep 3	Rep 1	Rep 2	Rep 3
Raminad St. 3	98	132	128	214	200	192	6	8	9
Zenith	7	2	6	3	5	0	0	0	0
Kataktara	0	0	0	0	0	0	0	0	0
Chokoto	0	0	0	0	0	0	0	0	0
Peta	114	125	145	223	261	247	34	29	24
KTH	157	136	176	159	161	170	48	43	40
Tetep	0	0	0	0	0	0	0	0	0
Usen	93	152	134	230	216	249	12	6	7

[a]Rep = replication.

inoculum. Many lesions appear on a susceptible cultivar because it is infected by most of the races, or by most of the spores in the inoculum. The broader the spectrum of resistance, the fewer the lesions. The phenomenon will be examined further in a discussion of quantitative resistance to blast.

I therefore assume that a constant change of races in the pathogen and a broad spectrum of resistance in varieties may constitute the basis for developing stable resistance to blast.

HORIZONTAL RESISTANCE, FIELD RESISTANCE, BROAD-SPECTRUM VERTICAL RESISTANCE, AND QUANTITATIVE RESISTANCE TO BLAST

Horizontal resistance

The value of vertical or race-specific resistance for disease-resistance breeding has been frequently challenged. Some researchers have sought horizontal (general) resistance to rice blast. In a seminar on horizontal resistance to rice blast, Van der Plank (1975a) made six suggestions.

The first was for "determining horizontal resistance as field resistance." He said:

> In the absence of vertical resistance, resistance is horizontal resistance. Therefore, if one can exclude all vertical resistance, one can simply compare cultivars or lines in the field, and the comparison will measure horizontal resistance alone.
>
> The method, then is to expose lines (or cultivars) to infectton in the field by virulent races to which the lines are (vertically) susceptible. The resistance that remains is horizontal resistance.
>
> The difficulty arises here. The lines must be exposed to a race virulent on all of them or to several races, each of which is virulent on all of them. False results are given whenever lines are exposed to a mixture of races, some of which are virulent on some races[2] but avirulent on other races[2] (Then vertical resistance enters and confuses the results).

In the blast fungus there are many races (race groups). Some infect a few cultivars, others many; no race infects all. All cultivars are resistant to a certain number of races, some to a few, others to many. Even the most susceptible is resistant to a few races. Knowledge of the number of resistant genes in rice varieties is inadequate. Under such conditions, the suggestion of Van der Plank is difficult to apply. In fact, researchers in Japan, pursuing the work on "field" resistance, discussed below, had Van der Plank's idea, but failed to find horizontal resistance because of the difficulty he mentioned and others.

Van der Plank's second, third, and fourth suggestions were to select cultivars (a) that are more difficult to infect, (b) in which the period from inoculation to sporulation is longer, and (c) on which sporulation is less abundant.

Yorinori and Thurston (1975), as well as Rodriguez and Galvez (1975), attempted what Van der Plank suggested: They said:

> The object of the study was to determine if consistent differences in characteristics which have been shown to be allocated with general resistance in other crops such as potato could be found in rice inoculated with *P. oryzae.* These characters might be useful in measuring

[2] Probabaly a typographical error; he must mean lines.

> and identifying relative levels of general resistance in rice. Characters studied were (a) size of lesions, (b) color of lesions, (c) time of sporulation, (d) number of spores produced, and (e) time of ingress.

They found that lesions of the resistant type are smaller, are a more intense brown, take longer to sporulate, and produce fewer spores than do the susceptible types. All of these symptoms are proportions to the degree of resistance or susceptibility. The findings confirmed what was already known. Yorinori and Thurston said:

> One of the most difficult problems encountered in making this study was the diversity of lesion types observed on the varieties and selections used. Even using a single isolate, a wide variation in lesion types occurred on the same variety in both detached leaf and greenhouse inoculations. Reactions varied from highly resistant to highly susceptible.

In addition to the variability of lesion types, they said, there was another basic problem: vertical interaction between the fungus isolates (races) and the host cultivars. Among the three cultivars (Peta, Binato, and Saturn, for which there were complete data) and five isolates, there were distinctly different isolate (race)-cultivar interactions. (Table 19, 20). As Van der Plank has said, one cannot evaluate horizontal resistance in the presence of vertical resistance.

Finally, Yorinori and Thurston said: "No conclusions can be made as to what might represent general resistance in rice to *P. oryzae* from the results of these studies. In addition, the results obtained were highly variable."

The other two suggestions of Van der Plank were to accumulate horizontal resistance by breeding, and to combine horizontal with vertical resistance. Because horizontal resistance has not been identified, these suggestions need no further discussion.

During the last few years, the Institut de Recherches Agronomiques Tropicales et des Cultures Vivieres (IRAT) in the Ivory Coast of West Africa tried *"to select horizontal resistance to avoid 'boom and bust' varieties"* by measuring the rate of disease development (*r* value) during the neck-blast phase of the disease. *"A very good value of r was observed in Moroberekan (0.146), Blue-Bonnet (0.167), IAC 25 (0.146), IRAT 78 (0.146), and IRAT 79 (0.112). A very high rate was found in Jaya (0.387)."* (IRAT 1977).

Among the cultivars with a low *r* value (higher horizontal resistance, HR), Blue-Bonnet is known not to have a high level of resistance. It has a relatively narrow spectrum of resistance in the International Blast Nurseries' tests and had been "busted" by showing nearly 100% neck-blast in Latin America and a near-complete killing by leaf blast in other locations. Other cultivars with a low *r* value are to be tested.

Different race groups of the blast fungus are present in different localities and seasons. The *r* value of various cultivars measured at a location will depend upon the race group present. The *r* value measured in a locality does not indicate HR.

Table 19. Range of lesion types on detached leaves of 13 rice varieties inoculated with 3 isolates of *P. oryzae* (Yorinori and Thurston 1975).

Variety or selection	Number of isolates, lesion type[a]				
	US5	27	59L13	68L4	68T1
IR8	1	1	–	–	–
Taichung (Native 1)	1	1	–	–	–
IR5-47-2	1, 2	1	1 - 2	1	–
Fortuna	1, - 2	–	1 - 2	–	–
Padma	–	1 - 3	1 - 2	1 - 2	–
T-141	–	1 - 3	1	1	–
TKM6	–	1 - 3	1 - 2	1 - 3	–
PI215-936	1, - 3	1		–	–
IR154-61-1	1, - 3	–	1 - 3	1 - 3	1 - 4
Bluebelle	1, - 3	1		–	–
Bluebonnet 50	–	–	1 - 4	1 - 4	1 - 4
Peta	1, - 4	1 - 2	1 - 3	1 - 3	1 - 2
Binato	2, - 5	1 - 3	1 - 4	1 - 4	2 - 5
Saturn	1, - 3	1	1	3 - 5	2 - 5

[a]Lesion types: 1 = highly resistant, 2 = resistant, 3 = moderately resistant, 4 = susceptible, 5 = highly susceptible, – = materials not available.

Table 20. Size of lesions measured 8 days after inoculation of detached leaves of rice with *P. oryzae*[a] (Yorinori and Thurston 1975).

Variety or Selection	Size of lesions[b] (mm^2) in leaves inoculated with				
	US5	27	59L13	G8L4	68T1
Padma	–	2.87 (3)[c]	1.07 (2)	1.06 (2)	–[d]
T-141	–	3.78 (3)	1.00 (1)	0.77 (1)	–
TKM6	–	5.11 (3)	1.86 (2)	7.11 (3)	–
IR154-61-1-1	9.37 (3)	–	2.77 (3)	1.45 (3)	4.22 (4)
Bluebonnet 50	–	–	13.42 (4)	14.26 (4)	13.25 (4)
Peta	5.70 (4)	3.09 (2)	11.37 (3)	6.33 (3)	1.14 (2)
Binato	25.74 (5)	6.25 (3)	8.08 (4)	12.70 (4)	16.90 (5)
Saturn	1.53 (3)	1.00 (1)	1.00 (1)	29.99 (5)	25.46 (5)

[a]Varieties, isolates and interaction differed at 1% level (see Table 10) [b]Av. of 12 lesions/isolate. [c]Number in parentheses indicates the lesion type; 1 = highly resistant, 2 = resistant, 3 = moderately resistant, 4 = susceptible, 5 = highly susceptible [d]Not tested.

Some rice varieties were found to have *r* value that was low in 1975 and then higher in 1976, while the reverse was true for other rices (IRAT 1977). This could be due to the races changing during the two seasons.

HR and vertical resistance (VR) are primarily the genetic relationship between the host and the parasites; HR is generailly polygenic, while VR is controlled by a major gene. Van der Plank extended this relationship to epidemiology; the disease development in cultivars with HR is slow and the *r* value is low, while the disease development of rices with VR is rapid once the virulent race occurs.

Measuring the *r* value only without considering the presence or absencc of virulent genes of the parasite is not adequate to indicatc HR, particularly in the case of the blast fungus, which is extremely variable and whose race groups differ from location to location and season to season.

Another attempt to identify horizontal resistance was made at IRRI. During the first screening for blast resistance of the first 10,000 accessions of the IRRI germ plasm collection, about 400 entries showed type-3 lesions. The plan was to see how many of those 400 entries would maintain that level of resistance after exposure to the races of the International Rice Blast Nurseries (IRBN). Because IRBN could not accommodate so many entries, the 400 were screened further. After 5 tests, the very susceptible group was discarded, the number had been reduced by about one-half; 212 were entered into IRBN in 1969. Within about 3 years, all were reported very susceptible at one or more test locations. To date, no variety has been identified to have horizontal resistance to blast.

Field resistance

Considerable effort had been made in Japan to find what researchers there called "field resistance" to blast. The work was started in about 1966 and more than 20 papers have been published (Toriyama 1975). In those papers, however, the real meaning in Japan of the term "field resistance" is not entirely clear.

Obviously, the objective of the study was to find in rice a field resistance to blast comparable to the field resistance to late blight—previously discovered in potatoes—a kind of resistance recognized as "horizontal" or "general" resistance by many others. Apparently Takase (c.f. Ezuka 1972) introduced the term field resistance into Japan in 1962 before other terms in common use. Kozaka (1975) referred to it as horizontal resistance. Sakurai and Toriyama (1967) apparently had the same idea in mind.

They found, however, that varieties with a high level of "field resistance" did not have a field resistance like that of potato to late blight or like the horizontal resistance of Van der Plank. Toriyama (1975) altered the definition of their "field resistance":

> Horizontal resistance has been called either field resistance or generalized resistance. In this report, the term "field resistance" will be employed in place of the term "horizontal resistance", because resistance showing horizontal reaction in a strict sense by Van der Plank has not been observed in any rice varieties by the Japanese investigators up to date. Rice breeders in Japan, therefore, classified blast resistance into two categories: "true resistance" and "field resistance". In this sense, true resistance is specific and qualitative rcsistance characterized by hypersensitivity to the pathogen. On the other hand, field resistance is recognized to be remainders of resistance other than true resistance. For clarifying an essential nature of field resistance, main efforts of investigation in Japan have been paid on the basis of information on true resistance with the procedure of elimination method.

It seems to imply there is true (vertical) resistance, that there is horizontal resistance, and that there is "field resistance"–*sensu* Japanese. The nature of this third type of resistance is not clear.

Workers in Japan give different definitions or meanings of "field resistance." Ezuka (1972) reviewed those definitions:

The meaning of the term "field resistance" is not always clear. Its original meaning was undoubtedly the resistance which could be recognized only in the field but not apparent in the laboratory or glasshouse. Recent studies made it possible to evaluate the "field resistance" to blast even in the glasshouse by seedling inoculation. The original definition does not fit present conditions.

Takase first introduced to Japanese rice breeders the view of dividing varietal resistance into "true resistance" and "field resistance" in reference to the breeding of potato varieties for resistance to late blight. According to his definition, field resistance implies all sorts of resistance proper to plant bodies except for hypersensitive reactions caused by the R-gene. He considered true resistance to be qualitative or major gene resistance, and field resistance to be a quantitative or polygenic resistance.

Yamada explained that true resistance was race-specific and controlled by a small number of major gene(s), while field resistance was controlled by polygenes, evaluated with quantitative characteristics, increasing with the advance of host age. Niizeki proposed to define field resistance to be evaluated with the number or size of lesions under the existence of fungus races against which the major gene(s) of the given variety do(es) not operate.

Sakurai and Toriyama stated that racial resistance (or true resistance) was characterized by hypersensitive reaction, while field resistance implied all types of resistance other than hypersensitivity controlled by major gene(s). Toriyama later defined that true resistance was specific and qualitative resistance characterized by hypersensitivity to the pathogen, while field resistance was the remainder of resistance other than true resistance.

Kozaka described that true resistance (or hypersensitivity) was the characteristic concerned in affinity or inaffinity between varieties and fungus races, while field resistance was the difference in disease severity when the affinity exists between host and pathogen. Ezuka explained simply for popular understanding that true resistance was concerned in whether a variety could be or could not he attacked by a fungus strain, while field resistance was concerned in the relative severity of the attack when a variety was attacked by a fungus strain.

Definitions of true and field resistance mentioned above are similar but do not always mean the same thing.

Kiyosawa gave reviews on the classification of varietal resistance to disease in general, and indicated that true resistance had much in common with protoplasmic resistance, hypersensitivity, specific resistance, seedling resistance, major genes resistance, qualitative resistance, and vertical resistance, while field resistance in common with morphological and functional resistance, tolerance, non-specific resistance, adult resistance, polygene resistance, quantitative resistance, and horizontal resistance, although there were some differences among these terms in the criterion for distinguishing two categories in pairs.

Kiyosawa advised the necessity of giving common and distinct definition to the terms, "true resistance" and "field resistance," in order not to introduce confusion into the discussion of varietal resistance. He gave careful consideration to every possible criteria for distinguishing two categories. For instance, specific resistance is not equal to true resistance because field resistance is occasionally known to be specific. It is difficult to certify a given resistance to be non-specific unless testing with a large number of fungus strains. Major gene resistance is not equal also to true resistance because field resistance may occasionally be controlled by major gene(s). It requires laborious work of gene analysis to determine the gene(s) controlling a given resistance.

Kiyosawa concluded from these and the other considerations that the height or intensity of resistance was at present the most adequate criterion for dividing true and field resistance. He gave this definition: true resistance is a high resistance which can be detected with testing procedures for differentiation of pathogenic races or other similar methods, while field resistance is a lower resistance which cannot be detected with these methods, but can be seen in the field.

Ezuka et al employed for blast resistance similar definition to Kiyosawa's as follows: true resistance can be apparently recognized through the difference of lesion type on the seedlings inoculated by spraying or injection method, while field resistance comprises the remainder of hereditary resistance except for the true resistance.

Kiyosawa gave the following equation for epidemiological analysis of blast infection:

$$l = l_o e^{\lambda t},$$

where l_o is the number of lesions at the initial time ($t = 0$), l is the number of lesions at a given time t, and λ is the rate of increase of the lesion number. Kiyosawa proposed to define true resistance and field resistance with the variables of the equation. According to his definition, true resistance is the resistance which affects l_o and does not affect λ, while field resistance mainly affects λ, and affects l_o only when the given field resistance is specific.

It is not common in Japan to divide resistance into vertical resistance and horizontal resistance. Kiyosawa and Toriyama noticed that these terms were not adequate for the classification of blast resistance, because field resistance to rice blast was not always non-specific to the strain of the causal fungus.

To identify "field resistance," Japanese researchers used field tests, blast nursery, artificial inoculation, and so forth (Ezuka et al 1969; Yunoki et al 1970b). They even tested "field resistance" by one or a few selected isolates of the fungus in the greenhouse (Yunoki et al 1970a). They found that "field resistance" changed greatly because of fungus strains (races) (Yunoki et al 1970b). Many of these methods and concepts differ from the conventional understanding of field resistance.

Several rice cultivars were identified as possessing high levels of "field resistance" (Japanese type), and varieties St 1 and Chugoku 31 as being among the best. The researchers also identified a dominant gene, *Pi-f,* for the high level of "field resistance" in these two cultivars and they studied the linkage relationships.

What is the value of these highly "field resistance" varieties to blast-resistance breeding? It may be revealed by few passages quoted from Toriyama.

When St 1 and Chugoku 31 were inoculated by the spray method with the virulent strains which showed pathogenicity by the injection and the spray method, only a few susceptible lesions were usually observed, and a few numbers of the susceptible lesions of these varieties were also observed in blast nursery beds. Therefore, it was considered that both St 1 and Chugoku 31 had the highest grade of field resistance. However, it was reported that St 1 was severely diseased in Fukushima Prefecture when these varieties were widely tested to ascertain their high field resistance by the blast nursery method all over Japan. Therefore, Yunoki et al tried to ascertain whether field resistance varied with the fungus strains or not.

Such breakdown of high resistance of St 1 and Chugoku 31 was apparently due to the specific reaction to the fungus strains, and this phenomenon is similar to the breakdown or vertical resistance.

Recently, it was found that high field resistance in these upland rice varieties was specific to the fungus strains, because some fungus isolates could develop a number of susceptible lesions on these upland varieties (Sekiguchi, personal communication). This was quite unexpected evidence for the Japanese rice breeders. Up until this finding, they believed that the Japanese upland rice varieties should be a favorable gene source for high field resistance. because the Japanese upland rice had been planted for many years in Japan and had exhibited stable resistance to blast.

As a result, it is concluded that field resistance of ricc varieties varied with the fungus strains, so the term field resistance does not coincide strictly with the term horizontal resistance (Toriyama, 1975).

Recently, it was reported by Yunoki et al. that some fungus strains could severely attack the varieties possessing *Pi-f* and produce many susceptible lesions. This means that the field resistance due to *Pi-f* gene is specific resistance, not horizontal resistance.

Since field resistance includes the resistant reaction controlled by a major gene (or genes), field resistance is specific and is not the same as horizontal resistance. Specific resistance should include both true resistance and field resistance. Any difference between true resis-

tance and field resistance is only due to the testing methods. Some resistance genes that are found to be true resistance genes by the spray method are sometimes not recognized to be true resistance genes in the injection method employing the same fungus strains. For example, the gene for true resistance to C-6 race in Homare-nishiki showed a hypersentitive reaction when inoculated by the spray method. Conversely, this resistance gene could not express its hypersensitivity against C-6 in the injection test.

This phenomena lead to the conclusion that non-specific field resistance, i.e. horizontal resistance in a strict sense, to blast disease will be difficult to find in rice varieties in Japan although some possibilities remain (Toriyama, 1972).

The so-called "field resistance" in Japan is definitely not the horizontal or general resistance that researchers set out to find. It appears St 1 and Chugoku 31 have wide spectrums of vertical resistance. (Table 12 shows the spectra of resistance of St 1 and Chugoku 31 and other resistant varieties in the IRBN tests.)

The "field resistance" of Japan creates confusion. The term "field resistance" is "borrowed" from the resistance in potato late blight. The original meaning should be kept. If the meaning is changed, another term should be created and the term "field resistance" should not be used. Van der Plank (1975b) condemned the terminology "true resistance" and "field resistance" also, for other reasons.

In evaluating the Japanese concept of field resistance, the work of Ezuka (1972) is helpful. He described the method of evaluating field resistance by 1) eliminating the effect of true resistance, 2) evaluating field resistance in the paddy field, and 3) evaluating field resistance in the blast nursery.

Basically the idea is similar to Van der Plank's (1975a), that if vertical resistance is eliminated, the rest is horizontal resistance. Though the concept appeared sound, two assumptions are questionable. First, it is assumed that the genetic composition of varieties in each group is known. Such knowledge is in fact incomplete. The fact that groups of varieties may have a common gene or genes does not mean they have no other resistance genes. Both Ezuka (1972) and Toriyama (1975) mentioned that the groups may be further divided by additional fungus strains. So far, only seven isolates have been used to group cultivars. There are many more in the field. For instance, Shin 2 was considered to have no resistance gene for a long time. But when it was tested with a Philippine isolate, a new allele, Pi-k^s, was found. Many other cultivars may be found to have many other vertical genes when more fungus isolates are tested. The classification of varieties is premature for use in screening for field resistance. The topic is discussed further in the next section "Genetics of blast resistance." Ezuka's second questionable assumption appears when he suggests using a fungus "known to be virulent to all the varieties under test." He underestimates the variability of the fungus. Both in the field and with artificial inoculation, there are many races with different pathogenicities to the varieties of each group. Toriyama (1972) stated :

In general, the composition of fungus races is not constant. It varies with year, location, and season. For example in Fukuyama, the strains belonging to N race propagate in the early part of the rice growth, then the strains belonging to the C race follow. This pheno-

menon of race change is repeated every year. Major fungus strains of the N racc collected in Fukuyama fields probably belonged to N-2 race because they showed virulence to the *Pi-a* gene of Aichiasahi type but did not attack the *Pi-i* gene of Ishikarishiroke type. The strains of the C race which followed the N race were estimated to belong to C-8 race, because they were virulent to *Pi-k* and *Pi-a* and were avirulent to *Pi-i.* In Fukushima, the fungus situation was different. Gene *Pi-k* did not express a resistant reaction was different. Gene *Pi-k* did not express a resistant reaction in the early stages of rice growth. In contrast, gene *Pi-a* showed moderate resistance because the major fungus strains in Fukushima were virulent to *Pi-k* and avirulent to *Pi-a.* Reactions in the field, therefore, did not directly indicate field resistance itself because of the complex reaction against races.

If the varieties have not only one or two common resistance genes, as a classification test indicates, but other unknown resistance genes as well, and if the fungus used for test has many races, it will be difficult to identify field (horizontal) resistance, as Van der Plank has warned.

A group of 63 cultivars from Japan (provided by Dr. Yoshimura, Dr. Ezuka, and others) with known level of "field resistance" and known R-genes (*Pi-a, Pi-i, Pi-k, Pi-z, etc.*) were tested at the IRRI blast nursery, 1977. The results (Table 21) confirm two points discussed above: 1) The cultivars with "field resistance" in Japan do not have field resistance in the conventional sense, i.e. horizontal resistance. Many of such "field resistant" cultivars are completely killed by leaf blast, whether in *rr* or *r* group. They are race-specific. 2) Among the cultivars in each group of specific R-genes, *Pi-a, Pi-i,* etc., some showed a susceptible reaction while others showed a resistant reaction, indicating other genes are involved besides the known specific genes. Field resistance cannot be measured by using a fungus strain virulent to *Pi-a* or *Pi-i* gene alone.

Table 21. Reaction of cultivars from Japan with "field resistance" and R-genes, tested at IRRI blast nursery, 1977.[a]

Variety	Gene type for true resistance	Field resistance[a] (Japan)	Reaction in blast nursery[b] (Philippines)
1. Chiyohikari	+	*rr-r*	*m*
2. Ou 247	+	*rr*	*m*
3. Tokai 26	+	*rr*	*m*
4. San'in 63	+	*rr*	*s*
5. Harima	+	*rr*	*m*
6. Murasaki-ine	+	*rr*	*ss*
7. Suzuhara-mochi	+	*rr*	*ss*
8. Rikuto Norin 24	+	*rr*	*m*
9. Ishloka 3	+	*rr*	*ss*
10. Sensho	+	*rr*	*s*
11. Fukuton	+	*rr*	*s*
12. Rikuto Norin Mochi 4	+	*rr*	*m*
13. Rikuto Norin Mochi 26	+	*rr*	*m*
14. Rikuto Norin 12	+	*r*	*m*
15. Honen Wase	+	*r*	*s*
16. Kansai 6	+	*r*	*m*

continued on opposite page

Table 21 continued

Variety	Gene type for true resistance	Field resistance[a] (Japan)	Reaction in blast nursery[b] (Philippines)
17. Saikai 90	+	*r*	*ss*
18. Tangin Bozu	+	*r*	*s*
19. Ukon Nishiki	+	*r*	*ss*
20. Kogane Nishiki	+	*r*	*ss*
21. Ginga	+	*r*	*s*
22. Kogane-masari	+	*r*	*s*
23. Koshiji Wase	+	*r*	*m*
24. Miyama Wase	+	*r*	*s*
25. Yamaji Wase	+	*r*	*s*
26. 65A-8	*Pi-a*	*rr*	*s*
27. Heiroku-mochi	*Pi-a*	*rr*	—
28. Rikuto Norin 11	*Pi-a*	*rr*	*m*
29. Shin Hakaburi	*Pi-a*	*rr*	*m*
30. Hideri-shirazu	*Pi-a*	*rr*	*m*
31. Kuroka	*Pi-a*	*rr*	*m*
32. Kirishima	*Pi-a*	*rr*	*m*
33. Kahei	*Pi-a*	*rr*	*m*
	Pi-a	*rr*	*m*
35. Yamabiko	*Pi-a*	*r*	*ss*
36. Fujiminori	*Pi-a*	*r*	*s*
37. Reimei	*Pi-a*	*r*	*s*
38. Homare Nishiki	*Pi-a*	*r*	*ss*
39. Tohonishiki	*Pi-a*	*r*	*m*
40. Himehonami	*Pi-a*	*r*	*m*
41. Mutsu-kogane	*Pi-a*	*r*	*m*
42. Sawanishiki	*Pi-a*	*r*	*s*
43. Asagiri	*Pi-a*	*r*	*ss*
44. Yoneshiro	*Pi-i*	*rr*	*m*
45. Joiku 232	*Pi-i*	*rr*	—
46. Kuiku 9	*Pi-i*	*rr*	*m*
47. Kukei 22	*Pi-i*	*rr*	*m*
48. Toyoma Wase	*Pi-i*	*rr*	*m*
49. Isao-mochi	*Pi-i*	*rr*	*ss*
50. Sorachi	*Pi-i*	*rr*	*rr*
51. Fujisaka 5	*Pi-i*	*r*	*s*
52. Todoroki Wase	*Pi-i*	*r*	*m*
53. Akishino-mochi	*Pi-a Pi-i*	*r*	*m*
54. Takasago-mochi	*Pi-a Pi-i*	*r*	*m*
55. Shinsetsu	*Pi-a Pi-i*	*r*	*m*
56. San'in 68	*Pi-k*	*r*	*rr*
57. Tatsumi-mochi	*Pi-k*	*r*	*rr*
58. Matsumae	*Pi-k*	*r*	*rr*
59. Ishikari	*Pi-a Pi-k*	*r*	*rr*
60. BR No. 1	*Pi-a Pi-k Pi-m*	*rr*	*rr*
61. Hokushin 1	*Pi-a Pi-k Pi-m*	*r*	*rr*
62. Ou 244	*Pi-z*	*rr*	*rr*
63. 54BC-68	*Pi-z*	*rr*	*rr*

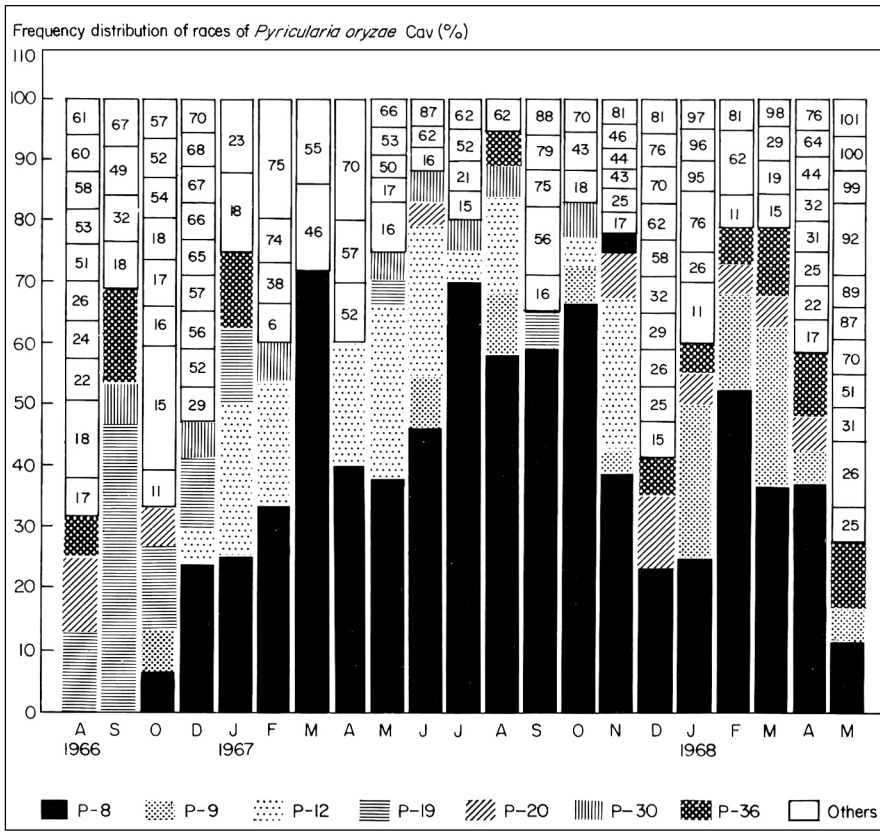
[a] *rr* (=1) = highly resistant, *r* (=2) = resistant, [b] *m* (=3,4) = moderately resistant, *s* (=5.6) = susceptible, *ss* (=7,8,9) very susceptible. Figures in parentheses are the numerical scale 1 to 9, adopted for international testing

Broad-spectrum vertical resistance

It is common knowledge that rice cultivars resistant in one country may be susceptible in another. The varying reaction may also be observed among different regions of a country. Table 22, presenting a portion of the data from the 1970–71 International Rice Blast Nurseries, demonstrates the point clearly.

Varietal reactions may also differ tremendously when tested at different times or seasons of a year in the same locality. Figure 4 illustrates the race composition from month to month in the IRRI blast nursery at Los Baños. Philippines. When the first 10,000 germplasm collection was screened at IRRI, 1,400 cultivars were highly resistant. The number decreased each time they were retested in succeeding months, and after five tests the number was about 400.

The fungus is, pathogenically highly variable. However, the survival and prevalence of a race depends upon the availability of host cultivars. Thus, after long association, *japonica* rices are readily infected by the prevailing race groups in Japan while many *indicas* are resistant. The reverse is true in the tropics.



4. Population of the Philippine race groups of *Pyricularia oryzae* Cav., sampled monthly at the blast nursery, Los Baños, Laguna, Philippines, from August 1966 to May 1968 (Quamaruzzaman and Ou 1970).

Table 22 International uniform blast nurseries, 1970–71 results[a] (R = resistant, M = moderate, S = susceptible, – = no data).

Variety	Iran 11	India 1234	Nepal 112	Ceylon 12312142	Bangladesh 1	Thailand 12343546354
1 CI 7787 (Zenith)	RR	MRMM	–––	R–––––––	R	RRRR––––RRM
2 CI 8985 (Lacrosse)	MR	SM–R	–––	S–RMRMRR	R	RMMRRRMRRMM
3 CI 1661-1 (Caloro)	SR	SS–R	–––	––M–––MM	R	MSSMMMSMRRS
4 CI 8970 (Purple)	––	–––––	–––	––––––––	–	––––––––––
5 CI 8970 (Straw)	S–	SS–S	–––	S–MMR–––	R	MSRSR–S–R–S
6 CI 5309	SM	SS–R	MS–	R–M–M–R–	R	RRRMRRM–R–M
7 PI 18006-1	SM	SSSS	SSR	S–MMMSSS	R	RRRMMRMMRRM
8 PI 201902	SM	SM–S	SSR	M–MMMMMM	M	RRRRSRMRRRM
9 PI 231128	RR	MMSM	–––	RRRMRRRR	R	RRRMMRMRRRM
10 PI 231129	MR	SRSR	–R–	RRMRMRRM	R	RRRMRRMRRMM
11 Tetep	SR	MRRR	–R–	RRRRRRRR	R	RRRRRRRRRRR
12 Tadukan	MR	MRRR	–––	RR–––RRR	R	RRRRRRMRRRM
13 Usen	SR	SR–S	–––	S–R––SMM	R	MSMSSRSRMSS
14 Chokoto	SM	SS–M	–––	R–MMM––	–	RRRM––––R––
15 Yakeiko	SR	S–R	–––	S–––––––	R	RRRM––M–RRM
16 Kanto 51	S–	S––M	–––	S––––MMM	R	RRRM–RMSRSM
17 Ishikari Shiroke	S–	–M–R	–––	S–––––––	–	RMRM–R–M–S–
18 Homare Nishiki	RR	M––R	–––	R––––RMM	R	RMMM–RMRRRM
19 Ginga	MR	MR–S	–––	M––––RMM	R	RMMMRRMRRM–
20 Norin 22	MR	SM–M	–––	–––MR–––	R	RMR––RM–RRM
21 Aichi Asahi	MR	SR–S	–MR	S–––RRMM	R	RSSMMRMRMRM
22 Norin 20	M–	SMSR	–––	M–––––––	R	RMM–––––RRS
23 Taichung 65	S–	SMMR	–––	S–––––––	–	MMR–––––––
24 Taichung 171	SR	SSSR	MS–	S–MMMSSS	R	MSSMSSSRMMS
25 Chianung Yu 280	MR	SSSM	MS–	S–MMMSSS	R	RSSMSMSMMSS
26 Chianung Yu 242	S–	SM–R	–––	S–––––––	R	RSRM––M–RMM
27 Kwang-fu 1	MR	SM–S	–––	M–––RR–––	R	RRRM––M–RRM
28 Pai-kan-tao	R–	MR–S	M––	––––––––	–	––R–––––––S-
29 Taichung Line 33	SR	SM–S	MS–	S––––M––	R	MRRMMRMSRMM
30 Kao-chio-liu-chou	SR	SSSM	SS–	S–MMMM––	R	SSSMSSSRSSS
31 Kaohsiung Ta-li-chen-yu	MR	SSSM	–S–	S––MMSSS	M	SSSMSMSMSSS
32 Taichung Ti-chio-wu-chen	SR	SS–R	SS–	S–RMM––R	R	MSSM–SMMSMS
33 Custugulcule	SR	SS–R	RR–	S––––MSM	R	RMR––M–MRMS
34 Natala	SS	SM–R	–MR	S––MM–––	R	MMMMRMMMRMS
35 Kanto 51	SM	S––S	–––	S–MMMSMS	M	RRR––RM–R–M
36 Nung-lin 21	RR	SM–R	–M–	M––M––SM	R	RSS–SMMRMSM
37 Senaho	RR	SM–S	RMR	S––M–SM	M	RMS–SRSR–––
38 Kung-Shan-wu-shen-ken	S–	MS–R	–––	S––––M––	R	MSS–––––––––
39 B 401	SM	SSSS	RMR	S–––––SS	R	MSS–SRSRRRM
40 C33-18	RR	MRSM	–––	RRMMSRRR	R	RMM–MRMMRMM

(*continued on next page*)

There have been numerous varietal resistance tests in many countries in the past; tests conducted in single countries, however, cannot assure us of the resistance levels of varieties.

Obviously international cooperative tests are the best means of identifying cultivars with broad resistance to blast. The idea of international blast nurseries was conceived in the late 1950's by the International Rice Commission (IRC)

Table 22 continued

Variety	Indonesia 12	Malaysia 1233	Taiwan 111	Philippines 1122222222222	Korea 11112222334
1 CI 7787 (Zenith)	RR	–RR–	RR–	MMRRRR–M–RMRR	RRR–RRRR–R–
2 CI 8985 (Lacrosse)	MM	RS–M	MMR	MMMSRRM–R–MR–	RRRRRRRRRMM
3 CI 1661-1 (Caloro)	SM	SSRS	SSR	RSSSSSSSSSSMM	SRRRSRRRM–M
4 CI 8970 (Purple)	––	----	---	----------	----------
5 CI 8970 (Straw)	SS	–SR–	SS–	SS–SS–S–S–S––	S–S–S------
6 CI 5309	RR	SS–S	SMR	MR–RR–MRRR–RS	SSRRM---R–S
7 PI 18006-1	MR	SSRS	SMR	MRRRRSRRRRRR–	MSMMRMMMMMS
8 PI 201902	RR	MSRS	SRR	RRRRRRRRRRRMM	RMRMMMRMMRM
9 PI 231128	RR	MMRM	RRR	RRMSSSSSMSMSM	RRRRRRRRRRR
10 PI 231129	RR	RRMM	RRR	RRMSSSSSMSMMS	RRRRRRRRRRR
11 Tetep	RR	RRMR	RRR	RRRRRRRRRRR–	RRRRRRRRRR
12 Tadukan	RR	RRRR	RRR	RSRRRRMRRRMRM	RRRRRRRRRR
13 Usen	RR	SMRS	MSS	SSMSSSSSMM–M–	RRRRRRRRRMR
14 Chokoto	––	–SR–	SM–	SSSRR–RR–RR––	S–RRR----M–
15 Yakeiko	RR	–SM–	SS–	MRRSRRR–RR–RM	SSS–SMSM–M–
16 Kanto 51	RR	–S––	SM–	SRRRRRRR–RR–R	SSMSSMSMM–M
17 Ishikari Shiroke	MR	–M––	SS–	MRRSSM––SSSS–	SMR–RMMMM––
18 Homare Nishiki	MR	SMRM	SSM	SMMSMMS–RSSS–	SSMMSMSMS–S
19 Ginga	MR	SM–R	SSS	MMRSMM––RSMMR	SSMSSMSMS–M
20 Norin 22	MR	–SRM	SSS	SMMSMMS–RSMRM	SSS–SSSS––R
21 Aichi Asahi	MR	MM–M	SSM	MMMSSSS––MMR–	SSMRSSSMSSS
22 Norin 20	RR	–SM–	SM–	MMMSMS––R––RR	SSM–SMSM–S–
23 Taichung 65	MR	–M–R	SS–	SMMSM––SMM–S–	S–M–S-----
24 Taichung 171	MM	SSMS	MSM	SSMSSSSSSSS–	MMMRRRRRMSM
25 Chianung Yu 280	MM	SSSS	RSM	MSSSSSS–SSSSS	RSMRRRRRMSR
26 Chianung Yu 242	MR	–RMR	SS–	RRSSSS–S---M–	RRR–RRRM–R–
27 Kwang-fu 1	MR	MR––	SSM	RRRSMRMSRMRRR	RRR–RRRR–M–
28 Pai-kan-tao	––	– – –	R––	RSRSR–M–RRMR–	––R–R------
29 Taichung Line 33	MM	MR–S	SSM	MRSSSS–S–SSR	RMRRRMRMMRM
30 Kao-chio-liu-chou	SM	SSSS	SSM	RSSSSSSSS–SR–	SSMMSSSSSSM
31 Kaohsiung Ta-li-chen-yu	MR	SSSS	SSS	SSSMSSSSSSSSS	MSSSSMSMSSR
32 Taichung Ti-chio-wu-chen	SS	SSSS	SSM	SSSSSSSSMSS–S	SSSMSMSSMSM
33 Custugulcule	MR	MRMS	SS–	MRSSSSS–MSSR–	MSR–MMSS–M–
34 Natala	SM	MRSS	SMR	MRSSSSS–SSS––	RRRRRRRRRRR
35 Kanto 51	RR	SSMM	MMR	SRRMS–RRRR–M–	SSMMS---MMS
36 Nung-lin 21	MR	SSRS	SMR	SSSSSSSS–SRSM	RMRRMRR–MMR
37 Senaho	MR	SMMM	MMR	SSRSSSS––SSMR	–MRRRRRRMMR
38 Kung-Shan-wu-shen-ken	MS	–SM–	SS–	SSRSSR-------	SSSSSSSS–S–
39 B 401	MM	SSSS	SSS	SSSSSSSSSSM–	RSSSRRMRSSR
40 C33-18	RR	MMMM	MMR	RMRRSRMRRRRRR	RRRRRRRRRMR

(continued on opposite page)

Working Party. After a symposium on rice blast in 1963, IRRI assumed the responsibility of coordinating the program, multiplying seeds, and compiling results. The data were published in the IRC Newsletter every 2 years until 1971, when the volume of data could not be accommodated in the Newsletter.

The program started with 258 cultivars, including 38 race differentials; some cultivars were known to be resistant or susceptible; most were commercial

Table 22 continued

Variety	Guyana 111111	Colombia 1111112	Panama 1	Brazil 11	Peru 11	Bolivia 1	Italy 11	Liberia 1
1 CI 7787 (Zenith)	SSS––R	M–––RR	–	MR	SS	S	–M	S
2 CI 8985 (Lacrosse)	SSSRRR	MSRMMRR	–	MR	SS	S	MM	M
3 CI 1661-1 (Caloro)	SSMRSS	SSSSSSR	–	MR	SS	S	MM	M
4 CI 8970 (Purple)	–––––	– – – – –	–	––	––	–	––	–
5 CI 8970 (Straw)	S–––SS	––––– MR	–	–M	SS	S	–S	–
6 CI 5309	S–––MR	R–RMR––	–	––	MS	–	RR	–
7 PI 18006-1	MMSRMR	MSRMMMR	R	SR	SS	S	MR	S
8 PI 201902	RSRRMR	SMRMMRR	M	MR	MS	S	MM	M
9 PI 231128	RRRRRR	RRRRRRR	M	MR	RR	M	RR	R
10 PI 231129	RRRRRR	RRRRRRR	M	RR	RR	M	RR	M
11 Tetep	RRRRRR	RRRRR––	R	R–	RR	R	RR	R
12 Tadukan	RSSRMM	MSRRRRR	R	RR	MR	R	RR	M
13 Usen	SSSMSS	SSMSS––	–	MR	MS	S	R–	S
14 Chokoto	R–––––	– – – – RR	–	–R	SR	S	–M	–
15 Yakeiko	RMM––R	RMR––RR	–	M–	MM	–	M–	M
16 Kanto 51	RMM–MR	RMRM–RR	S	MR	MM	S	MM	M
17 Ishikari Shiroke	SSM–––	MMSS–SM	M	M–	SS	S	M–	R
18 Homare Nishiki	MSMMMM	SSSRMRR	–	M–	MR	S	S–	M
19 Ginga	MSMMMM	SSMSRRR	–	S–	–R	S	M–	–
20 Norin 22	MSM–SM	RSMR–RR	–	M–	–R	S	M–	M
21 Aichi Asahi	RSMSR	SSMMM–	M	M–	SS	S	M–	M
22 Norin 20	RSS–SM	SS– – – –	–	RR	MM	–	SM	–
23 Taichung 65	S–––S–	– – – – – R	M	–R	MM	S	––	–
24 Taichung 171	SSSRSS	SSSSSSM	M	SR	MS	S	RR	S
25 Chianung Yu 280	SSSMSS	SSSSSSR	M	SR	MS	S	MR	S
26 Chianung Yu 242	SSS–SS	SS–––SM	M	M–	SS	S	R–	M
27 Kwang-fu 1	SMS–SS	SSM––RR	R	RR	RR	S	RM	R
28 Pai-kan-tao	–––––	– – – – SR–	–	–R	––	S	–M	–
29 Taichung Line 33	MSSSSS	SSSS–SR	M	SS	SS	S	MM	R
30 Kao-chio-liu-chou	SSSSSS	SSS––SM	S	SS	SS	S	MS	S
31 Kaohsiung Ta-li-chen-yu	SSSSSS	SSSSSSR	S	SM	MS	S	SS	S
32 Taichung Ti-chio-wu-chen	SSSSSS	SSS–SSM	S	SM	MS	S	MM	S
33 Custugulcule	SSSSSS	SS—SSR	M	SR	MS	S	RM	M
34 Natala	SSSSSS	SSSSSS	S	SR	SS	S	MM	R
35 Kanto 51	M–––RR	R–RMM––	R	––	RR	–	R–	–
36 Nung-lin 21	SSSMSM	SSSSSRR	S	SR	MS	S	RM	S
37 Senaho	SSSRSM	SSSSSSM	M	SR	MS	S	RM	S
38 Kung-Shan-wu-shen-ken	SSS––S	SS–––SS	S	S–	SS	S	S–	S
39 B 401	SRSSSS	SSSSSSM	S	SM	MS	S	MS	S
40 C33-18	RSSRMR	RMRRMRM	R	RR	RR	S	RR	S

(continued on next page)

cultivars. Those were the group I cultivars. In 1965, an additional 321 cultivars were included, selected from repeated tests of the first 10,000 germplasm acquisitions of IRRI. They constituted the group II cultivars. Both groups were used to identify the most resistant cultivars. Many susceptible cultivars were deleted and others were added in subsequent years. The program now also evaluates new cultivars and promising lines.

The program now involves some 60 test stations in 30 countries and has

Table 22 continued

Variety	Nigeria 123211	Senegal 1	Egypt 1	S. Leone 11	TOTAL R	TOTAL M	TOTAL S
1 CI 7787 (Zenith)	MSRMRM	M	R	M–	39	16	8
2 CI 8985 (Lacrosse)	SSRS–M	S	R	M–	37	30	15
3 CI 1661-1 (Caloro)	S–S–SS	S	S	SS	17	18	46
4 CI 8970 (Purple)	−−−−−−	−	−	−−	−	−	−
5 CI 8970 (Straw)	S–SRSS	S	S	S–	8	5	38
6 CI 5309	MMRMSR	M	S	S–	31	18	17
7 PI 18006-1	SSSSMS	S	M	SS	29	30	33
8 PI 201902	MSRRMR	M	M	MS	40	37	15
9 PI 231128	RMRMRR	R	R	MS	61	19	9
10 PI 231129	RMRMRR	R	R	RR	64	19	9
11 Tetep	RRRRMR	M	R	MR	82	5	1
12 Tadukan	RMRRRR	R	R	RR	71	13	4
13 Usen	SSMSMS	S	R	MM	22	20	37
14 Chokoto	– SMM – –	−	M	M–	20	13	12
15 Yakeiko	MMRMSR	S	S	MR	27	21	15
16 Kanto 51	RSRMSR	M	R	MM	29	28	17
17 Ishikari Shiroke	SMMMS–	S	S	MR	11	24	24
18 Homare Nishiki	SSMMRM	M	M	MR	20	35	23
19 Ginga	MRM–M–	M	R	R–	11	33	19
20 Norin 22	MSM–R–	M	M	R–	20	26	21
21 Aichi Asahi	SSMMM–	M	S	SM	15	34	31
22 Norin 20	MSM–MR	M	S	M–	14	23	17
23 Taichung 65	MS – – MR	S	M	R–	8	18	17
24 Taichung 171	SSSSSS	S	R	MS	14	23	54
25 Chianung Yu 280	–SSS – –	S	M	MS	14	21	52
26 Chianung Yu 242	SSS–RM	S	R	M–	20	15	26
27 Kwang-fu 1	MRMMRM	S	M	R–	38	21	14
28 Pai-kan-tao	–S– – – –	−	R	− −	15	5	7
29 Taichung Line 33	SSS–M–	S	M	SS	17	25	37
30 Kao-chio-liu-chou	SSSSSS	S	M	SM	5	15	65
31 Kaohsiung Ta-li-chen-yu	SSSMSS	S	M	MM	4	19	66
32 Taichung Ti-chio-wu-chen	MSS–SS	S	M	MS	5	21	59
33 Custugulcule	MSS–SS	S	S	MM	16	22	38
34 Natala	SSS–SS	S	M	SS	21	19	43
35 Kanto 51	RSMRSR	−	R	MM	26	24	16
36 Nung-lin 21	SSS–SS	S	R	MS	21	21	41
37 Senaho	SSM – SM	M	M	MS	21	27	34
38 Kung-Shan-wu-shen-ken	SSSRSS	S	S	SS	5	5	46
39 B 401	SSSRSS	S	S	MS	14	13	16
40 C33-18	RSMRRR	S	R	RS	55	25	10

[a]Testing localttles In each country: IRAN – Rasht; INDIA 1-Ponnapet, 2-Anakapalle, 3-Nellore, 4-Rajendranagar; NEPAL 1-Khumal, 2-Powanipur; CEYLON 1-Karapincha, 2-Peradeniya, 3-Karadian Aru, 4-Bombuwela; BANGLADESH – Joydevpur; THAILAND 1-Sako Nakorn, 2-Rajburi, 3-Bangkhen, 4-Kuan Gut, 5-Khon Khaen, 6-San Patong.
INDONESIA 1-Bogor, 2-Hampegan; MALAYSIA 1-Bumbong Lima, 2-P.E.S. Bt. Merah, 3-Paya Paloh; TAIWAN – Chiayi; PHILIPPINES 1-Tibak, 2-IRRI; KOREA 1-Milang, 2-Suwon, 3-Iri, 4-Radiation Research Center.
GUYANA – Timber, COLOMBIA 1-La Libertad, 2-Corinto; PANAMA – Divisa; BRAZIL – IRGA; PERU – Yurimaguas; BOLIVIA – Saavedra; ITALY – Vercelli; LIBERIA – Suakoko.
NIGERIA 1-IITA, Ibadan, 2-FDAR, Ibadan, 3-Badeggi; SENEGAL – Cosamonce; EGYPT – Sakha; Sierra Leone-Njala.

collected more than 300 test results. The cultivars tested have been exposed to a wide range of fungus races in all of the major rice-growing areas of the world for more than 10 years. The program provides the most extensive tests ever made for blast resistance.

The summary of results (Table 23) shows that no cultivar has been resistant in all tests, nor has any cultivar been susceptible at all locations. The spectrum of resistance, however, has varied from 98% in Tetep to about 20% in Fanny. (The range of resistance reactions may well represent the spectrum of resistance to the fungus races.) There have been graduations between. The broader cultivar's spectrum of resistance, the higher its level of resistance: very few races can infect the cultivar. Susceptibility means a very narrow spectrum of resistance: most races can infect susceptible cultivars.

The resistance of such cultivars as Tetep is vertical; Tetep is susceptible in a few tests and to a few isolates (Table 13).

I believe also that the resistance in Tetep and other resistant varieties is multigenic, and will discuss that statement later. I accept the concept of Nelson (1973) that the accumulation of vertical resistance in a single genetic background may contribute to or confer horizontal resistance. Aa was shown earlier (Fig. 3. Table 13), Tetep has behaved as if it has horizontal or general resistance.

Relationship between qualitative and quantitative resistance

"Qualitative" is used here to indicate whether a cultivar is resistant or susceptible. The type of lesion, not the number, is considered in determining qualitative resistance. All studies on race differentiation, and most varietal resistance

Table 23. Varieties most resistant to blast (International Blast Nursery. 1964-73 results summary).

IRRI acc. no.	Variety	Varieties (no.)			Total tests	Resistant (%)
		R	M	S		
Group 1		*1964–73*				
	Tetep	272	24	6	302	98.0%
	Nang chet cuc	210	70	12	292	95.9%
	Tadukan	249	43	17	309	94.5%
	Trang cut L. II	186	62	15	263	94.3%
	Pah Leuad III	199	44	15	258	94.2%
	C-46-15	240	48	19	307	93.8%
	Mekeo White	166	90	20	276	92.8%
	H-5	225	65	23	314	92.7%
	R-67	189	80	22	291	92.4%
	Ram Tulasl (Sel)	217	56	24	297	91.9%
	C1 7787	185	70	23	278	91.7%
	Ta-poo-cho-z	217	36	24	277	91.3%
	D-25-4	214	50	28	292	90.4%
	M-302	206	74	30	310	90.3%
Susceptible varieties						
	Kung Shan-wu-shen-ken	26	34	186	246	24.4%
	Fanny	27	22	203	252	19.5%

continued on next page

Table 23 continued

IRRI acc. no.	Variety	Varieties (no.) R	M	S	Total tests	Resistant (%)
Group 2		*1966–73*				
3441	Mamoriaka	178	44	5	227	98.8%
5993	Carreon	190	31	6	227	97.4%
178	Ram Tulasi (Sel)	158	31	5	194	97.4%
6786	C 46–15	177	46	6	229	97.4%
7802	Dissi Hatif	172	45	6	223	97.3%
181	Ram Tulasi	174	31	6	211	97.2%
5735	Ahmee Puthe	107	25	4	136	97.1%
6350	Ca 435/b/5/1	154	45	6	205	97.1%
6274	Thava Lakkanan P+ b 9	171	44	7	222	96.9%
4619	Huan-sen-go	186	22	8	216	96.3%
175	Pah Leuad 29-8-III	159	51	10	220	95.5%
5827	Macan Tago	101	47	7	155	95.5
6349	Ca 902/b/2/1	168	36	11	215	94.9
154	M-302	166	54	12	232	94.8
6347	Ca 902/b/3/3	170	44	12	226	94.7
3700	NP-97	138	74	12	224	94.7
9011	R-67	148	43	11	202	94.6
3311	C1 2011-1	138	36	10	184	94.6
6382	Ca 902/b/2/2	165	42	12	219	94.5
8517	DZ 193	153	65	13	231	94.4
8593	DL-5	164	45	13	222	94.2
5753	Badshabhog	164	45	13	222	94.2
6433	T-23	152	55	13	220	94.1
3375	Secano Do Brazil	150	56	13	219	94.1
6836	S 39 J.K.W.	152	47	13	212	93.9
3440	Unblatuzi Valley Sugar Co	149	65	14	228	93.9
3392	T1	141	75	14	230	93.9
7368	PI 184675-4	154	58	14	226	93.8
3716	N-12	149	62	14	225	93.8
6734	Pi 3	127	36	11	174	93.7
6951	E1 Gopher	150	44	13	207	93.7
8350	Pusur	145	62	14	221	93.7
3717	N32	133	72	14	219	93.7
6294	T1	134	76	14	224	93.7
8375	DNJ-60	134	76	14	224	93.7
3702	NP 130	116	92	14	222	93.7
3694	T3	152	66	15	233	93.6
6447	Basmati T3	159	54	15	228	93.4
8599	DL-12	152	56	15	223	93.3
5801	W.R.C. No. 4	150	41	14	205	93.2
4132	BMT 53R 3540	148	65	16	229	93.0
6062	Leter 08	122	65	14	201	93.0
6822	S 20 J.K.W.	170	39	16	225	92.9
3443	Somavari	159	51	16	226	92.9
6394	Amritsari HR 22	149	58	16	223	92.8

tests in the past have used that qualitative concept. Quantitative resistance takes into account the extent of disease or the number of lesions. After examining the data from the many resistance tests, I believe that resistance to blast

Table 24. The relative numbers of race blast lesions caused by *Pyricularia oryzae* on three rice lines in the field, and the reactions of the lines to 105 field isolates of 26 races in greenhouse (Ou et al 1975).

Rice line	Lesions (no./ tiller) In field	Lesions (no./10 seedlings) in greenhouse	Pathogenic isolates (no.)	Pathogenic races (no.)
IR442	73	35	94	24
IR2031	36	11	68	18
IR1514	3	1	6	3

should be measured not only qualitatively but more importantly, on a quantitative scale.

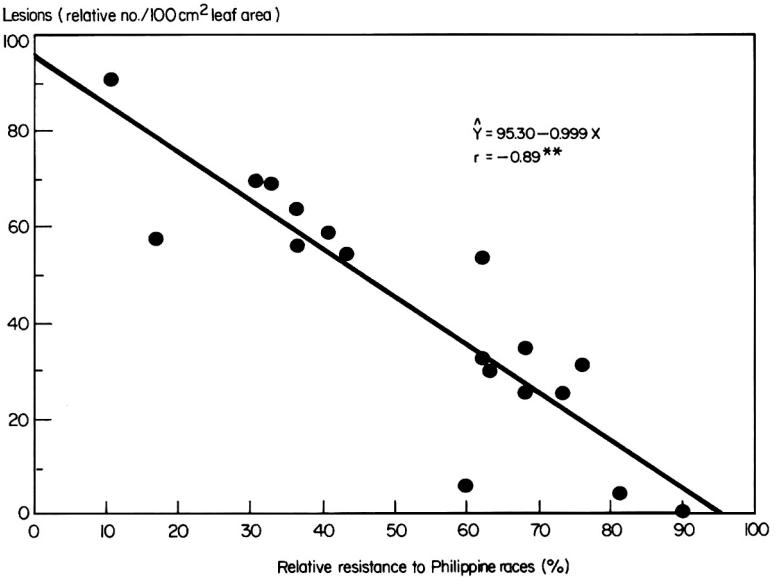
Recently a close relationship has been found between qualitative and quantitative resistance. We found all varieties in an upland varietal trial affected by blast (Ou et al 1975); qualitatively they were all considered susceptible. However, they differed greatly in the number of lesions on the leaves (quantitative); some had few lesions; some had so many that the leaves were killed. To discover why, an experiment was conducted with three rice lines. One line represented the cultivars or lines that show few lesions; another, those with intermediate numbers of lesions; and still another, those with many lesions. One hundred and five single lesions were collected at random from the field, isolated, and used to inoculate the 3 lines and 12 Philippine differential cultivars to identify the races.

The rice lines with the fewest lesions were resistant to most of the 105 isolates and most of the 26 races identified (Table 24). Thus, the quantitative difference in resistance observed in the field (number of lesions) reflected the qualitative (race reaction) resistance of the rice lines.

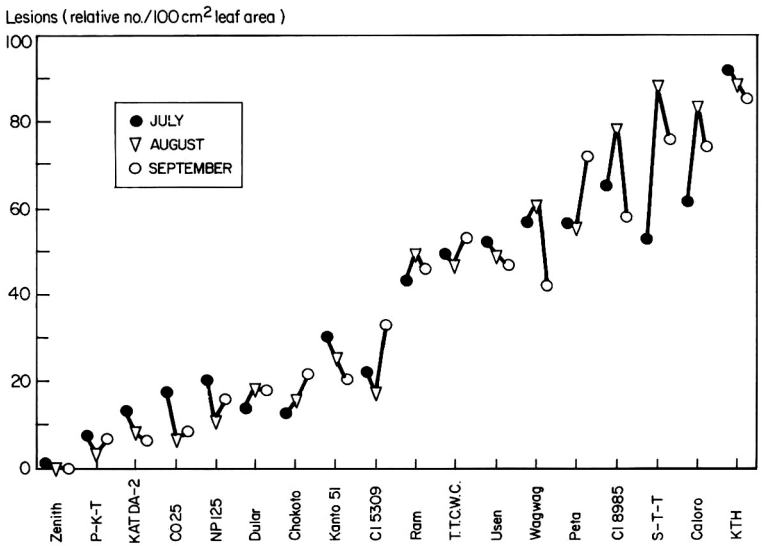
During the last 12 years, at IRRI we have inoculated more than 3,500 isolates of the blast fungus from the Philippines, and 262 races have been identified using the 12 Philippine differentials. The eight international differentials have also been involved in 242 of the races. Thus, we know how many races can infect each of the 18 differential varieties (2 of the varieties in each set being the same), and we know the percentage of resistance to the 242 races. For instance, if variety A is infected by 25% of the 242 races, its susceptibility is 25% or its resistance is 7%.

The 18 varieties were exposed to the blast nursery, and the lesions on each variety were counted. A close negative correlation existed between the percentage of resistance to races and the number of lesions (Fig. 5). The higher the percentage of resistance to races, the fewer the lesions. Further tests will show even closer correlation, as not all races are present at any one time. Monthly (seasonal) fluctuation in the numbers of lesions (Fig. 6) may also have been due to the presence of different race groups at different times. The trend, however, remained unchanged.

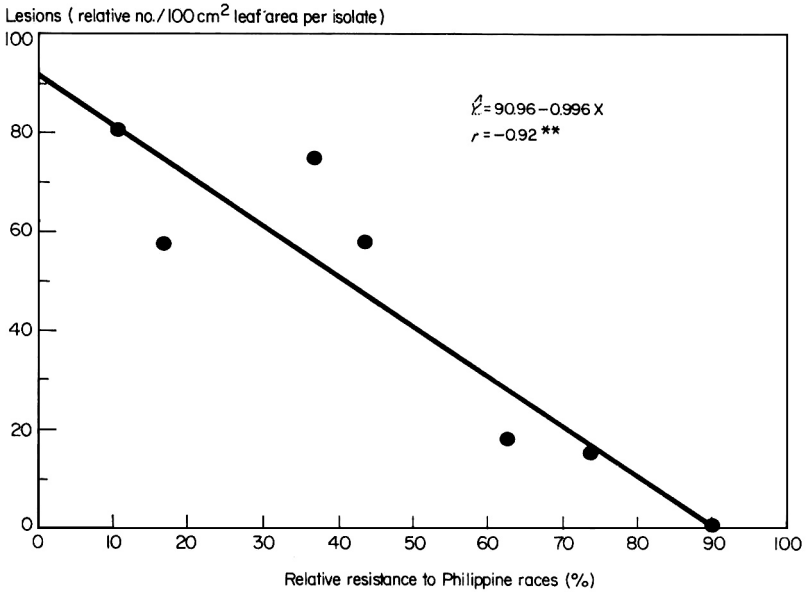
Forty isolates also were obtained from a blast nursery. They were artificially inoculated on seven of the differential cultivars that have various percentages



5. The relationship between quantitative resistance of 18 varieties in IRRI blast nursery and qualitative resistance (%) to the 242 identified Philippine races of *Pyricularia oryzae.* (Average number of lesions from 6 times of exposure.)



6. Number of susceptible type lesions of each of 18 varieties under natural infection in IRRI blast nursery in 3 tests in July, August, and September 1976.



7. The relationship between quantitative resistance in artificial inoculation with 40 isolates of *Pyricularia oryzae* and qualitative resistance to 242 Philippine races.

of resistance to known Philippine races, and the lesions were counted. The average number of lesions on each variety again was negatively and closely correlated with its resistance (Fig. 7).

These experiments indicate:

1. there are many races in a field;
2. many resistance genes are involved, although the genes have not yet been exactly identified;
3. the level of resistance to blast may be high if many resistance genes are present in a variety.

GENETICS OF BLAST RESISTANCE

Summary of information

Scattered genetic studies on blast resistance started before physiologic races were generally recognized (Takahashi 1965). Many such studies used specific races (see Ou 1972a). From these studies it appears that resistant varieties carry one, two, or, occasionally, three genes for resistance, and most of the genes are dominant. Those scattered studies have yielded fragmentary information that has given many people the false impression that resistance to blast is simple. It is true that inheritance of resistance to a specific race or isolate is relatively simple, but the problem is complicated by the large number of races in the field and their ability to change.

The most extensive work has been done during the last 10 years by Kiyosawa and his colleagues in Japan. Some 20 or more major papers have been published, the earliest being a study by Yamasaki and Kiyosawa (1966). Some of them are reviews (Kiyosawa 1967, 1971, 1972, 1974, 1976).

Kiyosawa and other workers have constantly used seven fungus strains, p-2b (race N-2), Ken 53-33 (T-1), Ina 72 (C-3). Hoku 1 (N-1), Ken 54-20 (N-2), Ken 54-4 (N-3), and Ina 168 (N-4), and tested them on many parental cultivars and on progenies of various cross-combinations. They also have some mutants

Table 25a. Resistance genes identified in some exotic varieties (Kiyosawa 1971).

Origin	Variety	Genes identified	Literature
Korea	Doazi chall	*Pi-i*	Kiyosawa 1968a
	Jae Keum	*Pi-a*	Klyosawa 1967c
	Pal tal	*PI-a*	
China	Usen	*Pi-a, others*	Kiyosawa 1967c
	Yakei-ko	*Pi-k*	Kiyosawa 1968b
	Reishiko	*Pi-k*	
	To-to (short grain)	*Pi-k, Pi-a*	
	Choko-to	*Pi-k, Pi-a*	
	Hokushi Tami	*Pi-k, Pi-a, Pi-m*	
	Pe Bi Hun	*Pi-a*	Kiyosawa 1967c
	To-to (long grain)	*Pi-k*^{s}	Kiyosawa 1969a
	Taichung 65	*Pi-k*^{s}	
	Sha-tiao-tsao	*Pi-k*^{s}	
	Oka-ine	*Pi-ta*	Kiyosawa 1969c
	Pai-kan-tao	*Pi-ta,* others	Kiyosawa et al 1971
Philippines	Tadukan	*Pi-ta* and/or *Pi-ta*^{2}	Kiyosawa 1966a, 1967b, 1969c
India	HR-22	*Pi-k*^{p}, others	Kiyosawa and Murty 1969
	CO. 25	*Pi-z*^{t}, *Pi-a,* others	Kiyosawa and Yokoo 1970
	TKM 1	*Pi-z*^{t}, others	Yokoo and Kiyosawa 1970
	Charnack	*Pi-k*^{s}, others	Kiyosawa, unpublished
	CO. 4	*Pi-z*^{t}	Fujimaki and Yokoo 1971
Pakistan	Dular	*Pi-k*^{s}, others	Kiyosawa, unpublished
	Pusur	*Pi-k*^{p}, *Pi-a,* others	Kiyosawa 1969b
Vietnam	Te-tep	Pi-k^s, others	Kiyosawa, unpublished
Malaysia	Morak Sepilai	*Pi-z*^{t}	Fujimaki and Yokoo 1971
	Kontor	*Pi-z*^{t}	
Thailand	Leuang Tawing 77-12-5	*Pi-z*^{t}	Fujimaki and Yokoo 1971
	Chao Leuang 11	*Pi-z*^{t}	
U.S.	Zenith	*Pi-z, Pi-a*	Kiyosawa 1967a
	Caloro	*Pi-k*^{s}	Kiyosawa 1969a
	Lacrosse	*Pi-k*^{s}	
	Blue Bonnet	*Pi-a*	Kiyosawa 1967c
USSR	Roshia No. 33	*Pi-k*^{a}	Kiyosawa, unpublished

[a]Any allele at the *Pi-k* locus.

of the seven fungus strains and of a Philippine strain, Ken Ph-03.

Yamasaki and Kiyosawa (1966) first found three genes: *Pi-a* from the Aichi Asahi type of variety, *Pi-i* from the Ishikari Shiroke type, and *Pi-k* from the Kanto 51 type. They concluded, "Dominance of these blast resistance genes is not absolute. Gene *Pi-k* and *Pi-i* show variation from complete dominance to incomplete dominance according to environmental conditions, although gene *Pi-a* shows complete dominance. It seems that the three genes behaved independently."

As more varieties were tested, more genes were found, such as *Pi-ta* from Tadukan and *Pi-z* from Zenith. Also, many allelic genes were found. To date 13 genes have been identified, 3 of them in 3 independent loci. 4 (*Pi-k,* Pi-k^{s}, *Pi-k^{p}, Pi-k^{h}*) at locus *Pi-k,* 2 (*Pi-ta, Pi-ta*^{2}) at *Pi-ta,* and 2 (*Pi-z, Pi-z^{t}*) at *Pi-z.* To which chromosome the three loci belong is not known (Kiyosawa 1976). Shinoda et al (1971), however, reported that *Pi-k* is located on *la* linkage group of chromosome 9, *Pi-ta* on *fs* linkage group of chromosome 1, and *Pi-z* on *wx* linkage group of chromosome 6. In addition, Toriyama et al (1968) identified a single dominant gene, *Pi-f,* for "field resistance" of cultivars St 1 and Chugoku 31.

Among the 13 genes, only 2 were found in japonicas: the others came from exotic cultivars. Kiyosawa (1971) summarized the information on genes found in exotic rice varieties (Table 35a). Table 25b supplements his list and includes genes from Japanese and other cultivars. Kiyosawa (1976) summarized the information (Table 26) on the basis of the resistance gene of the host and the reaction to eight fungus strains.

Kiyosawa (1967) also studied linkages of the genes for genes known up to that time (Fig. 8).

Rice varieties were classified into groups on the basis of reactions to the seven fungus strains (Table 27a, b; Kiyosawa 1967).

Table 25b. Resistant genes identified in some *japonica* rices and those not included in Table 25a.

Origin	Variety	Genes identified	Literature
Japan	Aichi Asahi	*Pi-a*	Yamasaki and Kiyosawa, 1966
	Ishikari Shiroke	*Pi-i*	
	Kento 51 (Toto-China)	*Pi-k*	
	Shen 2	*Pi-k^{s}*	Kiyosawa 1969
	Chugoku 31, St 1	*Pi-f*	Toriyama et al 1968
Indica	BL8 Tjina	*Pi-b*	
	Milek Kuning	*Pi-b*	
	Tjahaja	*Pi-b*	Kiyosawa 1972
	BL10 (Tjahala), Bengawan	*Pi-b, Pi-t*	
USA	Dawn	*Pi-a, Pi-i, Pi-k,* others	Kiyosawa 1974
Korea	Most of 86 varieties	*Pi-a, Pi-i, Pi-k*	Cho and Kiyosawa 1973
China (Taiwan)	Taichung Glu. Yu 26	*Pi-a, Pi-i*	Ezuka 1973

Table 26. Resistance genes so far found and their functional range (Kiyosawa 1976).[a]

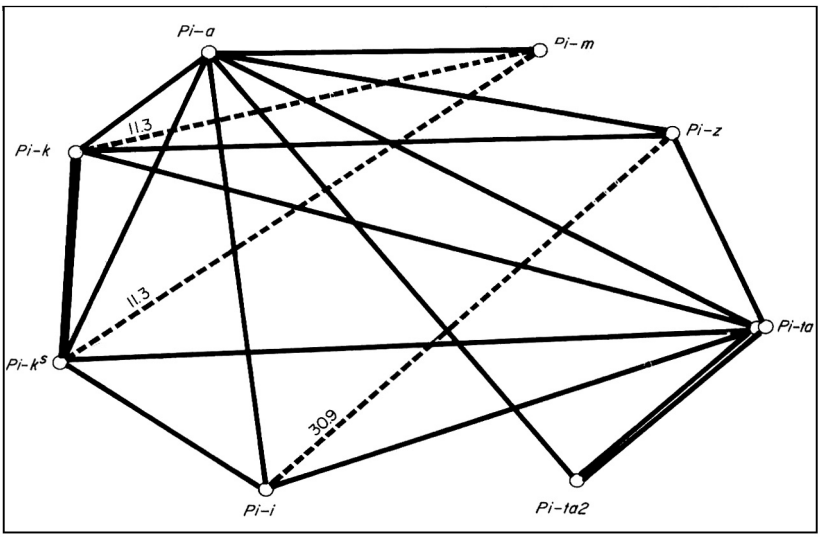
Resistance gene	Fungus strain							
	P-2b	Ken 53-33	Ina 72	Hoku 1	Ken 54-20	Ken 54-04	Ina 168	Ken Ph-03
Pi-a	S	S	R	S	S	S	R	S
Pi-i	M	S	M	S	MS	MR	M	S
Pi-k	MR	S	S	R^h	R^h	R^h	R^h	R^h
Pi-k^a	S	S	S	S	S	S	S	R^h
Pi-k^p	S	S	S	R	R	R	R	R
Pi-k^h	MR	S	S	R	R	R	R	R
Pi-ta	S	S	M	MR	M	MR	S	S
Pi-ta^2	S	M	MR	R	MR	R	M	
Pi-z	M	M	M	MR	M	MR	M	
Pi-z^t	R^h	R^h	R^h	R^h	R^h	R^h	R^h	
Pi-b	MR	M	M	MR	M	MR	MR	
Pi-t	R	M	M	MR	M	M	M	
Pi-m		S	S		M	MR	M	
	Avirulence (without +) or virulence (with +) genes							
	Av-a$^+$	*Av-a*$^+$	*Av-a*	*Av-a*$^+$	*Av-a*$^+$	*Av-a*$^+$	*Av-a*	*Av-a*$^+$
	Av-i	*Av-i*$^+$	*Av-i*	*Av-i*$^+$	*Av-i*	*Av-i*	*Av-i*	*Av-i*$^+$
	Av-k	*Av-k*$^+$	*Av-k*$^+$	*Av-k*	*Av-k*	*Av-k*	*Av-k*	*Av-k*
	Av-ks$^+$	*Av-ks*$^+$	*Av-ks*$^+$	*Av-ks*$^+$	*Av-ks*$^+$	*Av-ks*$^+$	*Av-ks*$^+$	*Av-ks*
	Av-kp$^+$	*Av-kp*$^+$	*Av-kp*$^+$	*Av-kp*	*Av-kp*	*Av-kp*	*Av-kp*	*Av-kp*
	Av-kh	*Av-kh*$^+$	*Av-kh*$^+$	*Av-kh*	*Av-kh*	*Av-kh*	*Av-kh*	*Av-kh*
	Av-ta$^+$	*Av-ta*$^+$	*Av-ta*	*Av-ta*	*Av-ta*	*Av-ta*	*Av-ta*$^+$	*Av-ta*$^+$
	Av-ta^{2+}	*Av-ta*^{2}	*Av-ta*^{2}	*Av-ta*^{2}	*Av-ta*^{2}	*Av-ta*^{2}	*Av-ta*^{2}	
	Av-z	*Av-z*	*Av-z*	*Av-z*	*Av-z*	*Av-z*	*Av-z*	
	Av-zt	*Av-zt*	*Av-zt*	*Av-zt*	*Av-zt*	*Av-zt*	*Av-zt*	
	Av-b	*Av-b*	*Av-b*	*Av-b*	*Av-b*	*Av-b*	*Av-b*	
	Av-t	*Av-t*	*Av-t*	*Av-t*	*Av-t*	*Av-t*	*Av-t*	
		Av-m$^+$	*Av-m*$^+$		*Av-m*	*Av-m*	*Av-m*	

[a]Reaction by the injection method for inoculation and recording (Kiyosawa 1974b)

Goto (1970), using the sheath-inoculation method, studied the cumulative effect of resistance genes. He inoculated two crosses with three fungus isolates. In Sensho × H-79 he found three independent genes—Rb1, Rb2, and Rb3—whose alleles contributed different degrees of resistance cumulatively when the virulence of an isolate and the host plant's resistance to it were each estimated by an index. He contends that "the reaction of a host plant to a pathogen may be due to the sum of resistance potential of host genes as compared with the virulence potential of pathogen genes." There is some similarity between his concept of resistance and the quantitative resistance mentioned above.

Kiyosawa (1972), Takahashi (1965), and Goto (1970), with some modifications, advocated the concept of a gene-for-gene relationship between the fungus strains and host varieties.

These recent studies have provided a great deal of new information; and they encompass all we know about blast resistance. The work of Kiyosawa and his colleagues seems to indicate that when more varieties are studied,



8. Linkage relationship among genes (Kiyosawa 1967).

Table 27a. Classification of rice varieties on the basis of resistance to blast (Kiyosawa 1967).

Variety	Classification of rice varieties		
	Kiyosawa	Goto and Yamanaka	Iwata and Narita
Zenith	Zenith type	I	A
Te-tep, Tadukan	Shimokita type	II	
Pi No. 1			
Pi No. 3	Pi No. 4 type		
Choko-to	To-to type	III	B
Yakei-ko, Reishiko, Kanto 51, Kanto 53	Kanto 51 type	IV	C
Ishikari Shiroke, Norin 34	Ishikari Shiroke type	V	E
Fujisaka 5		VI	
Homare Nishiki, Shuho, Ayanishiki	Aichi Asahi type		F
Aichi Asahi, Norin 17		IX	
Ginga	Shin 2 type	VII	G
Norin 22, Shin Yamabuki		VIII	
Norin 20		X	
Shinsetsu[a]	Shinsetsu type		D

[a]This variety was not tested by Department of Physiology and Genetics, National Institute of Agricultural Sciences, Hiratsuka, Kanagawa, Japan.

Table 27b. Rice varieties belonging to each type of blast resistance (Kiyosawa 1976).

Japanese varieties		Foreign varieties
Designated varieties[a]	Others	
Shin 2 type Shin 2, Norin 25, Norin 1, Tozan 38, Norin 22, Norin 8, Omachi, Koshiji Wase, Shin Yamabuki, Shirogane, Hatsunishiki, Tedori Wase, Koshihikari, Honen Wase, Norin 6, Norin 24, Norin 36, Norin 7, Norin 10, Norin 23, Norin 20, Norin 29, Obako Wase, Norin 12, Norin 30, Norin 31, Norin 32, Norin 35, Norin 37, Norin 38, Norin 43, Norin 44, Norin 47, Norin 48, Homasari, Futaketori, Yacht-kogane, Tone Wase, Shimotsuki, Bizennishiki, Kiyosumi, Akibae, Yaeho, Chikuma, Kokeshi Mochi[w], Yama-kogane, Shioji, Chiyohikari, Yomo-hikari, Shintsuru Mochi[w], Seto-honami, Natsuminori, Yamase-shirazu, Rikuto Norin 12.	Rikuu 132, Wase Sen-ichi, Alchi Wase Asahi, Kyushu 8, Zenkoku Wase, Kamenoo, Mokoto, St No. 1, Shinriki, Aikoku 1, Takenari, San-in 45, Toyosato, Sachiwatari, Wase Aikoku 3, Gin Bozu Chusei, Ta Sensho, Shinju, Wase Asahi 2, Togo, Kokuryo Miyako, Senichi, Bozu 6.	Caloro.
Aichi Asahi type Aichi Asahi, Senbon Asahi, Norin 17, Kinmaze, Norin 18, Norin 41, Towada, Sasashigure, Jukkoku, Eiko, Noun 27, Asakaze, Fujiminori, Zuiho, Yuki-mochi[w], Sasa-honami, Fukuminori, Norin Mochi 45[w], Hamayu. Yamabiko, Otori, Norin 21, Norin 16, Hoyoku, Norin 2, Norin 9, Norin 13, Norin 33, Nakate Shin Senbon, Mihonishiki, Gin-masari, Fukusuke, Nagiho, Akikogane, Wakasa, Toyo-chi kara, Hatsukine, Asuwa, Norin 28, Asahi, Norin 14, Norin 19, Norin 42, Norin 46, Norin 49, Norin 50, Norin 51, Norin Mochi 5[w], Kotobuki Mochi[w], Hashiri Mochi[w], Hatsuminori, Kaganishiki, Hatsushimo, Azusa, Yama-tedori, Benisengoku, Akebono, Oirase, Megumi-wase.	Ayansihiki, Yutaka Senbon, Okute Eiko, Tsurugi, Shinriki 11, Koganemaru, Haruta Asahi, Chikara Senbon, Tokai Senbon, Shuho, Kyoto Asahi, Futaba, Takara, Kaori, Mikawa Nishiki, Chusei Honen, Takane Asahi, Shiro Senbon, Homare Nishiki, Kogane Mochi[w], Zensho 26, Akage, Hashiri Boru, Hyoroku-mochi[w].	

continued on opposite page

more resistance genes are found. New studies may find new resistance genes. Rice has rich genetic resources; there are now more than 35,000 collections in the IRRI germplasm bank.

It is also possible that when more fungus strains are used, more genes may be identified. For a long time, cultivar Shin 2 was considered to have no resistance gene, but when a Philippine strain was used, an allelic gene, *Pi-k*^{s}, was found (Kiyosawa 1969).

The international blast nurseries have identified several highly resistant cultivars, and those varieties are being used or will be used in breeding programs. If they are extensively tested against many tropical fungus races, the genetic information that results will be directly useful in breeding programs in the tropics.

Table 27b continued

Japanese varieties		Foreign varieties
Designated varieties[a]	Others	
Kanto 51 type Kusabue, Mangetsu-mochi, Ugonishiki, Fukei 69, Hatsuiwai-mochi[w], Senshuraku, Tatsumi-mochi[w]. Tsuyuake.	Kanto 51, Kanto 53, Kanto 59, Chugoku 31, Imochi-shirazu.	Reishiko[w], Ya-kei-ko.
Ishikari Shiroke type Norin 34, Fukuyuki, Yoneshiro.	Ishikari Shiroke, Fujisaka 5, Seki-yama 2. Hokuriku 12, Fukumo-chi[w], Wase Bozu.	Butamachi (Doazi chall).
Ta type	K1.	
Pi No. 4 type	Pi No. 4, Pi No. 3, Pi No. 5.	
Fukunishiki type Fukunishiki.	54BC–68, Ou 243, Ou 244.	
To-to type Tei ne, Yukara, Kagura-mochi[w], Tsukiyi-mochi[w], Koshihibiki, Oyodo, Sanpuku, Minehikari.	BR No. 1 (Kongo), Hokushin.	To-to, Choko-to, Hokushi Tami.
Shinsetsu type Shinsetsu, Miyoshi, Takane Nishiki.		
Shimokita type Shimokita.	Pi No. 1, Pi No. 2.	
Zenith type	Fukei 67, Fukei 73.	Zenith.

[a]Excellent varieties that were registered in the Ministry of Agriculture and Forestry until 1967 and were being designated in prefectures in 1967. w = waxy endosperm.

Classification of rice varieties by resistance genes

Since genetical studies were begun, Yamasaki and Kiyosawa (1966) attempted to classify cultivars into groups on the basis of the resistance gene(s) they possess, e.g., the Aichi Asahi type, which has *Pi-a* gene; the Ishikari Shiroke type, which has *Pi-i* gene; etc. Many other cultivars were later tested and more genes were found. More cultivars were then classified and reclassified. Such classification (Table 27a, b) tells what gene(s) each variety possesses and is extremely useful in breeding and other studies. However, that information may not be sufficient to permit identifying the field resistance discussed above. Because only seven fungus strains were used in the testing, the results do not mean that varieties in a particular group possess only a particular gene or genes; some varieties in a group may possess additional genes.

The rationale is similar to that for grouping fungus isolates into races by using differential varieties; seven isolates (differential races) rather than differential cultivars, have been used. It is commonly known that when the number

of differential cultivars is increased for race identification, more races may be differentiated from the same number of isolates. In other words, isolates that are identified as belonging to the same races when tested on a few differential cultivars may separate into more races when more differentials are used. The isolates actually differ in pathogenicity. Similarly, when only seven fungus strains are used, a group of varieties may react similarly, indicating that they have the same gene(s). But use of more fungus strains may show that the cultivars also possess additional resistance gene(s), as reported by Ezuka et al (1969), who stated :

> Some varieties and strains were further tested for resistance to other fungus strains inoculated by spraying or injection, in order to correct a few defects of Kiyosawa's classification. The results suggest that Kanto 51 type of Kiyosawa may be divided into three genotypes. *"Pi-k", "Pi-i, Pi-k",* and *"Pi-i, Pi-m";* To-to type may be divided into three genotypes, *"Pi-a, Pi-k", "Pi-a, Pi-i, Pi-k",* and *"Pi-a, Pi-k, Pi-m";* and Pi No. 4 type may be divided into two genotypes, *"Pi-ta*^{2} " and *"Pi-a, Pi-ta*^{2} ". Some new or unidentified genes seemed to be present in several varieties and strains.

Ezuka (1973) further reported:

> The inoculation experiments with different fungus races revealed that the reaction of Akishino-mochi and Takasagomochi did not agree with that of Aichi Asahi carrying *Pi-a* alone ... have another gene *Pi-i* beside *Pi-a.* ...

Takeda and Takahashi (1970) also concluded :

> However, based on the fact that at times when these varieties are actually planted in the field they suffer severe damage from rice blast and the fact that in certain varieties which are considered to have the same genic constitution for resistant major genes, when their disease reaction is rated by "leaf lesion number" or "leaf lesion area" etc., a varietal difference which is a so-called quantitative character appears. ...

The first portion of the above statement shows that there are more races in the field than have been considered and the later portion indicates that there are more resistant genes in the cultivars than have been known.

Recently Goto (1976) reported that variety Fukunishiki and its parent Zenith were confirmed to have the major resistance gene *Pi-z* in common, but the former was less resistant to blast than the latter. In addition to *Pi-z,* Fukunishiki has one recessive gene and Zenith one modifier Rb_6, to control resistance to the fungus strain Ken 53-33.

In the study of "field resistance" in Japan, mentioned above, the workers apparently considered the varieties in the same group as having exactly the same resistance gene(s). This is perhaps a misconception that led to the unsuccessful attempt to find field (horizontal or general) resistance.

A basic question on method in studying the genetics of resistance

The focal point in studying the genetics of resistance is how to classify the disease reaction (R, M, or S) of a test-plant population to an inoculated fungus strain (race), since disease reaction is the basis for genetic interpretation.

Table 28. Materials and methods used by Kiyosawa's group for evaluating resistance of plants after artificial inoculation in their genetic studies (Cho and Kiyosawa 1973).

Some native varieties, leading varieties during past 60 years, materials developed recently and their parents as shown in Tables 1 and 2 were employed. These varieties were sown in a wooden box. After growing for about 3 weeks in a greenhouse, spore suspension of seven fungus strains, P-2b, Ken 53-33, Ina 72, Hoku 1, Ken 54-20, Ken 54-04 and Ina 168, in a concentration of 20,000 spores/ml, which was prepared using oatmeal decoction sucrose agar medum, was injected with a hypodermic syringe to the plants at the 4.3 ~ 5.5 leaf stage counted from incomplete leaf stage. Seventeen plants of each variety were alloted for injection of a fungus strain.

About 7 days after inoculation, lesion formed was divided into four types, b (brown spot), bg [small (less than 2 mm in a long diameter) gray or white lesion margined with brown color], bG ,[large (2 mm or larger lesion in a long diameter) gray or white lesion margined with brown color] and pG (large lesion with purple or uncolored margin). The number of each type of lesions was counted.

Resistance of each variety to each fungus strain was evaluated in the following way on the basis of the average number of lesions for each type in each variety-fungus strain combination.

I. Two thirds or more plants have no lesion.		R^h
II. Less than 2/3 plants have no lesion.		
1 Average number of lesions per plant is less than one.		R^h
2. Average number of lesions per plant is 1 or more.[a]		
A. Mode is in b type lesion,		
a b ³ 2 (bg + bG + pG)		R
b (bg + bG + pG) £ b < 2(bg + bG + pG)	a	R
	b	MR
c b < (bg + bG + PG)	a	MR
	b	M
B. Mode is in bg lesion.		
a (b + bg) ³ (bG + pG)	a	MR
	b	M
b (b + bg) < (bG + pG)	a	M
	b	MS
C. Mode is in bG lesion,		
a (b + bg) ³ (bG + pG)	a	MR
	b	M
b (b + bg) < (bG + pG)	a	M
	b	MS
D Mode is in pG lesion,		
a (b + bg + bG) ³ pG	a	M
	b	MS
b. (b + bg + bG) < pG	a	MS
	b	S

[a] a = average number of lesions is 7 or less b = average number of lesions is over 7

Table 28 illustrates the methods used by Kiyosawa and his colleagues throughout their studies. They recorded a variety of types and numbers of lesions. The classification is obviously arbitrary. The genetic ratios of R and S or R, M, and S are much dependent on the subjectivity of the observers.

As discussed above, if lesion type is the expression of a genetically controlled interaction between the host and the fungus, mixtures of lesion types indicate the presence of different spore genotypes in an inoculum. If that is the case, can the genetics of resistance be interpreted by simple ratios? Perhaps it should be studied by biometric methods or by population genetics? Takahashi (1965)

stated: "For all reasons we have stated, genetic research on blast resistance may have to be conducted by quantitative method of analysis, rather than by qualitative method." The geneticists have much to contribute to the area.

When there were different types and numbers of lesions on plants of a particular population, the results were interpreted on the basis of the type to which most of the lesions belonged. The genetic information obtained from these studies may therefore have indicated only that the gene(s) responded to the majority of the genotypes of the spores in the inoculum. It may show only the "tip of the iceberg."

General discussion

Although our knowledge of the genetics of blast resistance is incomplete, recent information has helped in the development of the important concept that resistance to blast involves many genes. To have high resistance, varieties must have a large number of these genes. The concept seems basic and necessary for breeding programs.

BREEDING FOR BLAST RESISTANCE

Breeding for blast resistance in various countries has been reviewed (Atkins et al 1965 ; Chang et al 1965 ; Dasananda 1965 ; Ito 1965 ; Padmanabhan 1965 ; Toriyama 1972). In all those breeding programs, the level of resistance in new varieties was increased but the spectrum of resistance was not broad enough to be stable. Discussions here will be brief and related to the development of concepts concerning 1) the level of resistance in donor parents, 2) the procedures for hybridization. 3) the procedures for testing and selection of progenies, and 4) the multiline varieties.

Resistance of donor parents

Donors for blast resistance should have a broad spectrum of resistance. Presumably, they will have large numbers of resistance genes, as discussed above. Most donor parents used in the past were selected locally and are resistant to the races prevailing in their native areas. Those parents may not be resistant to races that are not found in the area. The fungus can produce different races, but only those races that find host varieties prevail. Cultivars resistant only to the races prevailing in a locality cope inadequately with the capability of the fungus to produce races; new races will soon attack them. Thus, cultivars selected from the international blast nurseries (IBN) are valuable donors. Table 12 compares the level of resistance of various donors with that of a few resistant cultivars selected from IBN.

To increase the level of resistance of the existing donor cultivars at IRRI, we intercrossed many resistant cultivars. Although the progenies were resistant in all tests at IRRI, they require further international testing to determine whether they have wider spectra of resistance than their parents.

Hybridization procedures

In the many attempts to breed for blast resistance, almost always only simple crosses were made with a resistant donor. Resistance tests and selections were made to recover the resistance among the progenies. Such a procedure is successful only when a single gene or a few resistant genes are involved. It has been shown, however, that blast resistance involves many genes: we believe many more genes may be involved. The chance of recovering the whole complement of resistance genes in the progeny from a single cross is small. Some of the progeny may have more resistance genes, some less: some may have one set, some another. Dr. Takahashi told me that in the early years the cultivar Sensho, an upland rice resistant to blast in Japan, was often used as a donor for blast resistance breeding in Japan. But new resistant varieties developed from it were never as resistant. Recently in Colombia, Tetep, among others, was used as a donor to cross with some of the best newly developed varieties. Resistance tests and selections were made at each generation. Many resistant and agronomically desirable lines were selected. When they were tested in an epidemic area, however, resistance failed. Tetep remained resistant in the same test. It appears in both cases that the whole complement of resistance genes in the donors had not been transferred to the progeny.

Such cultivars as Tetep and others have high levels of resistance to blast, and the resistance will probably be maintained (i.e., remain stable) for the reasons I have given. The question now is how to transfer all the resistance genes to new cultivars. Since many genes are involved, multiple-cross combinations are necessary. While the geneticists and the breeders may each formulate different ways of crossing, several general approaches may be suggested.

The resistance of several donors may be introduced to a common variety (or varieties) at the beginning, and the resistant progeny from each may be selected as resistant lines. Those resistant lines may be intercrossed, because one line may have a certain set of resistance genes and other lines may have other sets: or the multiline approach may be tried, using those lines. Resistance may also be introduced at different stages in different ways.

Procedures for testing and selecting progeny

Testing progeny for resistance is a crucial step. Since the prevailing fungus races differ in different localities, the logical procedure is to test breeding materials in various localities; progeny selection can then be based upon overall reactions. In this way progeny with broad-spectrum resistance may be identified. Cooperation among interested nations is necessary. One weakness in past breeding programs may have been the lack of multilocation tests.

Multilines

Theoretically, multiline varieties would be useful in dealing with multiracial pathogen. However, no data are available. How the potential advantage of multiline varieties over single resistant varieties can be exploited remains to

be seen. Chiu and Teng (1975) and Chu (1976) tried the so-called composite and multiline varieties. Their experiments actually compared the original susceptible varieties with selections from crosses of resistant varieties. They have not demonstrated the advantage of multilines or composite varieties over single resistant lines.

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EXPERIENCES IN BREEDING FOR RESISTANCE TO DISEASES OF OTHER CROPS

BREEDING POTATOES FOR LATE BLIGHT RESISTANCE

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IN THIS PAPER THE TERMS "specific resistance" and "general resistance" are used with the meanings Hooker (1967) gave them in discussing the genetics and expression of rust resistance: "specific resistance (race specific) which functions against certain races or biotypes but not against others: and generalized resistance (race non specific) which functions against all biotypes." More than 16 terms have been used to describe generalized resistance (Thurston 1971). Most of them describe either the nature of the resistance or the manner of its inheritance.

GENERAL RESISTANCE

Jones et al (1912) were probably the first to publish information on the nature of general resistance in potatoes to *P. infestans.* They studied three susceptible cultivars of *S. tuberosum* subsp. *tuberosum* and found differences in the number of infections in the field when plants had equal opportunities for infection. They also inoculated leaf tips, and found differences in the rate of invasion by the fungus by measuring the development of lesions. General resistance was the only type available for many years, and it was observed to reach fairly high levels. Many investigators, including Stuart (1905), Ito (1918), Reddick (1928), Bonde (1932), and Stevenson et al (1937) noted general resistance in potatoes during the period before the utilization of specific resistance derived from *Solanum demissum.*

An important question about general resistance to *P. infestans* is whether it is stable, or whether it will be lost in time with the appearance of more aggressive (Knutson and Eide 1961) races of the fungus. The history of cultivars with general resistance in the field indicates that such resistance is stable. Large (1940) mentions the general resistance of the potato Champion, which was grown in Ireland for more than 50 years. Toxopeus (1956), De Bruyn (1951), and Davidson (1928) noted a loss in the level of resistance of Champion.

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According to Davidson, Champion was introduced into Ireland in 1877, and had begun to lose its resistance by 1885. However, in 1898, 70% of the area planted to potatoes in Ireland still was planted to Champion, and in 1926, 22%. In 1928, Champion's resistance was about equal to that of the cultivar President. President still has a high level of general resistance. Davidson claimed that when virus-free seed of Champion was obtained, it recovered its original level of resistance. Decline in acreage was probably due to virus infection, not to a loss in blight resistance. Paxman (1963) discussed the "apparent" loss of resistance of Champion and concluded that the evidence was not sufficient to show that it indeed had a "loss of resistance." Muller and Haigh (1958) and Muller (1958) in 1953 found that Champion had a very high level of general resistance both in the field and in the laboratory.

Bonde et al (1940) stated, "President, Sebago, and several seedling varieties have been grown for a period of 10 years in Aroostook County with no evidence that they have lost their resistance to late-blight infection." President and Sebago have general resistance to *P. infestans.* Niederhauser and Cervantes (1956), Niederhauser (1962), and Cervantes (1965) found that Mexican potato cultivars for many years have maintained the same level of general resistance in the presence of severe late blight. More recent information (pers. comm. with J. S. Niederhauser, International Potato Center, Mexico) indicates that a slight decline was noted in certain clones durinz 20 years of field-testing in Mexico, but no sudden breakdowns or losses in the level of blight resistance occurred. A study by Caten (1974) of intraracial variation and adaptation to field resistance in *P. infestans* led him to conclude that although some field resistance was lost, a complete loss of resistance is unlikely. Although it is difficult to obtain reliable information on the relative general-resistance levels of potato cultivars over a long period of time, the best evidence in the literature indicates that neither in nature nor in the laboratory has such resistance been suddenly lost. Van der Plank (1963, 1971) made the same conclusion in his review of the literature. In both Europe and North America, potatoes with known levels of general resistance still have that resistance by comparison with other cultivars with known levels of general resistance.

After 8 years of observations, Thurston et al (1962) stated that the relative general-resistance levels of *S. tuberosum* subsp. *tuberosum, S. tuberosum* subsp. *andigena,* and *S. phureja* clones did not change from year to year when compared with those of standard cultivars. Twenty-two years later there is still no evidence to change this statement. The variety Monserrate, first tested against *P. infestans* in 1954 and susceptible to all blight races, including race O, continues to maintain an extremely high level of general resistance in Colombia under severe epiphytotics of *P. infestans* (pers. comm. with N. Estrada, International Potato Center, Lima, Peru).

The discovery of simply inherited (specific) resistance to *P. infestans* in *Solanum demissum* by Salaman (1949), Muller (1928), and others; and the subsequent discovery of additional *R*-genes and races of *P. infestans,* which

would attack them; as well as the elucidation of their inheritance by Black et al (l953), Black (1960), and others are well reviewed by Gallegly and Niederhauser (1959), Gallegly (1968), and Van der Plank (1963, 1968, 1975).

For several decades, almost all potato breeders dropped their work on general resistance and concentrated on obtaining commercial potato varieties with *R*-genes. In the absence of specific races of *P. insfestans* capable of attacking them, those varieties were completely free from disease in the field. Unfortunately, the fungus produced new races faster than breeders could produce cultivars, although occasionally a 2- or 3-year period of freedom from blight occurred after the introduction of a potato cultivar with specific (*R*-gene) resistance. Currently, only those *R*-gene cultivars also possessing high levels of general resistance are of practical value where late blight is severe. In the Toluca Valley of Mexico, no clone of *S. demissum* tested was immune to blight (Niederhauser and Mills 1953). Niederhauser (1968) further stated that no tuber-bearing *Solanum* species has been found immune to *P. infestans* in Mexico. The subsequent discovery of the sexual stage of *P. infestans* in Mexico helped explain the extraordinary variability of the fungus there. Potato breeders and plant pathologists have reawakened to the value and importance of general resistance as specific resistance has failed to give lasting resistance in nature.

Many studies have been made, primarily with *S. tuberosum* subsp. *tuberosum,* to elucidate the nature of general resistance after the early study of Jones et al (1912). Vowinckel (1926) found that *P. infestans* had a longer incubation period and produced smaller lesions on cultivars with general resistance. Schaper (1951) found marked differences between different German cultivars (*S. tuberosum* subsp. *tuberosum*) and even greater differences between clones of *S. tuberosum* subsp. *andigena* in the time necessary after inoculation to produce sporangia in a moist chamber. Muller (1953) and Muller and Haigh (1953), by inoculation of leaf discs of English cultivars with a dilute zoospore suspension, estimated the "probability of becoming infected per unit area of foliage." They correlated that with the reaction of the same cultivars to *P. infestans* in the field. It is noteworthy that Champion had the highest degree of general resistance in both field and laboratory tests. Deshmukh and Howard (1956) obtained results similar to Muller's, but also found that the fungus had a longer incubation period and produced less sporangia on cultivars with general resistance.

Van der Zaag (1959) considered that general resistance consisted of these components: 1) the chance of infection: i.e., the chance that a spore can penetrate a leaf; 2) the extension rate of mycelium in leaf tissue; 3) rate (or speed) of sporangial production; and 4) the quantity of sporangia produced per leaf area. Subsequent workers had similar results. Umaerus (1959, 1960, 1963), Lapwood (1961, 1963), Lapwood and McKee (1966), Guzman (1964), Guzman et al (1960), Knutson (1962), Jeffrey et al (1962), and Hodgson (1962) made detailed studies of the phenomena as they affected general resistance. Thus, general resistance consists of several host plant- *P. infestans* interactions.

which can be measured independently.

Guzman et al (1960) studied clones of *S. tuberosum* subsp. *tuberosum, S. tuberosum* subsp. *andigena,* and *S. phureja* which had low, intermediate, and high general resistance to *P. infestans* in the field. They found less sporulation, slower sporulation, smaller lesions, and fewer lesions produced per given quantity of inoculum on resistant clones. Subsequently, Guzman (1964) expanded that work with similar results with the same species and subspecies. Greenhouse-grown foliage and field-grown foliage gave essentially the same results.

Thurston et al (1962) reported the results of 8 years of field observations on general resistance to *P. infestans* in the Colombian potato collection. In 1961 the collection consisted of 750 clones of cultivated and wild species, including 263 clones of *S. tuberosum* subsp. *andigena* and 200 clones of *S. phureja. Solanum tuberosutn* subsp. *tuberosum* clones were also included in the field observations. Very high levels of general resistance were found in *andigena* and *S. phureja;* those levels gave almost complete control of *P. infestans* in the field under severe blight conditions. The commercial cultivar Monserrate (a cross between *tuberosum* and *andigena,* with a very high level of general resistance), has been grown in Colombia since 1954 (Estrada et al 1959), and has maintained its resistance to the present. When grown in the Toluca Valley of Mexico, Monserrate also had a high level of partial resistance.

Grechushnikov (1939), Kammermann (1951), Kedar (1959), and Umaerus (1959, 1960) found a positive correlation between peroxidase activity in the foliage and general resistance to *P. infestans.* According to Umaerus (1959), that correlation exists only in clones of *S. tuberosum* subsp. *tuberosum,* and not in hybrids derived from *S. demissum.* Henniger and Bartel (1963) did not find the peroxidase test suitable for testing for general resistance. Fehramann and Dimond (1967) stated, "a positive and striking correlation was found between peroxidase activity in different organs of the potato plants and resistance to *P. infestans."* Sakai and Tomiyama (1964) found no correlation between peroxidase activity and blight resistance in the early stages of growth.

Factors that influence testing for general resistance to *P. infestans*

Numerous factors influence the expression of general resistance to *P. infestans.*

Plant age. Investigators disagree on the effect of age on susceptibility to blight. Most workers cited by Beaumont (1934) found older plants most susceptible. Grainger (1956) and Lowings and Acha (1959) found plants to be susceptible when very young and, again, after flowering during tuber production; plants were most resistant during the intermediate stage of vigorous foliage development. However, Beaumont (1934) attributed changes in susceptibility at different ages to differences in the microclimate caused by differential foliage density at different stages of plant growth. Toxopeus (1958) reported a close correlation of general resistance to late maturity, to the point that it was difficult to combine earliness with a high degree of resis-

tance. Umaerus (1963) has reviewed the work on plant age in relation to general resistance.

Nutrition. Awan and Struchtemeyer (1957) indicated that lesion size can be influenced by nitrogen, phosphorus, and potassium, although in the field only excessive nitrogen affected (increased) lesion size. Grainger (1956) found newly emerged and tuber-producing plants most susceptible to blight. This high susceptibility was correlated with a high ratio of total carbohydrate in the whole plant to the residual dry shoot weight. Lowings and Acha (1959) reported that high nitrogen was correlated with increased blight resistance under some conditions. Main and Gallegly (1964) found that when plants grown under conditions of normal nutrition were inoculated with *P. infestans,* those with general resistance survived, whereas when the same clones were grown under conditions of high nutrition, all plants, both those with and without general resistance, were killed.

Day length. Kammermann (1951) and Umaerus (1959) reported day length as a factor that influences general resistance. Umaerus gave as one example the observation that in Mexico the cultivar Alpha (with short days) is considered susceptible, whereas in Minnesota (with long days) it has a high level of general resistance. The difference may be due to environmental factors other than day length. He further stated that light conditions influence peroxidase levels; i.e., when days are short there is less peroxidase activity. Pohjakallio et al (1957) also reported that lesions appeared earlier on the foliage of potato varieties grown with short daily exposures to light. Garcia (1973) found *andigena* more susceptible to *P. infestans* when days were short than when days were long.

Light intensity. Victoria and Thurston (1974) found that leaves of potato plants grown under different light intensities had significant variations in size of lesions caused by *P. infestans.* Lesions were larger on plants grown at lower light intensities than on plants grown under stronger light. A field study by Schumann and Thurston (1977) indicated that infection rate and sporulation were higher under low light intensities.

Leaf position. Leaf position is an important consideration in testing for general resistance to *P. infestans.* Lowings and Acha (1959) and Hodgson (1961) found the top leaves most resistant, the middle leaves intermediate in resistance, and the bottom leaves most susceptible. In the field, Bjorling and Sellgren (1955) found that 7 or 8 times as many lesions occurred on upper leaves. Mooi (1965) found the lower, and sometimes apical, leaves more diseased than the upper leaves.

Plant source. Knutson (1962) found that greenhouse plants were more resistant than plants grown in the field. He found less sporulation and fewer lesions on greenhouse plants. Muller (1953) reported that greenhouse plants are usually more susceptible.

Virus infection. Muller and Munro (1951) reported that plants infected with virus X or virus Y were more resistant to infection. Richardson and

Doling (1957) found plants infected with leaf-roll virus also more resistant than uninfected plants. Diaz Moreno (1965) reported that plants infected with the potato vein-yellowing virus were more resistant to *P. infestans* than virus-free plants. A study by Pietkiewicz (1974) found that plants infected with potato viruses-X, Y, S, and leaf roll were more resistant to *P. infestans*. Cubillos and Thurston (1975) found that plants infected with potato viruses X, Y, leaf roll, and potato spindle tuber viroid were more resistant to *P. infestans* than healthy plants in both field and greenhouse. All of those factors should be considered in programs of testing for general resistance to *P. infestans.*

GENETICS OF RESISTANCE

It is often difficult to determine from the literature whether investigators were working with general or specific resistance. The mode of inheritance of such resistance appears to be the primary consideration; a second consideration is whether the reaction to blight was a hypersensitive reaction. The hypersensitive reaction generally was considered as evidence of specific or simply inherited resistance. That may not necessarily be the case.

Stevenson et al (1937) studied the progeny of a cross between two cultivars (No Blight and Erkishiazu) possessing general resistance and found that all were more resistant than Green Mountain (susceptible check), with 16.7% showing no infection and only 7.3% more heavy infected than either parent. They concluded that "blight resistance in the cultivated varieties is inherited as a recessive character probably controlled by multiple genes," that is, quantitatively inherited. Black (1954) stated, "Genetically speaking, field resistance is presumed to be controlled by a series of minor genes which determine the degree of susceptibility in susceptible varieties and the extent of necrosis in field-immune forms. In contrast with field immunity, field resistance gives partial protection against all the specialized races of the parasite that have arisen in the search for hypersensitive varieties." Black and Gallegly (1957) defined field (general) resistance as "all forms of inherent resistance that plants possess with the exception of hypersensitivity as controlled by *R*-genes." They attributed the inheritance of field resistance to the operation of a polygenic system. They also stated, "In extreme cases, polygenic resistance alone may be high enough to produce only resistant offspring under standard test conditions." Subsequently, Black (1960) defined field (general) resistance as "the degree of resistance exhibited by a plant towards all races of the parasite capable of causing more than a hypersensitive reaction on it." Malcomson and Black (1965) found their standard greenhouse test slightly less severe than the field test in the Toluca Valley of Mexico; their detached-leaf test was more severe. Their progeny test showed that field resistance is inherited in a polygenic fashion. They also stated, "In certain breeding lines, however, virtual immunity was obtained in two generations from a susceptible-by-resistant parent. Results suggest complementary factors in the parents." It

is not clear what reaction is meant by "virtual immunity." Gallegly (1960) also found that general resistance to *P. infestans* in tomatoes is quantitatively inherited.

Graham (1962) studied the inheritance of general resistance in *Solanum verrucosum,* and concluded that resistance was inherited on a quantitative basis. He (1963) later studied the partial (general) resistance to *P. infestans* of *Solanum bulbocastanum* Dun. and *S. verrucosum,* diploid Mexican species. After analyzing the reaction to race 1, 2, 3, 4 of the F_1, F_2, and F_3 progeny of crosses between resistant and susceptible clones within each species, he concluded that inheritance of resistance is quantitative. Some of the resistant progeny gave hypersensitive reactions to the fungus and there seemed to be no clear distinction between that type of reaction and one indicating partial resistance.

Toxoperus (1958) reported that field (general) resistance is combined with late maturity, but other investigators have not always found this true (Schick et al 1958; Simmonds and Malcomson 1967). He (1959, 1961) also found that the degree of field (general) resistance of the material he studied seemed to be governed by a series of minor genes.

Probably the most difficult problem in evaluating the literature on general resistance to *P. infestans* is interpreting and correlating the disease reactions given by different investigators. Many phrases such as "highly resistant," "virtually immune," and "high levels of resistance," are used, or disease reaction is given as percentage of defoliation. Many schemes have been proposed (Anon. 1947; Black 1960; Graham 1963; Simmonds and Malcolmson 1967: Thurston et al 1962) for evaluating resistance, but few precisely describe lesion size under standard conditions, and hypersensitive reactions are usually not included. However, Graham's system (1963) does so. When no infection occurs it is often not known whether the reason is that plants have escaped contact with the pathogen or that they have shown a hypersensitive reaction. A precise definition of terms for use by late-blight workers and a standard scheme for evaluation of resistance are needed.

Discussion

There are similarities between *P. infestans* and *Pyricularia oryzae.* Both fungi are highly variable with many races ; *P. oryzae* appears to outdo *P. infestans* in that respect. Both are airborne pathogens which can rapidly spread to epidemic proportions in the field. Their success as pathogens depends much on environmental factors. Both pathogens attack crops of worldwide importance that have been studied intensively, so knowledge of the crops, the pathogens, and the diseases is broad. Potatoes are asexually propagated; when one desirable tuber is obtained in a breeding program it can be multiplied rapidly to constitute a new variety. That is an advantage of potatoes over rice in breeding for general resistance.

I find it useful to think of the inheritance of general resistance as similar

to other polygenically inherited traits of the potato such as yield, dry-matter content, and after-cooking darkening. According to Plaisted (1960), polygenic inheritance has the following attributes:

1. Many genes contribute small and individually indistinguishable effects upon the phenotype.
2. Interactions between the different genes are quite likely.
3. The expression of the genotype is modified greatly by the environment.

General resistance in the potato is modified by many factors that would also affect yield, such as nutrition, light intensity, day length, temperature, and moisture. If these and other factors are not given adequate consideration, testing-programs for general resistance are confounded and their results may be meaningless.

To adequately measure or evaluate general resistance, there must be standard sets of varieties with known resistance to which breeding material can be compared. The lack of such standard clones has caused considerable confusion in the literature on general resistance in potatoes.

The experience to date with potatoes indicates that general resistance is long lasting. Whether more aggressive races of *P. infestans* can develop over time to break down general resistance is unknown.

During the process of breeding for specific resistance to *P. infestans* in potatoes, some general resistance is lost. That is the "Vertifolia effect" of Van der Plank (1963) and should be kept in mind in the operation of all breeding programs for resistance.

In the USA only the potato variety Sebago has an appreciable level of general resistance to *P. infestans.* After the rapid breakdown of specific resistance to *P. infestans,* potato breeders drastically reduced breeding for resistance to *P. infestans. P. infestans* in the US is controlled by fungicides and not by genes for resistance.

It is to be hoped that rice scientists will have better success in breeding for general resistance in rice. According to data from the Food and Agriculture Organization of the United Nations (1975), approximately 10 kg pesticides/ha are applied annually in Japan compared with 0.2 kg/ha in India. The report concludes, "Thus, if the Japanese example is followed throughout, for the 78 1/2 million hectares of rice grown in Southeast Asia a total of about 785,000 metric tons of pesticide would be applied in the region each year. This is more than the present-day total world production of pesticides." It is obvious from such figures that every effort must be made to develop adequate resistance in rice to *P. oryzae.*

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BREEDING FOR RESISTANCE TO SOME COMPLEX DISEASES OF CORN

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BY ORIGIN AND MAJOR AREA OF CULTIVATION, corn is an American crop. It has been cultivated in Central America and adjacent areas of Mexico and South America for thousands of years. Many races and varieties have been selected. Two of them, a northern Hint and a southern soft-dent, hybridized in the grasslands and woodland clearings when American settlers moved into the US Middle West. Many productive, open-pollinated dent corn varieties were selected by farmers and early seedsmen in the area. Those varieties have provided the germplasm pool for US hybrids. At first, double crosses, produced by crossing four inbred lines, were widely grown. Now a majority of US hybrids are single crosses, produced by crossing two unrelated inbred lines.

Corn breeding consists essentially of three major stages: 1) improvement of source populations of inbred lines. 2) extraction of inbred lines from source populations by selfing and selection, and 3) performance testing of various hybrid crossings between inbred lines to identify superior combinations.

Many parental inbred lines of hybrids are developed by corn breeders in state or federally supported agricultural experiment stations. They are known as public lines. Breeders in commercial seed companies also develop inbred lines. Public lines are available for use by everyone; the use of private lines is controlled by the seed companies. Hybrids grown by farmers may be made from public lines, private lines, or from combinations of the two. In most instances, the farmer does not know the pedigrees of the hybrids he grows.

Many inbred lines are used to produce commercial hybrids in the US Corn Belt, but only a few lines and their modified versions are used widely at any one time. The identity of the most widely used lines changes from year to year. Those popular lines provide the basic genetic structure of commercial hybrids. Less widely used lines contribute genetic diversity.

More than half of all the corn produced is grown in the US.

More than 25 significant diseases occur on corn. The pathogens are several kinds of fungi, bacteria, viruses, spiroplasmas, and nematodes. All parts of

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the plant can be affected. Some diseases, unless controlled, can cause severe losses over large areas; others can be destructive in localized areas; still others have never become destructive. The Helminthosporium leaf blights and common rust are potentially the most important diseases in the US Corn Belt.

Selection and breeding for genetic resistance, the primary means of disease control, keeps corn diseases from limiting production. The 1970 corn blight epidemic (Hooker 1971, 1972; Tatum 1971) was an exception, but recovery from it was rapid; 2 years after that epidemic the corn crop was again protected by resistance. A wide array of genetic mechanisms for resistance to various pathogens exists in corn (Hooker 1978).

NORTHERN CORN LEAF BLIGHT

Etiology, symptoms, and importance

Northern corn leaf blight is caused by the fungus *Helminthosporium turcicum* Pass. The perfect stage *Trichometusphaeria turcica* Luttrel is not believed to be significant to the persistence of the fungus in nature. The disease is called northern corn leaf blight in the US because it occurs most frequently in the northern part of the country.

The disease occurs primarily in the leaves. Plants may be infected at any stage, but in the northern US Corn Belt, plants are usually infected at anthesis or later. Lesions on susceptible plants are 5.0 to 15.0 cm long and 1.25 cm wide, elliptical, first grayish green and later tan. They form first on the lower leaves and, as the season progresses, move higher. Lesions may merge or be so numerous that entire leaves or plants die. Blighted plants are predisposed to root- and stalk-rot infections (Fajemisin and Hooker 1974a, b; Kim et al 1974).

H. turcicum survives locally between crop seasons in infected plant tissue. It is seldom seed carried. Conidia spread through the air, germinate on the surface of the leaf, and penetrate directly (Hilu and Hooker 1964; Jennings and Ullstrup 1957). Hyphae grow between and through cells, and in 3 or 4 days reach the xylem. If the leaf is susceptible, hyphae grow extensively in the xylem. After about 6 days, the hyphae grow from the xylem and into the surrounding mesophyll tissue. That infected tissue collapses, wilts, and becomes necrotic. When conditions are humid, abundant conidia form on the lesions. They cause the secondary spread within fields. Only after the upper leaves are blighted does the fungus spread from field to field.

The disease is favored by cool-and-humid conditions; it can cause yield losses of 30% or more over large areas (Elliott and Jenkins 1946). Losses in individual fields of about 50% were common in the US Corn Belt when susceptible hybrids were grown. Before the widespread usage of disease-resistant hybrids, the disease occurred sporadically, depending largely on environmental conditions. Yields of plants severely infected at midseason can be reduced by as much as 68% (Ullstrup and Miles 1957). If repeatedly inoculated, seedlings can be killed. As with all leaf diseases, the magnitude of grain loss depends

upon the severity of infection and the stage of plant development at which infection occurs (Fisher et al 1976: Ullstrup and Miles 1957). As the grain-filling period nears completion, loss of leaf area assumes little importance. In general, if not more than 30% of the leaf tissue is blighted earlier than 4 weeks after anthesis and the lower leaves are damaged, grain yields will be nearly normal (Fisher et al 1976).

Expression and types of resistance

Techniques have been developed to measure resistance to *H. turcicum* in seedlings and in adult plants. Inoculation procedures in the greenhouse usually involve spore suspensions in water. When cultured for a long time in the laboratory, the fungus often sporulates poorly on synthetic media or loses its ability to infect plants.

I have found that infected leaf tissue is a satisfactory source of inoculum. Sections of leaves bearing lesions are collected from the field or from greenhouse-inoculated plants. The tissue is dried in a plant press and stored dry at 10 °C. When inoculum is needed, leaf sections are placed on moist paper in covered dishes for 48 to 96 hours. Spore suspensions are prepared from the tissue and sprayed over seedlings, and the plants are incubated overnight in a mist chamber. Inoculations in the field require a large amount of inoculum. I have solved the problem by using milled, infected leaf-tissue as inoculum (Hooker 1954, 1955). Infected leaves are collected in the field in early September; they are air dried and ground in a feed mill. A small amount of the leaf tissue is placed in the leaf whorl, when plants are about 45 to 60 cm tall. Sometimes two or more inoculations are made, approximately at weekly intervals.

Reaction to *H. turcicum* is expressed in both kind and degree of infection. Individual lesions differ in form and size. Some forms of resistance are expressed as chlorotic lesions (Hilu and Hooker 1963; Hooker 1961). The lesions are usually smaller than those on susceptible plants and are wholly chlorotic, or are necrotic with some type of chlorotic margin. The size, type of pigmentation, and degree of necrosis will vary with plant genotype. Another resistant expression seen in seedlings is the chlorotic-fleck type (Hilu and Hooker 1965). The chlorotic-lesion and chlorotic-fleck types of resistance are best expressed by young or actively growing plants. Plants may also vary in degree of infection. That will be referred to as lesion-number resistance even though lesion size may also be a factor in the resistance. Such ratings are meaningful only on adult plants. The ratings are taken as percentage of leaf tissue infected. Disease ratings taken only once should be taken about 2 weeks after anthesis. Weekly ratings from anthesis to maturity allow the development of disease progress curves.

The nature of disease resistance has been studied (Hilu and Hooker 1963, 1964; Lim et al 1968, 1970; Obi 1975). Histological studies have shown that penetration and early stages of establishment are the same in all susceptible

and resistant plants. Subsequent hyphal development is different in resistant and susceptible plants and in plants having various resistance types. Hyphae grow poorly in the xylem of plants whose resistance is expressed as few lesions. Lesion development is delayed, but subsequent sporulation is abundant. In plants with the chlorotic-fleck type of resistance, the invading hyphae are limited to the few cells near the initial point of infection. Hyphae rarely invade the xylem of plants having the chlorotic-lesion type of resistance, and lesion enlargement is limited to slow-growing hyphae in the mesophyll tissue. Sporulation is substantially delayed or suppressed, but some spores are produced. Sporulation suppression varies, depending upon the original source of the chlorotic-lesion resistance and upon the inbred or hybrid containing it. The fewest spores are produced in lesions where necrosis of leaf tissue is least and on plants having both the lesion-number and chlorotic-lesion forms of resistance. The chlorotic-lesion form of resistance, in the field in the US Corn Belt, is adequate to keep pathogen development and spread to trivial amounts.

Toxic compounds that inhibit fungal spore germination and hyphal growth are produced in plants that have the chlorotic-lesion but not the chlorotic-fleck form of resistance when infected with pathogenic but avirulent isolates of *H. turcicum* (Lim et al 1968, 1970). Those phenolic compounds, called phytoalexins, are not found in either uninfected plants or infected susceptible plants. The quantity of toxic compounds, rapidity of their formation, and perhaps their chemical identity will vary with different corn genotypes (Obi 1975). Phytoalexins form most rapidly and in greatest amounts in plants that subsequently show the greatest degree of sporulation-suppression. Nonpathogenic *H. turcicum* isolates and virulent *H. turcicum* biotypes, against which the chlorotic-lesion resistance fails to be expressed, do not induce phytoalexin production in corn.

Sources of resistance

One of the most important objectives of the program is the identification of suitable sources of resistance to disease. A diversity of types is needed. Many corn lines and varieties have the lesion-number form of resistance to *H. turcicum.* They can be found in the Corn Belt as well as in other areas. They were identified by first screening a wide range of germplasm, then crossing promising sources with susceptible inbreds. The latter crosses were evaluated for disease reaction and again crossed with susceptible inbreds. The level of resistance in the F_1 crosses and the frequency of resistant plants in the segregating populations are most helpful in identifying sources of resistance useful in breeding work.

Associates and I (Hooker et al 1964) have identified more than 50 sources of resistance expressed in the form of resistant-type lesions. The sources include dent-, flint-, pop-, and sweet-corn endosperm types and corns from most areas of the world. Resistance has also been obtained from teosinte, and recently a form of chlorotic-lesion resistance that segregates as a single

dominant factor has been incorporated into corn from *Tripsacum floridanum*. The chlorotic-fleck resistance is present in inbred B1138T from Africa and selections derived from it (Hilu and Hooker 1965). When the genes for resistance are found in unadapted plant types, they are introduced into adapted Corn Belt plant types by backcrossing. The stocks are then used in further research and breeding work.

Genetics of host-pathogen interactions

The inheritance of resistance to *H. turcicum* in corn embraces a wide array of patterns.

The lesion-number form of resistance is polygenic in inheritance (Findley and Leffel 1962; Hughes and Hooker 1971; Jenkins and Robert 1952, 1959, 1961; Jenkins et al 1954). In some hybrids resistance is partially dominant; in others it is nearly intermediate between the two parents. In populations studied at the University of Illinois, additive genetic variance was the most important, but dominance and epistasis were also evident (Hughes and Hooker 1971).

The chlorotic-lesion form of resistance is usually monogenic dominant (Hooker 1961, 1962b, 1963b,c). Some 34 sources that were studied have dominant genes at the *Ht* locus on chromosome 2. Within the group, some genes confer a higher level of resistance than others when backcrossed to recurrent parental inbreds. Inbred NN14 from Australia has two dominant genes: one is at the *Ht* locus and the other is an independent gene, *Ht2* (Hooker 1973, 1977). The chlorotic-lesions conditioned by *Ht* and *Ht2* are slightly different in phenotype. Plants homozygous for *Ht2* are more resistant than are heterozygous plants. The gene from *Tripsacum floridanum* segregates independently from *Ht* and *Ht2* and may provisionally be designated at *Ht3*. Gevers (1975) has reported on resistance from the Mexican variety Pepitilla, conditioned by the dominant gene *HtN*. Lesions on *HtN* plants are unlike those on *Ht, Ht2,* or *Ht3* plants.

Some sources of resistance expressed as chlorotic lesions with suppressed sporulation are not inherited as single dominant genes. In some lines the resistance is recessive. In one line, expression of a single dominant gene seems to be suppressed by a modifier gene (Thung 1974). In the absence of the modifier gene, the dominant gene conditions an excellent level of resistance.

A diversity of sources of resistance has enabled Illinois workers to study the interaction of different forms of resistance. The lesion-number (polygenic) form of resistance enhances the expression of genes *Ht, Ht2, Ht3,* and *HtN*. Gene *Ht,* when combined with the lesion-number form of resistance, also reduces the amount of tissue blighted (Hooker and Kim 1973). When combined in the same plant, genes *Ht* and *Ht2* for chlorotic-lesion resistance result in smaller lesions and less necrosis than does either gene alone. The chlorotic-fleck and chlorotic-lesion forms, when combined, result in an excellent level of resistance.

Resistance conditioned by gene *Ht* is specific in that biotypes of *H. turcicum* virulent to plants having the gene are known (Bergquist and Masias 1974; Lim et al 1974). The corn- *H. turcicum* host-pathogen system meets the requirements of the gene-for-gene model (Flor 1955). The dominant genes *Ht* and *Ht2* for resistance to *H. turcicum* are part of the system studied (Lim et al 1974). When *H. turcicum* isolates virulent to plants having *Ht* were crossed with isolates avirulent to plants having *Ht,* and single-ascospore cultures from the cross were tested for virulence on corn plants having gene *Ht, Ht2,* or neither gene, the virulence and avirulence to plants having gene *Ht* segregated in a 1:1 ratio. All ascospore progeny from the cross were virulent to plants having neither *Ht* nor *Ht2,* but were avirulent to plants having gene *Ht2.* It is evident that disease reaction is conditioned by the interaction of those corresponding genes in host and pathogen.

A wide array of normal and male-sterile cytoplasms containing the same nuclear genotypes have been studied for reaction to *H. turcicum.* No differential reactions due to cytoplasm have been detected.

Pathogen variability

H. turcicum is variable. When grown apart from its host, *H. turcicum* varies in cultural characteristics (Hilu 1964; Robert 1952). The vegetative mycelium is generally multinucleate, and all nuclei of a cell divide simultaneously (Knox-Davis and Dickson 1960). Genetically unlike nuclei can persist in individual fungus isolates (Masias and Bergquist 1974). In addition to heterokaryosis, aneuploidy may be common. The fungus is heterothallic, and ascospores can be produced in laboratory cultures by mating compatible isolates (Lim et al 1974).

The fungus shows two types of physiologic specialization. It has physiologic races or forms that are pathogenic to corn, Sudan grass, sorghum, or Johnson grass, or their combinations (Bergquist and Masias 1974; Masias and Bergquist 1974; Robert 1960; Rodriguez and Ullstrup 1962). It has physiologic races distinguished by monogenic, resistant corn lines (Bergquist and Masias 1974; Lim et a1 1974). Two races having the virulence-formula numbers US1 and US2 have been designated (Lim et al 1974). Isolates of the fungus with the virulence formula number US1 have the virulence formula *HtHt2/0* and those with the formula number US2, the virulence formula *Ht2/Ht.* The virulence formula designates the identity of the genes effective or ineffective for resistance.

Rarely is *H. turcicum* virulent to corn plants having gene *Ht* in nature even though the virulence is due to a single gene. Avirulence to *Ht* is the common situation. When gene *Ht* was discovered it was recognized that its effectiveness in agriculture would depend upon the absence or rarity of races of *H. turcicum* virulent to it. Consequently, several studies were made. In seedling tests, some 166 isolates of *H. turcicum* from 13 host species in different locations in North America and in 16 other countries were avirulent to plants having gene *Ht* (Hooker et al 1965). Since that time many isolates from the US Corn Belt

have been studied and all have been avirulent to plants having gene *Ht.* A wide variety of matings among *H. turcicum* isolates were made and all ascospore progeny were avirulent. Successive passages of mixtures of *H. turcicum* isolates through plants having gene *Ht* did not increase virulence (Kinsey and Hooker 1973). *Ht* genes from 30 sources have been introduced into common genetic backgrounds by backcrossing. The stocks were distributed to cooperators in many parts of the world. When these corn stocks were exposed to infection in the field over a 2-year period, biotypes of *H. turcicum* virulent to gene *Ht* were not detected in Argentina, Brazil, Canada, Egypt, Ethiopia, Greece, Italy, Japan, Kenya, Korea, Mexico, Nigeria, Peru, Romania, South Africa, mainland US, or Yugoslavia. A large portion of the corn in the US and European Corn Belts is produced from hybrids having gene *Ht.* Biotypes of *H. turcicum* virulent to gene *Ht* have yet to be detected in those hybrids. In December 1972, however, virulence to *Ht* was seen in a seed-production field in Hawaii. Biotypes having that virulence have persisted on the island of Kauai since that time, but seem to be less aggressive than biotypes avirulent to plants having *Ht.*

H. turcicum also varies in aggressiveness. Isolates maintained for a long time on nutrient media may become nonpathogenic or weakly pathogenic. They vary in aggressiveness to polygenic-resistant corn (Nelson et al 1965, 1970; Robert and Sprague 1960). Such pathogen variability has not resulted in the loss of resistance to northern leaf blight in agriculture.

The factors that determine the prevalence of different genes for virulence and avirulence in *H. turcicum* in nature are not known. Biotypes virulent to plants having gene *Ht* conceivably should have a selective advantage over avirulent biotypes in areas of the world where gene *Ht* is extensively deployed in the commercial crop. Why such biotypes have failed to occur is not known.

Breeding for resistance

Several approaches to breeding for resistance to *H. turcicum* are used. The lesion-number form of resistance should receive major attention in the establishment and improvement of source populations. Such resistance is highly heritable. Recurrent selection has proven to be effective (Jenkins et al 1954). The chlorotic lesion *HtN* and perhaps the chlorotic-fleck forms of resistance could be integrated into source populations. Inbred lines should be developed in the presence of northern leaf blight, and susceptible segregates discarded. Selection for resistance should be adequate. There seems to be little value in invoking more complex breeding procedures to use the nonadditive types of gene action available for lesion-number resistance at present. Gene *Ht* has been incorporated into a large number of inbred lines by backcrossing. Inbred lines are usually developed, and if gene *Ht* is absent, the gene is incorporated after the line has proven to be useful in hybrids. Gene *Ht2* is now being incorporated into inbreds. The gene is being backcrossed into lines or versions of lines that do not have gene *Ht.* Hybrids will probably be produced first

with one parent contributing *Ht* and the other *Ht2,* since the resistance is dominant. Inbreds and hybrids homozygous for both genes *Ht* and *Ht2* for resistance would be the most effective. Gene *HtN* is not used now in the US Corn Belt but may be a useful form of resistance. Breeding procedures that use the maximum number of effective genes for resistance to *H. turcicum* should be the most effective.

Use of resistance in agriculture

Areas of the world where *H. turcicum* is endemic and where environmental conditions favor the disease can be considered risk areas. In those areas hybrids should be produced from inbreds that either have adequate lesion-number resistance, or are homozygous for *Ht* or *Ht2,* or both. My observations and test data would indicate that a leaf-blight rating of 30% or less leaf area infected 4 weeks after silking is satisfactory to prevent economic losses from northern leaf blight in the US and comparable areas of the world (Fisher et al 1976). So far, in the US Corn Belt, gene *Ht* in any background provides adequate resistance in the field.

It is anticipated that northern leaf blight will continue to be a problem in the US Corn Belt because of changes in hybrids grown and in potential changes in the pathogen. A wide array of sources of resistance are available to the corn breeder for use in agriculture. More sources of resistance should become available in the future. It should be possible to prevent *H. turcicum* from causing severe grain losses by implementing current research findings.

SOUTHERN LEAF BLIGHT

Etiology, symptoms, and importance

Southern corn leaf blight is caused by the fungus *Helminthosporium maydis* Nisikado and Miyake (*Cochlioholus heterostrophus* Drechsler). The disease is widespread in tropical and subtropical areas. In the US the disease has the common name southern leaf blight because its distribution is limited to the southern and warmer regions of the country where corn is grown.

The disease is recognized as a leaf blight or leaf spot. Symptoms produced on most susceptible corn by race O of the pathogen are lesions about 0.6 cm wide and 1.2 to 1.9 cm long. On corn with *cms*-T cytoplasm for male-sterility, race T produces lesions not only on leaves but on the leaf sheath and husk, and a gray-to-black ear-rot. Leaf lesions are usually larger than those produced by race O; they are spindle-shaped, tan, and usually have chlorotic borders. Seedling blight can result from infected seed.

Inoculum is from infected plant-refuse or diseased plants. Conidia are produced in large numbers, are air borne, and infect when free moisture is on the leaf for 6 to 10 hours. The lesions form and under favorable conditions resporulate within 5 to 6 days.

The disease is favored by warm, humid weather and can be very destructive.

In 1970 it reduced yields by 50% in each of several southern states and by 25% in such Corn Belt states as Illinois (Hooker 1971). Losses caused by race O are much less.

Expression and types of resistance

Plants can be inoculated in the greenhouse or in the field. In the greenhouse spore suspensions are sprayed over seedlings which are then incubated overnight in a humidity chamber. Field inoculations are made by placing spore suspensions cooked sorghum seed having the fungus growing in it, or pulverized, infected corn-leaf tissue collected from the previous crop into the leaf whorls.

Resistance to *H. maydis* is expressed in various ways, including percentage of plant tissue blighted, lesion size, lesion type, fungus sporulation in the lesion, resistance to the pathotoxin produced by race T, and magnitude of yield loss. Some of the characteristics can be measured in seedlings; but older plants in the field best express resistance. Lesion number is a quantitative character. So is lesion size. Lesion size and percentage of leaf area infected are important, intercorrelated resistance components. A chlorotic-lesion form of resistance is known (Craig and Daniel-Kalio 1968; Crais and Fajemisin 1969; Smith 1975). Lesions on resistant seedlings and on older plants in the field consist of chlorotic flecks, while those on susceptible plants are large and necrotic. Resistance to race T pathotoxin is readily seen when primary roots from germinating seeds are placed in contact with the pathotoxin: primary roots from seeds containing the susceptible *cms*-T or *cms*-P cytoplasm are markedly inhibited and often killed (Lim et al 1971).

Susceptibility to race T is related to the pathotoxin produced by race T, and the predominant site of action is the mitochondria (Hooker 1974a: Miller and Koeppe 1971). The inner membranes of the mitochondria from susceptible but not from resistant cytoplasm break down when in contact with race-T pathotoxin. Since the deoxyribonucleic acid in the mitochondria determines in part the structure of the inner membranes of the mitochondria, a general explanation for the cytoplasmic inheritance of disease reaction can be given.

H. maydis hyphae ramify the chlorenchyma cells between the large vascular bundles of susceptible leaves (Jennings and Ullstrup 1957). In resistant leaves of inbreds C103 and MoG the invading hyphae were confined about the point of penetration. Resistance in C103, as shown later, is polygenic in inheritance.

Fungus sporulation within lesions varies among plants having nuclear or cytoplasmic resistance to *H. maydis*. It is most strikingly inhibited by plants expressing the chlorotic-lesion resistance. When incubated at high humidity, the chlorotic lesions on seedlings and older plants having gene *rhm* sporulate sparsely if at all. Under the same conditions, lesions of the same age on susceptible plants sporulate abundantly (Smith 1975).

Yield data on tolerance to southern leaf blight caused by race T have been obtained. Kernel weights from genotypes in *cms*-T and in normal cytoplasm

inoculated with race T were obtained. Plants in normal cytoplasm are highly resistant. An estimate of disease tolerance was calculated. It is essentially the ratio of the yield of infected susceptible plants to that of uninfected plants. Its use indicated that yields of resistant inbreds were not markedly reduced by the disease. Corn hybrids with *cms*- T cytoplasm that had high disease tolerance produced about twice as much grain as those with low tolerance (Lim et al 1974).

Phytoalexins do not seem involved in resistance to *H. maydis* (Obi 1975).

Sources of resistance

Resistance to the common race O of *H. maydis* exists in many inbred lines and varieties of corn. It is the lesion-size and lesion-number form of resistance. Inbred lines developed in areas where the disease is endemic commonly have more resistance than do lines developed in areas where the disease formerly did not exist. *H. maydis* resistance, however, is not confined to lines developed under selection pressure for resistance. Only one source of chlorotic-lesion resistance is known (Craig and Daniel-Kalio 1968: Craig and Fajemisin 1969; Smith and Hooker 1973).

Cytoplasm is the most important factor in resistance to race T (Hooker 1972, 1974a; Smith et al 1971). Resistance to race T of *H. maydis* is common. In fact, only plants having *cms*- T and similar types of cytoplasm for male-sterility are susceptible. All normal (not male-sterile) cytoplasms tested have given high resistance under field conditions. They include a set of 42 cytoplasms having the same nuclear genotype, and which are diverse and distinguishable only in that the cytoplasm originated from divergent varieties or races of corn grown for many years in different areas of the world. Many cytoplasms for male-sterility react like normal cytoplasms in that they are highly resistant to race T. Thirty sources of male-sterile cytoplasm were tested against race T in inoculated field plots at Urbana, Illinois, in 1970; they subsequently were tested in the seedling stage in the greenhouse (Smith et al 1971). Only *cms*- T and related types were susceptible. Other sources have subsequently been studied and found to be resistant.

The fertile, open-pollinated varieties Golden June and Hastings Prolific, which are susceptible to *H. maydis* race T, were widely grown in the southern states of the US before hybrid corn was developed. The *cms*- T cytoplasm was discovered in Golden June. In University of Illinois tests, varieties having *cms*- T cytoplasm were as susceptible to *H. maydis* race T as were the hybrids with the same cytoplasm that replaced them.

Nuclear resistance to race T, as seen when interacting with *cms*- T cytoplasm, ranges from tolerant to very sensitive. Many inbreds having *cms*- T cytoplasm, and crosses of more than 250 inbred lines and genetic stocks made onto *H. maydis* -susceptible testers in both normal and *cms*- T cytoplasm versions have been evaluated for resistance. Based on field and seedling data, 19 inbreds

that are superior in resistance have been identified and have been used in further genetic and breeding work.

As an additional attempt to locate nuclear genes that might restore resistance to race T when acting in *cms*-T cytoplasm, a large quantity of seed was produced in 1970 in isolation, with a mixture of genetically broad-based synthetics used as pollen parents. About 350 out of 600,000 plants exposed to *H. maydis* race T infection in the field in 1971 were selected as having some resistance, and were harvested. Subsequent studies with progenies of those plants have shown that many progenies have superior forms of resistance to both race T and race O of *H. maydis.*

Genetics of host-pathogen interactions

Resistance to *H. maydis* race O is based on nuclear genes, while resistance to race T also involves cytoplasmic factors. Several studies of the inheritance of resistance to *H. maydis* have been made (Fisher 1976: Hooker 1971, 1972; Lim 1974, 1975a,b). The factors studied were lesion type, lesion size, and percentage of leaf area infected.

Chlorotic-lesion resistance to *H. maydis* is a qualitative character. Resistant lines, developed from an introduction from Nigeria, have a single recessive gene for resistance (Smith and Hooker 1973). The gene has been assigned the symbol *rhm.*

Lesion size and percentage of leaf area infected are quantitative in expression and polygenic in inheritance. Various sources of resistance have been studied and various statistical techniques used to partition and to estimate the magnitudes of the different components of genetic variance. Most of the genetic variance is additive, and heritability values for resistance are quite high. Dominance and epistatic effects usually account for less than 10% of the total variation observed in segregating populations. Recently, indications of dominant-gene resistance have been seen in some of the lines obtained from the screening of diverse composite populations.

The inheritance of pathogenicity and pathotoxin production was studied by crossing Illinois isolates of race T and race O in the laboratory and isolating ascospore recombinants (Lim and Hooker 1971). The specific pathogenicity of race T to plants with *cms*-T cytoplasm and the specific pathotoxin production of race T showed monogenic inheritance. The two characteristics were highly associated. The degree of pathogenicity or aggressiveness of the cultures and the amount of pathotoxin produced varied quantitatively among the ascospore cultures of both race-O and race-T types.

Pathogen variability

H. maydis varies in virulence, aggressiveness, and other characters.

Two races, O and T, have been distinguished (Hooker et al 1970a,b: Smith et al 1970). They differ in cytoplasm specificity, production of toxins, symptoms

produced, reproductive rate, relation to environment, and other characteristics (Hooker 1972; Hooker et al 1970a,b; Lim and Hooker 1972a,b).

Race T was first described in 1970 from infected corn plants collected in Central Illinois in 1969. The susceptibility of *cms*-T cytoplasm plants was noted in breeding nurseries in the Philippine Islands between 1957 and 1961 (Mercado and Lantican 1961; Villareal and Lantican 1965). Tests made in 1963 in Illinois of the same inbreds and cytoplasm source used in the Philippine Islands, however, failed to show evidence of race T. For many years before 1963, open-pollinated varieties and corn hybrids having *cms*-T cytoplasm were grown in the US without noticeable infection by race T, which was the predominant race of the fungus in the US in 1970 (Fisher and Hooker 1975; Nelson et al 1970). Since 1970 it has occurred in Europe, Australia, Brazil, Central America, Nigeria, and elsewhere.

The spectrum of variation to other cytoplasm is not known. To date no specificity to the *cms*-C, *cms*-S, or normal cytoplam has been detected (Fisher and Hooker 1975).

Nor has virulence to gene *rhm* been detected. Several hundred isolates of the pathogen have been studied. Resistant plants have also now been exposed to *H. maydis* in the field in many areas for several years. Again there is no evidence of virulence in *H. maydis* to plants having *rhm.*

Breeding for resistance

Recurrent selection has effectively improved corn populations for resistance to *H. maydis.* The University of Illinois has developed several composites—some in normal cytoplasm with resistance to race O and some in *cms*-T cytoplasm with resistance to race T. Such work with normal cytoplasm is also being done by others.

Backcross breeding has involved the incorporation of gene *rhm* for chlorotic-lesion resistance into 37 elite inbred lines representing different maturities and breeding groups, and the incorporation of several sources of high polygenic lesion-number-and-size resistance into a few lines. Sixteen sources of resistance and 9 recurrent parental inbred lines are involved in the latter program. With each recurrent parental line, backcrosses of the more resistant plants have been made for three or four generations. We have found that intercrossing among families within and among sources of resistance has been a useful technique for concentrating the genes for resistance.

Selection for reaction to *H. maydis* is being made in some corn breeding programs. In the main US Corn Belt, however, southern leaf blight is not a major hazard to corn production, and breeders pay little attention to the pathogen.

Use of resistance in agriculture

The most practical means of controlling *H. maydis* race T is to avoid using *cms*-T cytoplasm in seed production. After the 1970 corn blight, the US seed

industry and its counterparts in other affected areas of the world quickly shifted to normal cytoplasm and detasseling. By 1972 the disease was under control in the US Corn Belt. Some countries have imposed legal restrictions on the planting of seed having *cms*-T cytoplasm. In the US Corn Belt. *cms*-C and *cms*-S cytoplasms released from Illinois and the US Department of Agriculture in 1970 are now being used to a significant extent in seed production.

Regional deployment of male-sterile cytoplasm (Hooker 1971, 1979) seems epidemiologically sound but has not been implemented. Except for the 50:50 blend of sterile and normal cytoplasm, multiline hybrids with different cytoplasm components are not used to any appreciable extent.

Nuclear resistance is used where the disease is a significant factor. Gene *rhm* has not reached commercial hybrids. Many hybrids and their parental inbred lines grown in the northern US Corn Belt are quite susceptible to *H. maydis.* Those hybrids are not now exposed to infection in their area of production, and the susceptibility seems to be no serious deficiency. Many sources of resistance that appear to be broadly functional against *H. maydis* are available. By proper breeding and seed production practices, it should be possible to keep *H. maydis* under control.

HELMINTHOSPORIUM LEAF SPOT

Etiology, symptoms, and importance

Helminthosporium leaf spot is caused by *H. carbonum* Ullstrup (*Cochliobolus carbonum* Nelson), which infects the leaves, ears, and stalks of the plant. Leaf lesions are oval and about 1.5 cm long. Ear rot is dark black and usually confined to the tip of the ear. Stalk rot is a dry rot of the lower stalk late in the season. The pathogen overseasons in infected plant-refuse. Continuous corn, tillage that leaves refuse on the soil surface, early-season infection, and wet weather all favor the disease.

Helminthosporium leaf spot was formerly a minor disease, but in recent years it has occurred with greater severity in the northern US Corn Belt. Damage has been greatest in seed-production fields, where losses up to 50% of some inbred lines have been reported. Hybrids have been much less damaged although susceptible hybrids can be reduced in yield by 30% (Fisher et al 1976). A few inbred lines are supersusceptible to race 1 of *H. carbonum,* and plants can be destroyed (Ullstrup 1944). Stalk infections are widespread, but are not believed to damage yield.

Expression and types of resistance

Plants can be inoculated in the greenhouse or in the field with spore suspensions of the pathogen. Seedlings in the greenhouse are incubated in mist chambers for 12 to 16 hours after inoculation. Cooked sorghum seed having *H. carbonum* growing through it makes an effective inoculum when placed in the leaf whorl.

Resistance is expressed as differences in lesion type, lesion size, and percen-

tage of leaf tissue blighted. Some lines show chlorotic flecks or small necrotic spots. Lesion-size differences on seedlings are highly correlated with percentage of leaf tissue blighted in adult plants in the field. Resistance to race 1 of the pathogen is associated with insensitivity to the pathotoxin produced by the fungus (Comstock and Scheffer 1973).

Sources of resistance

Nearly all corn grown is resistant to the highly destructive race 1 of the pathogen. Only a few inbreds and hybrids made from the inbreds are susceptible.

A wide array of reactions to race 2 and other biotypes of the fungus exists among inbreds and hybrids (Hooker 1974b.c.d. 1975). A few lines are susceptible to race 2 and similar biotypes. Other lines are highly resistant.

Genetics of host-pathogen interactions

Resistance to race 1 is monogenic dominant (Ullstrup and Brunson 1947). A second locus for resistance in also known, and a series of alleles have been reported (Nelson and Ullstrup 1964), to condition the various degrees of resistance that are expressed in different plant stages.

I have worked with a range of inbreds and genctic stocks that condition resistance to a biotype (Hooker et al 1973) of *H. carbonum* that was recently found to be causing damage in seed production fields in the northern Corn Belt. Genetic studies in the greenhouse show that several stocks carry single dominant genes for resistance (Hooker 1973). The studies are not complete, but several loci may be involved. Segregations for plant reaction is less distinct in the field than in seedlings in the greenhouse.

Genetic studies show that the pathogenicity and tosin production of race 1 segregates as a single-gene character (Nelson and Ullstrup 1961). In my studies with another race, ascospore progeny from crosses between isolates virulent to a monogenic-resistance corn line and isolates avirulent to that line segregated in a single-gene ratio.

Pathogen variability

H. carbonum is composed of several races. Race 1 was described by Ullstrup (Ullstrup 1944) on the basis of its specific virulence and high pathogenicity to certain inbred lines. Isolates unlike race 1 were grouped as race 2, which was weakly pathogenic.

Recently, other biotypes of *H. carbonum* have been found to cause damage in the northern part of the US Corn Belt and in the northeastern and eastern states (Hooker et al 1973). My isolates of *H. carbonum* are similar to race 2 but seem to cause more damage to the corn plant. In the eastern US, race 3 has been distinguished (Nelson et al 1973) on the basis of spore size and the production of long, linear lesions. More studies of virulence and avirulence to monogenic resistant corn are needed with the pathogen.

H. carbonum is also variable in other respects. Its isolates differ in aggressive-

ness if that feature is measured as lesion size at a given number of days after infection. Isolates vary in spore size.

Breeding for resistance

Breeding for resistance to *H. carbonum* has less priority than breeding for resistance to *H. turcicum* and *H. maydis*. Most breeding programs do not consider the disease. In other breeding programs, finished inbred lines are checked for susceptibility to race 1 because of its destructive nature.

At Illinois, we have used backcross breeding to incorporate dominant-gene resistance into inbred lines. Pollinations are made in the field, but disease reactions and selections for resistance are determined in the greenhouse.

Use of resistance in agriculture

Resistance to race 1 in hybrids, in use in the US Corn Belt, presumably is conditioned by a single, dominant gene. That fact, however, has been verified in only a few instances. So far, the resistance has not failed.

Resistance to race 2 and similar biotypes rests now on partial-to-nearly-complete resistance contributed by one or both parents of hybrids. Highly susceptible lines are not used in combination with each other but in combination with resistant lines. The disease causes only minor damage in commercial hybrids.

COMMON RUST

Etiology, symptoms, and importance

Common corn rust is caused by *Puccinia sorghi* Schw. It is distributed throughout the temperate areas of the world wherever corn is grown. In tropical areas it is a high-elevation disease.

Symptoms consist of brick-red pustules on the leaves. If infection is severe, entire leaves or entire plants die late in the season.

P. sorghi is believed to overwinter in the southern regions of the North American continent and spread northward each summer. The alternate host, *Uxalis* spp., is common in the US but is rarely infected. In Mexico, however, it is heavily infected each year (Borlaug 1964).

Under favorable conditions, the disease can cause significant losses. Cool weather with overnight moisture-condensation on leaves favors disease development. Through the use of nearly isogenic resistant and susceptible hybrids, losses in grain yield of 6.3 to 23.5% has been measured (Hooker 1962b; Russell 1965). Infection is greatest on certain susceptible inbred lines in breeding nurseries or seed-production fields. In 1976, and in years before, common rust was widespread in Illinois, Indiana, and Ohio.

Disease losses are not great in commercial hybrids in the US Corn Belt even though nearly all conditions are favorable for an epidemic. Vast areas

of corn are grown from the US Corn Belt southward to Central America. *P. sorghi* is present nearly every year. It has a high reproductive capacity; the urediospores are released readily and can spread long distances through the air. Most corn grown would be susceptible to *P. sorghi* in the seedling stage. Resistance that functions in the adult plant protects the crop.

Expression and types of resistance

Resistance to *P. sorghi* is clearly of two types: general and specific. Both have been studied in our laboratory and elsewhere.

General resistance is expressed by the adult plant and is usually seen in the field. The resistance is quantitative and bad on the number of pustules, or percentage of leaf tissue infected. If there are any pustules, they are few on highly resistant plants, while on susceptible plants they cover most of the leaf surface. Plant reaction is continuous between the two extremes. The nature of the resistance is unknown, although there is some association between the number of spores that germinate and infect, and the percentage of leaf tissue infected.

General resistance is believed to function against all rust races, because plant reaction is similar from year to year and functions against natural inoculum that is composed of several races.

Pustule type indicates the specific resistance in seedlings and older plants. On highly resistant plants chlorotic flecks or small necrotic spots, or sometimes, small pustules surrounded by chlorosis or other resistant reactions appear. In corn specific resistance to rust pathogens, like that in other crops, is based on host-plant hypersensitivity (Hilu 1965; Van Dyke and Hooker 1969b).

Specific resistance functions against some *P. sorghi* biotypes but not at all against others. Plant reaction does not appear to change with plant age. Some sources of specific resistance condition resistance to a large number of *P. sorghi* isolates. In a 2-year international test, however, no single source conditioned resistance in all areas of the world. A few sources of resistance were broadly functional in that they conditioned resistance in most areas of the world. Some sources conditioned resistance in only a few areas.

Sources of resistance

An adequate level of general resistance can be found within the germ-plasm pool of Corn Belt corn (Hooker 1955, 1962b, 1967a). When inbred lines were first developed in the US at the beginning of hybrid-corn production, corn breeders had many of these lines available and they selected heavily against rust susceptibility. Those rust-resistant lines, in turn, provided the germ-plasm base for the newer inbred lines that now make up many of the modern hybrids. Consequently, high susceptibility to *P. sorghi* is quite rare among US inbreds even though many corn breeders pay little attention to rust reaction. Some inbred lines are very susceptible, as are some narrow-base composites. Inbreds that happen to be susceptible are usually crossed with more resistant inbreds,

because of heterotic patterns for grain yield and not because of rust reaction, so the hybrids grown on the farm have an adequate degree of disease resistance.

Natural rust epiphytotics have occurred in several years and serve to identify inbred lines with mature-plant resistance to rust. For example, in 1953, 405 established inbred lines in the dent-corn breeding nursery at the Iowa Agricultural Experiment Station were evaluated for rust reaction during a natural epiphytotic. The standard percentage-of-infection scale used for cereal rusts was followed. Approximately one-half of the lines had 1% or less rust infection. About 28% of the lines had 5% or more infection. When 160 of the field-resistant lines were tested in the greenhouse against 18 individual rust cultures, most were susceptible to all (Hooker 1955). The lines that showed no resistance in the seedling stage but were relatively rust-free in the field in 1953 also were relatively rust-free in inoculated field-plots the following year. Subsequent epiphytotics have shown that relatively few lines are highly susceptible to *P. sorghi* in the adult-plant stage.

Numerous sources of specific resistance are known. Most of those reported in the mid-1920's (Mains 1926, 1931) were lost. My associates and I more recently have located more than 100 sources of resistance expressed in the seedling stage by empirically screening many corn inbred lines and open-pollinated varieties from many parts of the world (Hooker 1962a,b; Hooker and LeRoux 1957; Hooker et al 1955). Specific resistance is also expressed by teosinte and by Tripsacum (Malm and Beckett 1962). Interestingly, no open-pollinated corn variety was uniformly homozygous for any rust-resistant gene. Only one major gene for rust resistance was usually found in any one inbred or variety source. Furthermore, the frequency of plants with specific resistance in each variety was usually low. Only rarely was there a high frequency of resistant plants.

Genetics of host-pathogen interactions

The inheritance of mature-plant resistance to *P. sorghi* was studied over a 3-year period in the field at Urbana, Illinois (Hooker 1962b, 1967a,b). The study involved parents and segregating F_2 populations of 5 susceptible × susceptible, 15 resistant × resistant, and 44 resistant × susceptible crosses. The field arrangement consisted of 10 plants in each plot with all plots pertaining to a cross together but in a random order. Hills of susceptible corn at the ends of each plot were inoculated with a mixture of *P. sorghi* biotypes, and the fungus was allowed to spread naturally into the test plots. Late in the season, while the plants were still green, approximately 100 plants of each segregating population and 10 plants of each nonsegregating population were scored individually for percentage of leaf area infected. Four crosses were advanced to the F_3 generation and scored for percentage of rust infection on the basis of a progeny row from each F_2 plant.

Rust reaction varied widely among the F_2 plants. The variation ranged from the rust scores of one parent to those of the other parent. Sometimes

rust scores of individual F_2 plants exceeded the parental scores, indicating transgressive segregation. Variation among the F_2 plants of each cross was continuous, and the frequency distribution of individual plant scores approximated a normal distribution. A majority of the F_2 plants had scores near the population mean. The mean score of each F_2 population was near the calculated average score of the two inbred parents. The mean score of the F_1 of each cross usually was between that of the F_2 and that of the most resistant parent. The latter may indicate partial dominance for resistance or manifest heterosis.

The results from the F_2 were supported by data from the F_3. Individual progenies more resistant or more susceptible than either parent were obtained.

Variation among the inbred or F_1 plants usually was quite small. That indicates that the intensity of rust infection throughout thc test plot was quite uniform and that only a small amount of variation among plants in the various populations can be attributed to nongenetic differences.

Heritability values were calculated by determining the ratio of the total genotype variance to the phenotypic variance. The heritability estimates ranged from 17 to 98% and averaged 84.3% for the 64 crosses.

Those data indicate that many genes, but perhaps not a large number, condition mature-plant resistance to *P. sorghi.*

Considerable data on the inheritance of specific resistance are available (Hagan and Hooker 1965; Hooker 1963a, 1967b; Hooker and Russell 1962b; Hooker and Saxena 1967, 1971; Hooker et al 1955; Lee et al 1963; LeRoux and Dickson 1957; Mains 1926, 1931; Malm and Hooker 1962; Rhoades and Rhoades 1939; Saxena and Hooker 1968; Wilkinson and Hooker 1968). Mains (Mains 1926, 1931) showed that such resistance could be inherited as a single dominant gene. A cytological study showed that the gene in GG208R is located on the short arm of chromosome 10 (Rhoades and Rhoades 1939; Rhoades 1935). It is the first gene for disease resistance in any crop to be located on a chromosome. Seed of GG208R was maintained and the chromosomal location confirmed through the use of chromosome translocation stocks (Russell and Hooker 1962). The gene was first designated as *Rp* (Rhoades and Rhoades 1939), later as *Rp1* (Russell and Hooker 1959), and more recently as $Rp1^a$ (Hooker 1963a). Subsequent genetic studies of other rust-resistant corn lines from various regions of the world have shown the existence of a large series of alleles at that locus (Hagan and Hooker 1965; Hooker and Russell 1962b; Lee et al 1963; Russell and Hooker 1959; Wilkinson and Hooker 1968; Table 1).

Subsequent work (Hooker and Saxena 1971; Saxena and Hooker 1968) has shown that some of what we called "alleles" of *Rp* are in fact very closely linked genes. A line carrying one allele was crossed with a line carrying another presumed allele, and the resulting F_1 was crossed with a homozygous, susceptible tester. A seed-color marker from the tester excluded the effects of unwanted pollen. Large populations of those test crosses are easily produced in corn and were evaluated in greenhouse seedling tests to detect genetic recombination

for disease reaction. In the first series of experiments, a single rust culture avirulent on both resistant parents was used. With that testing scheme, only susceptible recombinations could be detected. A susceptible seedling, however, could also result from a gene mutation or from deletion of a chromosome. Therefore, the occurrence of those phenomena was estimated by testing the F_1 of a resistant inbred × a susceptible inbred for disease reaction. A statistical test was used to determine if the frequency of susceptible seedlings in the test cross was significantly different from the frequency of susceptible seedlings in the check used to estimate the number of susceptible seedlings that might result from the combined effect of mutation and chromosomal deletion. In test-cross populations of 4,000 to 9,500 plants, recombinations of 0.27% and 0.37% were found between *Rp1*^{a} and *Rp1*^{k} and between *Rp1*^{g} and *Rp1*^{l}, respectively. Those values were significantly greater than what would be expected to result from mutation and deletion. The crosses *Rp1*^{d} × *Rp1*^{k} and *Rp1*^{a} × *Rp1*^{d} were also tested but. by the same measure of statistical significance, no recombinations were detected.

In a subsequent experiment, rust cultures giving reciprocal reactions on the two reistant parents in the test cross were available. Through use of a mixture of two cultures, it was possible to identify both the susceptible and resistant recombinants in a test cross. To confirm the phenotypes of those rare plants, detached leaf sections were inoculated with each culture separately. The rare plants also were saved for seed production and progeny testing. Both resistant and susceptible recombinations in a 1:1 ratio were detected in the test crosses. In test-cross populations of almost 14,000 to 19,000 individuals for each cross, the recombination values of 0.16, 0.10, and 0.22% were observed between *Rp1*^{c} and *Rp1*^{k}, *Rp1*^{b} and *Rp1*^{f}, and *Rp1*^{a} and *Rp1*^{c}, respectively. The genotypes of the recombinants were confirmed in progeny tests.

In rust-reaction tests using numerous single biotypes of *P. sorghi,* the resistant recombinant progenies were resistant to all biotypes for which either of the two parental components conditioned resistance. If neither parental component conditioned resistance, the recombinant expressed no resistance. In other words, the resistance conditioned by the recombinant was that of the additive effects of the two components. That is what would be expected on the basis ofthe gene-for-gene hypothesis.

The resistant *Rp1*^{c}-*Rp1*^{k} recombinant as well as genes *Rp1*^{c} and *Rp1*^{k} were among the nearly isogenic lines grown in various areas of the world in an international survey of virulence genes in *P. sorghi.* In all tests in which either *Rp1*^{c} or *Rp1*^{k}, or both, remained rust-free, the recombinant remained rust-free. In no test where virulence to both *Rp1*^{c} and *Rp1*^{k} was detected did the recombinant remain rust-free.

The genes *Rp1*^{c} and *Rp1*^{k} have been separated and recovered from the recombinant. Each gene has retained the spectrum of resistance of the original gene.

Thus, it is believed that *Rp1* is a complex locus about 0.40 map units long.

Each "allele" has a functional gene and one or more nonfunctional genes closely linked. On the basis of the broad pattern of resistance conditioned by *Rp1*^{d} and the fact that no recombination was obtained between *Rp1*^{d} and the other alleles, it is believed that *Rp1*^{d} probably consists of two or more functional genes closely linked. Perhaps a series of tandem duplications of the original gene gave rise to the *Rp1* locus, and during evolution the different elements differentiated to provide the specificity of reaction to different biotypes of *P. sorghi.* If that is true, various combinations of functional and non-functional genes could give rise to what, at first study using conventional genetic procedures, appeared to be different alleles at a single locus.

Other genes for rust resistance also occur on chromosome 10. Locus *Rp5,* first identified in corn line 191 from Uruguay, is linked to *Rp1* by about 1.1 ± 0.2 crossover units. Locus *Rp6* in corn line 597 from Turkey is linked to *Rp1* by about 2.1 ± 0.4 crossover units and to *Rp5* by about 3.3 ± 0.6 crossover units. Thus, *Rp1* must be situated between *Rp5* and *Rp6* (Hagan and Hooker 1965; Wilkinson and Hooker 1968). In addition to those three genes or gene clusters for resistance to *P. sorghi,* gene *Rpp9* for resistance to *P. polysora* Underw, is on chromosome 10 and is linked to *Rp1*^{d} by about 1.6 crossover units (Ullstrup 1965).

Dominant genes for resistance to *P. sorghi* also occur on chromosomes 3 and 4 (Russell and Hooker 1962). Locus *Rp3* is on chromosome 3. The locus was first identified in a series of Australian inbred lines (Hooker 1963a). Corn lines from South Africa, Mexico, and Yugoslavia also have single, dominant alleles at that locus (Table 1; Wilkinson and Hooker 1968). The genes of *Rp3* in the Australian inbreds NN14 and M16 exhibit a reversal of dominance to two biotypes of *P. sorghi* (Hooker and Saxena 1967). Either gene *Rp3* exhibits a dosage effect, or closely linked dominant and recessive genes occur at or near that locus in those resistant lines. However, those putative genes were

Table 1. Genes for resistance to *Puccinia sorghi* identified from various sources.

Gene	Source	Country	Gene	Source	Country
Rp1^{a}	GG208R, G Glow, G. King, 777	USA	*Rp1*^{m}	PI163563	Guatemala
Rp1^{b}	B38, B216, B217	USA	*Rp1*^{n}	BZU-20	Yugoslavia
Rp1^{c}	K148, B. Y. Dent, Syn. A.	USA	*rp2*	13b	USA
Rp1^{d}	Cuzco	Peru	*Rp3*^{a}	25	Australia
Rp1^{d}	Kitale, Njoro	Kenya	*Rp3*^{b}	M16	Australia
Rp1^{e}	B49	Argentina	*Rp3*^{b}	E697	South Africa
Rp1^{f}	PI172332	Australia	*Rp3*^{c}	NN14	Australia
Rp1^{g}	PI163558	Guatemala	*Rp3*^{d}	Leon 127-4-1	Mexico
Rp1^{h}	Guanajuato 29-157A	Mexico	*Rp3*^{e}	Hidalgo 3-5-1	Mexico
Rp1^{i}	PI163558	Guatemala	*Rp3*^{f}	PI251653	Yugoslavia
Rp1^{l}	Queretaro VI366	Mexico	*Rp4*^{a}	Queretaro V260-1	Mexico
Rp1^{k}	Queretaro V231-5	Mexico	*Rp4*^{b}	PI193906	Ethiopia
Rp1^{j}	PI163558	Guatemala	*Rp5*	PI186191	Uruguay
			Rp6	PI172597	Turkey

not detected by techniques similar to that with *Rp1;* if they do exist they occur within a map distance of 0.06 units (Saxena and Hooker 1974).

Locus *Rp4* is on chromosome 4. Corn lines from Mexico and from Ethiopia have alleles at that locus (Table 1). Allele $Rp4^b$ in line 906 from Ethiopia expresses a Z reaction type to *P. sorghi* (Van Dyke and Hooker 1969a). In that reaction type, the young leaf sections, especially those portions still chlorotic and just emerging from the leaf whorl at the time of inoculation, are rust-susceptible, but the older distal area of the leaf is resistant, as are all the older leaves.

In addition to dominant genes, recessive genes and modifying genes for resistance are also known (Hooker 1962b; Malm and Hooker 1962). Such forms of resistance have not been studied in as much detail as has dominant-gene resistance. Resistance may be due to one, two, or three recessive genes in a corn inbred line. In inbreds Amargo 47 and Midland 125, high resistance is conditioned by the complementary effect of three recessive genes at independent loci. The importance of modifying factors is not known, but modifying genes for resistance to *P. sorghi* have been detected in several studies.

Many sources of specific resistance to *P. sorghi* have not been studied adequately in inheritance tests. It is evident, however, from the backcrossing program used to develop nearly isogenic lines (Hooker and Russell 1962a) that many of the unstudied sources have single dominant genes. Enough work has been done to show that a large number of loci and alleles for rust resistance exist in corn. The genes frequently function as single genes, but may interact in various ways. They occur on at least four chromosomes. Some loci are closely linked.

There is only limited work on the inheritance of virulence in *P. sorghi* (Flangas and Dickson 1961). It was done before the nearly isogenic lines became available and before the present number and diversity of rust-resistance genes became known. The ability to culture *P. sorghi* through its full life cycle on detached leaves of its hosts in the laboratory may expedite the studies (Hooker and Yarwood 1966).

Pathogen variability

P. sorghi is composed of many races (Hooker 1962b; LeRoux and Dickson 1957; Stakman et al 1928) although a set of differential varieties has never been formally established. At the University of Illinois we have used numerous cultures of *P. sorghi* with various combinations of virulence. To identify those biotypes as races and to differentiate and describe the large number of races that are possible, based on the number of genes for resistance in our collection, seems to be an academic exercise. Furthermore, the differentiation of *P. sorghi* biotypes and groups of biotypes into physiologic races seems to serve no useful purpose.

The gene-for-gene relationship (Flor 1955) that prevails for other rusts presumably exists in the *Zea mays-Puccinia sorghi* host-pathogen system. All

available data support such a concept. If the relationship exists, an international survey for virulence genes in *P. sorghi* indicates that genes for virulence do not occur at random throughout the world. Furthermore, many genes for virulence occur in Peru, Korea, and Ethiopia, while a few genes for virulence occur in Northeastern US and adjacent Canada, Central US, Hawaii, India, and Italy. The interpretation, however, may be faulty, if some of the "genes" recognized as individual genes do, in fact, represent functional units composed of two or more closely linked genes. University of Illinois workers have failed to find recombination between any gene tested and *Rp1*^{d}, for example, and virulence to *Rp1*^{d} is rare. If *Rp1*^{d} is a complex locus, what we fail to find is combined virulence to all the components of the locus.

In other host-pathogen systems, the genotypes of the host crop grown in an epidemic area influence to a considerable degree the prevalence of genes for virulence in pathogens of that crop. When the host population is heterogeneous, a greater diversity of avirulence genes will persist. When the host population is uniform for specific resistance, virulence genes corresponding to the genes for resistance tend to predominate. It is not known how many and what genes for specific resistance might be in the corn crop in different areas of the world. In the US Corn Belt, genes for specific resistance are rare.

P. sorghi biotypes may vary in aggressiveness. At least I find that spore production of some biotypes is greater than that of other biotypes on the same susceptible host and under other comparable conditions.

Breeding for resistance

Corn breeders and pathologists have no significant testing program for rust reaction, and rust resistance is not an important objective in US corn-breeding programs.

Various forms of recurrent selection should be effective in improving corn populations used as sources of inbred lines. The adult-plant form of resistance is highly heritable.

If new lines are developed under epiphytotics of *P. sorghi,* there should be little problem in selecting and fixing in a stable condition the general form of resistance.

Backcrossing has been used to incorporate genes like *Rp1*^{d} into a few widely used but rust-susceptible inbred lines. The method is highly efficient, and if the genes for resistance are not widely used in the crop, the resistance should be effective.

Specific resistance is rare among the widely used inbred lines. Gene *Rp1*^{d} is used in a few lines and supplements the mature-plant resistance that is conditioned by the other parents of the hybrids. Virulence to *Rp1*^{d} has never been detected in the US.

The extensive use of numerous inbred lines with mature-plant resistance is the primary reason that corn rust is not an economic disease in the US Corn Belt. Rust infection occurs but is kept continuously at a low level during the

critical growth period of the crop. There is little or no selection for virulent races of the pathogen that can develop in epiphytotic proportions on plants with only narrow bases of specific resistance.

GENERAL ASSESSMENT

The US corn blight epidemic of 1970 heightened worldwide awareness that all major food and feed crops are vulnerable to plant diseases and other hazards. World food and energy shortages, as well as good business sense, demand that every effort be made to keep losses due to plant disease at a minimum.

Plant diseases are preventable. The ideal method of prevention is the use of genetic resistance. The method is not costly to the farmer, requires few daily management decisions, and causes little concern for the quality of the environment. Such a program of disease control embraces eight facets: vigilance, problem assessment, reaction to disease, sources of resistance, nature and expression of resistance, inheritance of resistance, breeding for resistance, and utilization of resistance in agriculture.

Plant-disease problems change. New problems continue to arise and old problems recur because of changes that take place in cropping practices, germ plasm in use, and pathogen populations. Through vigilance, potentially important problems can be detected. In the US, however, there are no organized programs for detecting new disease problems or new races of current pathogens. Monitoring is done by people whose primary activities are in other areas. Fortunately, corn pathogens are not highly variable.

Assessments must be made to determine which problems need attention, what resistance is needed in the crop, and how much and what kind of resistance is needed to protect the crop. Potential losses should receive as much as or even more consideration than actual crop losses.

It is important to know the reaction to common diseases of crop varieties and hybrids. It is especially important for new varieties and hybrids about to be released from breeding programs.

Techniques are needed to adequately evaluate crop germ plasm for reaction to disease. Resistance to the same pathogen may be expressed in several ways. The resistance mechanisms need to be identified and, if possible, equated to specific genes for resistance so that they can be selected during plant breeding. An understanding of the physiology, histology, and biochemistry of disease resistance is interesting, but unless such information has selection value in a screening program or is useful in the identification of specific genes, it is not of major importance in a breeding program for disease resistance. US corn-breeding programs devote extensive effort to evaluating new hybrid combinations for yield, maturity, and standability. Less effort is given to determining reactions to specific diseases. Hybrids with serious weakness, however, usually express those weaknesses through poor yield. International testing is a powerful tool in testing for disease reaction.

Designing a breeding program to use a resistance component becomes easier when the mode of inheritance is known. Much of the disease resistance in corn is polygenic in inheritance. Some effective resistance is monogenic.

Breeding for resistance in US corn-breeding programs is one of improving source of populations, selecting during inbred-line development, and using backcrossing where resistance is needed and monogenic. Various forms of recurrent selection are used to improve source populations. Not all breeding programs, however, devote much attention to population improvement. Disease resistance is considered during selfing and selection as inbred lines are developed. Frequently, inbred lines are developed and, after establishment, are tested for disease reaction. The most susceptible lines are discarded. Backcrossing is used extensively to incorporate gene *Ht* for resistance to *H. turcicum* into inbred lines.

Most US hybrids have at least moderate resistance to the important diseases. Much improvement, however, is possible.

The current level of resistance is quite effective in disease control. The 1975 season was a high-rainfall season and should have favored many leaf diseases in the US Corn Belt. Few disease problems were encountered and grain yields were the highest on record.

Diversity occurs in the US Corn Belt. While some inbred lines are widely used, they are used in combination with other lines in hybrids. Various modifications of standard lines have also been produced, and these are used by seedsmen. Breeding programs are productive, and new hybrids are continually being introduced. While some pedigrees are popular, a diversity of hybrids are offered to the farmer. The American seed industry is competitive and capable of making rapid changes, if necessary. Nearly all firms have winter seed-production programs. Even though the commercial crop is hybrid, susceptible cytoplasms or inbred lines can be replaced in the crop in 1 or 2 years. A wide array of sources of resistance is available in breeding programs. No doubt more sources await discovery. It is to be hoped that the resistance will be adequate in the years ahead.

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available data support such a concept. If the relationship exists, an international survey for virulence genes in *P. sorghi* indicates that genes for virulence do not occur at random throughout the world. Furthermore, many genes for virulence occur in Peru, Korea, and Ethiopia, while a few genes for virulence occur in Northeastern US and adjacent Canada, Central US, Hawaii, India, and Italy. The interpretation, however, may be faulty, if some of the "genes" recognized as individual genes do, in fact, represent functional units composed of two or more closely linked genes. University of Illinois workers have failed to find recombination between any gene tested and *Rp1^d,* for example, and virulence to *Rp1^d* is rare. If *Rp1^d* is a complex locus, what we fail to find is combined virulence to all the components of the locus.

In other host-pathogen systems, the genotypes of the host crop grown in an epidemic area influence to a considerable degree the prevalence of genes for virulence in pathogens of that crop. When the host population is heterogeneous, a greater diversity of avirulence genes will persist. When the host population is uniform for specific resistance, virulence genes corresponding to the genes for resistance tend to predominate. It is not known how many and what genes for specific resistance might be in the corn crop in different areas of the world. In the US Corn Belt, genes for specific resistance are rare.

P. sorghi biotypes may vary in aggressiveness. At least I find that spore production of some biotypes is greater than that of other biotypes on the same susceptible host and under other comparable conditions.

Breeding for resistance

Corn breeders and pathologists have no significant testing program for rust reaction, and rust resistance is not an important objective in US corn-breeding programs.

Various forms of recurrent selection should be effective in improving corn populations used as sources of inbred lines. The adult-plant form of resistance is highly heritable.

If new lines are developed under epiphytotics of *P. sorghi,* there should be little problem in selecting and fixing in a stable condition the general form of resistance.

Backcrossing has been used to incorporate genes like *Rp1^d* into a few widely used but rust-susceptible inbred lines. The method is highly efficient, and if the genes for resistance are not widely used in the crop, the resistance should be effective.

Specific resistance is rare among the widely used inbred lines. Gene *Rp1^d* is used in a few lines and supplements the mature-plant resistance that is conditioned by the other parents of the hybrids. Virulence to *Rp1^d* has never been detected in the US.

The extensive use of numerous inbred lines with mature-plant resistance is the primary reason that corn rust is not an economic disease in the US Corn Belt. Rust infection occurs but is kept continuously at a low level during the

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STRATEGIES IN COMBATING CEREAL DISEASES IN EUROPE, WITH SPECIAL REFERENCE TO YELLOW RUST OF WHEAT

J. C. ZADOKS

RESEARCH ON YELLOW RUST (*Puccinia striiformis* Westendorp) of wheat (*Triticum aestivum* L. emend. Thell. subsp. *vulgare* Mackay) (2 n = 6x = 42) is well organized in Europe. Races are identified and varieties are tested by at least three major stations—the Institute of Phytopathological Research (IPO), Wageningen, in the Netherlands; the Biologische Bundesanstalt (BBA), Braunschweig, in the Federal Republic of Germany, and the National Institute of Agricultural Botany (NIAB), Cambridge, in the United Kingdom. Exchange of seed, rust isolates, and information among these institutes is routine. A new international set of standard differentials for worldwide use has been developed in close collaboration with American workers on yellow rust in the northwest Pacific area of the USA (Johnson et al 1972). There is a continuous exchange of information between Europe and the USA, and mutual visits are frequent. By agreement made, Europe uses a supplemental differential set for detailed screening ot European isolates.

The First International Congress of Plant Pathology in London, in 1968, accepted a resolution to initiate an International Survey of Factors of Virulence of *P. stiiformis,* a system for the early detection of new virulences. The system was implemented by the IPO (Stubbs et al 1974). Some 250 nurseries are now grown in Europe, Asia, and Africa, and in North, Central, 2nd South America. Races are identitied at the IPO, in close cooperation with the BBA. The three stations mentioned give service and provide training to colleagues from many developing countries. Regular contacts are maintained with the International Maize and Wheat Improvement Center (CIMMYT) and with field workers engaged in national wheat programs in developing countries.

Scientific results are discussed and new ventures initiated at the European and Mediterranean Cereal Rusts Conferences. These meetings, to which non-Europeans are also welcome, began in Braunschweig, Germany, in 1956 and have continued to be held at 4-year intervals. The most recent conference was in Switzerland in 1976. Part of the organizational netwrk is the *Cereal Rusts*

Laboratory of Phytopathology, Agriculture University, Wageningen, The Netherlands.

Bulletin published by the European and Mediterranean Cereal Rusts Foundation, a private, nonprofit foundation with headquarters in Wageningen, The Netherlands.

The conference structure, based on mutual understanding and close ties of friendship, is completely voluntary, without formal international agreements or funding.

This paper reviews the status of yellow stripe rust (*Pucciniastriiformis*) on wheat in Europe. It discusses recent developments in the ideas on control strategies, with emphasis on the period from 1972 through 1976; mentions possibilities of adult plant resistance, "vertical" resistance, "horizontal" resistance, and eventual combinations, with and without chemical control: and explains the pattern of international cooperation. It also refers to relevant research in other cereal crops.

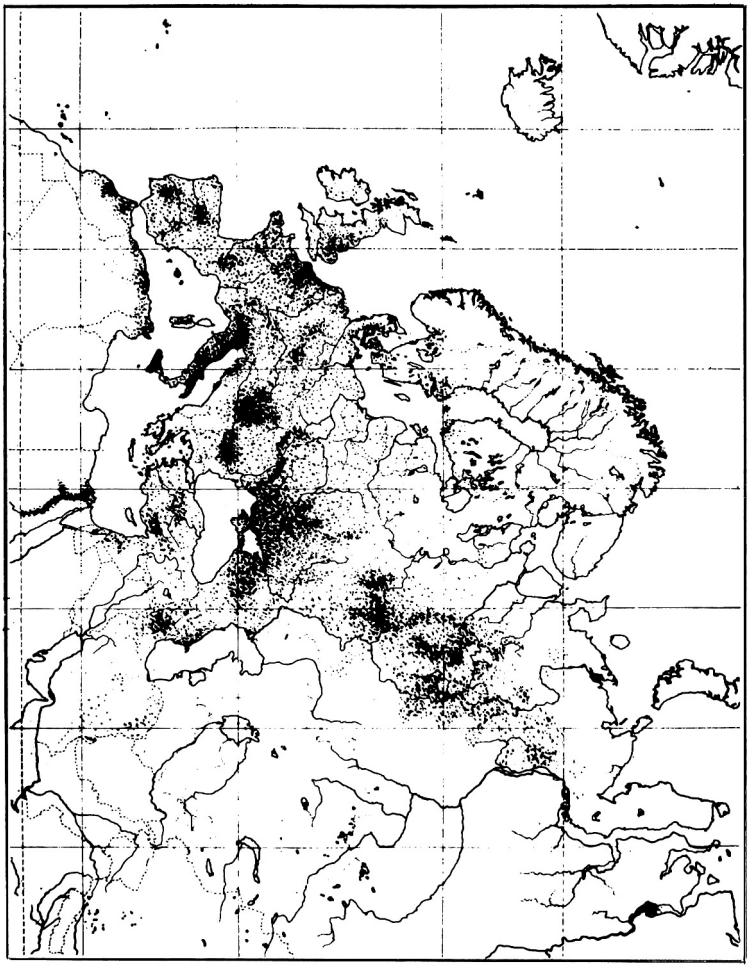
As a starting point for the scientific aspects of this paper, I use the excellent publication of Macer (1972), and as the end point the proceedings of the European and Mediterranean Cereal Rusts Conference in Switzerland, 1976.

THE EUROPEAN SCENE

Wheat is grown with varying intensity nearly everywhere in Europe (Fig. 1). It occupies about 26 million hectares in areas with a population approaching 500 million. Yellow rust is found nearly everywhere. From west to east the climate of Europe changes from temperate atlantic, with mild winters and relatively cool summers, to continental, with cold winters and hot dry summers. From north to south the climate changes from boreal to subtropical. Wheat cultivars and agricultural practices are adapted to the variations in climate and a broad variety of soils. In view of such diversity of environments, it would be unthinkable to have European wheat research directed by a single institute; many plant breeding stations are needed (Fig. 2). Only one wheat crop is grown in a year, but from north to south the vegetation period varies from 11 to 4 months, roughly; the wheat-free period varies from 1 to 8 months, accordingly. The longer the wheat-free period, the more difficult is the carry-over of the yellow-rust inoculum from one year to the next.

Northwest Europe is usually under the influence of cyclonic weather patterns, which distribute wind-borne spores freely. Rust races originally found in one country spread readily over others. In addition to the rapid exchange of great amounts of inoculum within northwestern Europe, there may be a less conspicuous influx of new genes for virulence by way of the Near East. At first glance, the Near East does not satisfy the criterion "moist and cool," but wheat is grown there in winter, and yellow rust can become a problem even in countries like Egypt. Yellow rust thrives when nights are cool and dew is ample, a situation that can exist in all the Mediterranean countries. In otherwise uncongenial climates, irrigation furthers attacks by yellow rust.

Figure 3 shows the average annual losses due to yellow rust in Europe and

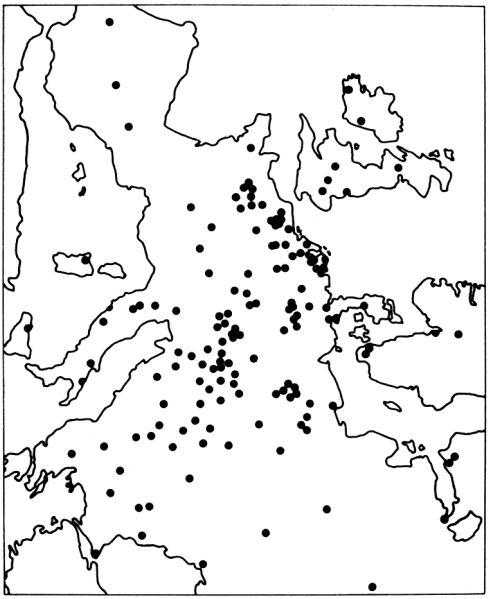


1. Wheat cultivation in Europe. Total area under wheat, about 26 million ha. Each dot represents 2,000 ha (de Casseres 1959).

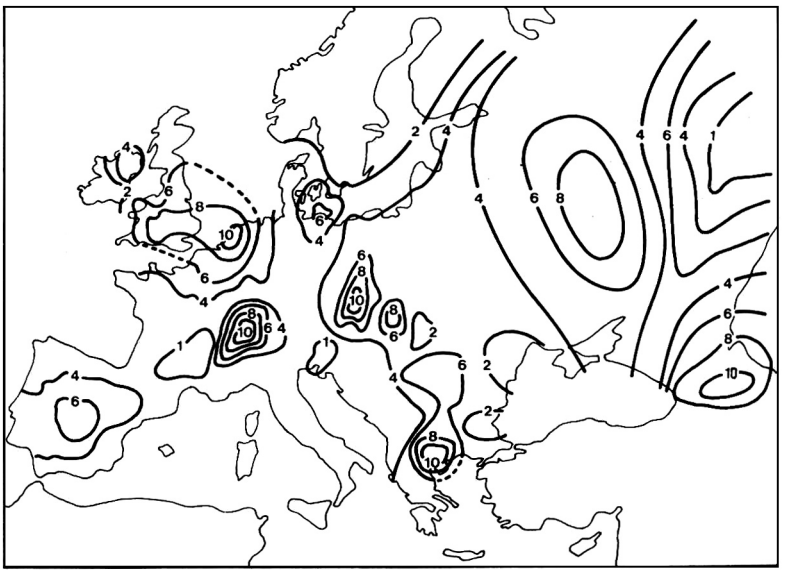
Figure 4 the climatic risks incurred when susceptible cultivars are grown. If the average wheat losses are low in areas with high climatic risks, credit must be given to the plant breeders for effective use of resistance to control loss.

EVOLUTIONARY ASPECTS OF YELLOW RUST

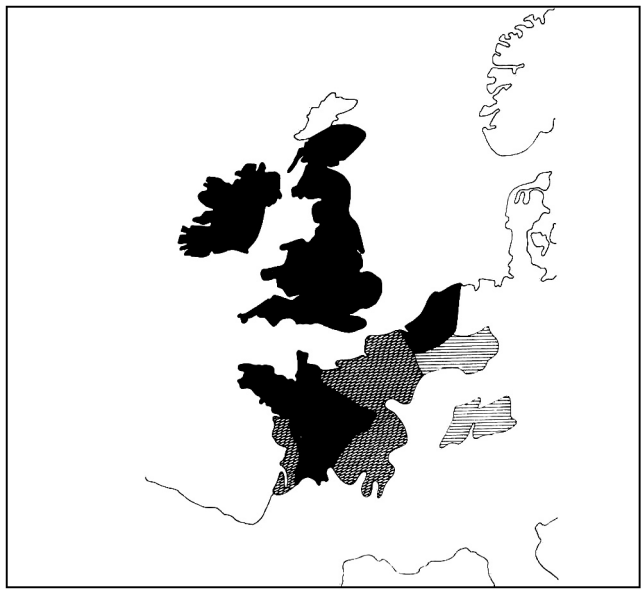
Yellow rust, as a species, has probably evolved together with its grass hosts in the mountain areas of the Near and Middle East. It has spread over the world except Australia. In the tropics, where wheat is grown at high altitudes,



2. Breeding stations for winter wheat in Europe (Broekhuizen 1969).



3. Provisional map showing average annual losses (%) due to yellow stripe rust (*Puccinia striiformis*) on wheat (Rijsdijk and Zadoks 1977).



4. Provisional map showing the climatic risks caused by yellow stripe rust (*Puccinia striiformis*) on wheat when susceptible cultivars are grown (Rijsdijk and Zadoks 1977). Black = high risk, cross-hatched = medium risk, hatched = low risk, white = no data yet available

climatic conditions are suitable for the rust. When cereals were selected by man, the originally graminicolous rust became also cerealicolous. In some areas of the world the graminicolous and cerealicolous phases of the rust alternate regularly (Zadoks 1961), but in the old agriculture of Europe some types of the rust became purely graminicolous. The rust specialized to the extent that in Europe *formae speciales* could be identified on wheat, barley, rye, and *Dactylis glomerata* (Eriksson and Henning 1896). The latter f. sp. has now received varietal rank (Manners 1960). Within the f. sp. *tritici* physiologic specialization has proceeded. There are many physiologic races on record that are more or less specialized on wheat; among them are the northwestern European races, which no longer alternate with grasses. The races are characterized by differences in virulence to various cultivars; in addition, there are numerous indications that they differ in ecological preferences (Schroeder and Hassebrauk 1964), temperature resistance, survival ability (Zadoks 1961), competitive fitness, and other characters, but such matters will not be pursued here.

The pattern of development of physiologic races is typical of that in "man-guided evolution" (Johnson 1961), as it is in so many cereal rusts. Sexual recombination is unknown. Mutation is a source of new races, as has been

shown experimentally (Stubbs 1968). The rust is a dikaryotic fungus in which somatic or parasexual recombination of nuclei can lead to new races (Taylor 1976). Though exchange of nuclei apparently is easy, exchange of chromosomes during mitosis is improbable because the nuclear envelop remains intact during the metaphase, the crucial phase for chromosomal exchange (Wright 1976). Field observations suggest that under severe infection pressure many new races appear that are abortive because they lack fitness (Zadoks 1975). So a paradoxical picture emerges; successful races are stabilized and protected against genetic disintegration, and, at the same time, new races can appear at the bewildering rate of about one per year in northwestern Europe.

New races apparently originate monotopically, develop under high selection pressure when new and disease-free cultivars are introduced, and spread rapidly over hundreds of kilometers per year, as can be detected from the International Yellow Rust Nurseries (Stubbs et al 1974; Zadoks 1961). Stubbs (1972) states that the potential of yellow rust to develop new races seems to be the same all over the world, and documents his statement with an example of convergent evolution. In Chile, where European cultivars are grown and used in breeding programs, rust races similar to those found in Europe develop. From the point of view of the fungus, the problem is not to be able to produce new variants, but to be able to exploit them (Parlevliet and Zadoks 1977).

RESISTANCE

Vertical resistance

Resistance testing is currently done in the greenhouse, using primary leaves of seedlings. Conditions are commonly standardized at 15°C and a light intensity of at least 10,000 lux (Johnson et al 1972). With the usual techniques, using monosporic isolates (uredospores are one-celled and dikaryotic), repeated testing, and a standardized method of scoring response types (reaction types), a gene-for-gene system could be identified (Macer 1972), as in so many other host-parasite systems. Some genes have been classified and numbered, and several others await that classification.

The host plant is not equally susceptible to the rust at all growth stages (Zadoks 1961; Dutlu and Prescott 1976). A simplified representation is given in Table 1. The R-S types will not be discussed here because they appear sporadically. The S-S types can be readily discarded by a combination of greenhouse and field trials. The R-R and S-R types are of special interest. Most trials by most plant breeders have been and still are field trials in which only adult-plant resistance is tested. Whether that resistance is also expressed in seedlings can be ascertained only by greenhouse tests. If it is, we have R-R resistance, also called overall resistance. Because the selection pressure exerted by intensive greenhouse screening came into being after 1960, most current resistances are of the S-R type. Relatively few reliable R-R gene donors have

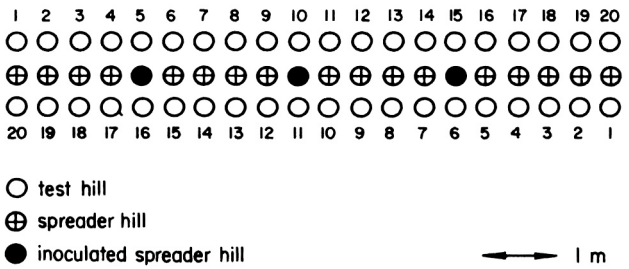
Table 1. Yellow rust *(Puccinia striiformis)* **on wheat: responses at seedling and adult stages.**

Response[a] combination	
Seedling	Adult
S	S
S	R
R	S
S	S

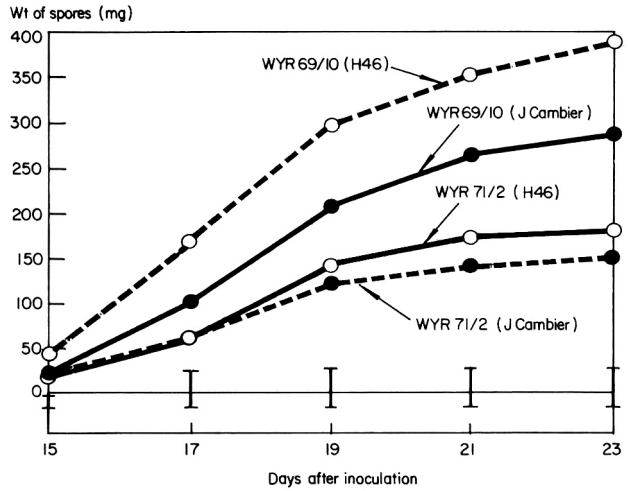
[a] S = susceptible, R = resistant

been found, but they are enough at least to demonstrate the operation of a gene-for-gene system for overall resistance.

Some epidemiological phenomena that could not be explained by the then-current race surveys based on work with seedlings in the greenhouse were observed in the field in the 1950's. Consequently, the "race nursery" technique was developed (Zadoks 1972b). A race nursery is a differential set of mature plants grown in the field (Fig. 5); recently, race nurseries have also been grown under polythene tunnels for improved separation of isolates and better conditions for infection (Priestley and Doodson 1976). With this technique, current races could be subdivided into subraces, called "field races," to distinguish them from races differentiated in the greenhouse (Zadoks 1961). The idea of field races, which were to be regarded as the basic epidemiologic units (Zadoks 1966), was readily accepted in continental Europe but initially rejected in the United Kingdom. Person-analysis (Person 1959) showed that adult-plant resistance (S-R type) could well be governed by a gene-for-gene system (Zadoks 1961). That evidence favored the idea that selection pressure on the rust was exerted mainly in the later phase of the epidemic, when adult-plant resistance



5. Design of a rust race nursery for continuous monocylic testing of resistance to one race of *Puccinia recondita* in 70 wheat cultivars. Cultivar clump diameter = 25 cm. distance between centers of clumps = 40 cm (Zadoks 1972b).



6. Cumulative weights of uredospores of two isolates of *Puccinia stiiformis* collected from 100 seedlings of wheat cultivars Hybrid 46 and Joss Cambier. Vertical bars are least significant differences at the 5% level. Both isolates belong to race 104 E 137; nevertheless they are genetically distinct (Johnson 1972).

had already come to expression. After severe epidemics in the 1970's, researchers in the United Kingdom also accepted the idea of field races (Priestley and Doodson 1976).

Two technical problems remain to be discussed: 1) how to recognize mature-plant resistance in the greenhouse, and 2) how to recognize races that have virulence to mature-plant genes, using seedlings in the greenhouse. Question 1 can be answered in part. The effect of mature-plant resistance genes cannot be seen in seedlings at the usual 15°C test temperature. However, a decrease in the response of a seedling with increasing temperature can indicate the presence of mature-plant resistance (Lewellen and Sharp 1968: Stubbs 1964). Mature-plant resistance can manifest itself in the seedling at elevated temperatures. To answer question 2, British workers tested two isolates, one virulent and the other avirulent to adult plants of a particular cultivar, on seedlings of that cultivar. Both isolates gave type-4 responses (the highest) on the seedlings. The two isolates differed in rate of sporulation, the isolate virulent to the mature plant sporulating at the higher rate (Fig. 6; Johnson 1972).

Adult-plant resistance and virulence toward adult-plant resistance are now well-established facts. Available evidence suggests that the reservoir of resistance genes conditioning adult-plant resistance is far greater than the stock of overall-resistance genes. Fortunately, under the relatively moist conditions of wheat-growing in northwestern Europe, seedling resistance is not necessary; it is far more important in such semiarid areas as Washington state (USA)

or Turkey, because seedling infection leads to root damage, which depresses yield more severely under dry than under wet conditions (Stubbs and De Bruin 1970; van der Wal et al 1975; Celik 1976).

Horizontal resistance

Adult-plant resistance can be typical vertical resistance, but there are other possibilities. When major-gene resistance of either type—adult or overall—is overcome by a new rust race, the breakdown of resistance is rarely complete. A breakdown that is partial, compared with that in the most susceptible cultivars known, will usually be found (Zadoks 1977a). With partial breakdown, a serious epidemic can develop; nevertheless, about one half of the original resistance remains (Table 2). The measurable remaining resistance can be called "residual resistance," a term without genetic implications. (The term "rest resistance" [Zadoks 1961] seems to be less appropriate.)

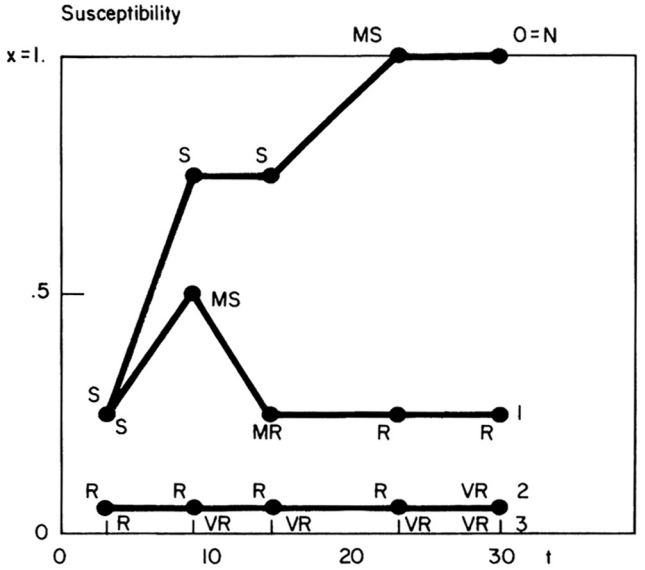
In the USA, Sharp and coworkers (Lewellen and Sharp 1968; Sharp 1976) demonstrated a type of resistance in which genes expressed relatively small but additive effects. The effect of one such gene could be demonstrated only by applying high temperatures to seedlings in the growth chamber. The presence of two such genes could be demonstrated in the same way; the two genes also conditioned a fair degree of resistance in adult plants in the field. Three such genes provided practically complete adult-plant resistance in the field. Pope (1968) found evidence for at least five loci where genes with small and additive effects could be.

In Europe, Stubbs (1972, 1977) provided strong evidence that residual resistance is based on an additive gene-system. Figure 7 shows the development of an epidemic in the field on Sharp's lines, with 0, 1, 2, or 3 additive minor genes; Figure 8 compares numerous races on Sharp's lines. The additive effect of minor genes is largely (but not completely) independent of the cultivar-race combination and of the year of testing (and the weather). One cannot escape the conclusion that Sharp's additive genes, when tested against numerous

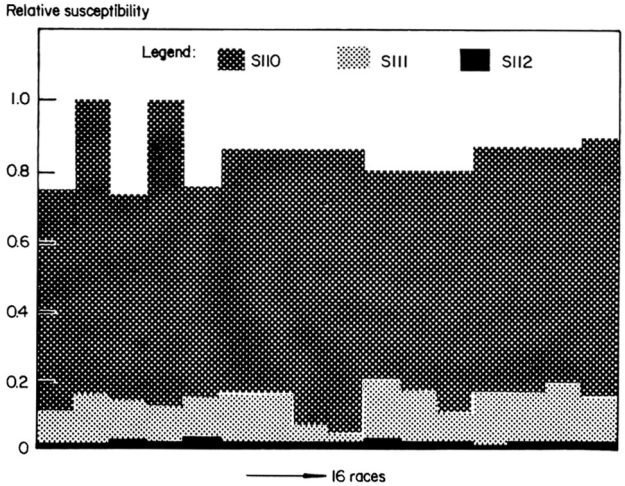
Table 2. Relative rust resistance of wheat cultivars when the original high level of resistance is overcome by a new physiologic race of yellow rust, *Puccinia striiformis*. Data from Stubbs et al (1974).

Cultivar[a]	RRR[b]	Culivar[a]	RRR[b]
Etolie de Choisy[c]	0.69	Heines VII	0.58
Leda	0.67	Probus[d]	0.58
Flamingo	0.66	Cleo	0.47
Opal	0.63	Alba	0.46
Peko	0.63	Dippe's Triumph	0.34
Falco	0.60	Michigan Amber[e]	0.01

[a]Netherlands cultivars tested repeatedly in the Netherlands, except[c] and[d], [b]Relative rust resistance (RRR) = 1 – CI 100; CI is the compatibility index determined by observation of mature plants in race nurseries (Zadoks 1961). [c]French cultivar-race combination rested in The Netherlands. [d]Swiss cultivar-race combination tested in Switzerland (Zadoks 1961). [e]Susceptible control



7. Disease-progress curves of yellow rust (*Puccinia striiformis*) on wheat lines with different numbers of additive minor genes (0, 1, 2, and 3) for resistance. The four wheat lines with N minor genes were selected by Sharp in the USA and tested by Stubbs in The Netherlands. Disease on the susceptible control (N = 0) proceeds rapidly to its maximum value (x = 1). With N = 1, disease is intermediate; with N = 2 and N = 3, disease is equally low but response types differ (after Stubbs 1972).



8. Field tests of Sharp's additive minor genes for resistance of wheat to yellow rust (*Puccinia striiformis*) in The Netherlands (1976) with 16 physiological races (after Stubbs 1977). Relative susceptibility: S = .01 × average coefficient of infection, maximum susceptibility: S = 1, Entries: S 110 (no additive genes), S 111 (one additive gene), and S 112 (two additive genes).

European rust races, provide a high level of horizontal resistance. Because some cultivar-race interactions although small compared with the additive effects, seem to remain, it is not the strict 1963 definition but rather the more flexible 1968 definition of horizontal resistance that applies to the situation discussed (van der Plank 1963, 1968).

Technicalities of the study of horizontal resistance (e.g. components analysis), have been described elsewhere (Zadoks 1972a). Components that can be of interest in the study of details of partial resistance are: 1) germination, 2) appressorium formation, 3) penetration, 3) substomal vesicle formation, 5) colonization, 6) sporulation, and 7) infection (= sporulation) period. Detailed study can explain some aspects of "tolerance" or of "slow rusting or blasting," or both. Host-induced differences in spore germination have been found by Stubbs and Plotnikova (1972). Differences in spore deposition, germination, penetration, latent period, sporulation rate, sporulation period, lesion growth, apparent infection rate in the field, and rate of dispersal in the field can be studied (Russell 1976; Young and Powelson 1976: Zadoks 1961) and exploited in breeding programs.

CONTROL STRATEGIES

The control strategies available are genetic control (breeding for resistance), chemical control, and combinations of the two. Chemical control is difficult because of the semisystemic growth of the yellow-rust fungus. Only systemic chemicals, or at least systemic eradicants, are good enough. At this moment only triadimefon and, possibly, benodanyl, show promise, whereas tridemorph has given some relief. Breeding for resistance is still the major strategy and should remain so because the constant use of systemic fungicides on a staple food crop is objectionable in terms of human health and environment.

Vertical resistance

Sources of vertical resistance, especially the adult-plant type, are still available, and new sources of resistance are being found in wild species. The problem is not a lack of resistance sources but a lack of durable resistance. The durability (longevity, stability, or span of usefulness) has becomc so short that a new, vertically resistant cultivar often loses its resistance within about the first year of its commercialization. In western and eastern Europe, instability of resistance to yellow rust, brown rust, and mildew of wheat can be found. Accumulation of vertical genes, which might delay changes in the fungal population, is possible. However, the most appropriate technique, backcrossing, is not much favored in Europe because compared with current crossbreeding techniques it delays improvement of yields by about 4 years. The attitude of the breeder in northwestern Europe is that one should improve yield and agricultural characteristics (e.g., harvestability), and take care of at least five major diseases, some minor diseases, and physical damage. It is not usually

easy to tell what vertical resistance is present in the end-product. The rust seems to easily accumulate virulence genes, especially when it can do so step by step.

Gene deployment

Gene deployment—the use of different resistance genes in different regions—has been advocated by Johnson (1958) and later authors. Gene deployment was used unconsciously before the European Common Market was established. In that period every country fostered its own policy on registering new cultivars. During the great 1961 epidemic, The Netherlands were protected from yellow rust by the mechanism of gene deployment, as Belgium was protected in 1955. Conscious gene deployment within the Common Market, which favors competition among private breeders, seems to be an illusion.

Multiline cultivars

Multilines use vertical genes in a sensible way. In an endemic disease with slow focal development, which yellow rust often is, foci are effectively suppressed by cultivar mixtures (Zadoks 1972b), and the same can be expected for multilines. There are "clean" and "dirty" multilines. Dirty multilines are composed of lines that have low infectability but will show some pustules of high-response type; in oats, dirty multilines against oat-crown rust (*Puccinia coronata*) (Browning 1974) are now planted on millions of hectares in the USA. Clean multilines are composed of lines that are all fully resistant at the time of introduction. Private breeders now shark an increased interest in multilines, although the disadvantage of the backcrossing technique, mentioned earlier, applies here. One multiline cultivar is ready now, and negotiations have begun about registration and other legal aspects, a complicated affair under Common Market regulations. Promising results have been obtained with varietal mixtures of barley against mildew (*Erysiphe graminis*), a fungus that spreads faster than yellow rust.

Hybrid wheat

Another way to apply vertical genes is to use them in hybrid wheat. Technically, hybrid wheat is feasible, and the introduction of different resistances into the hybrids by way of different parents is possible. As with multilines, but to a lesser degree, the system allows flexibility; an obsolete gene can be replaced. Early hopes were high (Macer 1972), but interest has waned because of commercial problems connected with low fertility.

Horizontal resistance

As a reaction to the disappointment with vertical genes, the search for stable or durable resistance has begun. Durable resistance, although an objective, has not been defined in operational terms. Much attention is now given to such phenomena as partial resistance and slow rusting (Zadoks 1972a), the

inheritance of which is now being studied. Good advances in barley resistant to brown leaf rust (*Puccinia hordei*) have been made.

Slow rusting in barley is determined by three factors: low infectability, low spore production, and long latent period. Prolongation of the latent period is highly effective, has a high heritability, and is polygenically determined with probably at least five additive genes (Parlevliet 1976). Low infectability is also effective, has a moderate heritability, and is possibly polygenically determined (Parlevliet and Kuiper 1977). To assess the farm value of those "alternative" forms of genetically determined resistance, the classical techniques of the plant breeder should be abandoned and another technique pursued: the technique of "microfields" (Zadoks 1972b). All such unconventional work, including that on the additive resistance to yellow rust, is still in the experimental phase. New principles have appeared, but practical results in the form of cultivars with unconventional resistance cannot be reported.

Laboratory techniques and field methods go hand in hand, and give essentially the same results; e.g. in the barley-brown rust system and in the tomato-*Phytophthora* late blight system (Turkensteen 1973). Practical advances have been made independently by several researchers in ye1low-rust resistance—especially by Sharp (1976) in the USA. The essential approach is to start with lines that are evidently susceptible and that have been screened specifically for the absence of vertical resistance. By crossing those lines in various combinations, selecting the offspring for resistance, crossing and selecting again, etc.. high levels of resistance could be attained within a few years. In the wheat-black rust system, Wilcoxson (1976) obtained high levels of generalized resistance based on 3 to 15 genes, according to the cross. The genes operated independently of known vertical genes; the presence of vertical genes could, however, increase resistance somewhat even if the vertical gene were ineffective because of the presence of a matching rust race. Independently, and following different techniques, Knott and Brennan (1976) obtained similar results. They arrived at three lines with good additive adult-plant resistance that seems to hold out against more than one rust race. Whether that general resistance is true horizontal resistance remains to be seen. No horizontally resistant cultivars have yet been commercialized under that label, but the best examples of what could be called horizontal resistance are found in cereals (Table 3).

Integrated control

There is reason for moderate optimism about the use not only of residual resistance, partial resistance, general(ized) resistance, and other resistances but also of chemicals for the control of yellow rust in wheat. There is reason for great optimism about the combination of the two. Stubbs and De Bruin (1970) showed that a chemical that cannot control the rust on a highly susceptible cultivar can adequately protect a moderately susceptible cultivar. Their observation is important, because it points toward a new approach. Moderate resistance of wheat to yellow rust is good enough in most places and in most

Table 3. Examples of host or parasite systems or both where traditional vertical resistance has not been used.

Host	Parasite	Area	Author
Maize	*P. sorghi*[a]	USA	Hooker 1969
Maize	*P. polysora*	Africa	van der Plank 1968
Rye	*P. recondita*	Europe	Parlevliet 1977
Barley	*P. hordei*	Europe	Parlevliet 1975
Avena sterilis[b]	*P. graminis avenae*	Israel	Browning 1974

[a] *P.* = *Puccinia.* [b] A wild population

years. Only occasionally would it need support from a fungicide. The advantage is twofold: the breeder can hope to obtain durable resistance with reasonable effort; the health expert can allow chemical control in exceptional situations only. One condition, however, must be met: there must be a good warning system that takes into consideration the effects of weather, partial resistance, and chemical control (Zadoks 1975). Such warning systems are being developed.

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THE MULTILINE APPROACH TO THE CONTROL OF SOME CEREAL DISEASES

D. R. MACKENZIE

GENETIC DIVERSITY IS EXPECTED of wild species. Genetic uniformity is expected of cultivated crops. Herein lies our dilemma.

Genetically uniform crops offer no impediment to the spread of a plant pathogen. If one susceptible plant becomes infected, all plants are equally susceptible. Disease then spreads between plants, increasing exponentially and causing crop losses.

Genetically diverse wild species resist plant pathogens. Although a pathogen may become established on one susceptible plant, adjacent plants will not be expected to possess equivalent genes for resistance. Spread between plants is blocked. The epidemic is blocked.

This effect is sought in the multiline approach to the control of some cereal diseases. It is the focus of this paper.

Jensen (1952) first proposed a solution to the dilemma of the desire for crop uniformity for agronomic traits and for diverse genetic resistance to disease Borlaug (1959) later addressed the issues directly with specific breeding programs. Browling (1974) summarized much work in demonstrating the epidemiological advantages of multilines. As a result of such research, a significant acreage of oats in the midwestern USA is now planted to multilines. Specifically, multilines are mechanical mixtures of phenotypically identical component lines, each differing in its resistance to specific races of a target-pathogen population. Such resistance has been termed vertical resistance (Van der Plank 1963).

However, many questions must be answered before specific recommendations can be made to adopt multilines for particular crop-pathogen models. The first that I wish to address is the production and maintenance of component lines.

Specific genes for resistance to specific races of a plant pathogen are the ingredients necessary for the construction of multiline components. Back-

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crossing such genes into the recipient (the recurrent-parent) lines poses no insurmountable genetic problem. Of concern here is how component lines should be maintained.

LEMMA 1: *Mechanical mixtures of component lines will not maintain initial frequencies in bulk populations.*

Many studies of varietal mixtures (Frankel 1939: Harlan and Martini 1938: Klages 1936; Laude and Swanson 1942; Montgomery 1912; Suneson 1949) were reviewed by Jensen (1952) in his original paper on multilines. Perhaps the most commonly cited study was that conducted by Harlan and Martini (Harlan and Martini 1938). Seed mixtures of 11 barley varieties were grown at 10 North American experiment stations for up to 12 years, each succeeding generation the product of the previous year's yield (and hence selection). No variety could be identified as the best competitor for all locations. Different sites appeared to favor different component varieties. At a given site, one or two component varieties quickly dominated the population.

It can be argued that multiline-component competition will be reduced greatly below that of varietal-component mixtures. One would expect lessened competitive force in nearly isogenic lines. However, the study of Suneson and Stevens (1953) with barley suggests that unfavorable linkages may play a role in affecting the survival of particular genes. Two loci, in particular, speak directly to the issue. The smooth-awn locus and the black-lemma locus are thought to be competitively neutral with their respective alleles (hooded and white). However, over the 12-year period from 1925 to 1936, the black-lemma allele did not survive. Moreover, the smooth-awn allele gradually increased relative to the hooded allele in the population. Both patterns reflect known or suspected linkages with inferior agronomic types.

Two factors have been suggested to explain survival in mixtures of plants. Seed number (not weight) and the proportion of seed-producing mature seed in the next generation are each thought to contribute to component-line fitness. Disease-caused crop losses have also been demonstrated (Klages 1936) to significantly alter component-line frequencies. Such natural forces could cause a weakening of the multiline protection. Therefore, given the expectation of undesirable gene-linkages and possible selection pressures by the pathogen population, I make the following statement:

PROPOSITION 1. *Component lines must be maintained separately and blended just before use.*

That proposition has profound implications for the adoption of multilines in the developing world. If proven true, the cost of maintaining and distributing multilines could prove limiting if not prohibitive.

LEMMA 2: *The quantity of effective initial inoculum is reduced by the action of a multiline.*

By definition, pathogen inoculum landing on vertically resistant (in the sense of Van der Plank) plant tissue is rendered ineffective. Consider the simplest multiline—a two-component mixture. If only one component is susceptible

to the predominant race, then only one-half of the inoculum that lands on plant tissue is effective. The amount of initial disease (X_o) is reduced to one-half. on a 10-component multiline, X_o is reduced, by chance, to one-tenth that expected on a susceptible field. That is one of the attributes of a multiline. It is also one of the reasons why racial mixtures must be monitored systematically. Given sufficient time for differential selection to occur within a pathogen population. distorted virulence frequencies could result in the evolution of more complex races capable of attacking several or many of the components. The result would be a drastic decrease in the multiline's ability to limit the amount of initial inoculum that is effective.

For that reason I state:

PROPOSITION 2. *Virulence-gene fluctuations must be monitored constantly to countermand unintentional directional selection of more complex races of the pathogen.*

That proposition also has a number of implications for the adoption of multilines in the developing world. The complexity of race monitoring, as demonstrated by the time and effort of the North American stem rust surveys, speaks directly to the problems anticipated for other diseases such as rice blast. However, much can be done to unravel the race profile. In the next section I shall attempt to do so for *Puccinia graminis* f. sp. *tritici,* wqhich causes stem rust of wheat.

LEMMA 3. *Virulence-gene frequencies, once disturbed, remain so-indefinitely.*

Van der Plank (1968, 1975a,b) and others following his suggestion have argued that complex races (races having many genes for virulence) do poorly on simple varieties (varieties having few, if any, genes for vertical resistance). The forces of nature causing such action have been termed stabilizing selection by Van der Plank. That is a misapplication of the concept from population genetics. It unfortunately can lead some persons to assume that directional selection—the forces causing rapid burgeoning of a complex race on a complex variety (the only compatible reaction that works)—is matched by stabilizing selection as an "equal and opposite" force (Van der Plank 1975b).

The implications for the application of the multiline concept to disease control are apparent, especially when one considers that the evolution of complex races will inevitably result from the directional selection imposed by multilines. Groth (1976) even goes so far as to suggest that without stabilizing selection, multilines will not be successful. He argues that in the absence of stabilizing selection, complex races would dominate the pathogen population on a multiline, thereby soon negating the effect of the multiline. Data from old articles have been dredged up to demonstrate the effects of stabilizing selection. Exceptions have been excused as "weak genes." Few, if any, convincing, positive arguments have been offered. Recently Van der Plank (1975a) reanalyzed the data of Green (1971) to demonstrate that stabilizing selection was operating with *Puccinia graminis* f. sp. *tritici* in Canada from 1965 to 1969 (Table 1).

The alleged importance of stabilizing selection to the successful use of

Table 1, Isolates of *Puccinia graminis tritici* in Canada virulent on resistance genes *Sr 5*, *Sr 6*, and *Sr 9d*, singly and in combination. in 1965, 1967, 1968, and 1969. Van der Plank (1975a)

Resistance gene	Isolates (% of total) found during year[a]			
	1965	1967	1968	1969
Sr 5[b]	81.0	98.5	96.5	99.4
Sr 6[b]	10.4	13.5	18.8	8.1
Sr 9d[b]	65.1	80.2	79.2	91.3
Sr 5 + *Sr 6* expected[c]	8.4	13.3	18.1	8.1
Sr 5 + *Sr 6* found	7.5	13.5	18.8	8.1
Sr 5 + *Sr 9d* expected[c]	52.7	79.0	76.4	90.8
Sr 5 + *Sr 9d* found[d]	56.1	78.7	74.2	90.7
Sr 6 + *Sr* 9d expected[c]	6.8	10.8	14.9	7.4
Sr 6 + *Sr 9d* found[d]	0	0	0	0
Sr 5 + *Sr 6* + *Sr 9d* expected[c]	5.5	10.6	14.4	7.4
Sr 5 + *Sr 6* + *Sr 9d* found[d]	0	0	0	0

[a] Total number of Isolates: 1965 = 373; 1967 = 207, 1968 = 202 1969 = 172 [b] Data of Green (1971). [c] Expected on the assumption that the virulences on the resistance genes occur independently of each other in the fungus population and are selectlvely neutral. [d] Excluding race C25, which was found in 1965 and 1968, but not in 1967 or 1969.

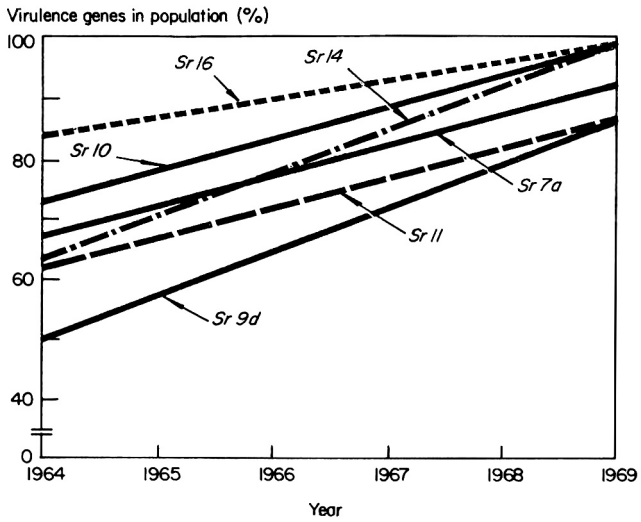
multilines is the primary consideration that led me to reanalyze the data of Green (1971) for a more complete examination of changes in gene frequencies. I conclude, as did Green, that there is no evidence that stabilizing selection operated in the Canadian *Puccinia garaminis tririci* population from 1964 to 1969.

Figures 1, 2, and 3 plot the virulence gene frequencies for the 14 resistance genes reported by Green (1971). Figure 1 gives the yearly virulence gene frequency for those wheat stem rust (*Sr*) resistance genes that showed significant increases in frequency from 1964 to 1969. It is interesting to note that, while *Sr 7a* and *Sr 9d* are important resistance genes in Canadian wheats, *Sr 11* "has not been important under field conditions." All six *Sr* virulence genes appear to respond to diretional selection, albeit the reasons for selection are not readily apparent.

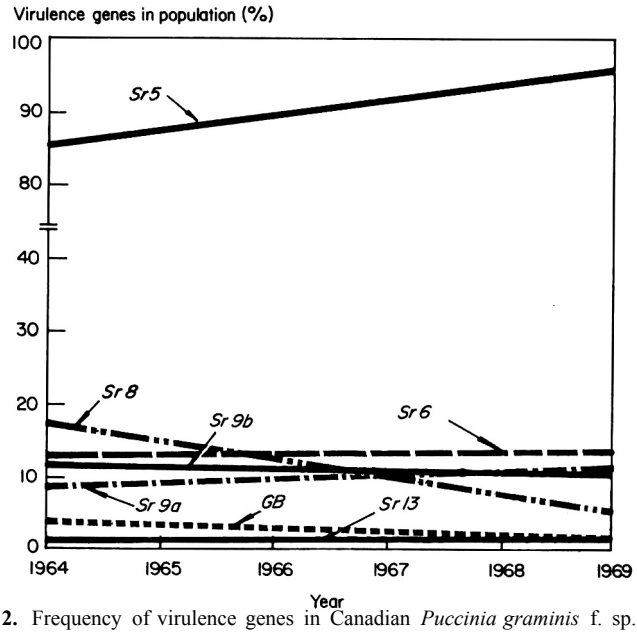
Figure 2 plots the frequency of seven additional *Sr* virulence genes during the same period. Regression analyses of the frequencies suggest that each gene was in equilibrium, with no significant increase or decrease during the period they were monitored.

Figure 3 gives the only possible example of stabilizing selection for single genes. Admittedly the evidence is weak. *Sr 15* is not considercd important in Canadian wheat varieties. Extrapolation of the rate at which *Sr 15* virulence is decreasing indicates that its frequency should drop to less than 1% in about 20 years.

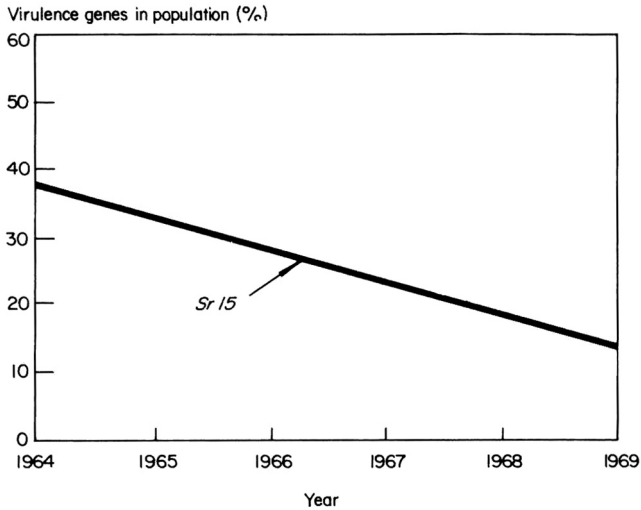
Table 2 gives the expected and observed frequencies of virulence genes on *Sr* resistance genes in combination with virulence on *Sr 6*. Slight deviations in Table 2 from the expected frequencies listed in Table 1 are due to the fact that Van der Plank (1975a) used observed frequencies on the Marquis isogenic



1. Frequency of virulence genes in Canadian *Puccinia graminis* f. sp. *tritici* populations from 1964 to 1969 for those genes found to increase in frequency, plotted as best-fitting regression lines. Coefficients of determination for *Sr 7a* = 79%, *Sr 9d* = 91%, *Sr 10* = 87%, *Sr 11* = 73%, *Sr 14* = 85%, *Sr 16* = 77%.



2. Frequency of virulence genes in Canadian *Puccinia graminis* f. sp. *tritici* population from 1964 to 1969 for the genes found to be in (near) equilibrium, plotted as best-fitting regression lines. Coefficients of determination for *Sr 5* = 25%, *SR 6* = 1%, *Sr 8* = 30%, *Sr 9a* = 2%, *Sr 9b* = < 1%, *Sr 13* = < 1%, *GB* = 48%.



3. Decreasing frequency of virulence on *Sr 15* in Canadian *Puccinia graminis* f. sp. *tritici* populations from 1964 to 1969, plotted as the best-fitting regression line. The coefficient of determination was 75%.

lines for calculating expected frequencies, whereas I used the expected frequencies derived by virulence formulas given by Green (1971) for the specific population sample.

The significance of Table 2 should not be lost in the volume of material. Note that the virulence genes on *Sr 9d, Sr 7a,* and *Sr 11* all have frequencies lower than expected. All three genes were identified as increasing in frequency (directional selection) from 1964 to 1969.

Four genes for virulence (on *Sr 8, Sr 9a, Sr 9b,* and *Sr 15*) were found in frequencies significantly greater than expected. One of those genes, virulence on *Sr 15* (Fig. 3), is thought to possibly demonstrate "stabilizing selection" (i.e., reversed directional selection).

However, when considered in relation to virulence on *Sr 6* and *Sr 15,* the combined frequency is unexpectedly high.

Note also that although virulence on *Sr 9d* is not found in combinations with *Sr 6* virulence (Table 1, 2), an extraordinarily high frequency (as indicated by chi-square tests) of virulence on *Sr 6* and *Sr 9a* and on *Sr 6* and *Sr 9b* was detected. Resistance at the *Sr 9* locus in wheat is a multiple-allelic series with alleles designated as subscript letters a, b, d. Is there a relationship between allelism in the host and combinations of virulence genes in the pathogen? I do not know! I would hesitate to draw firm conclusions about a population's "stabilizing selection" until many more questions have been answered.

The proponents of stabilizing selection often neglect to acknowledge the basic assumptions underlying genotypic selection. The primary consideration is that the population be randomly mating. Most of the foliar pathogens of

Table 2. Isolates of *Puccinia graminis tritici* in Canada virulent on resistant gene *Sr 6* and *Sr 9d, Sr 7a, Sr 8, Sr 9a, Sr 9b, Sr 11,* and *Sr 15.* Virulence on *Sr 6* in combination with virulence on *Sr 5, Sr 10, Sr 13, Sr 14, Sr 16,* and GB were found by chi-square analyses to be insignificantly different from the expected frequencies.

Virulence to specific resistance genes	Isolates (% of total) found during year[a]						
	1964	1965	1966	1967	1968	1969	c^2
Sr 6 + *Sr 9d* expected[b]	6.08	5.71	7.28	10.62	14.32	7.30	
found[c]	0	0	0	0	0	0	51.3**[d]
Sr 6 + *Sr 7a* expected	7.90	7.72	8.69	11.29	15.38	7.77	
found	7.4	4.8	8.0	0.5	2.0	5.2	35.6**
Sr 6 + *Sr 8* expected	1.70	2.11	1.48	0.27	0.68	0.94	
found	4.9	2.3	3.7	0.5	0.5	5.2	29.9**
Sr 6 + *Sr 9a* expected	1.75	0.62	0.26	1.76	3.24	0.66	
found	5.4	4.6	2.5	13.0	16.8	8.1	264.9**
Sr 6 + *Sr 96* expected	2.18	0.68	0.65	1.82	3.34	0.66	
found	8.4	5.6	6.2	13.0	17.3	8.1	311.6**
Sr 6 + *Sr 11* expected	7.18	6.84	8.82	10.62	15.00	7.21	
found	1.9	5.8	8.0	0.0	2.0	0.0	33.2**
Sr 6 + *Sr 15* expected	4.40	4.10	2.45	2.67	4.69	0.89	
found	8.7	5.6	6.2	13.0	17.8	8.1	145.5**

[a] Total number of isolates: 1964 = 370; 1965 = 373; 1966 = 163; 1967 = 207; 1968 = 202; and 1969 = 172. [b]Expected on the assumption that the virulences on the resistance gene occur independently of each other in the pathogen population, are selectively neutral and are given as the product of the individual frequencies. [c]Derived from Green (1971). Table 9. [d]%² significance at the 1% level.

cereal crops fail that requirement. Hence, the direct application of genetic theory to plant-disease control is restricted.

Consider the origin and increase of plant-pathogen races. I would suggest that most races arise by mutation. Race 15B-3 of *Puccinia graminis tritici* could rise from 15B-1 by simply mutating for virulence on *Sr 6.* Race 15B-3 is virulent on variety Selkirk. Race 15B-3, as noted by Green (1974), did not occur in Canada from 1964 to 1969. It did, however, occur in the United States (Stewart et al 1970). In the 1968 race survey, 57.2% of the isolates in south Texas were virulent on Selkirk. Of the isolates collected in Oklahoma and Kansas that year, 16.6% attacked Selkirk. And in the Dakotas, 10.4% of the isolates attacked Selkirk. Katsuya and Green (1966) suggest that the difference between 15B-1 and the more frequently collected and hence more fit Race 56 is due to differential response to temperature, with Race 56 predominant at warm temperatures (Canadian summers favor Race 56). The difference between Race 15B-1 and 15B-3 is due to a single gene: virulence on *Sr 6.* Mutation of Race 15B-1 to 15B-3 (and hence, to virulence on Selkirk) does not appear to alter this "ditferential reproductive potential." The addition of virulence on *Sr 6* would not, therefore, appear to affect isolate fitness. Race 15B-1 was not fit in Canada. Neither was race 15B-3. The unexpected low frequency of virulence on Selkirk is, therefore, more likely an accident of mutation than a result of stabilizing selection.

In the absence of stabilizing selection, and faced with the possibility of

directional selection toward more complex races on multilines, I state:

PROPOSITION 3. *Every attempt must be made to limit susceptible components in multilines.*

The acceptance of moderate levels of disease in multiline populations will magnify mutation and selection. Mutations are directly proportional to the number of individuals in a population. The more disease present, the greater the chance that mutations will provide the variation on which selection will operate, allowing superraces of increased fitness to evolve. From the viewpoint of a population geneticist, intentionally mixing a susceptible portion into a multiline to allow for stabilizing selection, as has been suggested (Groth 1976; Leonard 1969), would be disastrous.

LEMMA 4: *Multilines reduce the rate of epidemic buildup by blocking cross-injection between plants.*

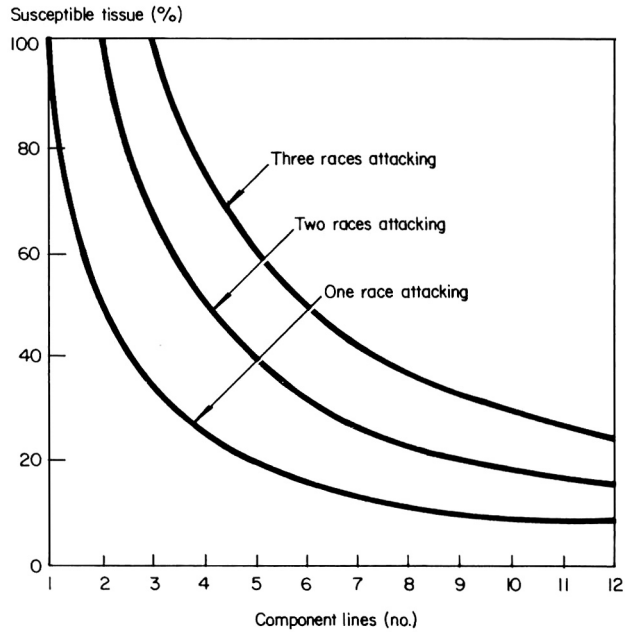
As I have pointed out, inoculum landing on resistant tissue is removed from the epidemic. The initial inoculum, i.e., the first that arrives in a field, cannot initiate an epidemic if no disease results. Such resistance is genetic sanitation. In a multiline, however, once an epidemic has been initiated, the "vertical" resistance is not strictly sanitation, for throughout the course of the epidemic vertical resistance would operate to restrict interplant spread. The effect would be to reduce the apparent infection rate. Let us examine the mathematics of that concept.

Consider again the simplest multiline variety with two components. If a single race attacks only one of its two lines, half of the effective initial inoculum is removed by genetic sanitation. Consider the secondary epidemic. One-half of all potential subsequent infections are lost to the epidemic since they, too, land on resistant tissue. A more complex multiline with 5 components would be expected to dissipate 80% of a specific race, for only 1 of 5 combinations (20 %) would be compatible and lead to more disease.

The return on investment in component lines follow the law of diminishing return (Fig. 4). The gain in disease control from each new component that is added to a multiline is smaller than the gain from the previously added component line. Suggestions that we construct complex multilines with many component lines fail to take account of that fact. Wanton incorporation of specific genes for resistance for "insurance" wastes a precious natural resource. Genes for resistance must be intelligently deployed to preserve that resource. On the basis of the evidence for Proposition 3, and the observation that vertical resistance limits epidemic buildup and does so subject to the law of diminishing return, I state:

PROPOSITION 4. *Resistance genes in multilines should be deployed only as necessary to limit an epidemic, with the remainder held in reserve for future use.*

That consideration could have profound effects on the application of multilines to the control of some diseases on some crops in some locations. Given a handful of genes for resistance, to be used in controlling a foliar



4. Theoretical relationship between number of component lines of a host mixture and the percentage of susceptible tissue when that mixture is attacked by pathogenic races.

disease in an area where epidemics rip along at high speed, satisfactory control may not be attainable without some supporting resistance.

Such complementary resistance for multiline varieties takes the form of an "apparent infection-rate (r) limiting" type of resistance. Some have used the term "slow rusting" while others have applied Van der Plank's term "horizontal resistance" to describe a reduction on the Van der Plankian r value of an epidemic.

If we use the notation of Van der Plank (1963) we can state that:

$$r = 1\ t[\ln(X_2\ 1\ -\ X_2)\ -\ \ln(X_1\ -\ X_1)] \qquad (1)$$

where r is the apparent infection rate, t is the duration of time and $1n(X\,1—X)$ is the logit of the disease proportion (X), with the subscripts referring to the time period for which X was assessed.

I said in Lemma 2 that it reduction of the initial inoculum is the equivalent of genetic sanitation. If the reduction of inoculum continues throughout an epidemic (as in a multiline), the effect will be to reduce r. Although the resistance ofa multiline is vertical, it functions as ifit were horizontal resistance.

Other genetic systems operate to reduce r (MacKenzie 1976). Consider an individual plant in a multiline mixture—a single plant infected with a compatible race. The bulldup of disease on that plant will proceed mostly through

self-infection. The rate of self-infection will characterize how much terminal disease will result from self-infections. Sufficient evidence has accumulated to suggest that the rate of self-infection differs among varieties. That suggests that some varieties would be more suitable for multiline conversion because once they have been infected, self-infection will proceed at a reduced rate.

LEMMA 5 : *Slowed-self-infection types of resistance complement the effects of multilines by limiting self-infection, therefore limiting spread to other component individuals and, in summation, restricting epidemic buildup in the multiline population.*

That lemma has been investigated at The Pennsylvania State University through mathematical modeling and computer simulation. The model is developed as follows :

The disease proportion (X) on any plant can be considered to be the result of self-infection projected as :

$$X_2 = X_1 e^{at} \tag{2}$$

where e is the base of the natural logarithm system, t is time (in days, usually), and a is the self-infection rate. That disease quantity (X) together with all "incoming disease" from neighboring plants that donate compatible inoculum constitutes a plant's disease intensity. The important consideration here is the effect of distance on the spread of disease. At The Pennsylvania State University we had previously used the model of Gregory (1968) given as :

$$\hat{y} = aD^{-b} \tag{3}$$

(or by transformation $\log_{10} \hat{y} = \log_{10}{}^{a-b} \log_{10} D$)

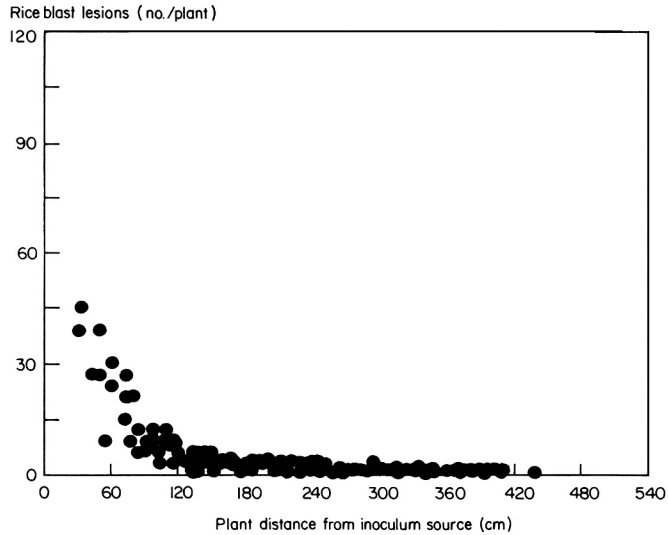
in which the predicted quantity of disease $\hat{y}$ equals the negative power $-b$ of distance D times a scaling factor a. That relationship has been the standard expression for disease spread for many years.

Recently Kiyosawa and Shiyomi (1972) offered a second model :

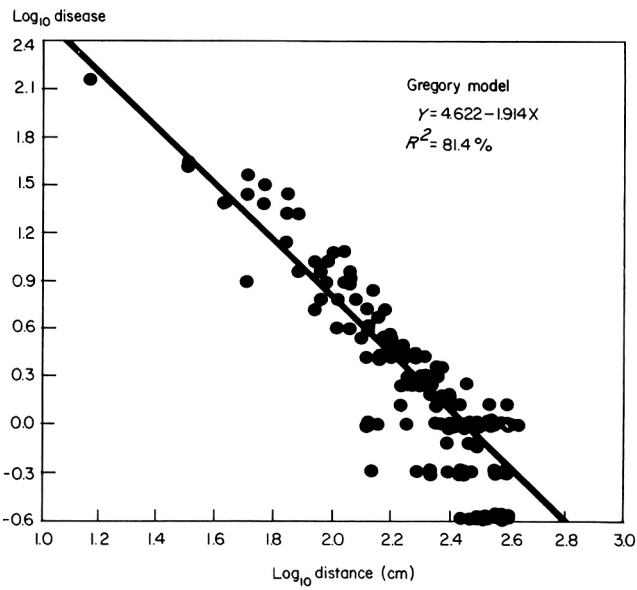
$$\hat{y} = ae^{-Db} \tag{4}$$

(or by transformation in y = 1n a – Db)

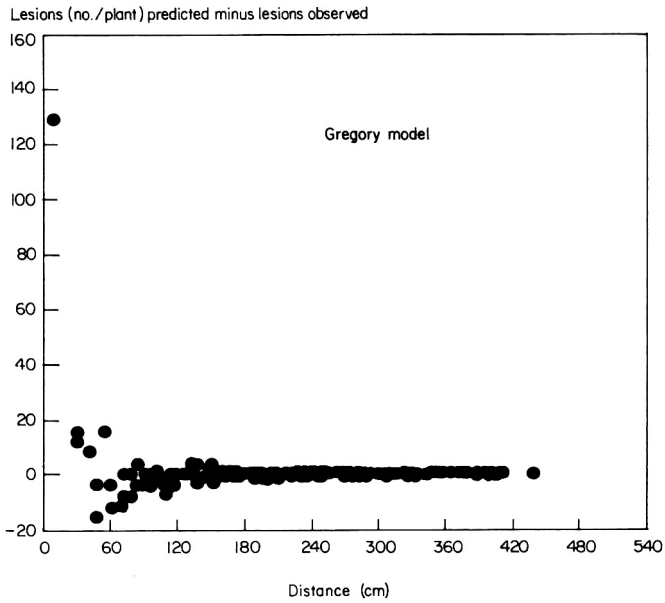
An associate and I (MacKenzie and Villareal 1977) have investigated those models, using blast of rice (caused by *Pyricularia oryzae*) to evaluate their "goodness-of-fit" to actual data. Figure 5 gives the primary spread of blast from a point source on blast-susceptible Tjere Mas seedlings. Figure 6 gives the regression values for the log 10 by log 10 transformed Gregory model. Figure 7 presents the Gregorian expected values minus the observed values, plotted against distance. Similar comparisons for the Kiyosawa and Shiyomi (1972) model by the transformation are given in Figures 8 and 9. A chi-square goodness-of-fit test of the models (analyses of Figures 7 and 9) showed the Gregory model to be superior to the Kiyosawa and Shiyomi model. However, neither could adequately describe the first meter of blast spread. The first meter is, I believe, the critical area for multiline stimulation, and more intensive investigation of that relationship is under way. Nevertheless, given the limitations of the Gregory model for dealing with disease spread, I argue that



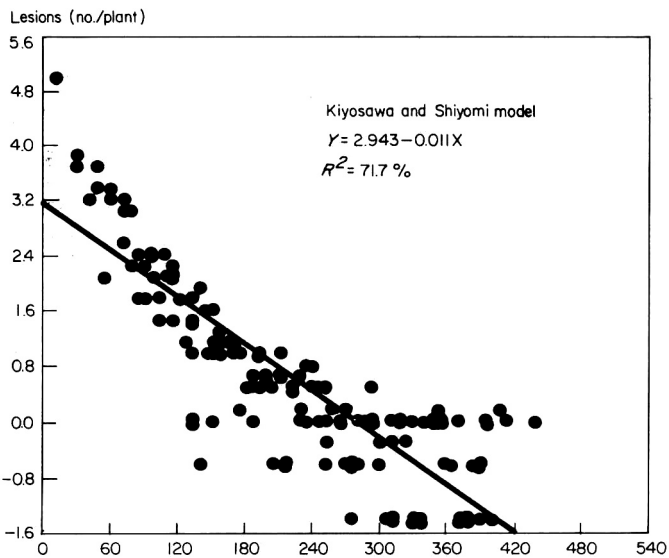
5. Number of rice blast lesions per plant of Tjere Mas seedlings at varying distance from a single-point source of inoculum. Disease was evaluated after 9 days' exposure and reflect, therefore, only primary spread from the point source.



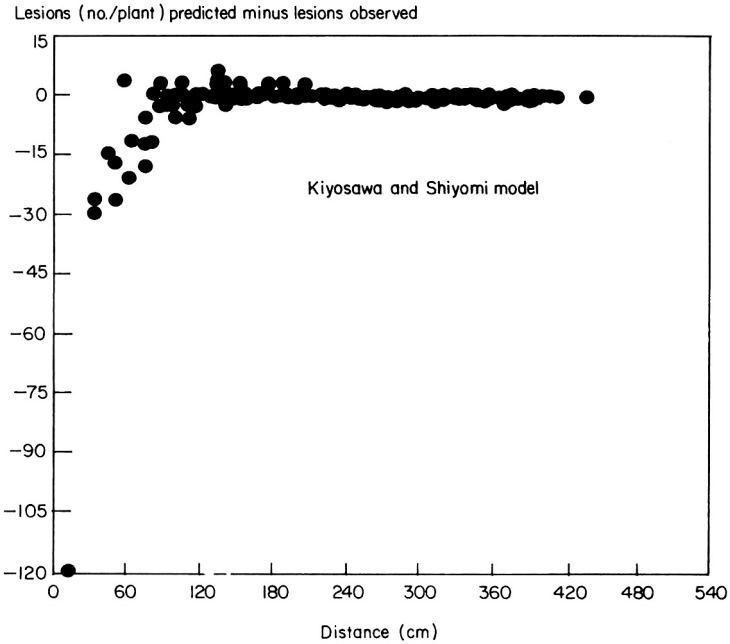
6. Data of Figure 5 plotted as the transformed relationship of the $\log_{10}$ of disease (number of lesions plant) by $\log_{10}$ of distance model (Gregory 1968) and tested for linearity by regression analysis (solid line).



7. Deviation of values expected according to the Gregory model (1968) from the observed number of blast lesions per plant from Figure 5.



8. Data of Figure 5 plotted as the transformed relationship of the ln disease by distance model of Kiyosawa and Shiyoma (1972) and tested for linearity by regression (solid line).



9. Plotted deviation of expected values by Kiyosawa and Shiyom model (1972) from the observed number of blast lesions per plan given in Figure 5.

disease at time 2 (X_2) on any plant is the sum of self-infection plus cross-infection :

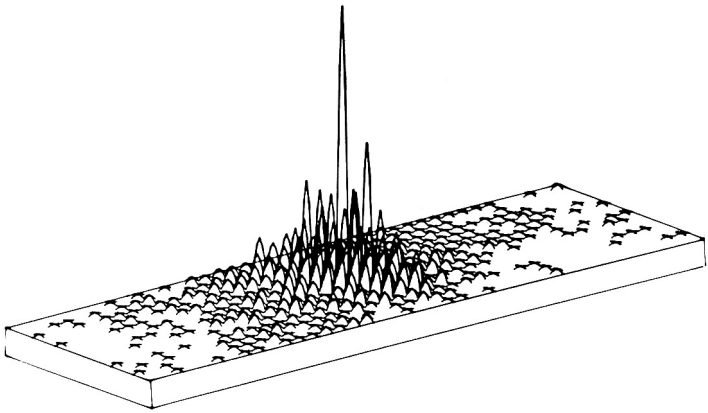
$$X_2 = X_1 e^{at} + \sum_{i=1}^{n-1} aD^{-b}. \tag{5}$$

and that the diseased proportion of the entire population of plants can be summed to give:

$$X_2 \text{ field} = \sum_{i=1}^{n} \left(X_1 e^{at} + \sum_{i=1}^{n} aD^{-b} \right). \tag{6}$$

Equation 6 tells us two things. First, infection in a field is not random but depends on the distance of the source of inoculum from the plant in question. Figure 10 is a three-dimensional graphic plot of the information given in Figure 5. Figure 11 is a similar study of the spread of blast from three point sources. A plant that is distant from the source of inoculum would not be expected to receive as much inoculum as would a plant next to the source. I earlier said that infection in an epidemic is random. Some authors insist that is the case. It does simplify the mathematics of epidemics but it completely misrepresents the action of a multiline.

Second, equation 5 tells us that the action of a multiline is complementary to slow-rusting types of resistance (horizontal resistance, if you must). The



10. Three-dimensional image of data given in Figure 5 for blast spread from a point source of disease.

two together slow the apparent infection rate r of the multiline population by complementation as given when we substitute equation 6 in equation 1:

$$r = \frac{1}{t} \ln \frac{\sum_{i=1}^{n} \left(X_1 e^{at} + \sum_{i=1}^{n-1} aD^{-b} \right)}{1 - \left[\sum_{i=1}^{n} \left(X_1 e^{at} + \sum_{i=1}^{n-1} aD^{-b} \right) \right]} - \ln \frac{X_1}{1 - X_1} \tag{7}$$

Herein lies the application of computer simulation to multiline varieties. Given the following information, computer simulation can be used to evaluate the optimum genetic strategy for a given crop-pathogen model. To set the strategy, we must know:

1. the potential for epidemic buildup in the target region,
2. the number of genes available for use in converting the recurrent parent to a multiline,
3. the self-infection rate of the recurrent parent and its rate of spread between plants, and
4. the expected duration of the epidemic (usually in days). Items 1 and 4 are related, as shown in a reformation of equation 1:

$$X_t = \frac{e\left(\ln \frac{X_1}{1 - X_1} + rt \right)}{1 + e\left(\ln \frac{X_1}{1 - X_1} + rt \right)} \tag{8}$$

where X_t is the terminal amount of disease.

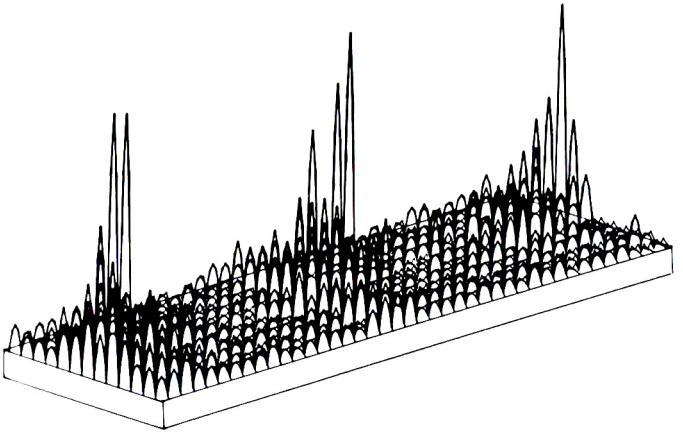
If we are told that a typical r value for the target region is 0.4 unit/day and epidemic starts 10 days after flowering, we can set the initial disease to $X_0 = 0.001$ (i.e., 0.1%). When we do so, the terminal disease level X_t is projected to be 99.4% after 30 days (at crop maturity). Thus, if $r = 0.4$ unit/day, the target region is characterized as a fast-epidemic area. Regions of crop production can, thus, be classified as fast-epidemic areas (such as $r = 0.4$) or slow areas where r might be 0.1 (and the terminal disease severity X_t after 30 days would be less than 2%). Different strategies of gene deployment would then be employed.

Given 14 genes for use in converting the recurrent-parent line to a multiline, we must attempt to balance gene use with genes in reserve. The slack in level of disease control, if any, is to be taken up by using a recurrent-parent line that has an appropriate level of restricted self-infection.

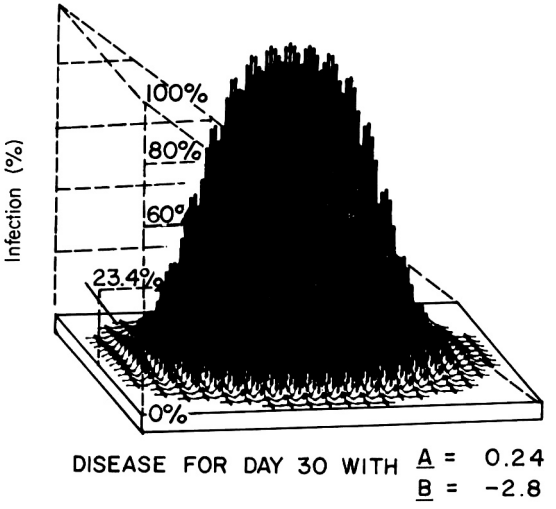
It is for those reasons that I state:

PROPOSITION 5: *Only varieties possessing a measure of slowed apparent self-infection rate should be used for conversion to a multiline.*

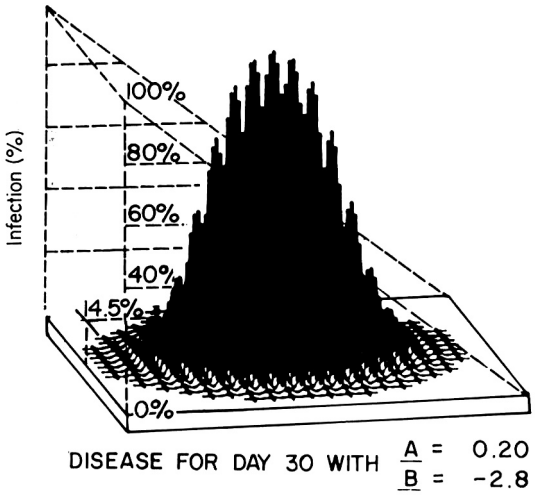
Balancing the many considerations to formulate an optimum strategy for disease control is mathematically complex. The Pennsylvania State University workers have employed computer simulation to demonstrate the concepts and to evaluate potential options. The results of their effort have been made into a 17-minute sound movie. Figures 12 and 13 show typical graphic displays from the film.



11. Number of rice blast lesions per plant of Tjere Mas seedling at varying distance from three distinct point sources of inoculum. Disease evaluated after 9 days exposure to point source of inoculum reflecting primary spread. Data plotted as three-dimensional image.



12. Computer graphics terminal display of stem-rust epidemic simulation of susceptible wheat cultivar (i.e. rapid rate of self-infection given as *A* value) after 30 days of epidemic buildup. Image from a frame of the multiline simulation movie S.M.A.R.T. The *B* value is the mathematical expression for rate of spread over distance.



13. Computer graphics terminal display, as in Figure 12, simulating stem rust of wheat epidemic with self-infection rate (*A*) significantly reduced below value for Figure 12. Although spread within plot is equal (*B* = -2.8), the epidemic is retarded (disease incidence drops from 23.4% to 14.5% after 30 days because of lower self-infection).

Attention at The Pennsylvania State University is now turning to field documentation of the computer simulation for further elaboration of the multiline model. To date it has been learned that multiline deployment, if done properly, will demand rational and intelligent genetic planning. For if we have learned anything from the past 75 years of breeding for disease resistance in small grains, we have learned that the net effect on the *P. graminis tritici* population has been to render 6 of our limited number of useful genes for resistance unavailable for use in multilines in northern America. Genes for virulence on *Sr 16, Sr 10, Sr 14, Sr 7a. Sr 11,* and *Sr 9d* were all present at a frequency of 90% or higher in the Canadian population as late as 1969. That will severely restrict any effort to utilize multilines for the control of stem rust of wheat in Canada. This dilemma is a result of a general ignorance of the principles of population genetics. It is time to pay greater attention to the laws of nature. It is time to protect and use our genetic resources wisely for crop protection.

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CONCLUDING REMARKS

P. R. JENNINGS

ON BEHALF OF ALL of the participants, I should like to extend to Director General Nyle C. Brady and Dr. S. H. Ou our appreciation for the opportunity to participate in this blast workshop.

I personally wish to cite the excellence of the papers presented. They set the background for constructive discussion. The reports of experience with variable pathogens of potatoes, maize, and small grains were expecially useful to us, rice scientists, in that they widened our vision and enhanced our ability to grapple with the blast disease.

What has come out of this workshop? I, for one, would conclude: a great deal. A clearer picture of the blast problem has emerged. The Latin American and African production areas have fundamentally similar problems with blast, which represents the major restraint to stable rice production.

In tropical Asia, for some reason, blast is somewhat less critical except in certain recognized areas. In temperate nations such as Japan, blast would appear to have more in common with blast in other continents with that in tropical Asia.

A second result of the workshop was the emergence of distinct strategies for combating the problem.

That led to the third contribution of the workshop: the identification of some basic needs in the research system that must be satisfied to permit exploration of one or more ofthe defense strategies.

The fourth product of the workshop, although not discussed directly, issues from the fact that blast is not the only problem of its kind in rice. It is not an exaggeration to say that the discussion of blast is equally appropriate to that of the brown planthopper and other sources of biological pressure which have relied heavily on simple Mendelian inheritance patterns of resistance. Thus, justification for development of alternative breeding strategies for rice is broader than merely their utility in dealing with the blast problem.

What are the strategies that have emerged? There are four. It is emphasized that different strategies will have distinct values in different blast situations.

It is not possible or desirable to rank them in the order of utility. Common sense will dictate which might be best for any given area of production.

Rice workers accustomed for decades to one way of tackling the problem must pursue a positive attitude toward alternatives. Failure is guaranteed if one is convinced at the outset that any given alternative is valueless. No guarantee is offered that all the proposed strategies, even with concerted, open-minded study, will result in total success. Nevertheless, they are sufficiently powerful to demand fresh attitudes—to demand honest study and fair trial. The probabilities seem good that at least one approach will be more effective than the present efforts to control blast.

Involved is a question of timing. Most breeders, I hope, realize that the continuation of breeding for vertical-gene resistance keeps us in a pitfall that continually deepens. Logic would suggest new routes, and the sooner it does, the better. A sudden substitution, however, for vertical-gene resistance, of any alternatives, could involve an element of potential calamity. Timing becomes important. At the outset full emphasis must be placed on methods, which in any breeding effort will be the key to success. As methods are refined and used in the pursuit of alternative strategies, useful varieties will emerge that can be eased into the rice production system, weaning us gradually from dependence upon the existing vertical resistance.

What are the alternatives at our disposal?

1. single-gene addition,
2. pyramiding of genes,
3. horizontal resistance, and
4. multiline varieties

SINGLE-GENE ADDITION

The present policy is to add new resistance to agronomically desirable lines. The genetics of the new resistances are unknown. They may be based on single genes or groups of genes. Present policy covers both strategy 1 (single-gene addition) and strategy 2 (pyramiding of genes). The experience in Latin America suggests that vertical genes are added one or only a few at a time.

The strategy of single-gene addition is based on the idea of using as few resistance genes as possible. A new resistance gene is added to an agronomically valuable germplasm only when needed. Single-gene addition techniques can give highly stable resistance in some cases, unstable resistance in others.

Eventually resistance is lost by a stepwise increase of virulence in the pathogen. By careful monitoring of the pathogen population, the correct timing for inserting a new resistance gene can often be ascertained.

From the breeder's point of view, the single-gene addition strategy is economical. Its usefulness in practice, depends on the ecosystem where it is applied. Vast acreages of wheat in Australia and Canada are protected by methods that fall into the category of single-gene addition. The strategy is

very powerful in general, and may be good for rice in at least some areas of the world where there are appropriate ecosystems.

A prerequisite for its use is knowledge of individual resistance genes. The methodology for obtaining such knowledge is available: back-crossing to susceptible parents, diallel crossing, race testing and—in a later stage—virulence-gene testing. The ingredients of the methodology can be created for rice.

PYRAMIDING GENES

One approach to breeding for blast resistance could be the pyramiding of genes for resistance to *Pyricularia oryzae* in varieties with agronomic acceptability. The objective would be to develop multigene resistant varieties with sets of 3 to 5 genes for specific resistance, and multicomponent resistant varieties that combine specific resistance and partial field resistance. Those varieties would have resistance based on genes with major as well as minor effects. They would have complex modes of inheriting resistance to *P. oryzae.* Furthermore, it is proposed that several such varieties or groups of varieties should be developed—each with different genes for resistance, if the genes are known, or at least with resistance obtained from different donors or sets of donors. It would be desirable to maintain the diversity of the genetic base in a succession of varieties that should emerge through the breeding program, each having different genes or sets of genes.

For successful pyramiding, the following materials and information are needed:

- numerous sources of resistance with known or unknown genes.
- some concept of the frequency of genes for virulence and avirulence in *P. oryzae* in different epidemiology areas.
- knowledge of the genetics of resistance, and
- an evaluation program to properly identify plant material to be used in the crossing program and to select from the breeding program.

Various approaches to achieve that breeding objective could be taken. Opportunities should be available to scientists of the International Rice Research Institute (IRRI) to develop and explore novel approaches to the problem. The conventional methodology of plant breeding with, perhaps, a greater amount of intercrossing among selections from a given set of original crosses would be used. An attempt would be made to identify those genes for specific resistance that condition resistance to a wide spectrum of biotypes of *P. oryzae,* and to secure or to develop a set of *P. oryzae* cultures, each containing a simple gene for avirulence that would identify the corresponding resistance genes. Data would be obtained from the blast nursery, the International Rice Blast Nursery (IRBN), and national field trials and nurseries. Various promising lines with different breeding backgrounds would be provided to national programs so national breeders could select among various alternatives for further breeding or for varietal release.

Because of the variation of the fungus, no single variety would be expected to be resistant in all areas or resistant for a long period of time in any one area. The opportunities are available for certain varieties to be useful in certain areas for quite a long time and, one hopes, to be replaced before severe blast infection results.

HORIZONTAL RESISTANCE

One approach to developing stable and long-lasting resistance to any biologically flexible pest or pathogen, such as the blast fungus or the brown planthopper, is to breed for horizontal resistance. The recent text by Robinson[1] should be read for definitions of terms and concepts. The definitions and concepts of horizontal and vertical resistance are precise but generally are poorly understood. The confusion has lead to the suspicion that horizontal resistance, as precisely defined, is ephemeral or not useful in breeding. Moreover, there is an almost universal reluctance to breed for horizontal resistance because it requires the use, as parents, of cultivars that previously have been classified as "susceptible." Adherence to classical genetic principles is still necessary in a program of breeding for horizontal resistance, but the emphasis shifts toward quantitative inheritance. Working with simply inherited vertical genes (that show the classical 3:1 ratio) tends to reduce the ability to detect and use horizontal resistance.

In the pursuit of horizontal resistance, it should be clearly recognized that such resistance was used successfully many years ago in potatoes, that it is the major type of resistance to disease that occurs on maize in the tropics, and that programs have recently begun breeding for it in wheat and coffee, and in rice in Japan and West Africa.

Methods already exist for making use of horizontal resistance in rice, but the use of seedling tests combined with a search for single-gene donors has precluded their employment and invited the "Vertifolia" effect and "breakdown of resistance." Methods for utilizing horizontal resistance require that all parents be "susceptible" to at least one vertical pathotype.

A set of susceptible cultivars (with known vertical resistance) serves as a check, as a monitor, and in spreader rows. Sets can be manipulated to provide the level of disease pressure that will permit expression, during the postseedling stage, of horizontal resistance. Large numbers of crosses. preferably random polycrosses, are required. Some innovative and flexible research on methods is required for each pathosystem that is to be studied (e.g., blast, and the brown planthopper). Each approach must be tied to the ecosystem where the problem pathosystem exists naturally, and successful transfer of an approach from one ecosystem to another should not always be expected.

In the case of blast, some rice cultivars from Japan, West Africa, and the

[1] R. A. Robinson, *Plant pathosystems* (Springer-Verlag, Berlin, Hadelberg, New York, 1976).

USA are vertically susceptible to known pathogen-genes and can be used to index a series of parents that can provide a wide genetic base for initial selection and crossing. The key to the method is to preclude errors of 'resistance estimation' due to vertical genes. It is best to follow the approach used in potatoes.

MULTILINE VARIETIES

Multiline varieties are mechanical synthetics of phenotypically similar component-lines, each line differing in its reactions to specific races of a target pathogen-population. It is suggested that multiline varieties be considered for areas where epidemics and crop damage are unusually severe.

The observation that wild species benefit from genetic diversity has been offered as an example of the best use of race-specific genes for resistance. By proper genetic manipulation, an agronomically acceptable variety can be converted to a series of component lines while retaining the necessary commercial uniformity.

On-going research at several institutions around the world indicates the importance of properly selecting the agronomically acceptable line that is to be converted. It is known that certain types of infection-rate-limiting disease resistance (e.g.. horizontal) greatly complement the protective action of a multiline. The implication is that any research strategy involving multilines must incorporate an integrated approach to the genetic control of foliar pathogens.

Multiline varieties for control of blast may have application in those rice-producing regions which are considered to have intense problems, year after year, with blast. An adequate number of genes for resistance is thought to now exist in the IRRI world collection of rices. Given those resources, and the knowledge and germplasm available from previous research efforts in the USA and Japan, it is proposed that multilines be given consideration as one strategy for the control of rice blast disease.

It is acknowledged that many unforeseen problems exist between the expression of this suggestion for a research effort and the ultimate distribution to rice farmers of multiline varieties. For that reason I propose that IRRI concentrate its technical resources only on those problems suited to its existing staff, money, and facilities. Sufficient interest exists in conducting research into the multiline control concept that, given encouragement, complementary research programs in other research institutions (e.g., in Japan, USA, national programs in Asia, Latin America, Africa, etc.) could focus tremendous effort on the resolution of the problems. Such encouragement should most logically come from IRRI.

RESEARCH NEEDS

Some needs in research to get on with the job appear paramount. There are desperate situations in Latin America and Afirica at this moment. The Inter-

national Center for Tropical Agriculture (CIAT) has competence in breeding but no support in pathology, and there is none in the rest of the continent. To ensure stabilization of past yield advances and to permit future progress, CIAT must have competent plant pathology input. Africa is similarly deficient in capability. The Ivory Coast has had an innovative pathologist now gone working in isolation. The International Institute of Tropical Agriculture (IITA) desperately needs a superior breeder-pathologist team to tie to national programs.

IRRI, as the recognized leader in rice research, would seem to have urgent need for a geneticist competent in the genetics of hosts, pathogens, and pests. We do not have anything that approaches fulfillment of our need for useful genetic information to guide the breeding team. That seems to be a serious weakness, but it is at the same time a golden opportunity for a person or persons to support and interact with breeders, pathologists, and agronomists to ensure achievement of the ultimate goals of yield stability and healthy rice crops.

I have expressed appreciation of the group to IRRI for inviting all of us to this workshop. In closing, I should now like to thank all the participants for their individual contributions that, when implemented, can change the course of disease and pest control in the years to come.